

Growth and Survival

Child Health Disparities and Early Life Adversity in
Sub-Saharan Africa

OMAR KARLSSON

LUND STUDIES IN ECONOMIC HISTORY 88 | LUND UNIVERSITY



Growth and Survival

Child Health Disparities and Early Life Adversity in
Sub-Saharan Africa

Omar Karlsson



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DOCTORAL DISSERTATION
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Sweden.

To be defended at EC3:210 on Friday 21 September at 10.15.

Faculty opponent
Bruno Schoumaker

Organization LUND UNIVERSITY Department of Economic History Author(s) Omar Karlsson	Document name DOCTORAL DISSERTATION
	Date of issue: 2018-08-31
	Sponsoring organization
Title and subtitle: Growth and Survival: Child Health Disparities and Early Life Adversity in Sub-Saharan Africa	
<p>Abstract</p> <p>Sound physical health is a critical component of the human development process, wherein early life and childhood are pivotal periods. Although child health in sub-Saharan Africa has been improving for the past few decades, the region still has the highest mortality rate for children under five as well as high levels of child morbidity. The most immediate causes of these poor health outcomes are infectious diseases and undernutrition, while more remote factors, such as resources available to parents, are also known to play a role in child health. Parents are further embedded in an economic, social and epidemiological environment, which commonly reflects the parent's own resources, as well as aiding them in or inhibiting them from ensuring the healthy development of their children.</p> <p>The primary aim of this dissertation is to first study the consequences of an adverse environment in infancy, such as exposure to infectious diseases, on human development; and secondly, to explore disparities in child health as they relate to several parental factors – maternal health, parental education, and religious affiliation. Specifically, the dissertation examines the overlap and interaction between these parental factors and contextual factors in determining child health. The results show that adversity in infancy negatively impacts subsequent human development, which is not mitigated in households with higher socioeconomic status. Secondly, contextual factors explain some of the differences in child health between different groups but do so to a varying degree. The relationship between child health and parental education and, especially, maternal health, appears to be persistent and strong, even independent of contextual factors and other measures of living standards. On the other hand, the link between religious affiliation and child health mostly reflects geographic clustering of religious groups and heterogeneity in living standards. As the contextual environment has improved, the association between parental education and maternal health and child health appears to have decreased. Specifically, the persistence of the intergenerational transmission of health from mother to child has attenuated as government spending on health care has increased, and the association between parental education and child health has diminished over time as the contextual environment has improved.</p> <p>Parental resources, health, and socioeconomic status are all vital components of human development in sub-Saharan Africa and other low and middle-income countries. The intergenerational transmission of disadvantage from parents to children is, however, reduced with improving environments.</p>	
Key words: Child health, under-mortality, sub-Saharan Africa, low-and-middle-income countries, parental factors	
Classification system and/or index terms (if any)	
Supplementary bibliographical information	Language: English
ISSN and key title 1400-4860	ISBN: 978-91-87793-50-9
Recipient's notes	Number of pages 307
	Security classification

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ISBN 978-91-87793-50-9 (print)
ISBN 978-91-87793-51-6 (pdf)
ISSN 1400-4860

Printed in Sweden by Media-Tryck, Lund University, Lund 2018



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Acknowledgment

I would like to thank Lund University, the Centre for Economic Demography, and the Department of Economic History for providing me with a supportive environment to take my first steps as a researcher.

To my main supervisor, Martin Dribe, I am grateful for the opportunity to do research in an inspiring environment. Your advice, instructions, and insightful comments on my work have been invaluable in my training as a researcher. I would also like to thank my assistant supervisor, Therese Nilsson, who provided me with a wealth of useful comments and valuable suggestions.

I would like to thank Harvard University and SV Subramanian for granting me the opportunity to spend a year at the Harvard T.H. Chan School of Public Health. I would further like to thank SV Subramanian and Jan-Walter De Neve for fruitful cooperation which gave me invaluable experience in conducting and publishing research.

I thank Kirk Scott and Volha Lazuka for a thorough assessment of this dissertation and suggestions made at my final seminar. Also thanks to Joyce Burnette, Tommy Bengtsson, and Astrid Kander for their comments. Thanks to participants at the 2015–2018 Population Association of America’s annual meetings, the 2018 European Population Conference, the 2015 Africa Population Conference, Lund University’s Department of Economic History weekly seminar, and the Harvard Landmark meetings who commented on early versions of the papers in this dissertation.

Writing this dissertation would also not have been possible without the generous funding provided by the Swedish Research Council. I would also like to thank Handelsbanken and the Tom Hedelius Foundation for the generous Hedelius Scholarship which funded my stay at Harvard University 2016–2017. I am thankful to Stiftelsen Landshövding Per Westlings Minnesfond for supporting my travel to the 2018 Population Association of America’s annual meeting, and Knut and Alice Wallenberg Foundations for funding my trip to the 2015 African Population Conference.

Many thanks to Mats Olsson, the head of the Department of Economic History at Lund University, and the current and previous Directors of Graduate Studies Patrick Svensson and Astrid Kander. Thank you, also, to the administrative staff, Anneli Nilsson Ahlm, Madeleine Jarl, Kristin Fransson, Birgit Olsson, and Tina Wueggertz.

My time in the Ph.D. program was made more enjoyable by all my colleagues. Frank van der Most and Anna Missiaia; words cannot describe how grateful I am for safely securing me through all my ups and downs, on difficult ascends and high falls, whether it was on slabs or overhangs. I would like to thank my Ph.D. cohort at Lund University, Kathryn Gary and Viktoras Kulionis, who have been a great company during these years. I would like to thank Krisztina Gero, Masayoshi Zaitsu, and the other researchers at Harvard T.H. Chan School of Public Health for making my stay in the United States even more enjoyable. I was lucky to have four great office mates during my time at Lund University; thank you Zeyuan Chen, Anna Tegunimataka, Yannu Zheng, and Cecilia Larsson. I would finally like to thank everyone at the Centre for Economic Demography and the Department of Economic History for contributing to an excellent environment during my time as a Ph.D. student at Lund University.

List of papers

- I Karlsson, Omar (2018). Scarring and selection in sub-Saharan Africa: The effects of adverse environment in infancy on health and education. *Unpublished manuscript.*
- II Karlsson, Omar and Dribe, Martin (2018). Maternal height and child development in sub-Saharan Africa: Underlying mechanisms and the role of public spending on health. *Unpublished manuscript.*
- III Karlsson, Omar; De Neve, Jan-Walter; and Subramanian, S.V. (2018). Weakening association of parental education: analysis of child health outcomes in 43 low- and middle-income countries. *International Journal of Epidemiology.*
- IV Karlsson, Omar (2018). Child health disparities by religious affiliation in West and Central Africa. *Unpublished manuscript.*

Introduction

Motivation and aim

Individuals' well-being is related to what they are capable of being and doing. According to the capability approach pioneered by Amartya Sen (1990), development entails an expansion of capabilities that enable individuals to live good lives and achieve desirable outcomes. Capabilities are embedded in individuals in the form of skills and capacities, as well as in their living contexts, where capabilities manifest as opportunities and freedoms. Skills and capacities accumulate throughout an individual's life course with particularly important periods being those of early life, childhood, and adolescence (Barker, 2004; Case and Paxson, 2008; Gluckman et al., 2010). While survival, health, and primary education are fundamental capabilities of intrinsic value, they are also instrumental in acquiring further capabilities and achieving desirable outcomes (Heckman and Corbin, 2016; Nussbaum, 2011). Children born to parents with sufficient resources, in a food-secure environment with good public health, and access to health care and education, have opportunities to develop skills and capacities and to grow into healthy individuals.

There have been major improvements in well-being, globally, reflected in increased life expectancy, better health, and the availability of more opportunities (Deaton, 2013). Beginning in the eighteenth century, improved nutrition and reduced exposure to infectious diseases in Europe resulted in a significant reduction in the death rate of children, marking the onset of the mortality transition. The health of survivors also improved, reflected in better health in adulthood (Floud et al., 2011; Hatton, 2013). Improvements in well-being are also underway in the developing world, where under-five mortality rates have shown a decline since the 1950s, and nutrition and school enrollments have improved more recently (Deaton, 2013, 2007; Soares, 2007). However, large disparities in well-being persist between countries, with much of the developing world continuing to lag far behind the developed countries with regard to most fundamental capabilities (Victora et al., 2003).

The list of least-developed countries, compiled by the United Nations (UN), contains 47 countries, 33 of which are in sub-Saharan Africa (SSA) (UN, 2018,

2014). SSA has an under-five mortality rate of 78 deaths per 1,000 live births and accounts for half of the global deaths of children under 5 years old while, having one-fourth of global births (Liu et al., 2017, 2015; World Bank, 2017). Further, many surviving children are undernourished and unhealthy, as reflected in the high prevalence of stunted physical growth affecting 35% of the under-5 age group in this region (UN IGME, 2015; World Bank, 2018). The proportion of children out of school is 22%, which is higher than the corresponding percentage in any other part of the world (UNESCO, 2018; World Bank, 2017). Despite improvements, only a few countries achieved the Millennium Development Goal of reducing under-five mortalities by two-thirds during the period 1990–2015 (Way, 2015).

The main proximate determinants of child health in SSA are infectious diseases, although undernutrition and preterm birth complications also play substantial roles (Horton et al., 2008; Liu et al., 2017, 2015; Mosley and Chen, 1984; UNICEF, 2007). Within households, parents are often unable to provide sufficiently for their children because of a variety of factors associated with a lack of material means and other capabilities, such as health and education. Further, disadvantaged households are situated within environments where necessary infrastructure and services, such as health care and education, are lacking, and where the risk of exposure to harmful diseases and food insecurity is high (Fotso and Kuete-Defo, 2005; Griffiths et al., 2004; Kravdal, 2004; Stephenson et al., 2006).

The introduction of the Sustainable Development Goals (SDGs) led to renewed efforts to improve child health and human development, with the objectives of reducing the under-five mortality rate to 25 deaths per 1,000 live births, achieving universal primary and secondary education, and ending all forms of child malnutrition by 2030, and reducing stunting to less than 15% by 2025. The SDGs call for disaggregation of important indicators to combat inequalities associated with, for example, ethnicity and socioeconomic status (SES). Most countries in SSA need to accelerate their reduction rates relating to the above conditions and to tackle disparities in under-five mortality rates, stunting, and school enrolment to achieve the SDGs (UNICEF, 2018).

Significant disparities in child health between various groups within countries continue to be apparent, and many children inherit disadvantages from their parents. Studies have revealed an enduring association between child health and maternal health, parental education, and, to a less extent, religious affiliation, which respectively represent health, socioeconomic, and sociocultural factors (Caldwell, 1979; Gyimah, 2007; Monden and Smits, 2009). Poor maternal health, measured by height, has been found to negatively impact child health in most developing contexts, with the main suggested pathways being compromised human development in mother's early life (Perkins et al., 2016). Parental education has been widely studied as a determinant of child health, with causal

links posited to occur through various pathways, such as improved skills, and improved incomes (Mosley and Chen, 1984). It has been suggested that the relationship between religious affiliation and child health is rooted in differences in behaviors and attitudes prescribed by religious doctrines (Gyimah, 2007). However, other explanations lie in differential access to resources and living standards between these groups.

Although studies have found disparities in child health by maternal health, parental education, and religious affiliation, the extent to which group-level differences in child health overlap and interact with contextual factors is less well understood. Compared with lower-income households and their locations, those that are better-off are more likely to be found in places characterized by better living standards; better provision of services, such as healthcare and education; a less disease-prone environment, and more food security, with each of these factors independently influencing child development. Religious groups are also highly clustered geographically, which may explain observed differences in child health.

Further, parental factors may interact with the contextual environment in determining child health, given that many of the proposed pathways of parental factors, such as health and education, are related to contextual factors. For example, it has been suggested that maternal education impacts on child health by increasing the skills and knowledge of mothers regarding nutrition, hygiene, preventive care, and treatment of diseases (Mosley and Chen, 1984, p. 35). Therefore, the effectiveness of parental education, and, more broadly, parental resources in determining child health evidently varies according to the external environment, relating, for example, to economic development, underlying disease risks, and availability of health care, nutrition, contraceptives, and clean water (Jeong et al., 2018).

This dissertation is a compilation of four papers. The main aim of this thesis is to first, study the effect of early life adversity on human development, and second, examine broad relationships between parental factors and child health, in a large number of sub-Saharan African and other low- and middle-income countries (LMICs). Three out of the four papers further quantify the extent to which contextual factors, as well as various dimensions of living standards and demographic characteristics, account for the relationship between parental factors and child health.

The overarching research question is as follows:

To what extents do contextual factors explain the observed disparities in child health by maternal health, parental education, and religious affiliation?

Since parental factors also interact with contextual factors in their relationship with child health, the four sub-questions addressed in each paper are:

Are children in better-off households less affected by an adverse environment during infancy in SSA?

Does increased public spending on health reduce the intergenerational transmission of health from mother to child in SSA?

Does the association between parental education and child health change over time as contexts have improved in LMICs?

What are the implications of community-level religious composition for child health in West and Central Africa?

Considering the first sub-question, infants in better-off households may be less affected by adverse exposures as a result of greater investments in health before, during, and after they are exposed to an adverse environment. Considering the second sub-question, increased public spending on health may improve the public health environment and access to health care and therefore mitigate the negative consequences of poor maternal health. In relation to the third sub-question, the strength of the relationship between parental education and child health may change over time as education becomes less exclusive and other underlying factors change. Considering the last sub-question, the community-level religious composition may influence, for example, religiosity, and have implications for religious minorities, as well as reflecting living standards.

Measures of child health used in the dissertation are under-five mortality, neonatal, post-neonatal and child mortality, and anthropometric indicators, such as height-for-age and stunting. In addition to child health, two of the papers examine school attendance as an outcome. School attendance is a fundamental capability and an important dimension of the human development process (Nussbaum, 2011). It is also related to child health, as healthier children are more likely to attend school (Alderman et al., 2001; Brooker et al., 1999; Heckman, 2007; Heckman and Corbin, 2016). Three of the papers cover between 11–35 countries in SSA and one paper covers 43 LMICs, 25 of which are in SSA. The data used in these studies comprise pooled datasets extracted from the Demographic and Health Surveys (DHS) conducted between 1990 and 2016. In one paper, the DHS data have been supplemented with data from the Global Health Expenditure Database of the World Health Organization (WHO). Most analyses included children born up to five years prior to each survey, but in some of them, complete birth histories, beginning in the late 1950s, are used.

DHS data from 33 countries in SSA are used in the analysis presented in the first paper, which assesses the impacts of adversity experienced in infancy on child health, and school attendance. Because of data requirements, this analysis is confined to more densely populated and extensively surveyed areas. For the analysis of the association between maternal height, and under-five mortality and school attendance presented in the second paper, all available DHS surveys conducted in SSA are included to obtain a large sample size. Thus, the sample comprised 35 countries in SSA. The analysis of the association between parental education and child health, presented in the third paper, included all LMICs for which data from at least two DHS surveys are available to test for changes in the association over time. The analysis is based on 86 surveys from 43 countries. Finally, the analysis of differences in child health by religious affiliation, presented in the fourth paper, focuses on all religiously mixed countries in West and Central Africa for which data are available, or 11 countries. In all of the analyses, only children born to women who are respondents in the DHS surveys are included. Consequently, the substantial population of orphans in the region, especially in countries with a high prevalence of HIV/AIDS, such as southern Africa, and countries affected by civil wars, are excluded from the studies.

Sub-Saharan Africa and the developing world

SSA is a vast region encompassing 48 countries and extending over an area of 21.2 million square kilometers, with a total population of about one billion people that accounts for 12% of the world's population. This region is ethnically, culturally, historically, and environmentally diverse. Over 2,000 languages are spoken in the region, of which French and English are widespread. There is usually an overlap between languages and ethnic groups, which number several thousand. Despite the ethnic and cultural diversity of the region, most people practice some form of either Christianity or Islam (Lugo and Cooperman, 2011, 2010). The majority of the countries were formerly under colonial rule, with most of these nations gaining independence in the 1960s (Hunziker, 2005). Political turmoil has characterized many of the countries in SSA, and there have been civil wars with high deaths tolls within the region (Ghobarah et al., 2004).

The region is also diverse regarding its geography and climate. The northern part of the subcontinent has vast deserts and a hot and arid climate. Highlands with a dry and cool climate are located in the southern and eastern parts. Tropical forests occur in the central part of the region, which along with the western part, is generally characterized by a hot and humid tropical climate. Countries in SSA also vary vastly in size, ranging from 2.2 million square kilometers (the Democratic

Republic of Congo) to 1,000 square kilometers (Sao Tome and Principe). The continent also includes Nigeria, which is one of the most populous countries in the world with 186 million inhabitants as well as some of the smallest countries, worldwide, such as the Sao Tome and Principe with 200,000 inhabitants.

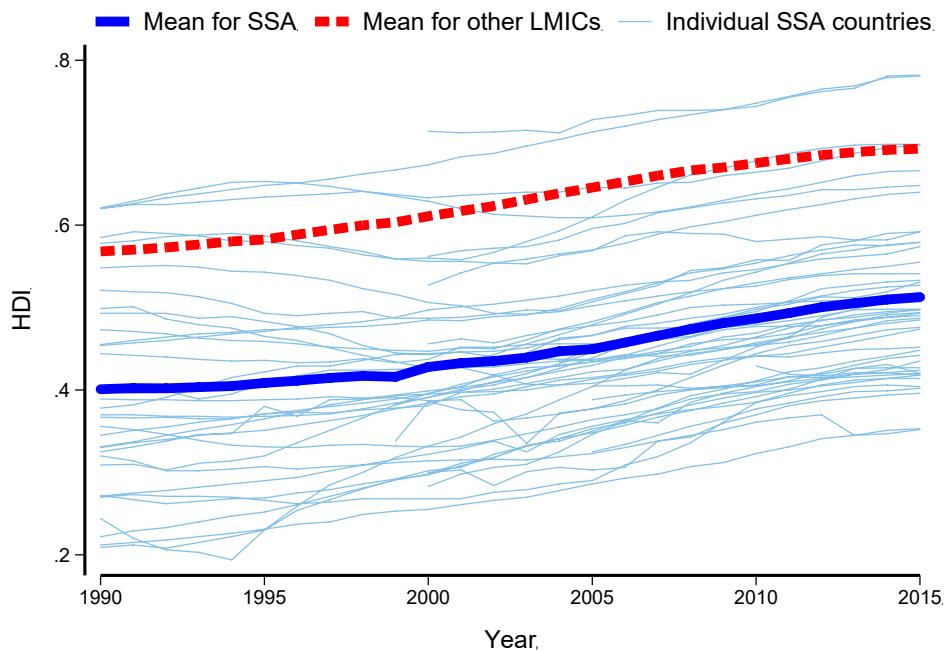


Figure 1. Human Development Index (HDI) in sub-Saharan Africa (SSA) and in other low- and middle-income countries (LMICs)

Note: The mean value calculated for sub-Saharan Africa and low- and middle-income countries is at the country level for each year. Means are unweighted. Source: World Bank (2017).

For many decades, living standards in SSA have been deficient, with this region ranking among the lowest, globally, for most measures of development and well-being (Malik, 2014). The Human Development Index (HDI) prioritizes capabilities as a measure of development and is composed of life expectancy at birth, mean years of schooling, and gross national income per capita (UNDP, 2018). An HDI of under 0.55 is generally considered to indicate a low level of human development, whereas one between 0.55 and 0.7 indicates a medium level of human development. Figure 1 shows that the mean HDI has been increasing for countries in SSA as well as other LMICs. However, the mean for SSA is considerably lower than that for other LMICs, with most countries in the region demonstrating low HDIs. Figure 2 shows that the under-five mortality rate, which is another development indicator, had started decreasing in 1960 in SSA and other

LMICs. The under-five mortality rate has remained considerably higher in SSA than in other LMICs.

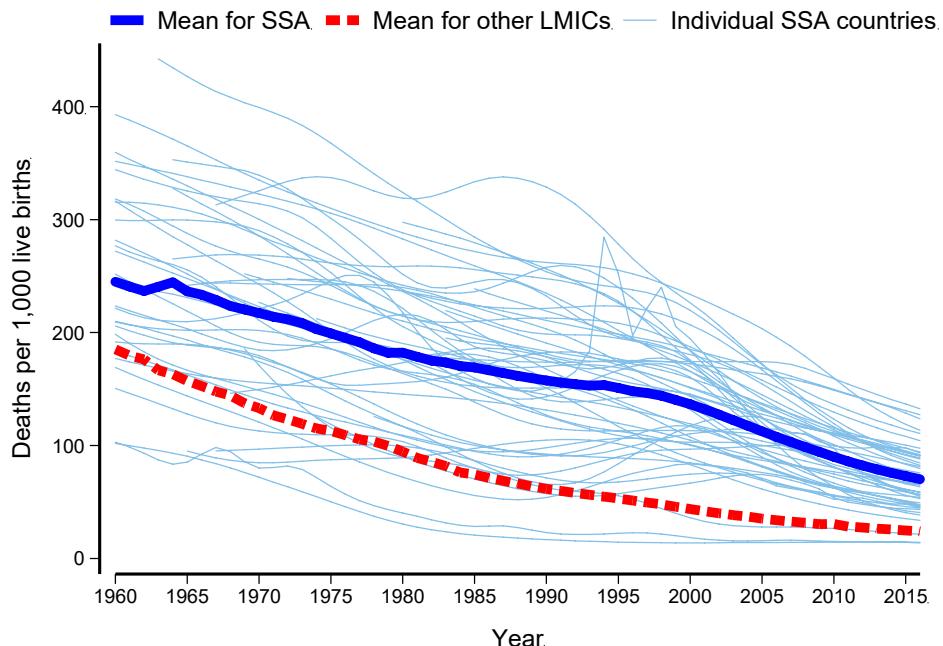


Figure 2. Under-five mortality rate in sub-Saharan Africa (SSA) and other low- and middle-income countries (LMICs)

Note: The mean value calculated for sub-Saharan Africa and low- and middle-income countries is at the country level for each year. Means are unweighted. Source: World Bank (2017).

The underlying cause of low HDI values is poverty. In many countries within the region, people are dependent on subsistence agriculture for their survival. Although a favorable climate for agriculture characterizes some parts of the region, food security is still a critical issue, with malnourishment being common among children and adults. A low level of technology within the agricultural sector reflected in primitive equipment and limited irrigation and fertilizer use undermine food security. Droughts and crop failures further complicate the situation. Integration of the region's markets with international markets is weak, and local markets are unstable. Poor infrastructure constrains the distribution of food, and inadequate storage facilities result in food spoilage and wastage (Devereux and Maxwell, 2001; Shively and Hao, 2012).

Governments in countries in SSA are generally weak leading to ineffective institutions, political instability, corruption, and in some cases, civil wars. Weak

states have limited capacities for raising revenue, which, in turn, results in limited financial support available for infrastructure programs, education, and healthcare. Unstable growth, large informal sectors, and high debt ratios, further constrain financing. Given minimal public expenditure on healthcare, facilities for the provision of health care are insufficient, inaccessible, and ineffective for many within the region, while infections and other health problems are common (Ghura, 1998). There is often an absence of safe water supplies and sewage plants to provide clean water and hygienic toilet facilities. Extensive exposure to infectious diseases, morbidity, and mortality among children in SSA may be primarily attributed to the lack of public initiatives by governments relating to the implementation, monitoring, and maintenance of facilities and infrastructure (Montgomery and Elimelech, 2007). Moreover, the hot and humid climate in some areas and the presence of jungles and rivers create ideal breeding grounds for pathogens (Kalluri et al., 2007).

Although the onset of a fertility transition is evident in SSA, this occurred later and proceeded more slowly than in other regions (Gerland et al., 2017). Whereas for most of the developing world the fertility decline began in the mid-1970s, it did not fully manifest until the mid-1990s in most countries in SSA. Further, differing from other regions, the shift in fertility in SSA began during stages of lower socioeconomic development lower incomes, less schooling, and less urbanization. Currently, however, fertility in SSA is higher than in other developing countries. Moreover, the level of fertility is higher than would be expected for the level of economic development. It appears that demand for children in SSA is higher than in other LMICs, given that contraceptive use is lower, and the desired family size is greater than in other regions (Bongaarts, 2017). There are, however, sub-regional differences. East African countries, such as Rwanda, Malawi, and Ethiopia, where family planning has been prioritized, have experienced a rapid uptake of contraceptives followed by a fertility decline. As a whole, western and central Africa lag behind southern and eastern Africa when it comes to the shifting fertility trend (Mbacké, 2017).

One consequence of declining mortality, with a lower decrease in fertility, is that Africa's population is growing at a fast pace and is predicted to rise from its current proportion of 13% of the global population to 17% in 2030 and 36% in 2100. The continent also has a large share (16%) of children under the age of 5 years, compared with the global share of this age group (9%) (UN, 2017). This youth bulge has led to hopes of a demographic dividend, which has historically contributed to economic growth and development in other regions in the world (Lee and Mason, 2006). Adequate investments in and care of children and youth through the provision of proper health care and education are necessary to ensure the development of human capital and to reap most of the benefits of a demographic dividend (Ahmed et al., 2016; Gribble and Bremner, 2012).

Under-five mortality

The year 2015 was an important year, as it marked the endpoint of the designated period (1990–2015) for achieving the Millennium Development Goal of reducing the under-five mortality rate by two-thirds (Unicef, 2015). This rate was reduced by 55%, globally, during the above period, thus falling short of the goal (WHO, 2015a). Figure 2 shows under-five mortality rates in SSA and other LMICs from 1960 onward. In SSA, the under-five mortality rate declined from 83 to 54 deaths per 1,000 births between 1990 and 2015, and the absolute number of deaths of children less than 5 years old decreased by 24%. In 2015, the region accounted for 50% of under-five deaths globally. After a setback caused by the HIV/AIDS epidemic in the 1990s was overcome after 2000, the declining trend in the mortality rate accelerated. Within SSA, Western and Central Africa have higher death rates for children less than 5 years old (99 per 1,000 births) compared with Eastern and Southern Africa (67 per 1,000 births). However, Angola (in southern Africa) had the highest under-five mortality rate at 157 per 1,000 births. All countries with an under-five mortality rate above 100 per 1,000 births are located in SSA: Angola, Chad, Somalia, the Central African Republic, Sierra Leone, Mali, and Nigeria. Moreover, all of the countries in these regions had under-five mortality rates above 25 deaths per 1,000 births. The SDGs for 2030 seek a reduction in the under-five mortality rate to fewer than 25 per 1,000 in all countries. To achieve this goal, 47 countries, of which 32 are in SAA, will need to accelerate their reduction rates (You et al., 2015).

The primary causes of under-five mortality are preventable diseases. WHO classifies the underlying cause of death according to its “International Classification of Diseases” (ICD-10) (WHO, 2015b). Globally, the causes of child death changed considerably as the rate declined between 2000 and 2015. In 2000, the leading causes of under-five deaths were pneumonia (17%), preterm birth complications (13%), and diarrhea (12%). The most significant reduction in deaths during this period occurred for deaths due to pneumonia, diarrhea, intrapartum-related events, malaria, and measles, all of which were reduced by over 30%, contributing to a 62% reduction in the under-five mortality rate. In 2015, the leading causes of death for children under the age of 5 years were preterm birth complications (18%), pneumonia (16%), and intrapartum-related events (12%) (Liu et al., 2017, 2015).

The share of deaths of children under 5 occurring in the neonatal period has been increasing over time, indicating a reduction in deaths due to infectious diseases. Neonatal deaths accounted for 45% of the deaths of children under 5 years in 2015, increasing from 38% in 2000. Consequently, preterm birth complications have become the leading cause of child mortality, not just among neonatal babies but also among children under 5. The leading causes of neonatal deaths are

preterm birth complications (16%), intrapartum-related events (11%), and meningitis or sepsis (7%). Pneumonia (13%), diarrhea (9%), injuries (6%), and malaria (5%) accounted for the highest proportions of post-neonatal deaths of children under 5 (Liu et al., 2017, 2015).

The mortality rate in SSA is not only higher than in other countries, but it also exhibits a different pattern. Through 2000 and 2015, the share of under-five deaths attributable to infectious diseases was considerably higher in SSA than in other regions, whereas the percentage of deaths in the neonatal period was lower. Over 90% of global deaths from malaria and HIV/AIDS occur in SSA, resulting in a higher prevalence of deaths from infectious diseases in the region. In 2000, malaria was the leading cause of child deaths in SSA, responsible for 17% of under-five mortalities in the post-neonatal period. Over the period 2000–2015, reduced rates of malaria, diarrhea, and measles accounted for 22%, 17%, and 17%, respectively, of the reductions in under-five mortality rate in SSA. In 2015, the leading causes of under-five mortalities were pneumonia (17%), preterm birth complications (12%), and intrapartum-related events (12%) (Liu et al., 2017, 2015).

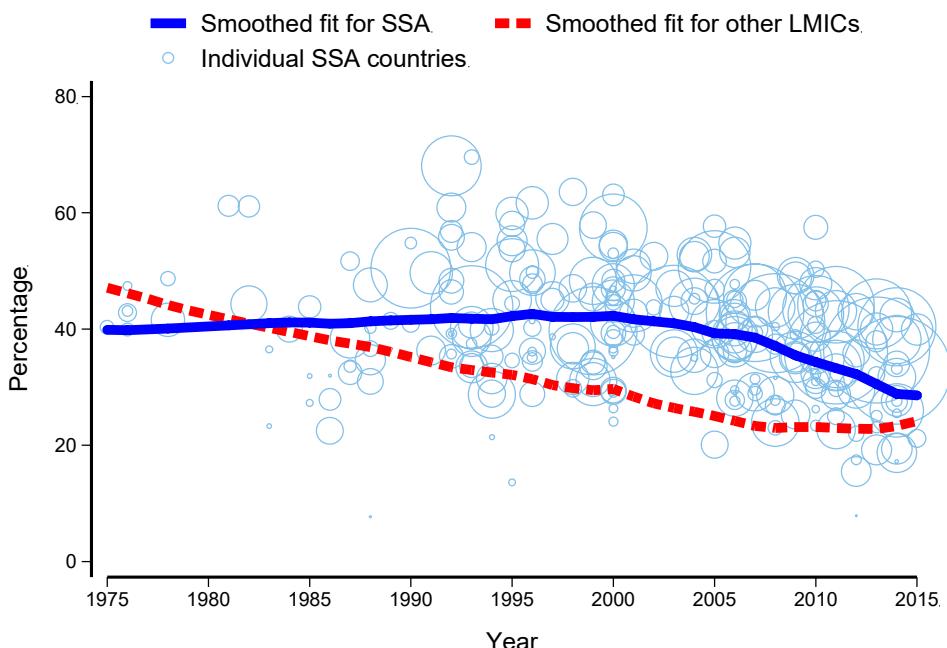


Figure 3. Prevalence of stunting in children under the age of 5 years in sub-Saharan Africa (SSA) and other low- and middle-income countries (LMICs)

The smoothed fit is obtained using locally weighted scatterplot smoothing. Source: World Bank (2017).

The HIV/AIDS epidemic has also had severe consequences for children in SSA. Mother-to-child transmission of HIV, which can happen during pregnancy, birth, and after delivery through breastfeeding, is the most common cause of HIV/AIDS infection among children. It has been estimated that about 60% of infected children die before the age of 5, and in 2002, HIV/AIDS may have caused up to 10% of deaths of children less than 5 years old in the region (Newell et al., 2004). The consequences of the HIV/AIDS epidemic for under-five mortality rate are apparent in Figure 2, reflected in a reduced pace in the mortality reduction from the 1990s up to 2000.

Although the WHO systematically selects a single cause of death, the reality is that under-five mortalities in developing countries are often caused by a sequence of infections and other types of harmful exposure, such as birth complications and undernutrition. Disease and nutrition are also interrelated. Malnourished children are more susceptible to infectious diseases, and infections, in turn, often prevent efficient absorption of nutrients (Ibrahim et al., 2017; Scrimshaw et al., 1968; Walson and Berkley, 2018). Malnutrition is believed to explain about 35% of diseases (Horton et al., 2008) and to be a significant contributing factor in about 50% of under-five mortalities (UNICEF, 2007).

Anthropometric outcomes

Improved survival leads to an increasing focus on the morbidity and healthy development of children. Anthropometric measures are widely used as indicators of child development. Similar to mortality, cumulative exposure to malnutrition and disease determine anthropometric outcomes. Child stunting is a commonly used indicator of chronic undernutrition and exposure to infectious diseases. Figure 3 shows the stunting trend from 1975 to 2015, which decreased rapidly after 2000 in SSA, although it remains greater in SSA than other LMICs. In the 1990s, the global prevalence of stunting was 40%, but this figure dropped to 27% in 2010. In 1990, within SSA, Southern Africa had the lowest prevalence of stunting (35%), followed by West Africa (38%), Central Africa (45%), and East Africa (48%). In 2010, Southern Africa still had the lowest prevalence of stunting, slightly declining to 33%. Western Africa showed no decrease but had the second lowest prevalence in 2010. Central Africa evidenced the most significant reduction in stunting (to 39%) in 2010. Although the highest prevalence of stunting was still in East Africa, it decreased to 45% (De Onis et al., 2012).

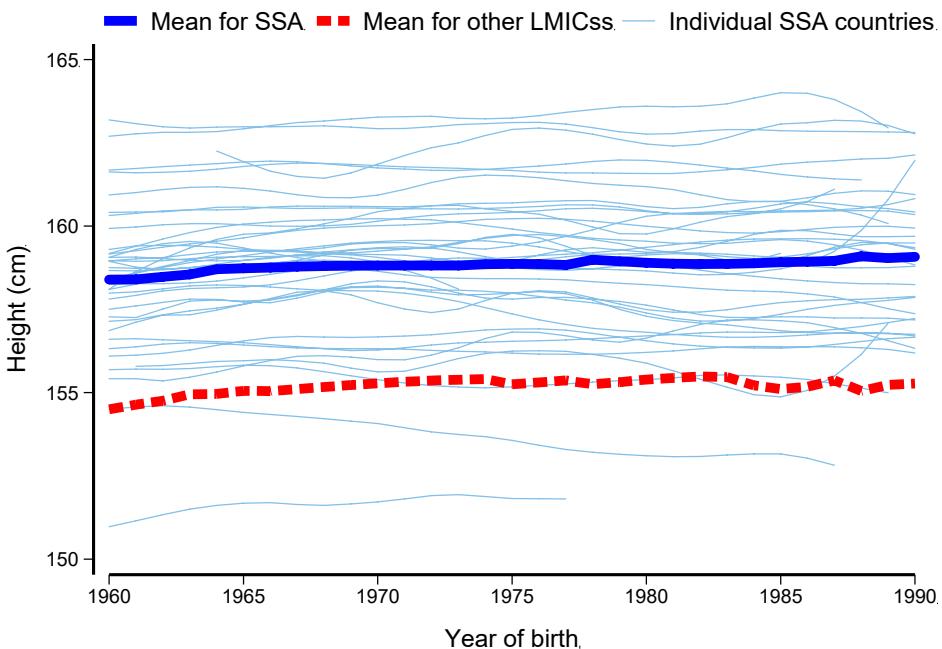


Figure 4. Height (in cm) of women in sub-Saharan Africa (SSA) and in other low- and middle-income countries (LMICs)

All available DHS data are used to calculate annual country-level means for SSA and LMICs. The means are weighted using sampling weights adjusted to sum up to one for each survey. Only women above the age of 21 are included. Source: DHS (2018).

Exposure to diseases and undernutrition in early life also impact on adult height, which is a widely used indicator of cumulative health and living standards in the field of economic history (Steckel, 1995a). Figure 4 shows height trends by birth cohorts for SSA and other LMICs. Although African women are shorter than women in developed countries, their heights exceed the predictions for the adverse environments in which they were raised, and they are considerably taller than women in other developing countries. The reasons for the surprisingly tall stature of African women remain unclear. Part of the explanation may be that their food is not predominantly vegetarian and is not as limited as that of women in developing countries in Asia. Infant mortality has been extremely high, indicating an environment with a high level of disease severity that has been suggested to lead to selection effects in early life, where children with lower health potential are more likely to die, leaving the surviving population taller. Countries in SSA are also less densely populated than many other developing countries, resulting in exposure to fecal germs that are not as chronic and reduced and slower human-to-human transmission of infectious diseases. The surprisingly tall stature of African

women may also be related to catch-up growth during adolescence since stunting rates are generally very high in childhood. A final suggested factor explaining the surprisingly tall stature of women in SSAs is genetic traits (Akachi and Canning, 2010; Deaton, 2013, p. 162, 2007; Moradi, 2010).

Overall, the heights of adult women belonging to all socioeconomic groups in SSA first increased and then decreased for cohorts born during the period 1940 to 1990. However, they varied slightly by region. On average, women in Southern Africa steadily grew taller and faster in urban as opposed to rural areas. Sahelian women showed no change over time in urban areas, but their heights decreased in rural areas. The heights of women in both Central and East Africa increased up to the 1970s but subsequently began to decline (Garenne, 2011; Moradi, 2010).

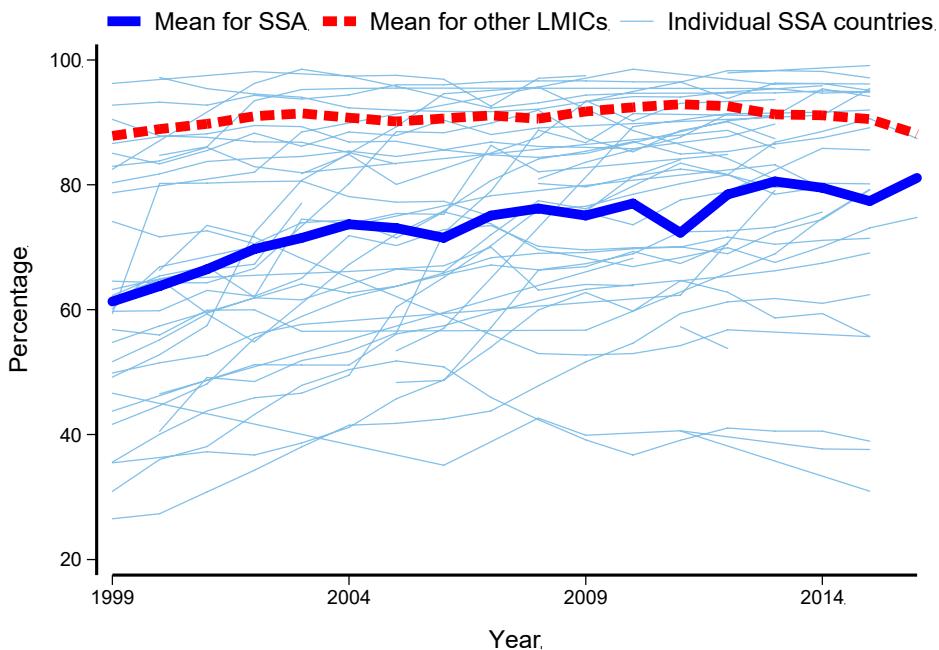


Figure 5. Net primary school enrolments in sub-Saharan Africa (SSA) and other low- and middle-income countries (LMICs)
Annual country-level means are calculated for SSA and LMICs. Means are unweighted. Source: World Bank (2017).

Education

Primary school enrolments have been increasing throughout the developing world, especially in the wake of initiatives such as the Millennium Development Goals

and Education for All. Many countries have abolished primary school fees, thereby making this level of education more accessible for all students. Figure 5 shows increases in net enrollments in primary education since 1999. Net adjusted enrolments in primary school increased from being just over 60% in 1999 to 77% in 2014. Although enrollment rates remain lower in SSA than other LMICs, the gap has been steadily decreasing. Increases in enrolment percentages have been higher for girls (76% in 2014 as opposed to 49% in 1990) than for boys. However, the increase over time in primary school completion rates was less marked (54% in 1990 and 69% in 2014).

The adult literacy rate showed a more modest increase from 57% in 1995 to 61% in 2014. Growing cohort sizes put pressure on the education system, and the rapid increase in the absolute number of students which was not met with sufficient increase in funding may have lowered the quality of education in some cases (UNESCO, 2011; World Bank, 2017). Although primary school attendance has generally reached high levels, school quality and outcomes remain poor and a minority of primary school students have acquired basic skills such as reading, writing, and basic arithmetic skills (ASER, 2015; Bietenbeck et al., 2018; Bold et al., 2017; Hungi et al., 2010; Malpel, 2016).

Interventions

It has been suggested that initial steps toward better health in now developed countries were achieved through better nutrition, improved sanitation, and clean water in parallel with rising incomes (Cutler and Miller, 2005; Fogel, 2002; Kremer and Glennerster, 2011). Most of the subsequent improvements in health that occurred in the twentieth century were the outcome of technological advances rather than income growth (Jamison et al., 2001; Preston, 1975). A large proportion of the reduction in under-five mortalities in developing countries was achieved through transfers of low-cost technologies that were easy to implement. Consequently, life expectancies are significantly higher in the currently developing world than they were in the now developed countries at similar income levels. Sub-optimal investments in public goods, such as water infrastructure, as well behavioral factors related to the uptake of various interventions are the reasons for the continued high rates of infectious diseases in many developing countries (Kremer and Glennerster, 2011).

Different interventions are required to improve child health, depending on the sources of adversity. Tuberculosis, malaria, diarrhea, and lower respiratory tract infections require environmental changes, such as pest control, clean water, and better sanitation. Medical treatments provided within healthcare systems are not sufficiently effective for addressing these threats. However, childhood diseases,

such as whooping cough, diphtheria, polio, measles, and tetanus, as well as perinatal complications and malnutrition, could be improved through better childcare, the provision of guidance before and after delivery, monitoring and vaccinating children, and the availability of health facilities for dealing with emergencies. Solutions to these problems require effective clinics and healthcare systems (Deaton, 2013, p. 121). Parental training in proper nutrition is also essential because of the higher risk of children becoming malnourished after weaning, and the synergetic relationship between infectious diseases and undernutrition (Kwena et al., 2003).

Public health in developing countries has advanced through new inventions, programs, and initiatives, often initiated by non-governmental organizations. In the 1950s, campaigns to promote the provision of penicillin to treat yaws, for example, were instituted. Vector control, aimed at the chemical elimination of disease carriers, such as mosquitos, was also successful, at least initially (mosquitos later became resistant and more powerful agents deemed too harmful to the environment). In the late 1950s, the UN extended the reach of campaigns already implemented in Europe to the rest of the world, aimed at reducing the incidence of tuberculosis, yaws, leprosy, malaria, and trachoma and supported a project aimed at the provision of improved sanitation and clean water. This was followed by other campaigns, such as the WHO's promotion of the DPT vaccine covering diphtheria, whooping cough, and tetanus as well as vaccines for measles, polio, and tuberculosis in 1974. The most recent campaign was launched in 2000, aimed at reinvigorating previous efforts. Some discoveries have helped to reduce infant mortality, such as oral rehydration therapy, entailing oral administration of a mixture of salt, glucose, and water to treat diarrhea as well as the introduction of low-cost mosquito nets to prevent malaria (Deaton, 2013, p. 104).

Theoretical background

Capabilities

The focus on survival, health, and schooling can be attributed to their importance for individual well-being and human development. All of these are integral to the capability approach, which views development as the expansion of capabilities (Robeyns, 2005; Sen, 1990). A set of capabilities enables individuals to achieve various desirable outcomes, referred to as "functionings," which they "value and have a reason to value." The capabilities possessed by an individual constitute a space for potential actions, whereas functionings are the actions materialized by the individual, depending on her or his preferences and norms (Heckman and

Corbin, 2016). Functionings comprise states of “beings and doings,” such as being healthy, working, and being well nourished. Capabilities can be of intrinsic value (functionings that are valuable in and of themselves), of instrumental value (the means for achieving further capabilities) or both. Education and good health are examples of capabilities that are of intrinsic value but can also be instrumental in, for example, securing a job and raising healthy children. In the same way, a child’s health is of intrinsic value but is also instrumental in relation to their school attendance and performance because healthier children are more likely to attend school and perform better than less healthy children (Miguel and Kremer, 2004; Mukudi, 2003). Internal capabilities comprise skills and capacities that are embedded in individuals, such as literacy and health, whereas external capabilities¹ are embedded in their living contexts that include, for example, healthy parents with sufficient resources, access to health care and education, and hygienic and disease-free environments with secure access to food. At birth, and throughout infancy and childhood, individuals have few internal capabilities and are dependent on external capabilities for their well-being and for developing internal capabilities. This dissertation is about the development of internal capabilities in the form of bodily health, survival, and education. External capabilities are considered as determinants that inhibit or aid the development of internal capabilities. Given that the full set of capabilities includes a vast array of skills, capacities, and opportunities, overlapping with individual preferences and needs, their measurement is inherently difficult. The outcomes considered in this dissertation, namely survival, health, and education, commonly feature in the human development literature, and scholars have argued that they are universal fundamental capabilities, independent of any preference (Nussbaum, 2011, 2001).

Human development and capabilities

Heckman offers a framework for studying the accumulation of internal capabilities, or skills and capacities, such as good health (Heckman, 2007; Heckman and Corbin, 2016). A skill production function can demonstrate the

¹ There is no standard definition for the concept of external capabilities (Jackson, 2014). The notion of capabilities as being either embedded in individuals or beyond the individual has been defined and conceptualized differently within the literature. In her earlier work, Nussbaum referred to internal and external capabilities (Nussbaum and Long, 1988). Others have used S-capabilities to denote skills embedded in individuals and O-capabilities to denote the options available to individuals to exercise these skills (Gasper, 1997; Otto and Ziegler, 2006). The idea of external capabilities, being abilities that individuals have to function with the help of interpersonal relations is conceptually different (Foster et al., 2008). However, a general notion of external capabilities as factors external to the individual that inhibit or aid the development or use of capabilities and outcomes (functioning) is well established (Heckman and Corbin, 2016).

process of accumulation of skills and capacities in childhood in relation to this dissertation, encompassing internal and external capabilities.

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

In the above equation h denotes parents' instrumental capabilities for child rearing, θ_1 denotes the initial stock of internal capabilities (endowments at conception such as genes), and I_1, \dots, I_t denotes sets of investments at each stage, that determine the set of internal capabilities at a subsequent stage θ_{t+1} . Investments are resources directed at children such as nutrition, schooling, health care, and illness control. Investments are provided by, for example, parents and governments. The instrumental capabilities for childrearing associated with the parents are, for example, knowledge about proper nutrition, hygiene and illness control, as well as health and material resources. Chronic exposure to diseases and malnutrition early in life has permanent negative effects on children's health and cognitive development (Barker, 1997; Gluckman et al., 2010; Steckel, 2008) and consequently affect their education and future incomes (Case and Paxson, 2006; Currie, 2008; Currie and Moretti, 2007). Because pregnancy and birth complications can also have long-term consequences (Van Handel et al., 2007), maternal health and abilities are of particular importance as they determine child health, in utero, during birth, as well as post-birth, because women are the primary caretakers of children in SSA (FAO, 2014).

Assuming that h is constant, the investments (I_1, \dots, I_{t-1}) and the initial endowments (θ_1) can be replaced by the accumulated stock of internal capabilities (θ_t) and investments (I_t) made at a previous stage t .

$$\theta_{t+1} = f_t(h, \theta_t, I_t)$$

h and I are external capabilities signifying the extent to which a child's circumstances enable her or him to develop internal capabilities in the form of skills and capacities. The stock of capabilities, θ_t , refers to internal capabilities that are instrumental in producing θ_{t+1} . The accumulated stock of internal capabilities augments internal capabilities in subsequent periods through self-productivity. Further, internal capabilities produced at one stage will raise the productivity of investments at later stages; and investments at later stages will also increase the returns to investments made at earlier stages. This is referred to as dynamic complementarities. Self-productivity and dynamic complementarities imply that investments are most efficient in early life (Heckman, 2007). A child who has received sufficient investments in the form of nutrition and disease prevention during infancy will have a stronger immune system. A stronger immune system is an internal capability that allows the child to fight off infections more effectively during subsequent periods, enabling further development of the

child's internal capabilities regarding cognitive abilities and health. If a school-aged child has reached sufficient cognitive development and good health, as a result of adequate investments during previous periods, then investments in the form of primary schooling will be more efficient, as healthier more able children attend schools more regularly and learn more.

Early-life conditions, human development, and anthropometric measures of health

The Heckman production function demonstrates synergistic accumulation of internal capabilities which is cross-fertilizing across periods, which implies that investments made early in life are the most efficient. Early life investments are also of major importance because adverse exposures in early life cause severe and irreversible biological and physiological damage. Consequently, other measures of well-being, such as health, income, and education are affected. Expressions of phenotypes, which influence health and cognition, are most sensitive to environmental impacts during critical periods of developmental plasticity². Critical periods of particular importance are the fetal stage and the first two years after birth when rapid physical growth and development take place (Bateson et al., 2004; Kuh and Shlomo, 2004). The thrifty phenotype hypothesis suggests that nutritional deficiencies in early life lead to reduced growth and a slower metabolism to increase the chances of survival. This adaptation may, however, have detrimental consequences later in life. Limited nutrition may also need to be allocated selectively within the body to save vital organs at the cost of the development of other organs (Barker, 2004; Gluckman et al., 2010; Low et al., 2012). Diseases and malnutrition can operate interactively, and malnourished children are more vulnerable to diseases, and exposure to diseases can influence the ability of their bodies to absorb nutrients. Disease exposure can result in an inflammatory response that may similarly reduce physical growth and cause permanent physiological damage. Energy can also be diverted from growth and development to combatting infections (Finch and Crimmins, 2004; Katona and Katona-Apte, 2008).

Environmental factors, as well as genetics and hormonal factors, also influence physical growth and height (Coutant et al., 2001). In an optimal environment, entailing sufficient nutrition and a low degree of exposure to infectious diseases, different genotypes can give rise to the same stature. Conversely, in contexts of frequent exposure to infectious diseases and poor nutrition, these same genotypes

² Developmental plasticity refers to the ability of genotypes to produce different phenotypes (observable traits) according to environmental cues.

may give rise to shorter but differing heights. In developed countries, it has been estimated that 10–20% of adult height is environmentally determined (Silventoinen, 2003). The environmentally determined component of height is likely to be higher in developing countries than in developed countries since standards of living are lower, and food scarcity and diseases are more severe, making height a good indicator of living standards in these countries (Fogel, 1994; Steckel, 1995b).

Exposure to diseases and undernutrition in early life reduces physical growth and is associated with low birth weight, growth restrictions during early childhood, and ultimately shorter adult height. In the context of early-life conditions, these anthropometric measures are related to human development, mortality, adult health, and consequently education and income (Case and Paxson, 2008; Currie, 2008; Deaton, 2007; Steckel, 1995b). Therefore, birth weight is a widely used indicator of fetal nutrition, growth, and stunting during childhood indicate childhood nutrition and exposures to diseases, and adult height indicates the cumulative health and the net nutritional history (Bozzoli et al., 2009; Currie and Moretti, 2007; Perkins et al., 2016; WHO, 1986).

In a context of impoverishment, children are born slightly below the growth standard set by World Health Organizations (WHO) and continue to diverge from this standard up to the age of 2. After this age, these children's growth is mostly parallel to, but below the growth standard, and ultimately, their adult height reached upon maturity is shorter (De Onis et al., 2006; Eckhardt et al., 2005). Even though deprivation in early life restricts growth, individuals can catch up in height during their adolescence, and as a result of an extended growth period, if nutrition is sufficient and exposures to infections rare. However, chronic and severe exposures in early life most likely always restrict growth and result in shorter adult height, which can be 10–15 cm in the most extreme conditions (Steckel, 1995b, 2009). Further, even though catch-up growth may occur, it is unclear whether it also leads to catch-up in relation to health and other negative consequences of early life adversity (Case and Paxson, 2008).

The negative effects of adverse exposures in early life and reduced physical growth may also have negative consequences across generations, which are examined in the second paper of this dissertation. One biological issue that is raised by these conditions is the growth of the fetus during pregnancy. There are indications that mothers whose birth weights were low, and who are of shorter stature, experience lower weight gain and a higher risk of developing hypertension during pregnancy which puts their children at risk of being born with low birth weights. Growth restrictions in utero also lead to the reduced size of the uterus and ovaries in a woman, which restricts the growth of the fetus, leading to lower birth weight (Ibáñez et al., 2000). Further, in general, the pelvises of shorter women are

narrower than those of taller women, increasing the risk of cephalopelvic disproportion and obstructed labor (Rush, 2000; Sokal et al., 1991). There have also been reports of the practice of “eating down,” or eating less during pregnancy, among smaller women in some cultures to keep the baby small and thereby avoid complications during delivery (Christian et al., 2006). Another potential biological mechanism is transgenerational epigenetic inheritance, entailing a process in which environmental influences on gene expressions are passed down generations (Heard and Martienssen, 2014).

Intergenerational transmission of attributes that can be influenced by conditions during early life appears to be more genetically determined in environments in which children have been able to live up to their genetic potential to a greater extent with less harm from exposures to infections and undernutrition. Turkheimer et al. (2003) found indications that variations in IQ were almost entirely genetically determined in well-off households, whereas they were contingent on the environment to a much greater extent among impoverished families. Similarly, the influence of genetic inheritance on a child’s birth weight was found to be reduced when fetal growth is restrained, for example in first pregnancies and when a pregnant mother smokes (Little and Sing, 1987).

It is unclear to what extent differences in height among distinct populations are environmentally or genetically determined. However, social classes that are economically advantaged within different populations and ethnic groups tend to have similar heights, and immigrants tend to reach similar statures to those of the original inhabitants within the United States and Europe within two generations (Malcolm, 1974; Martorell and Habicht, 1986; Steckel, 1995b). Regardless, the use of anthropometric measures is valid when comparing changes in living standards within populations over time. Genetic determinants of population height are mostly constant over relatively short periods because potential evolutionary changes act slowly, so observed changes in height over the past two centuries most likely reflect improved living conditions.

Numerous studies have linked adversity faced in early life to later life health outcomes, such as adult height. However, several different mechanisms have been suggested. Preston et al. (1998) suggest that the main mechanisms through which adversity in early life can impact on observed health outcomes are scarring, selective mortality, acquired immunity, and correlated environments. Scarring is a mechanism of primary interest as it captures the full extent of the negative health consequences of adversity in early life. Through scarring effects, the overall health of the surviving population, manifested, for example, in stature, shifts downward, resulting in a lower mean for health. Another mechanism is selective mortality, whereby individuals with underlying health problems and lower potential health, are more likely to die when exposed to adversity in early life, compared to

healthier individuals. When selective mortality dominates over scarring, the surviving population is observed to be healthier, as individuals from the lower end of the health distribution are not observed due to selective mortality. The third mechanism, acquired immunity, relates to individuals who survive adverse exposure to influenza, for example, for which immunity can be acquired. Such individuals have been observed to be healthier, as subsequent exposure does not have the same impact on their health. Last, the effect of exposure to adversity in early life may be exaggerated by correlated environments wherein individuals experience sustained exposure to adversity and deprivation over multiple periods and even across generations.

Measuring external capabilities

Infectious diseases, undernutrition, and perinatal complications are the leading causes of mortality and morbidity, obstructing child development in LMICs. However, the extent to which children are exposed to undernutrition and infections is related to the characteristics of the parents, and the communities in which they reside. Mosley and Chen (1984) proposed a framework that is useful for examining the determinants of child health in developing countries. They identified a set of proximate determinants that directly affect mortality or morbidity, such as faltering growth, and all other determinants, such as socioeconomic determinants, must operate through in their effects on child health. There are five broad categories of proximate determinants: maternal factors (e.g., age, parity, birth intervals); injuries; nutrition; environmental contamination (transmission of infectious agents); and illness control (preventive controls such as immunizations and curative controls, such as oral rehydration and antibiotics). Importantly, specific disease states are not treated as causes of illness or death, but rather as indicative of the operation of specific proximate determinants. Faltering growth, and ultimately mortality, is an ideal outcome for modeling such a process as it reflects cumulative exposures to harm rather than an acute phenomenon. In this sense, illnesses are inherently transitory and either result in complete recovery or irreversible consequences for physical growth and survival. Although SES may cause acute events, it primarily affects child health through cumulative exposure to harm via proximate determinants.

Mosley and Chen further suggest specific socioeconomic determinants operating through proximate determinants. For example, they suggest that maternal education affects proximate determinants directly by increasing the capacities of mothers in relation to their feeding practices and skills in preventing and responding to diseases. By contrast, they suggest that paternal education mainly operates via household wealth, although they suggest it also influence attitudes toward reduced fertility and investments in children. Household incomes influence

resources available for the provision of adequate nutrition and health care as well as more sanitary living environments. Additionally, they suggest sociocultural factors, such as religion, impact on traditions, norms, and attitudes, which can affect the value ascribed to children, the use of modern medicine, hygiene, food preferences, and fertility (Mosley and Chen, 1984).

A Review of the literature

Adverse exposures in early life and later life outcomes

A variety of approaches have been applied to demonstrate the critical implications of exposure to diseases and undernutrition in early life for later life outcomes. Studies conducted in the UK have shown that children who had suffered from severe illness and respiratory infections were shorter on average (Kuh and Wadsworth, 1989; Rona and Florey, 1980; Tanner, 1962). A history of disease in early childhood may, however, be endogenous in relation to later outcomes, and would not, therefore, reveal causal effects. Unexpected exogenous events, such as outbreaks of disease and famine often feature in estimates of the causal effects of exposure to adversity in early life on later life outcomes. For example, individuals who were in utero during the Dutch “Hunger Winter” of 1944–1945 when food supplies were cut off for eight months, evidenced higher rates of obesity (Ravelli et al., 1999), type two diabetes (Lumey et al., 2009), schizophrenia (Susser et al., 1996), and schizoid personality disorder (Hoek et al., 1996). Moreover, studies showed that women who were exposed in early gestation had children with lower birth weight (Lumey, 1992), and women who were exposed to the famine during their childhood had increased risks of breast cancer (Elias et al., 2004). Almond (2006) studied the impacts of an outbreak of a short and unexpected influenza pandemic in 1918 that affected roughly two-thirds of mothers giving birth that year and found that those who were in utero during the period of the pandemic had worse health, education, and income.

Another approach that has been used to estimate the effects of early life conditions on later outcomes entails an assessment of temporal variations in ecological indicators that capture or proxy disease environments and nutritional availability. Temporal variations in malaria rates in early life were found to affect educational attainment in the United States (Barreca, 2010), the economic status of men in India (Cutler et al., 2010), men’s productivity in Colombia, the United States, Brazil, and Mexico (Bleakley, 2010), and female education and literacy rates in Sri Lanka and Paraguay (Lucas, 2010). Infant mortality rate is also a commonly used proxy for adverse environments. Finch and Crimmins (2004) found that a high

infant mortality rate in the year of an individual's birth lowered her or his life expectancy by several years. The effects of exposure to adversity later in childhood were not as strong as those experienced in infancy, indicating that exposure to harm is more critical during the period of infancy than during subsequent life phases. Using data from nineteenth-century Sweden, Bengtsson and Broström (2009) found that a high infant mortality rate in the year of birth had a substantial negative effect on wealth accumulation as well as old age mortality. They did not find any evidence that early life adversity affected old-age mortality through acquired wealth, suggesting that physiological damage from infections was the leading pathway. Bozzoli, Deaton, and Quintana-Domeque (2009) found that the prevalence of post-neonatal mortality at birth had a substantial negative impact on adult height in Western Europe and the United States. The post-neonatal mortality rate at birth accounted for as much as 60% of variations in adult height, and most of the increase in stature over the period was attributable to a decline in post-neonatal mortality.

These studies have revealed scarring effects, that is, worse health outcomes in the surviving population caused by exposure to adversity in early life. However, studies have also found that populations exposed to adversity had better health outcomes than those not exposed, indicating that those with worse health potential were more likely to die as a consequence of adverse exposures, resulting in a higher observed mean health value for the surviving population. For example, a study found that individuals exposed to the Chinese famine in early life were taller as adults than those not exposed (Gørgens et al., 2012). A study conducted on adult women in SSA found a positive association with child mortality in the year of birth and adult height (Deaton, 2007). These findings have been attributed to the severity of these adverse exposures, which results in the dominance of selective mortality over scarring effects.

Intergenerational transmission of health

Adverse exposures in early life not only impact negatively on those who experience it but also on their descendants through intergenerational health transmission. Intergenerational transmission refers to the environment, conditions, and exposures undergone by one generation that influences the outcomes of the next, for example in the areas of health, growth, and development. Maternal health is a particularly important factor in determining the health outcomes of children, as it relates to pregnancy, birth, and childcare. As previously discussed, relatively short average heights in many developing countries reflect a history of disease and poor nutrition in early life. The protective role of maternal height on the outcomes of children has been widely reported in studies (Baird, 1964; Donnelly et al., 1964).

Many studies have examined the relationship between maternal health, as measured by height, and child health outcomes in contemporary developing countries. A large-scale study covering 54 LMICs found a robust inverse relationship between maternal height and child health outcomes. The association between maternal height and under-five mortality was strongest in the neonatal period, followed by infancy. This association was consistent, and it was statistically significant for mortality in 46 out of 56 countries studied (Özaltin et al., 2010). In a study entailing a large sample drawn from 38 developing countries, Bhalotra and Rawlings (2011) found a considerable level of intergenerational persistence in health outcomes. They reported that a standard deviation increase in mother's height decreased the risk of her child's mortality in infancy by 7.6% in relation to the average mortality rate in their sample. Adjusting for parental SES and demographic characteristics decreased the intergenerational correlation by 45%, indicating that much of the association is indirect.

Smaller-scale studies have also been conducted. For example, one study found that children of taller mothers are less likely to die before the age of 5 years in Nigeria (Enwerem et al., 2014). In India, maternal height was found to be associated with child mortality, stunting, wasting, and anemia (Subramanian et al., 2009). In Vietnam, Venkataramani (2011) found that a child's height increased by approximately 0.2 standard deviations for a single standard deviation in maternal height (while controlling for paternal height). Another study found an association between maternal height and both fetal growth and gestational age at birth in the Nordic countries (Zhang et al., 2015). The association between maternal height and birth weight was found to be primarily defined by genetics, whereas its association with gestational age at birth was not.

Several studies have examined the correlation between a woman's birthweight and that of her child. Currie and Moretti (2007) found a strong correlation between the birthweights of mothers born in California during the period 1970–1974 and those of their children. A woman born with a low birth weight (less than 2,500 grams) was 50% more likely to give birth to a child with low birth weight when compared to her non-low birth weight sister. Moreover, they found that both the intergenerational correlation in birth weight and the correlation between birth weight and future outcomes were stronger in disadvantaged areas. Thus, they concluded that early-life health shocks are more severe for mothers from economically disadvantaged backgrounds. Addo et al. (2015) found that both maternal and paternal birthweights and early childhood growth were associated with a child's birthweight in several LMICs. The association with maternal growth in early life was higher, indicating the importance of pregnancy-related pathways in accounting for the relationship with child's birthweight and growth.

Using a large sample of children born in the United States during the period 1989–2006, Almond et al. (2012) identified a direct linkage between the disease environment of a mother during her early life and growth and childhood outcomes of her children. They found that white children born to mothers who were exposed to a period of high post-neonatal mortality (a proxy for disease exposure in early life) had a lower birth weight than those born to mothers that were not exposed. The reverse was the case for black children. The authors suggested that this finding may reflect selection, due to greater severity of exposures for black mothers. In a study of a British cohort born in 1958, Palloni et al. (2009) examined the intergenerational persistence of poverty and how it could relate to health during early life. They found that intergenerational transmission of social class could partially be attributed to childhood health. These effects on childhood health also extend to the development of human capital. A further finding was that early-life health is related to the socioeconomic gradient observed in adulthood.

Two studies examined whether more favorable living standards reduced intergenerational health transmission in developing countries using maternal height as a health measure (Bhalotra and Rawlings, 2013; Monden and Smits, 2009). They found a substantially weaker association between maternal height and child mortality for educated mothers compared with less well-educated mothers. However, because maternal education, like height, is also influenced by living standards during early life and childhood, they may be determined simultaneously. Therefore, a more valid approach for testing the impacts of living standards is to explore how factors external to the household interact with maternal height. Monden and Smits (2009) adopted this approach by interacting maternal height with district- and national-level factors, such as GDP, female employment, and the prevalence of hospital deliveries. However, they found an absence of heterogeneity in the association. On the other hand, Bhalotra and Rawlings (2013) found that temporal variations in female education, immunization rates, and income at aggregate levels modified the association between maternal height and mortality. An increase in female education by one year attenuated the association by 17%, and a standard deviation increase in the log of GDP, and in immunization rates, decreased the association by 20%, and 19%, respectively.

Parental education and child health

Intergenerational transmissions of disadvantage are not only observed as health and biological characteristics, such as maternal height but they can also operate through various measures of SES, such as parental education. Parental education reflects past and present living standards, in addition to indicating their attitudes, resources, and skills for childrearing. Several studies have found a strong positive association between maternal education and child health that exceeds associations

for other measures of SES. Caldwell (1979) found a strong association between maternal education and child mortality in Nigeria. Caldwell suggested that maternal education enhanced skills and knowledge and positively influenced attitudes, and receptivity and adaptation to beneficial changes within a modernizing society. More recent studies have shown that maternal education is related to various measures of child health such as under-five mortality (Grepin and Bharadwaj, 2015), and child nutrition (Makoka and Masibo, 2015; Strauss, 1990), independent of other measures of living standards. On a national level, increased female education was found to be associated with improved nutrition globally (Headey, 2013) and to have a stronger association with improved child nutrition than did economic growth in Africa (Harttgen et al., 2013), explaining much of the decrease in the under-five mortality rate (Murphy et al., 2009). Education of girls also appears to be associated with child health beyond the developing context, with studies in the UK and Sweden linking maternal education to child health (Lakshman et al., 2013).

The pathways through which maternal education affects child health have been attributed to a variety of factors, such as increased access to and use of health-related products and services (Onsomu et al., 2015); knowledge, skills, income and SES (Cleland and Van Ginneken, 1988); information use (Handa, 1999; Thomas et al., 1991); assortative mating (Breierova and Duflo, 2004); reduced fertility (Grepin and Bharadwaj, 2015); and female empowerment and attitudinal changes (Caldwell, 1979). The importance of different pathways appears to vary. In some contexts, there is an income pathway relating to parental education (Frost et al., 2005), whereas in others there is none (Handa, 1999). The association of parental education with child health can also vary through its interaction with contextual factors. For example, contextual factors, such as access to health services have been found to be complementary³ to maternal education in one context (Barrera, 1990), substitutes⁴ in another (Thomas et al., 1990), and having no significant interaction with maternal education in a third context (Strauss, 1990).

The particular importance of maternal education above other measures of living standards has been disputed. Desai and Alva (1998) found a much weaker association between maternal education and child nutrition after adjusting for households' living standards and unobserved spatial variations. In their final model, the association of maternal education with child nutrition was only statistically significant in a handful of developing countries. However, they noted

³ I.e., children of educated mothers benefit more from access to health care than children of non-educated mothers, possibly due to greater uptake or better access for educated mothers.

⁴ I.e., access to health service reduces the health disadvantage of children born to non-educated mothers compared to children born to educated mothers, because mothers are to a less extent on their own in securing the health of their children.

that their control variables were inadequate for capturing all aspects of living standards and other possible confounders, both individual and contextual.

Mosley and Chen's study (1984) suggested that paternal education mostly affects child survival through increased income, especially in an urban setting, but a more favorable attitude on the part of educated fathers may also play a role. The role of paternal education in child health has not received as much attention, but several studies have compared this with maternal education. Some studies found an association between child health and maternal education but no association with paternal education (Aslam and Kingdon, 2012), whereas others found an association with both maternal and paternal education, sometimes of similar magnitudes (Breierova and Duflo, 2004; Semba et al., 2008). A large-scale study found a similar association between maternal and paternal education, and child nutrition after adjusting for contextual factors, the educational composition of the household, and interactions between maternal and paternal education (Vollmer et al., 2017a). According to the authors, maternal education reflects living standards more systematically than does paternal education.

It is also unclear whether parental education has a causal effect on child health or whether it is merely a marker of subtle differences in living standards, innate ability (Card, 2001), or genetic and childhood endowments (Behrman and Wolfe, 1987). The results of small-scale causal studies have varied, often depending on the context or the outcomes used (Currie and Moretti, 2003; De Neve and Subramanian, 2017a; Grepin and Bharadwaj, 2015; Güneş, 2015; Lindeboom et al., 2009; McCrary and Royer, 2011). In two studies, the educational reform in Zimbabwe was considered as an exogenous variation allowing causal effects of maternal education on child health to be identified. One study, which used under-five mortality as an outcome, found that children born to women with secondary education were substantially less likely to die (Grepin and Bharadwaj, 2015). The second study found no effect on child stunting using the same natural experiment, (De Neve and Subramanian, 2017b).

Because of the multitude of pathways, confounders, and interactions, it is not surprising that the strength of the association between parental education and child health is context-dependent (Jeong et al., 2018). The association between parental education and child health has been observed to vary between different populations (Desai and Alva, 1998; Jeong et al., 2018), but it is also likely to vary within populations over time, as the context changes. Caldwell (1979) observed that in the 1970s, the association between maternal education and child survival lessened over time. Bado and Sathiya Susuman (2016) found reduced disparities in under-five mortalities in relation to maternal education for later-born children in several countries in SSA born 1990–2015. Between 1992 and 1995, undernutrition of children in urban India declined, whereas inequalities between socioeconomic

groups increased (Kumar et al., 2015). In the state of Sao Paolo in Brazil, disparities in under-five mortalities by household wealth decreased, whereas those related to maternal education increased (Sastry, 2004). Among Norwegian children born during the period 1968–1991, the association between neonatal mortality and maternal education increased over time (Arntzen et al., 1996). It is therefore apparent that the association between parental education and child health not only varies by country, but it also changes within countries over time, and not necessarily in the same way as other socioeconomic measures.

Religious affiliation and child health

Studies on sociocultural factors, such as religion, and child health have been far fewer than those on maternal health and paternal education, and child health. Religion influences behaviors, traditions, and attitudes. Thus, religion and religious affiliation may directly influence proximate determinants, for example, through hygiene, feeding practices, and attitudes towards modern medicine, or through other determinants, such as parental education, productivity, or fertility. However, religious affiliation may also be unrelated to child health beyond merely reflecting social status and access to resources.

Studies have revealed that certain religious groups have significant health advantages over other groups, despite having lower living standards. This finding suggests that behaviors and attitudes that are rooted in religious doctrine may be beneficial for child health. In the nineteenth and early twentieth centuries, child mortality rates among Jews were lower than those of Christians despite Jews having lower living standards in Europe and the United States (Condran and Kramarow, 1991; Derosas, 2003). It has been suggested that these differences are related to the emphasis on personal hygiene in Judaism, which would be incidentally beneficial for child health outcomes (Preston and Haines, 2014). Others have suggested that social isolation of Jews reduced their exposure to infectious diseases, leading to reduced child mortality. Contemporary studies in India have similarly found lower child mortality among Muslims than among Hindus, despite Muslims having substantially worse living standards (Bhalotra et al., 2010). However, studies exploring differences based on community-level religious composition found that child mortality rates among Hindus and Muslims residing in communities with Muslim majorities were similar. It has been suggested that the reason for this finding is the lower rates of open defecation among Muslims, and in Muslim communities, which reduces mortality due to exposures to fecal germs (Geruso and Spears, 2018).

A study by Caldwell (1986) revealed an overlap between developing countries with mostly Muslim populations and those with high child mortality rates.

Caldwell attributed the poor outcomes in countries with significant Muslim populations to the role of women, who had lower levels of education, whose use of family planning was limited, and who had little access to employment outside of the home. It should be noted that Caldwell did not claim that these outcomes were inherent to Islam or that they were immutable; instead, Caldwell suggested that they were embedded in the contemporary cultural context of these regions. West Africa has large populations of both Muslims and Christians, but studies have found that children born to Muslims have worse health outcomes and that Muslim mothers are less likely to use health care. Two studies from Burkina Faso and Nigeria found that vaccination rates were lower among children born to Muslims than among those born to Christians (Antai, 2009; Soura et al., 2013). Utilization of maternal health services was found to be lower among Muslim mothers than among Christian mothers in Ghana. In both of these studies, SES only partially explained these differences, suggesting that they may be rooted in religious teachings. However, a study from Ghana found that SES entirely explained the lower survival of children born to Muslim women (Gyimah, 2007), suggesting that religion may merely reflect differences in living standards and access to resources.

Summary

The theoretical framework guiding this dissertation and the terminology used for the relationships under investigation are those of the capabilities approach pioneered by Amartya Sen (1990). This approach posits that the value of health and education is more than intrinsic, and illustrates the enormous breadth of the determinants of human development. Capabilities determine functionings, that is, what individuals are able to be and do. Whereas internal capabilities are embedded in individuals in the form of skills and capacities, external capabilities, namely freedoms and opportunities, extend beyond individuals, enabling them to achieve functionings both in the present and in the future. For example, in early life, external capabilities, such as parental resources, disease-free environments, and access to health care, are important determinants of both current and future well-being. Heckman's skill production function emphasizes that internal capabilities are acquired through a cumulative process and points to the efficiency of investments in early life, as internal capabilities are self-producing and dynamically complementary, so that "skills beget skills" (Heckman, 2007; Heckman and Corbin, 2016). The importance of investments during the early phase of life is further supported by a large body of literature that documents the importance of early-life nutrition and disease control for human development (Barker, 2004; Gluckman et al., 2010).

Sound health is a critical component of the human development process. Undernutrition and infections along with perinatal factors are the most common proximate causes of child mortality and morbidity in developing countries (Liu et al., 2017, 2015). More remote socioeconomic and sociocultural factors are also widely acknowledged as influencing child development. Mosley and Chen (1984) formalized how socioeconomic factors, such as parental education and sociocultural factors, such as religion, are operationalized in ways that influence child health through nutrition, disease prevention, environmental contamination, maternal factors, and injuries, which are proximate determinants of child health.

Sub-Saharan Africa contains most of the world's least developed countries, where many fundamental capabilities, such as survival, bodily health, and primary education, are far from guaranteed (UN, 2018; UN IGME, 2015; World Bank, 2018, 2017). The opportunity to develop skills and capacities is inhibited by frequent exposures to infectious diseases, food insecurity, lack of services such as health care and education, and lack of infrastructure such as clean water provision. As a consequence, parents and communities in SSA are to a greater extent left on their own in ensuring the healthy development of their children, in an environment that is more hazardous, compared to parents in developed countries, where nutrition is abundant, infections less severe, and health care and education available for most parents. Obstacles to healthy human development persist across generation, and as underprivileged individuals grow up, the toll on their health and socioeconomic status negatively impacts the healthy development of their children.

Previous research has used infant mortality in the year of birth as a proxy for infectious diseases environment and other proximate determinants of child health and demonstrated negative, scarring effects, on the health outcomes of the survivors in adulthood (Bengtsson and Lindström, 2003; Bozzoli et al., 2009). Studies have also indicated that selective mortality may have a more significant impact on the observed health of the surviving population when adversity is severe, such as in many contexts in SSA, biasing downward, or in the most extreme cases, indicating a positive association between early life adversity and health (Deaton, 2007; Preston et al., 1998). The first paper in this dissertation contributes to this literature by testing the impact of a novel measure of adversity experienced in infancy on outcomes observed earlier in the human development process than in adulthood in SSA.

Intergenerational transmission of health from mother to child has been observed in most developing countries, using maternal height as a measure for accumulated health (Bhalotra and Rawlings, 2011; Monden and Smits, 2009; Özaltın et al., 2010). Although a part of this association has been attributed to SES, residual association remains indicating that maternal height is a proximate determinant

impacting child health directly. However, it appears that intergenerational health transmission occurs to a greater extent in disadvantaged environments (Bhalotra and Rawlings, 2013; Currie and Moretti, 2007). The second paper tests whether maternal height is associated with neonatal, postneonatal, and child mortality as well as with school attendance in SSA, and quantifies the extent to which the observed association is accounted for by various dimensions of living standards, demographic factors, and contextual and paternal characteristics. Whereas previous studies focused exclusively on child health outcomes, this study also explores the impact on school attendance. Last, the paper contributes to the literature through its assessment of whether public spending on health in SSA reduces intergenerational health transmission.

Children do not only inherit the health disadvantages from their parents; parental education has also been found to impact on children's health outcomes. Numerous studies have identified a positive association between parental education and child health, attributing it to various pathways (Caldwell, 1979; Vollmer et al., 2017a). However, social determinants of child health, such as parental education, are dynamic and highly context-dependent (Desai and Alva, 1998; Jeong et al., 2018). With recent advances in child health, education, and related factors, the context in LMICs has changed, which may have an impact on the observed association. The third paper explores the association between both maternal and paternal education with child health in LMICs and assesses whether the observed association has changed over time, as LMICs have developed. Also, it contributes to the literature through quantification of the statistical impacts of various dimensions of living standards and demographic and contextual factors, both for the observed association between parental education and child health and changes in the associations that have occurred over time.

Sociocultural factors, such as religion, have received much less attention than parental health and education. Studies have suggested that parental religious affiliation may be causally linked to child health via religiously prescribed behaviors and attitudes (Caldwell, 1986; Gyimah et al., 2006; Jarvis and Northcott, 1987). However, other studies have suggested that the differences in child health between religious groups can be explained by differences in living standards and access to resources (Gyimah, 2007). Muslim mothers' utilization of health care facilities has been found to be less than that of Christian mothers; a difference that is not fully explained by observed differences in living standards (Antai, 2009; Gyimah et al., 2006; Soura et al., 2013). Conversely, differences in SES fully explained the worse health outcomes observed for children born to Muslims compared to Christians in Ghana (Gyimah, 2007). The third paper revisits the topic of the Muslim disadvantage in child health and explores differences in child health outcomes between Muslims and Christians in 11 West and Central African countries. The main contribution of this paper lies in its assessment of the

relevance of geographic clustering of these religious groups—which only share a living environment to a limited extent—in accounting for observed differences in child health. The paper further explores the implications of community-level religious composition, and the statistical impact of various dimensions of living standards, health care use, and demographic characteristics.

Data and methods

Data

The empirical analyses presented in this dissertation are based on microdata obtained from the nationally representative DHS surveys conducted in numerous developing countries. Standardized questionnaires and measures are applied in the surveys, which are comparable across survey years and between countries, to obtain reliable data on population health and nutrition as well as other characteristics. The sampling is based on stratified multi-staged sampling. Stratification is commonly based on the type of residence (urban or rural) crossed by administrative or geographic regions. The primary sampling units (PSU) are sampled within each stratum, based on a probability proportional to population size, and using a sampling frame of geographically constructed areas, which are often the enumeration areas of the most recent census. Households are sampled within the second-stage sampling frame, and women aged 15–49 years within these households are interviewed by trained interviewers about their health, birth history, children’s health, partners, and household characteristics. In most of the surveys, all women in the 15–49 year age group are interviewed. However, in a few cases, only married women are interviewed, and in others, females aged 10–49 years old are interviewed. The response rate in the DHS surveys is very high in SSA, being approximately 98% and 97% for households and individual women, respectively, during phases II and III (Vaessen et al., 2005). Non-responses are not replaced, but numerous measures are included to ensure high response rates. Smaller population segments are oversampled to acquire an adequate number of observations for analysis. Sampling weights are provided to adjust for oversampling, non-responses, and for greater precision. Weights applied to both respondents and households are calculated as the inverse of the probability of being included in the survey (Aliaga and Ren, 2006).

The DHS provides various types of data files (recode) arranged according to the units of analysis. The main type of recode used in this dissertation are birth recodes, in which each record is a single birth from the birth history of interviewed women, constituting the source of outcomes such as mortality and anthropometric

measures. The other outcome used in this dissertation, school attendance, was recorded in a household member recode, which contains basic information on each household member. In one analysis, a variable for community-level religious composition was calculated using the individual recode, entailing one record per respondent. The surveys are appended, and then the different recodes are merged using the identifiers provided in each survey as well a survey identification number attached to each data file.

Although efforts have been made to provide high quality and accurate data, there are some apparent deficiencies. Age heaping is a persistent problem, with respondents disproportionately reporting certain ages, such as ages ending in zero or five. On average, 5% of respondents misreported their ages, showing a digit preference (Lyons-Amos and Stones, 2017). Omissions of births from birth histories are another problem. It has been suggested that this problem is most common for births which occurred at younger ages among older cohorts. However, there have also been indications that omissions from birth histories may occur for more recent births, more commonly for deceased children and children of uneducated mothers (Schoumaker, 2011). Date-of-birth displacements have also been reported where birth dates are altered beyond a certain age threshold, for which more detailed information should be obtained. A detailed health questionnaire is administered for respondents with children below 5 years old, which may have motivated interviewers to displace birth dates that occur close to five years before a survey, to a date just after the threshold time for the detailed health questionnaire. Similarly, the ages of respondents may be displaced to below 15 years and above 49 years (Pullum, 2006).

The DHS surveys do, however, have numerous benefits. Primarily, they enable researchers to study a wide range of demographic and health-related topics in multiple developing countries where civil registration systems and health and education data are generally lacking. The major benefits of these surveys lie in their broad geographical scope and their use of a large number of standardized variables. Moreover, they are extensively used in the fields of demography, economics, epidemiology, and public health, thereby facilitating more efficient research and communication among scholars.

Methods

Outcomes

Anthropometric measures are applied as outcomes for children in three of the papers. The WHO provides standards for the growth trajectory of healthy children (WHO, 2006a). The 2006 WHO growth standards are constructed using data from the WHO Multicentre Growth Reference Study which include breastfed infants

and appropriately fed children growing up in optimal conditions and of various ethnic backgrounds (WHO and UNICEF, 2009). Height-for-age z-scores reflect the standardized distribution of height, by age and sex, indicating standard deviations from the WHO growth standards.

$$\text{height-for-age z-score}_{\text{as}} = \frac{\text{cm}_{\text{as}} - \bar{\text{rcm}}_{\text{as}}}{\sigma_{\text{rcm,as}}}$$

The height-for-age z-score of an a -month old child, of sex s , is calculated by subtracting the median height of the corresponding age and sex in the reference population (rcm), from the child's height (cm) and divided by the corresponding standard deviation of the reference population (σ). A z-score corresponds to a standard deviation from the reference median. Recumbent length is usually measured for children under 24 months old, while height is measured for older children.

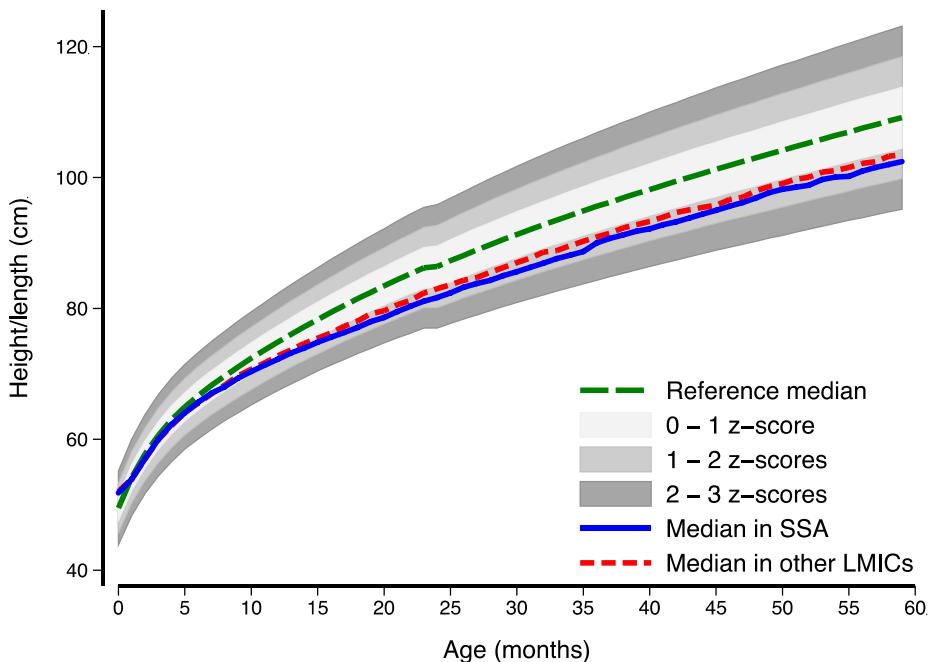


Figure 6. The 2006 WHO growth standard compared with those in sub-Saharan Africa (SSA) and other low- and middle-income countries (LMICs)

Medians are calculated using all available DHS data and are weighted using sampling weights adjusted to sum to one for each survey. The WHO growth standards released in 2006 show averaged growth for males and females. Source: DHS (2018); WHO (2006a).

Figure 6 shows the growth trajectory based on the WHO growth standards (2006) with the z-scores (or standard deviations) in relation to that trajectory. It shows that the height-for-age of children in SSA and other LMICs is slightly greater than the reference population in the first month, indicating that measurement errors or selective mortality may be a problem in relation to measures of neonates. Measurements for children in both SSA and other LMICs start to deviate from the growth trajectory as they advance in age, indicating cumulative exposure to infections and undernutrition. The deviation is greatest for children in SSA at around 24 months when they start catching up with the WHO growth standards, although only to a minimal extent.

Anthropometric measures are often dichotomized. Child stunting indicates chronic undernutrition, defined as two height-for-age z-scores below the reference median. Other common indicators are wasting, defined as two weight-for-height z-scores below the reference median, indicating acute malnutrition, and underweight, defined as two weight-for-age z-scores below a reference median, which is a composite indicator for chronic and acute undernutrition (WHO, 2010). Dichotomized measures are particularly useful in population-level comparisons and trends as they can be presented as prevalence measures. In analyses of individual data, continuous measures can be preferable, as exposure to undernutrition and diseases in early life shifts the entire distribution downward.

In two papers of this dissertation, school attendance is used as an outcome in relation to maternal height and adverse exposures in infancy. School attendance is a binary indicator of whether 7–16-year-olds attended school at the time of the survey. Children enter primary school when they are either 6 or 7 years old for the duration of 6 to 7 years (UNESCO, 2011). In three papers of this dissertation, under-five mortality is used as an outcome that indicates whether a child died between birth and the age of 59 months. In the paper on maternal height, all births from the compiled birth histories are included, whereas, in the respective papers on parental education and religion, only births occurring 0–59 months before a survey are considered. Information on health care use, such as whether or not a child was born in a health facility, is generally only recorded for births occurring less than 60 months prior to a survey. Because use of healthcare facilities is one of the mechanisms through which parental education and religion are thought to impact on child health, only births occurring less than 60 months before the surveys are considered for these papers.

As discussed above, the underlying causes of neonatal, post-neonatal, and child mortality are somewhat different. Therefore in the paper on maternal height, under-five mortality is separated into these three phases of mortality. Neonatal mortality is approximated as death within the first month, post-birth. Post-neonatal mortality refers to the death of a child who survived the neonatal period but died

between the ages of 1–12 months. Child mortality refers to the death of a child who survived the first 12 months but died between the ages of 12 and 59 months. Children below the age of one month at the time of the survey are excluded from the analysis of post-neonatal mortality, and children below the age of 12 months at the time of the survey are excluded from the analysis of child mortality because they had not entered the risk period. Descriptive statistics on mortality, especially child and under-five mortality are likely to be underestimated because not all children lived through the risk period.

Gelbach decomposition and Mundlak's fixed effects

Gelbach decomposition is used in three of the papers to estimate the statistical impacts of covariates on the relationships of interest (Gelbach, 2016). Estimates entailing the use of multiple regression models are common in social science research, beginning with a simple model and subsequently adding control variables, stepwise, to account for the statistical impacts of different covariates on the relationship of interest. The goal can be to comment on how much of the mean difference for a specific outcome between categories is attributable to category-level heterogeneity in the covariates. For example, the question of the extent to which observed differences in the height between children born to educated and non-educated mothers are due to differences in measures for fertility, SES, and use of health care facilities between the two groups can be considered⁵. The main problem entailed in the use of sequential addition of covariates to comment on the statistical impact of each of the added covariates on the relationship of interest is that the order in which variables are entered influences the results. To avoid this problem, Gelbach proposed a method of decomposition based on the formula for calculating omitted variable bias.

In practice, the decomposition is conducted by first running a restricted regression with outcome y , the exposure variable of interest e , and a vector x containing any number of basic control variables.

$$y = \alpha^{\text{basic}} + e\beta^{\text{basic}} + x'\rho^{\text{basic}} + \varepsilon^{\text{basic}}$$

Here the outcome y denotes height, exposure e denotes maternal education, and x denotes a vector of some control variables, e.g., the child's age and sex.

⁵ The interpretation of the decomposition is the same regardless of whether a correlation coefficient for years of education is decomposed or mean difference in height between children born to educated mothers compared to non-educated mothers.

Subsequently, another regression is run that includes an additional set of covariates:

$$y = \alpha^{\text{full}} + e\beta^{\text{full}} + \mathbf{x}'\boldsymbol{\rho}^{\text{full}} + \mathbf{z}'\boldsymbol{\gamma} + \varepsilon^{\text{full}}$$

Here \mathbf{z} contains some number of variables, for example, household income, number of siblings, and the number of vaccines received. The objective is to estimate the independent statistical impacts of each of the additional covariates from \mathbf{z} , which appear in the unrestricted regression only, on the parameter of interest β^{basic} .

The difference between the two estimates is calculated as:

$$\delta = \beta^{\text{basic}} - \beta^{\text{full}}$$

δ is the total impact of all the covariates in \mathbf{z} on the association between maternal education and child height, adjusted for \mathbf{x} . In this example, \mathbf{z} entails three variables, namely income, vaccines, and siblings. Therefore, $\mathbf{z}' = z_1, z_2, z_3$. Three auxiliary regressions are run, the first for household income, which is expressed as follows:

$$z_1 = \alpha_1 + \alpha_{1e} e + \alpha_{1x}' \mathbf{x} + \omega_1$$

Here z_1 denotes household income. The same process is followed for the other two covariates, siblings, and vaccines. In the above equation, α_{1e} denotes the association between maternal education and household income, independent of \mathbf{x} . The total impact on β^{basic} attributable to all three covariates is calculated as:

$$\delta = \beta^{\text{basic}} - \beta^{\text{full}} = \sum_{f=1}^3 \gamma_f \alpha_{fe}.$$

The statistical impact of the income measure on the association between maternal education and height is obtained using the following calculation:

$$\delta_1 = \gamma_1 \alpha_{1e}$$

Therefore, the impact of the income covariate is determined first by the strength of the association between income and child's height, independent of vaccinations, number of siblings, age, and sex (γ_1), and second by the strength of the association between maternal education and income, independent of age and sex (α_{1e}). The same applies to the other two variables in \mathbf{z} , namely vaccines and siblings.

The decomposition allows for any number of basic controls in \mathbf{x} and any number of covariates in \mathbf{z} . The impacts can easily be summed up for a set of covariates relating to similar mechanisms. For example, rather than using just the number of siblings to capture fertility, a set of variables, such as the number of siblings, maternal age at birth, and birth interval can be added. Consequently, the total statistical impact of the measures for fertility is the sum of the estimated impact of each of the covariates relating to fertility.

All of the analyses in this dissertation use fixed effects, which control for group-level heterogeneity, at the level of the survey, region-ethnicity, community (primary sampling units), mother, or father, respectively. Mundlak's fixed effects are used when conducting the Gelbach decomposition by adding a group level mean of all valid observations, for all independent variables (Antonakis et al., 2010; Mundlak, 1978). This procedure yields group-level parameters that can be used to decompose the statistical impact of between-group heterogeneity on the relationship of interest.

Two alternative methods are widely used to control for group-level heterogeneity, which are, however, not feasible when estimating the impacts of group-level heterogeneity using the Gelbach decomposition. The first method entails adding a dummy coded variable for the group. However, because the number of groups can number in the hundreds of thousands, this is not computationally efficient. A second conventional method of adjusting estimates for a large number of groups is to subtract a group-level mean of valid observations, from each independent variable. However, this method does not provide any group-level estimates, which are needed in the Gelbach decomposition to estimate the statistical impact of between-group heterogeneity on the relationship of interest. All three methods for controlling for group-level heterogeneity yield parametrically identical estimates for variables measured below the group level. Further, the impact of using Mundlak's fixed effects within the Gelbach decomposition is parametrically identical to that of decomposing a set of group-level dummy coded variables.

Summary of papers

Paper I: Scarring and selection in sub-Saharan Africa: The effects of adverse environment in infancy on health and education

High infant and child mortality, as well as the high prevalence of stunting, indicate the difficult conditions that infants and children are subjected to in SSA, where diseases and undernutrition are significant causes (Horton et al., 2008; Liu et al.,

2017, 2015; UNICEF, 2007). Other sources of adversity include, for example, indoor air pollution emanating from cooking fuel, which causes pneumonia (WHO, 2014). Human development is a process in which inadequate investments in critical areas, such as disease prevention and nutrition, during one life period, such as infancy, can have detrimental effects on outcomes during subsequent periods, and, ultimately, in adulthood (Bozzoli et al., 2009; Heckman and Corbin, 2016). There is extensive literature that links undernutrition and disease exposure in early life to outcomes in later life, such as cognitive abilities, earnings, education, and health (Almond, 2006; Barker, 1997; Bengtsson and Lindström, 2003; Case and Paxson, 2008; Finch and Crimmins, 2004). These studies indicate the occurrence of a scarring effect, whereby adverse exposures shift the entire distribution of health downward, resulting in a lower mean for health. Studies have also found evidence of a selection effect, whereby children with underlying health problems are more likely to die than healthier children when faced with adversity as a result of which the surviving population appears healthier (Deaton, 2007; Gørgens et al., 2012). It has been suggested that the selection effect may dominate in conditions of severe adversity, for example, in many countries in SSA.

Paper I tests the influence of an adverse environment during infancy on subsequent child health and school attendance in SSA. The contributions of this paper are threefold. First, a novel spatiotemporal indicator for adverse environment experienced in infancy is constructed using geocoded birth histories obtained from household surveys. Contrasting with previous studies that have relied on national-level measures of adversity, measured for calendar years, this indicator provides more variance in adversity as it is more geographically and period specific. Second, the effects of an adverse environment in infancy on observable outcomes during a part of the human development process that precedes adulthood are assessed. Previous studies have focused mostly on observable outcomes in adulthood, which may obscure some of the negative impacts of adverse exposures in infancy, which may be especially important in a context such as SSA, where selection effects may dominate scarring effects on health outcomes in adulthood. Third, the paper examined whether the observed impacts of an adverse environment in infancy are less severe in better-off households in SSA and whether there are differences in the effect by sex.

For the above analysis, data at the individual, household, and community levels extracted from DHS surveys conducted in 33 countries in SSA are used. The outcomes considered are height-for-age z-scores that are indicative of nutrition available for growth after accounting for disease, and school attendance. The incidence rate of postneonatal mortality (PNM) occurring within a 50 km radius of the place of birth during the period of infancy is considered an indicator of an adverse environment. The interactions of an adverse disease environment with household living standards are assessed to determine whether children in better-off

households are less impacted. OLS models with fixed effects at the levels of the survey, community, and mother are applied.

The results indicated that an adverse environment in infancy negatively affects the height-for-age of the surviving children. However, the magnitude of the effect, which is small, is only observed at the lower end of the PNM distribution. The association with school attendance is also found to be weak and negative at the lower end of the PNM distribution, but it is positive at higher levels. Households' socioeconomic status does not appear to modify the effect. Higher selective mortality at high levels of adversity may offer an explanation for the absence of negative effects at high levels of adversity.

Paper II: Maternal height and child development in sub-Saharan Africa: Underlying mechanisms and the role of public spending on health⁶

Intergenerational transmission of poor health from parents to children is well established within the academic literature. Breaking these negative intergenerational cycles can substantially improve the health of populations in developing countries. In poorer countries, such as those in SSA, children are at a higher risk of inheriting their parents' poor health because poor health as well as low levels of household resources are aggravated by rampant diseases and vulnerability to food insecurity; lack of services, such as health care; and a poor health environment. Although SSA accounts for 11% of the world's population, its health expenditure accounts for only 1% of global health expenditure. Given that 50% of under-five mortalities occur in this region and that it bears 24% of the worldwide disease burden (WHO, 2006b), the need for adequate health care in SSA is substantially higher than in other regions. Human development, adult health, and related outcomes are compromised by infections and undernutrition in early life, which reduces physical growth and are, reflected in short average stature of women in SSA (Moradi, 2010; Silventoinen, 2003; Steckel, 1995b).

Studies conducted in a large number of LMICs have used maternal height and under-five mortality to assess intergenerational transmission of health from mother to child (Bhalotra and Rawlings, 2011; Monden and Smits, 2009; Özaltın et al., 2010). Suggested pathways include physiological factors and compromised human development from harmful exposures in early life, which influence adult health, and which subsequently determine income and education levels (Currie and Vogl,

⁶ Paper II was co-authored with Martin Dribe. Both authors contributed to the study concept and design, the interpretation of findings, and the drafting of the manuscript. The author of this dissertation acquired the data and conducted the statistical analysis.

2012). Hence, the mechanisms underlying the association of maternal height and child health can reflect a variety of factors, such as income, education, accumulated health, and related factors as well as physiological factors entailing impacts on the growth of the fetus during pregnancy and risk during birth.

Studies have examined interactions between maternal height and modifiable factors at the household, district, and national levels. At the household-level, outcomes of children born to educated mothers were found to have a weaker association with maternal height. However, maternal education reflects mother's past living standards, which is also a major determinant of adult height, so maternal height and education are therefore determined simultaneously, to an extent (Bhalotra and Rawlings, 2013; Monden and Smits, 2009). A few studies have therefore focused on variations in modifiable measures of living standards beyond the household with mixed results. Monden and Smits (2009) found no interaction effects between national-level GDP, district-level health care use and economic status, and maternal height, for under-five mortality. However, Bhalotra and Rawlings (2013) found an interaction between maternal height and changes in aggregate national-level factors at the time of birth, with improvements in GDP, female education, and vaccination rates reducing the strength of the transmission.

This paper is aimed at advancing understanding of the intergenerational transmission of health, using maternal height, in two ways. The first entailed an exploration of potential mechanisms. The association of maternal height with various outcomes that have different underlying determinants is first estimated, and the extent to which the associations are attributable to measures likely to overlap with maternal height (i.e., maternal education, fertility, household SES, and community and paternal characteristics) is quantified. The second contribution entailed an examination of whether increased public health expenditure has led to decreased intergenerational transmission of poor health in SSA. The paper also contributes to the literature in other ways. The first is the focus on SSA, which is the most impoverished region in the world with the worst child health outcomes, the lowest health expenditure, and a high proportion of disadvantaged households. The second contribution relates to the consideration of other outcomes apart from health and longer-term consequences of maternal height for human development through the inclusion of school attendance as an outcome.

Data at the individual, household, and community levels are extracted from multiple DHS surveys conducted in 35 countries in SSA between 1992 and 2016. These data are linked to data on annual government health expenditure for the period 1995–2015, obtained from the WHO Global Health Expenditure Database (WHO, 2017). The findings of the analysis are in line with those of previous studies, demonstrating a consistent relationship between maternal height and under-five mortality that is statistically significant in all but five countries. The

association with neonatal mortality appeared to be direct to a greater extent than the association with mortality at a later stage in childhood. Of the covariates, community-level factors appeared to have the most explanatory power, followed by household SES and fertility. Paternal and household characteristics explained most of the association with child mortality for children in polygamous households, but they do not account for any of the association with neonatal and post-neonatal mortality. However, after accounting for covariates, a robust residual association remained for all mortality outcomes. There is also an association between maternal height and school attendance, which, however, is weak and not statistically significant in most countries, and mostly explained covariates for community-level factors, maternal education, and SES. Government health expenditure substantially weakened the association between maternal height and under-five mortality, but this is not the case for education. These results indicate that physiology is the most important underlying mechanism behind the association between maternal height and under-five mortality, but interventions such as public health expenditure around the time of birth can substantially reduce its persistence, especially for infant mortality.

Paper III: Weakening association of parental education: analysis of child health outcomes in 43 low- and middle-income countries⁷

The World Bank and the UN have recommended maternal education as a cost-effective way to improve child health in LMICs (Jamison et al., 1993; United Nations, 2014, 1994; Veneman, 2007). Studies have found that the association of child health with maternal education is stronger than for other measures of SES (Caldwell, 1979; Grepin and Bharadwaj, 2015; Harttgen et al., 2013; Headey, 2013; Lakshman et al., 2013; Makoka and Masibo, 2015; Murphy et al., 2009; Thomas et al., 1990). This association has been attributed directly to proximate determinants of child health, such as illness control and nutrition, whereas the effect of paternal education is mostly attributed to increased earnings (Mosley and Chen, 1984). The suggested pathways through which maternal education affects child health include SES (Cleland and Van Ginneken, 1988), skills (Handa, 1999; Thomas et al., 1991), assortative mating (Breierova and Duflo, 2004), reduced fertility (Grepin and Bharadwaj, 2015), use of health care facilities (Elo, 1992; Onsomu et al., 2015), female empowerment, and attitudinal changes (Caldwell, 1979).

⁷ Paper III was co-authored with Jan-Walter De Neve and S.V. Subramanian. All of the authors contributed to the study concept and design, the interpretation of findings, and the drafting of the manuscript. The author of this dissertation acquired the data and performed the statistical analysis. The paper has been published in the International Journal of Epidemiology (Karlsson et al., 2018).

Other researchers have shifted from an exclusive focus on maternal education, emphasizing the need to understand pathways and heterogeneity in the association (Subramanian and De Neve, 2017). The association between maternal education and child health demonstrates a very high degree of heterogeneity between countries and is rarely statistically significant for individual countries after accounting for community-level factors and household living standards (Desai and Alva, 1998; Jeong et al., 2018). Further, studies have found that the associations of each parent's education with child nutrition are of similar magnitudes (Vollmer et al., 2017a). Estimating a causal effect of parental education on child health is difficult because unobserved variables such as innate ability (Card, 2001) and other endowments (Behrman and Wolfe, 1987) determine both education and child health, leading to confounding bias. Education policy reforms have been exploited and used as "natural experiments" which provide causal estimates, but these studies have yielded conflicting results (Currie and Moretti, 2003; De Neve and Subramanian, 2017a; Grepin and Bharadwaj, 2015; Güneş, 2015; Lindeboom et al., 2009; McCrary and Royer, 2011).

As the literature suggests, the relationship between parental education and child health operates through multiple pathways, is likely to be confounded, and is highly context-dependent. Changing contexts in LMICs entail modernization and improvements related to child health and education as well as factors related to the suggested pathways and confounders. Therefore the relationship between parental education and child health is likely to have changed over time. A few studies have observed changes in the association between maternal education and child health. Caldwell observed attenuation over time in the association between maternal education and child survival in the 1970s (Caldwell, 1979). There was a modest decline in the differences in child undernutrition by maternal education between household surveys conducted in developing countries before and after 2000 (Vollmer et al., 2017b). The gap in under-five mortality rates between educated and uneducated mothers was lower for more recent birth cohorts in several countries in SSA (Bado and Sathiya Susuman, 2016).

A large sample of mutually comparable and nationally representative repeated cross-sectional studies from 43 LMICs are used to test changes in the associations of parental education with under-five mortality, stunting, wasting, and underweight status over time. The study explored the impact of measures of early-life conditions of mothers, assortative mating, fertility, households' living standards, use of healthcare facilities, urban-rural differences, and geographic clustering, in addition to differences between countries relating to changes over time. The results showed that the association of parental education has attenuated to a considerable extent over time. Underlying factors driving these changes appear to be related to the changing impact of fertility, households' living standards, and urban-rural differences on the association. Moreover, the

association of child health with nutrition is being increasingly driven by geographic clustering rather than by individual-level associations. However, an association remains between the education of both parents and all child health outcomes.

Paper IV: Child health disparities by religious affiliation in West and Central Africa

Religion is a major social force that plays a critical role in the lives of people in SSA. Over 90% of surveyed respondents stated that religion is very important in their lives (Lugo and Cooperman, 2010). Many countries are also religiously mixed with large numbers of both Christians and Muslims. Limited studies have been conducted on the sociocultural factors and child health, even though religion has an impact on norms, values, attitudes, and behaviors, while also reflecting SES. Some religions prescribe healthy behaviors relating to nutrition, hygiene, reproductive health, and substance use. Moreover, religion influences other known determinants of child health, such as education, productivity, and fertility (McQuillan, 2004). Differences in child health between religious groups have also been attributed to social and economic disparities (Gyimah, 2007; Jarvis and Northcott, 1987).

At the individual level, studies have found that Christian mothers demonstrate better health-related behaviors than Muslim mothers in West Africa and their children have been observed to have lower mortality rates (Gyimah, 2007; Gyimah et al., 2006; Soura et al., 2013). However, these studies have not fully accounted for geographic clustering of religious groups and community-level religious composition. Although Muslims and Christians coexist within national borders in West and Central Africa, these religious groups are highly clustered geographically and only share a living environment to a limited extent.

This paper studies the implications of geographic clustering of religious groups, as well as community-level religious composition, for child health. First, differences in outcomes between children born to Christians and Muslims are tested by comparing Christians and Muslims within religiously diverse communities. Secondly, children born to Muslims and Christians in communities that are religiously homogenous are compared. In addition, the implications of community-level religious composition are explored, for example, whether being a religious minority has any implications for child health. Last, the statistical impacts of living standards and demographic characteristics for the observed health differences are decomposed to identify the possible underlying factors linking religious affiliation and child health.

The results showed that in religiously mixed communities, there is no statistically significant difference in child health outcomes between Muslims and Christians, indicating that the observed discrepancies relate to geographic clustering of religious groups rather than to the direct effects of religious affiliation. All-Muslim communities have worse child health outcomes than all-Christian communities, which appears to be explained to a large extent by living standards, use of health care facilities, and fertility, although some residual differences remain.

Discussion and conclusion

Human development is aimed at expanding capabilities that enable individuals to function and lead a good life. Fundamental capabilities are survival, bodily health, and basic education. Internal capabilities, or skills and capacities, accumulate throughout the life course, with early life and childhood being particularly important phases of accumulation. External capabilities provide individuals with the freedom and opportunities to develop and use capabilities. Examples include a disease-free environment with a secure supply of food and access to health care and education, as well as parents with sufficient resources. Whereas fundamental capabilities are almost guaranteed for children in developed countries, children in the developing world face challenges which jeopardize their survival, bodily health, and basic education. As a region, SSA has the highest rates of under-five mortality and out-of-school children as well as high levels of stunted physical growth. The main proximate determinants of child health are undernutrition and infectious diseases, as well as perinatal complications. Moreover, parental factors have been found to be important, and significant health disparities persist between groups.

This dissertation aims to explore the implications of context for the relationship between parental factors, and child health and school attendance in SSA as well as in other LMICs. Many household-level factors are geographically clustered, and parents with more resources are more likely to reside in favorable environments that independently determine child health and school attendance. For example, healthier and more educated parents are more likely to live in an environment with available education, health care facilities, and nutrition. Contextual environments are also correlated over the life course and across generations. Further, many of the proposed pathways behind parental determinants are related to contextual factors. For example, Mosley and Chen suggest that mother's education relates to her child's health by "influencing her choices and increasing her skills in health care practices related to contraception, nutrition, hygiene, preventive care, and disease treatment" (Mosley and Chen, 1984, p. 35). Therefore, the effectiveness of

parental education, or resources and skills more broadly, in determining child health evidently varies in accordance with the external environment with regard to, for example, the availability of health care, nutrition, contraceptives, health care, and clean water.

The first paper shows that the incidence rate of postneonatal mortality, which is a proxy for adverse environment in infancy measuring a large number of proximate determinants, affect physical growth negatively. However, the effect is small and is only observed at the lower end of the adversity distribution. For school attendance, there is also a small negative effect at the lower end of the distribution, but this becomes positive with higher levels of adversity. This finding indicates that adverse exposures in infancy can influence outcomes later in childhood, but also that selection effects may dominate in SSA, even when adversity is at relatively low levels. This finding supports the suggestion that selection dominates over scarring in contexts entailing high levels of adversity, such as in SSA (Deaton, 2007). The second paper shows that in most countries in SSA, maternal height appears to have a direct negative association with under-five mortality, independent of covariates, which corroborates findings from previous studies (Bhalotra and Rawlings, 2011; Monden and Smits, 2009; Özaltin et al., 2010). Further, because the association is strongest and most direct in the neonatal period, it is likely to be related to pregnancy and perinatal factors. The relatively limited impact of covariates indicates that maternal height is mostly a proximate determinant of child health. However, the association between maternal height and school attendance is mostly explained by other measures of living standards. Although the proximate determinants considered in these studies, namely maternal height and an adverse environment in infancy, appear to have an association with health outcomes in early childhood, they do not appear to negatively influence school attendance consistently or strongly. This may be because of the inadequacy of school attendance for capturing skill formation; a better measure would be achievements in school, such as grades.

The third paper shows that there is also a consistent association between parental education and child health in most LMICs, which is generally stronger for maternal education than for paternal education. This finding is in line with previous studies (Caldwell, 1986; Jeong et al., 2018). Mosley and Chen (1984), Caldwell (1986, 1979), and other researchers have suggested that maternal education is a socioeconomic determinant of child health that is of particular importance, as it is directly related to various proximate determinants. By contrast, paternal education is suggested to operate mostly via income. However, for all child health outcomes, the statistical impacts of covariates follow very similar patterns for maternal and paternal education, both for the main associations and for changes in these associations over time, which may indicate that maternal and paternal education may operate similarly in their relationship with child health.

Differences may, however, lie in the residual association. The fourth paper shows that, overall, in West and Central Africa, children born to Christian mothers have better health outcomes than children born to Muslim mothers, which corroborates previous findings (Antai, 2009; Gyimah et al., 2006). However, the difference in outcomes appears to be between all-Muslim and all-Christian communities and is mostly explained by observable measures of living standards, fertility, and use of healthcare facilities, although some residual differences remain.

The overarching research question of this dissertation is: “To what extents do contextual factors explain the observed disparities in child health by maternal health, parental education, and religious affiliation?” The results of this dissertation show that contextual factors matter for all parental factors, but to varying degrees. Contextual factors, measured as community-level factors explained about 12% of the relationship between maternal height and under-5 mortality, and about 21% for maternal height and school attendance. Community-level factors have greater explanatory power regarding under-five mortality rates than households’ living standards, whereas for school attendance community-level factors have less explanatory power than household living standards. Further, community-level factors explained a significant portion of the association of maternal education with under-five mortality (25%), stunting (16%) and underweight (18%) in the oldest set of DHS surveys, increasing to 42%, 27%, and 33%, respectively, in more recent DHS surveys. Similarly, community-level factors also explain a significant portion of the association of paternal education with under-five mortality (16%), stunting (13%), and underweight, (15%) in the oldest set of surveys, increasing to 40%, 25%, and 28%, respectively, in more recent DHS surveys. Community-level factors account for more of the associations between parental education and mortality than households’ living standards but less than fertility, whereas households’ living standards account for a greater portion of the relationship of parental education with stunting and underweight. The statistical impact of community-level factors on the associations has, however, increased over time for all outcomes (although not statistically significant for all outcomes), while the explanatory power of households’ living standards and fertility has decreased. Last, Muslims are only found to have worse child health outcomes in religiously homogenous communities in West and Central Africa. No differences are found between Muslims and Christians residing in religiously mixed communities, indicating that observed health differences are related to contextual factors.

The four sub-research questions focus on how parental factors interact with contextual factors at various levels. In relation to the first sub-question, children in better-off households, approximated by maternal education, are not found to be less affected by adverse environments in infancy. This indicates that better-off households do not mitigate harmful effects by investing more in children before,

during or after exposure to adverse environment in infancy. The findings relating to the second sub-question show that even though maternal height has a very consistent relationship with mortality in SSA, this association is reduced with an increase in public health spending at the national level. This finding indicates that public health spending benefits mothers with poor health and facilitates the care of children in disadvantaged households.

The findings relating to the third sub-question indicate that although parental education is consistently associated with under-five mortality and child health in LMICs, this association has decreased over time. Parental education does, however, still have an association with all child health outcomes in most countries. Further, over the investigated period, an urban penalty relating to child health appears to have emerged, which suppresses the association between parental education and child health, thereby contributing to the attenuation in the association over time. Conversely, geographic clustering appears to have increased over time, especially in the case of stunting. Consequently, the observed association is increasingly driven by the clustering of educated parents and healthy children in the same communities. Finally, with regard to the fourth sub-question, religious composition appears to have implications for children born to Muslims in West and Central Africa, where children in all-Muslim communities have worse health outcomes than children born to Muslims in religiously mixed communities. Most of this difference is explained by household living standards and the use of health care, but some residual differences remain.

In conclusion, the primary results of this dissertation reveal that context partly explains the relationships between parental factors and child health, but to varying degrees. A substantial part of the association between parental education and child health is explained by community-level factors, whereas community-level factors have less impact on the relationship between maternal health and mortality. However, both parental education and maternal health show a strong protective association with child health independent of community-level factors and a wide range of other measures of living standards, indicating that they are important indicators of external capabilities which give children the opportunity to survive, grow, and accumulate internal capabilities. However, the link between religious affiliation and child health appears to be mostly related to geographic clustering of religious groups, which reflect living standards and access to resources. Further, parental factors, especially socioeconomic and sociocultural ones, but also proximate determinants such as maternal height, vary in their relationships with child health depending on the context of the household. Improved contexts appear to weaken the link between parental education and maternal health, and child health. These findings underline the importance of considering the external context in which parental factors operate. SSA, as well as other LMICs, are going through

a rapid societal, economic, and epidemiological transition, which may change how parental factors influence child health.

The SDGs are aimed at improving survival, health, and education as well as reducing disparities by, for example, SES and ethnicity (UNICEF, 2018). Under-five mortalities in SSA and other LMICs increasingly occur in the neonatal period, indicating a shift from infectious diseases as the most critical factors influencing child survival, to preterm birth complications and perinatal factors (Liu et al., 2017, 2015). Maternal health needs to be ensured not only during pregnancy but also in her early life because the accumulated health of mothers appears to have a direct influence on survival beyond the neonatal period in SSA. Parental education has intrinsic value and also continues to be positively associated with child health in most LMICs. Disparities in child health between Muslims and Christians in West and Central Africa can be reduced through improved material living standards and access to, and use of health care facilities within Muslim communities. Improving the public health environment and preventing adverse exposures to infectious diseases and undernutrition is crucial for human development.

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Paper I

Paper I

Scarring and selection in sub-Saharan Africa: The effects of adverse environment in infancy on health and education

Abstract

Infants in sub-Saharan Africa are exposed to adversity, such as diseases and undernutrition, which can have long-lasting consequences for human development regarding health and education. In this paper, an indicator for adverse environment in infancy is created, and its effect on height-for-age and school attendance explored. Further, effect heterogeneity by household socioeconomic status and sex is considered. The results show that adverse environment in infancy negatively affects height-for-age in ages 1–4 and school attendance in ages 7–15, but only at the lower end of adversity distribution. At high levels of adversity, a positive association is observed for school attendance.

Introduction

Adverse conditions facing infants in sub-Saharan Africa (SSA) are detrimental to their subsequent health and educational development. Even though SSA is home to only 11% of the world's population, it carries 24% of the disease burden, has 50% of under-five deaths, and has high rates of stunting from chronic undernutrition and exposures to infectious diseases (De Onis et al., 2012; You et al., 2015). Further, health care and public health interventions are inadequate, and the region represents only 1% of global health expenditures (WHO, 2006a). Infections spread easily in environments that often lack clean running water and modern toilet facilities. Hot and humid climates in some areas make ideal breeding grounds for disease vectors, such as malaria-carrying mosquitos. Food is often scarce and of poor quality (Bain et al., 2013). Undernutrition and infectious diseases are major causes of both mortality and stunting and operate in synergy as infections inhibit the body's uptake of nutrients and undernutrition increases the body's susceptibility to disease (Katona and Katona-Apte, 2008).

Human development is an accumulative and synergistic process where health and capacities acquired at one stage impact the production of health and capacities at subsequent stages (Heckman and Corbin, 2016). Underinvestment and harmful exposures in one period—such as infancy—can have detrimental effects on the outcomes in subsequent periods and adulthood. The process starts in utero, where adequate maternal nutrition and health investments play a crucial role in the development of the fetus (Barker, 1997). More importantly, after birth, undernutrition and inflammatory responses to infectious diseases can have irreversible consequences for health and cognitive development, and consequently affect income and education (Case and Paxson, 2008; Crimmins and Finch, 2006; Finch and Crimmins, 2004). Health at the schooling age can determine educational outcomes, as healthier children are more likely to attend school (Alderman et al., 2001; Brooker et al., 1999). Restricted growth (especially over the first two years after birth), and shorter adult height, reflects inadequate nutrition and frequent infections in early life (Case and Paxson, 2008).

An extensive literature has identified negative effects of adverse exposures, such as undernutrition and infectious diseases, in infancy—using ecological indicators such as infant mortality rate—on outcomes in adulthood including earnings, education, and health (Bengtsson and Lindström, 2003; Bozzoli et al., 2009; Case and Paxson, 2008). However, in regions with extreme levels of adversity, such as SSA, adversity in early life has been found to affect observed adult height positively (Deaton, 2007). This demonstrates that adversity not only takes a toll on the health of the surviving population through scarring, but can also cause selection effects where those with underlying health problems and lower health

potential are more likely to die from harmful exposures, leaving the observed population, on average, healthier (Bozzoli et al., 2009; Preston et al., 1998).

This paper studies the influence of temporal variations in adverse environment during infancy on survivors' physical growth and school attendance in SSA. The paper's first contribution is the construction of a new proxy for adversity in infancy using the incidence rate of post-neonatal mortality (PNM) over the period of infancy in a 50-kilometer radius from the place of birth. The second contribution is the assessment of outcomes observed in the human development process before adulthood, as previous studies did find negative effects of early life adversity on adult health in SSA (Deaton, 2007). A final contribution is the testing of whether there is heterogeneity in the observed impact of PNM in infancy according to household socioeconomic status, and sex.

The first outcome used is the height-for-age z-scores of children aged 12–59 months. These height-for-age z-scores indicate the nutrition available for physical growth after accounting for disease. The second outcome is an indicator of whether 7–15-year-olds attended school in the year of the survey. School attendance is likely to be related to accumulated health as studies have shown child nutritional status—measured by height-for-age—to determine school attendance in Ghana, Tanzania, and Pakistan (Alderman et al., 2001; Brooker et al., 1999). This paper uses OLS models with survey, community, and mother fixed effects.

Background

Several studies have found that children who suffered infections in early life have a lower height later in childhood than children that did not. According to a study on primary school children, born in 1972 and 1973 in England, who suffered a respiratory infection in the preceding year were 0.4 cm shorter, on average, than their peers (Rona and Florey, 1980). Children suffering severe illness in early childhood were found to be up to 2 cm shorter on average in a 1946 longitudinal study from the U.K (Kuh and Wadsworth, 1989). Another study conducted in a similar setting found that three-year-old children with a history of bronchitis and pneumonia were up to 2.5 cm shorter on average (Tanner, 1962). Disease history in early childhood may, however, be endogenous to later outcomes (Johansson, 2004).

A common approach used to estimate the effects of early life adversity on later life outcomes is to link ecological indicators approximating adversity to year of birth (or other periods in early life). These studies have focused on outcomes in adulthood such as wealth accumulation, life expectancy, height, and education. Temporal variation in malaria rates in early life has been found to affect the

following outcomes: educational attainment in the United States (Barreca, 2010), the economic status of men in India (Cutler et al., 2010), men's productivity in Colombia, United States, Brazil, and Mexico (Bleakley, 2010), and female education and literacy rates in Sri Lanka and Paraguay (Lucas, 2010).

Several researchers have used national-level infant, post-neonatal, or under-five mortality rates as proxies for an adverse environment. Many children suffer from infectious diseases, and a proportion of them die. Thus, mortality rates reflect the extent of the adversity caused primarily by infectious diseases (Bozzoli et al., 2009). Finch and Crimmins (2004) found that a high infant mortality rate in the year of birth reduces life expectancy by several years. The effects of exposure to adversity later in childhood were not as strong which indicates that harmful exposures in infancy are more critical. Using data from 19th century Sweden, Bengtsson and Broström (2009) found that infant mortality rate in the year of birth had an adverse effect on wealth accumulation and old age mortality. Bozzoli, Deaton, and Quintana-Domeque (2009) argued that post-neonatal mortality at birth is a better indicator for disease environment facing infants. They found it to have a substantial negative impact on adult height in Western Europe and the United States.

The four major mechanisms discussed in the literature on early life exposures and later outcomes are scarring and acquired immunity—which are direct effects of early life adversity on later health—and correlated environments and selection—which are indirect effects (Preston et al., 1998). Scarring refers to the long-term negative effects of adversity in early life on survivors, and scarring effects shift the whole distribution of health downwards. On the other hand, a direct consequence of exposure to infectious diseases for which immunity can be acquired, such as influenza, is better adult health for survivors. This is because subsequent infections will not have the same negative effects on those who obtained immunity due to early life infection as on those who have not acquired immunity. Acquired immunity is, however, mostly discussed in relation to adult mortality (Lee, 1997; Preston et al., 1998) and is not considered as important when studying, for example, adult height (Bozzoli et al., 2009), and may, therefore, be less relevant to this paper. Correlated environments suggests that children exposed to adversity in early life will continue to suffer adversity in later periods because their environment—measured by indicators such as household living standards or external epidemiological environment—often remains similar throughout their life (Elford et al., 1991; Mare, 1986; Preston et al., 1998). Not accounting for correlated environments would bias the observed effect between early life adversity and later health outcomes upwards.

Finally, in some cases, the observed association between adverse environment in infancy and later outcomes can be biased downwards, canceled out, or in the most

extreme situations, be positive, as a result of selective mortality. If those with lower potential health, due to underlying health problems are more likely to die due to a harmful exposure in early life, such as a famine or an epidemic, than those with better health potentials, the observed mean health of the surviving population is greater. For example, a selection effect was observed for survivors of the 1959-61 famine in China: survivors exposed in childhood were 1-2 cm taller as adults than those not exposed (Gørgens et al., 2012). Similarly, for women in SSA, there was a positive association between child mortality rate during the year of birth and adult height which suggests that selection dominated over scarring (Deaton, 2007). These findings are consistent with the idea that selection starts to dominate at high levels of adversity, such as those existing in SSA, while scarring dominates in more moderate contexts, such as in preindustrial Europe (Bozzoli et al., 2009). Further, Akachi and Canning (2010) found a lack of increase in adult height despite a substantial reduction in infant mortality in SSA, as is generally observed in the rest of the world. They argued that health is multidimensional, and the decline in infant mortality in SSA may be related to interventions that prevent infant deaths but do not improve nutrition and morbidity. For example, oral rehydration therapy and vaccination reduce infant mortality rates, but they do not prevent impairments to physical growth caused by undernutrition, acute respiratory infections, malaria, and diarrhea.

The current study

This paper makes three main contributions. First, it constructs a spatiotemporal indicator for adverse environment in infancy using geocoded birth histories from household surveys. The indicator is calculated as the incidence rate of post-neonatal mortality (PNM) over the period of infancy in a 50-kilometer radius from the place of birth. Previous research on developing countries has mostly used national-level proxies for adversity for each calendar year, which provides less variation in adversity than this paper's strategy of using an indicator that is period and area specific. As in previous studies, the PNM indicator provides variance in the intensity of adversity, which is exogenous to child health outcomes. However, not all children face adversity when PNM is high, and some children face adversity even when PNM is low, which has similar implications as non-compliance in randomized controlled trials, meaning that the estimates will reflect intention-to-treat (ITT) effects (Almond and Mazumder, 2011; Van den Berg et al., 2016). Further, although PNM mostly reflects infectious disease environment (Bozzoli et al., 2009), other negative exposures such as undernutrition, air pollution, or armed conflict cannot be ruled out as contributing to adversity. However, all of these factors contribute to restricting human development reflected in physical growth and school attendance (Currie and Vogl, 2012; WHO,

2014). The second contribution is that this paper assesses the effects of adverse environment in infancy on outcomes observable early in the human development process, before adulthood, i.e., childhood and adolescence. In most parts of the world, there is a negative correlation between early life adversity and adult health, but this has not been observed in SSA (Akachi and Canning, 2010; Deaton, 2007). In addition, adult height in SSA is closer to heights in privileged settings while growth failure during childhood is much prevalent, indicating that catch-up growth may occur between childhood and adulthood (Coly et al., 2006; Steckel and Ziebarth, 2016). Therefore, adult height may not adequately reflect childhood morbidity in SSA.

A third contribution is that we test whether there is heterogeneity in the effects of adverse environment in infancy according to socioeconomic status (measured by maternal education) and sex. Better-off households can invest more in the health of their children, which improves these children's accumulated health and makes them less susceptible to infections. Better-off households can also shelter their children during periods of elevated adversity by preventing and responding more effectively to infections. For example, better-educated mothers have been reported to use health services more often and to administer treatments—such as oral rehydration therapy to treat diarrhea—more efficiently (Taha, 2002). Greater post-exposure investments in better-off households may also ameliorate some of the negative impacts of adversity. Whether there is heterogeneity in the effects of adverse environment also depends on the nature of the adversity: where highly virulent, non-nutrition-dependent infectious diseases are the primary cause of adversity, children from better-off households are less likely to be sheltered (Hedefalk et al., 2017). Finally, socioeconomic heterogeneity may relate to the measure of adversity used in this study, reflecting ITT effects, and the actual exposure to adversity may differ by household socioeconomic status. As for heterogeneity by sex, scarring or selection mechanisms may be greater for males, as males generally have both higher mortality and of stunting rate due to biological frailty (Rutstein, 2005; Sawyer, 2012; Stinson, 1985; Wamani et al., 2007).

Data and Methods

The analysis of height-for-age is based on 83 Demographic and Health Surveys (DHS) from 33 countries in SSA, and for school attendance, it is based on 58 surveys from 29 countries. The DHS are nationally representative household surveys where 15-49 years old women are interviewed and data are collected on the topics of their health, household context, birth histories, and children's health. The DHS use two-stage sampling where first primary sampling units (PSU) are sampled and then households are drawn from each PSU. The PSUs are relatively small geographic units, such as a village or an urban neighborhood. Most DHS

surveys provide GPS¹ coordinates for these PSUs, which are used in this study to create the proxy for adversity in infancy.

Two outcomes are studied. The first is child height-for-age z-scores, which are a standard deviation from a median growth trajectory of healthy children according to the World Health Organization (WHO) 2006 growth standards (WHO, 2006b). Lower height-for-age is indicative of chronic undernutrition and high disease exposure. Typically, only children under five years old have their height-for-age measured in the DHS, while children less than twelve months old at the time of the survey are excluded as they have not yet lived through the risk period. This means that the children included in each survey are born within, at most, a four-year interval. The second variable is an indicator of whether 7– 15-year-olds attended school in the year of the survey.

Adverse environment is approximated using the postneonatal mortality² rate (PNM) in a 50-kilometer radius from the place of birth³ (PSU) over the period of infancy. According to the WHO (2015) “International Classification of Diseases” (ICD-10) the most common causes of death in SSA during the postneonatal period are malaria, diarrhea, measles, and pneumonia (Liu et al., 2017, 2015). Although the WHO systematically selects a single cause of death, mortality is usually a result of an accumulation of exposure to both infections and undernutrition. Undernutrition is believed to be a significant causal component in about 50% of under-five deaths and to explain about 35% of the disease load in developing countries (Horton et al., 2008; Liu et al., 2017, 2015; UNICEF, 2007). Food security may, however, be less relevant for infants as children are commonly breastfed for much of the period of infancy. Neonatal deaths are not included since they indicate preterm birth complications and perinatal factors rather than disease exposure (Liu et al., 2017, 2015).

¹ The GPS locations of the PSUs are displaced to protect anonymity. Urban clusters are displaced between 0–2 kilometers. Rural clusters are displaced between 0–5 kilometers. Further, a randomly selected 1% of rural clusters are displaced between 0–10 km (Burgert et al., 2013).

² Neonatal mortality is approximated as death in the first month of life rather than in the first 28 days.

³ Due to the lack of information on migration, we assume that the PSU of the survey is the same as the PSU of birth. This could cause misclassification if mothers migrated after giving birth. Of all respondents who answered the question “Years lived in place of residence,” 43% of births were from respondents who had never migrated and 89% were from respondents who did not migrate after giving birth to their first child.

The incidence rate, pnm_{cm} , is calculated for each cluster, c , and each month of birth⁴, b , as

$$pnm_{cb} = \frac{\sum_b^{b+12} \text{deaths}_{c<50\text{km}}}{\sum_b^{b+12} \text{at risk}_{c<50\text{km}}} \times 12\,000.$$

The numerator is the number of post-neonatal deaths occurring in a 50-kilometer radius from the PSU of birth over the period of infancy, and the denominator is the total person-months at risk in a 50-kilometer radius from the PSU of birth over the period of infancy⁵. Sensitivity analysis suggests similar results for 25- and 75-kilometer radii. The indicator is multiplied by 12 000 so that it indicates the number of deaths for every thousand children that survive infancy. Since the PNM distribution is right-skewed it is converted to a logarithmic scale which gives a normal distribution of PNM. When presenting the results, PNM z-score is calculated using the sample mean and standard deviation. In order to obtain more precise estimates, the indicator is limited to areas and periods with a minimum of 360 person-months (or 30 person-years) of exposure and five deaths. Consequently, the results reflect more densely populated and more extensively surveyed areas. However, sensitivity analysis indicates that the results are not sensitive to alternative restrictions on the denominator. Not restricting the number of deaths to at least two gives similar results for school attendance but non-significant results for height-for-age.

All DHS surveys from SSA that include GPS coordinates are pooled when calculating PNM. Further, sums of person-time of exposure and deaths do not necessarily occur within the same survey. Surveys which do not record the outcome variables used may still be used to calculate the PNM.

The models are specified as OLS:

$$y_{icb} = \alpha_c + \beta_1 pnm_{icb} + \beta_2 pnm_{icb}^2 + \boldsymbol{\theta}' \mathbf{x} + \varepsilon_{icb}, \quad (\text{Eq.1})$$

where y is one of the two outcomes, \mathbf{x} is a vector of controls, and α_c is a survey-, community-, or mother-specific intercept. A squared term for PNM is also included to allow for nonlinearity due to selection at high adversity levels. The predicted outcomes are calculated for Eq.1 holding all covariates at their means.

⁴ Most respondents (87%) gave complete information on their children's dates of birth, meaning that they specified both the year and month of birth. Further, there is no indication of age heaping for the children. There is, however, age heaping for information on children's age at death, every six months. Due to age heaping for age at death, deaths of children at 12 months old are classified as post-neonatal death.

⁵ See appendix A1 for details.

Further, the average marginal effect is shown across the distribution of PNM by taking the first-order partial derivative of Eq.1 with respect to PNM

$$\frac{\partial}{\partial pnm} = 2 \times \beta_2 \times pnm + \beta_1$$

for pnm between -3 and 3 z-scores.

The preferred model uses community fixed effects. A community-level mean is subtracted from all included observations for all independent variables, which controls for unobserved community-level heterogeneity and is equivalent to adding a dummy-coded indicator for each community. Similarly, fixed effects at the survey and mother level are used, where siblings born to the same mother or children measured in the same survey are compared. However, when using mother fixed effects and child height-for-age z-score as an outcome the sample may be less representative because only mothers that have had at least two children (who are not twins) in a four-year period are included in the models. When using survey fixed effects both temporal and spatial variation in PNM are used to identify the effects, but since some areas have persistently higher PNM rates the variation in exposure to PNM captures correlated environments and adversity more generally.

Table 1 shows descriptive statistics for variables used and their functional form in statistical models. The complete set of controls is only used in the survey fixed effects models. Maternal education and the number of siblings ever born are excluded from models using mother fixed effects as they do not vary between siblings. These covariates are related to child health so their inclusion in statistical models reduces standards errors. With the exception of age in months, these covariates are unlikely to reflect confounders. Not accounting for age may cause the effect of PNM in infancy to be overestimated. Older children have on average been exposed to higher levels of adversity since there has been a decline in postneonatal mortality in SSA over time. Further, in deprived contexts, children deviate away from the WHO growth standards until about 24 months of age (De Onis et al., 2006; Eckhardt et al., 2005). Therefore, older children have had more time to accumulate exposures, which decrease their height-for-age, and have on average been exposed to a higher level of PNM, due to the secular downward trend in mortality, which can bias estimates. Similarly, as mortality has trended downwards, height-for-age and school attendance have been trending upwards which may also bias the estimated effect of PNM on height-for-age and school attendance. A bias relating to age is more of a concern when analyzing school attendance since children in that sample can be born up to nine years apart, whereas a bias is less of a concern when analyzing height-for-age sample since children are born at most 4 years apart in that sample.

Testing whether the effects of adverse environment in infancy vary by socioeconomic status is explored by interacting PNM with an indicator for whether the mother of the child has had any education. Maternal education is chosen as an indicator of socioeconomic status as it is more frequently recorded in the DHS than, for example, paternal education or household wealth. The interaction models are specified as

$$y_{icb} = \alpha_c + \beta_1 pnm_{icb} + \beta_2 pnm_{icb}^2 + \beta_3(e \times pnm_{icb}) + \beta_4(e \times pnm_{icb}^2) + \gamma e + \boldsymbol{\theta}' \mathbf{x} + \varepsilon_{icb} \quad (\text{Eq.2})$$

The indicator for maternal education e is multiplied by pnm and pnm squared. The interaction terms give the difference in effect for children born to educated mothers compared to those born to non-educated mothers. A separate slope is calculated for children born to mothers with and without education when presenting the results. The interaction with sex is done in a similar way, where an indicator for being a female is interacted with PNM and PNM squared.

Results

Table 1 shows descriptive statistics. On average, children are -1.81 standard deviations (z-score) below the reference population and 77% attend school. PNM is about 56 deaths per 1,000 person-years of exposure in the height-for-age sample, and 66 per 1,000 in the school attendance sample. The main reason for these differences is that children in the schooling sample are born earlier. Birth intervals are considerably shorter for the sample of mothers that had at least two surviving children 12–59 months before the survey, and the children, therefore, included in the mother fixed-effects analysis of height-for-age. Figure 1 shows the study area. Although communities from most countries in SSA are included in the sample, it is clear that more densely populated areas and more extensively surveyed areas are overrepresented.

The functional form of the relationship between PNM is explored in Figure 2 which shows the predicted child outcomes by PNM using a non-parametric nearest-neighbor local linear smoother. Height-for-age is shown in Figure 2a. For most of the distribution, the between-survey variance in PNM is negative and mostly linear for much of the PNM distribution. For community and mother deviations in PNM, a U-shaped relationship is observed, with height-for-age declining at low levels and increasing slightly at high levels of PNM. The smoothed fit for school attendance is shown in Figure 2b. A general negative slope is observed for survey deviations in PNM for most of the distribution. The relationship appears to be highly non-linear when using community and mother

deviations in PNM. When using community and mother a fixed effect, school attendance seems to increase with increasing PNM at very low levels of PNM.

Table 1: Descriptive statistics

Sample:	Full	2 births	Full	2 births	
Outcome:	Height-for-age z-score		School attendance		Functional form
Outcome	-1.81 [1.61]	-1.78 [1.59]	0.77 [0.42]	0.77 [0.42]	Linear/Binary
PNM (per 1,000 survivors)	56.41 [31.11]	55.23 [29.82]	66.49 [33.61]	66.43 [32.52]	Natural log
Female	0.5 [0.50]	0.5 [0.50]	0.49 [0.50]	0.49 [0.50]	Binary
Child's age (months)	33.51 [14.10]	34.74 [15.74]	126.28 [30.74]	128.75 [30.54]	Natural log
Birth order	3.81 [2.50]	3.95 [2.36]	3.52 [2.25]	3.8 [2.22]	Linear
Birth interval (months)	38.26 [21.03]	30.18 [13.22]	35.91 [20.65]	33.43 [16.64]	Linear
Firstborn	0.2 [0.40]	0.12 [0.32]	0.21 [0.41]	0.14 [0.35]	Binary
Number of siblings ever born	4.22 [2.52]	4.61 [2.32]	5.79 [2.40]	6.28 [2.26]	Linear
Maternal age at birth (years)	26.66 [6.78]	26.31 [6.10]	25.64 [6.15]	26.11 [5.97]	Quadratic
Mother educated	0.51 [0.50]	0.51 [0.50]	0.56 [0.50]	0.54 [0.50]	Binary
First born twin	0.01 [0.12]	0.01 [0.11]	0.01 [0.12]	0.01 [0.12]	Binary
Later born twin	0.01 [0.12]	0.01 [0.11]	0.01 [0.11]	0.01 [0.10]	Binary
Communities (PSU)	19,576	10,031	19,465	16,598	
Mothers	133,579	22,050	168,893	86,639	
Surveys	86	86	58	58	
Countries	33	33	29	29	
Observations	157,894	45,018	299,941	216,827	

Notes: Functional form refers to the specification of variables in statistical models. PNM shows number of postneonatal deaths per 1,000 surviving children. Columns labeled '2 births' are for children born to mothers that had at least two births, which were not twins, in the samples and therefore include in models using mother fixed effects. Birth interval is adjusted for firstborns using dummy variables adjustment.

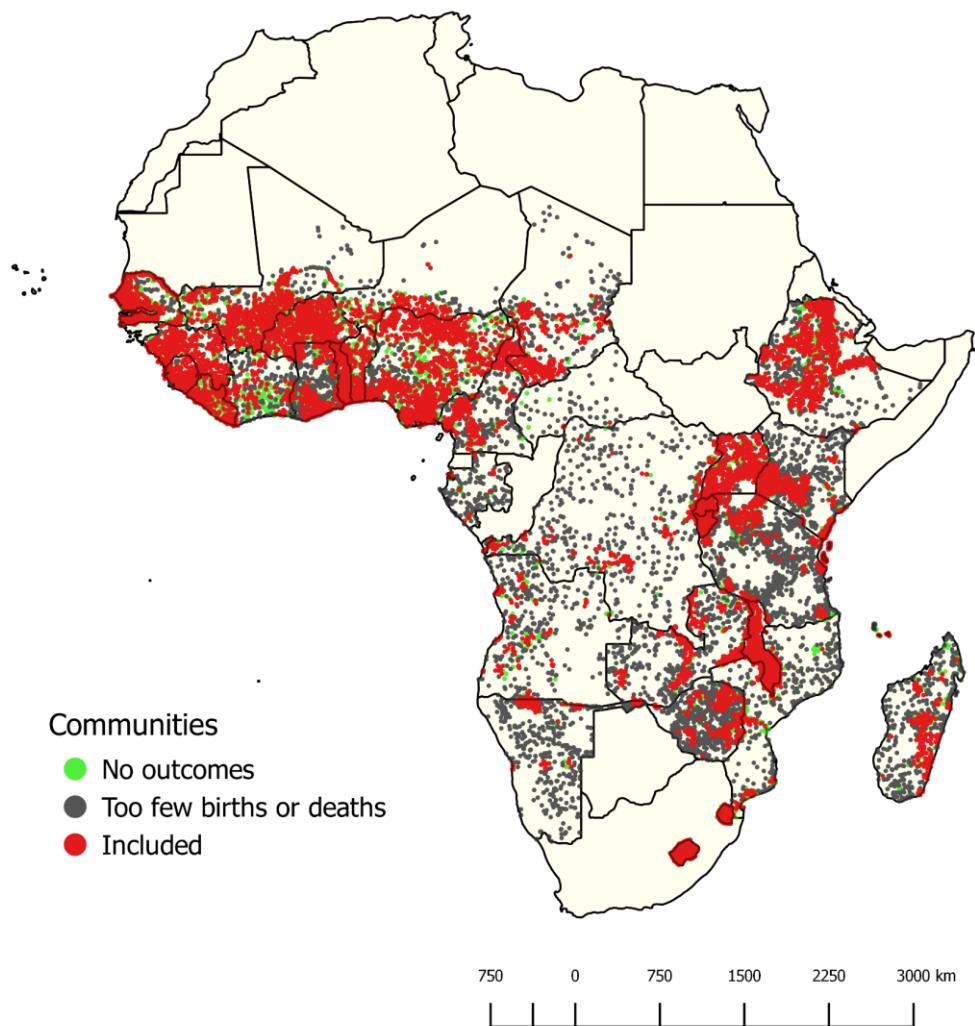


Figure 1. Map of the study area.

'No outcomes' refers to communities where no outcomes, i.e., school attendance or height-for-age z-score were available. The PNM is restricted to periods with at least 360 person-months of exposure and five deaths. 'Included' shows clusters (PSUs) included the analysis.

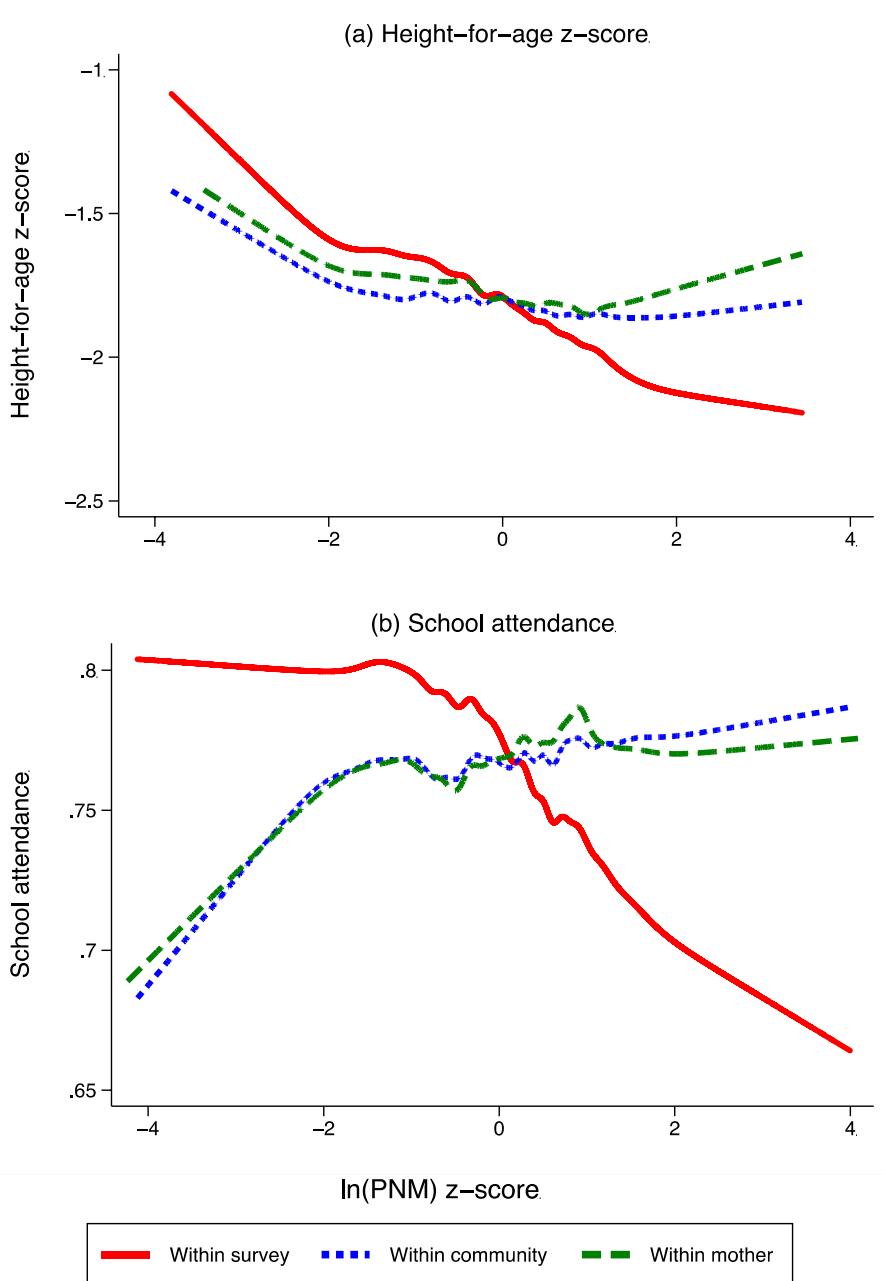


Figure 2 Smoothed fit of the relationship between PNM, and height-for-age and school attendance
 Adjusted using all independent variables as well as survey, community, or mother specific means of all independent variables. Obtained using the STATA program mrunning (Royston and Cox 2005).

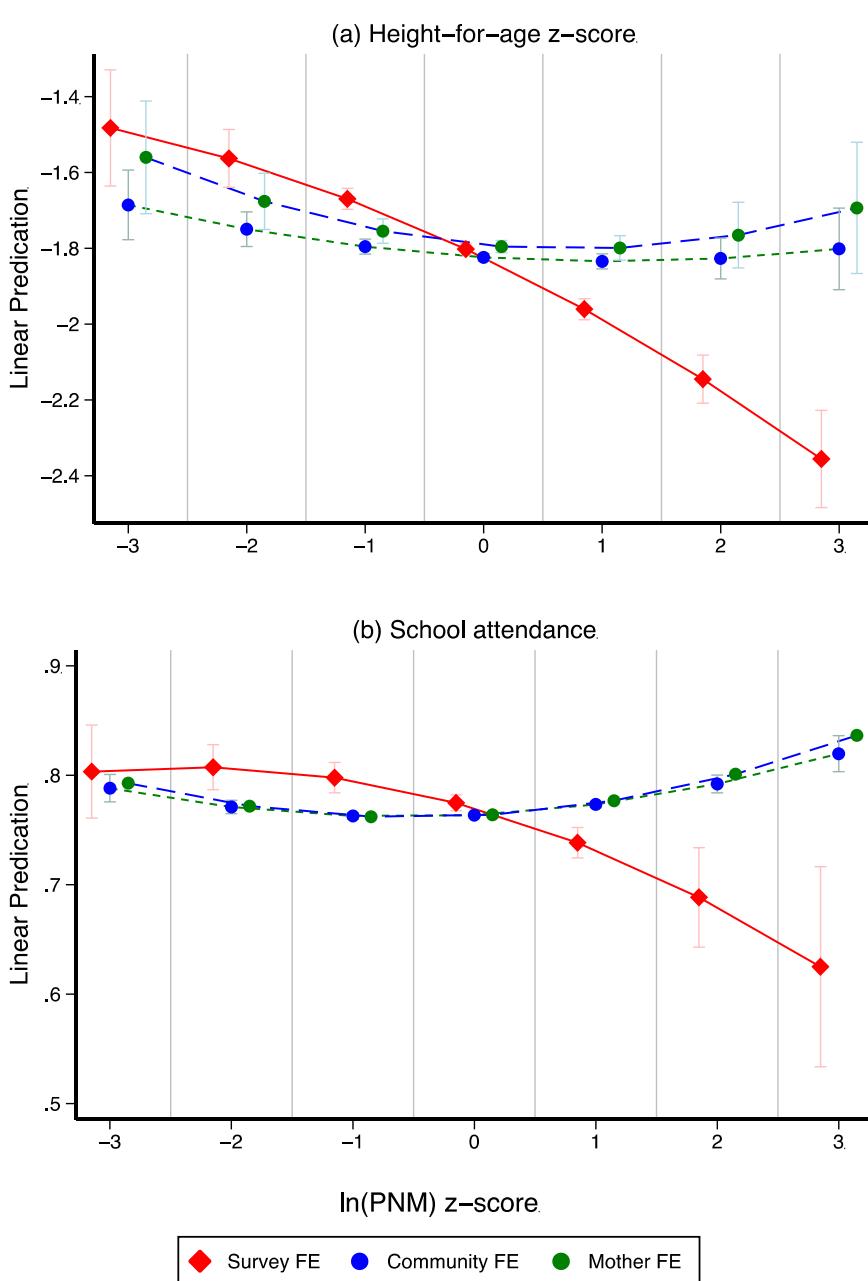


Figure 3. Predictive outcomes by PNM z-score using Eq.1 (95% confidence intervals)

Figure 3 shows the predicted outcomes from Eq1 for each level of fixed effects, Figure 3a shows results for height-for-age and Figure 3b shows results for school attendance. When using survey fixed effects, the predicted outcomes decline along most of the PNM distribution, although the slope is flat at low levels of PNM for both outcomes. For the community and mother fixed-effects models, for height-for-age, at low PNM levels, the predicted outcomes decline as PNM increases, while the slope remains nearly flat above mean PNM. With respect to school attendance, the slope also appears to be negative at very low PNM levels but increases sharply at higher PNM levels.

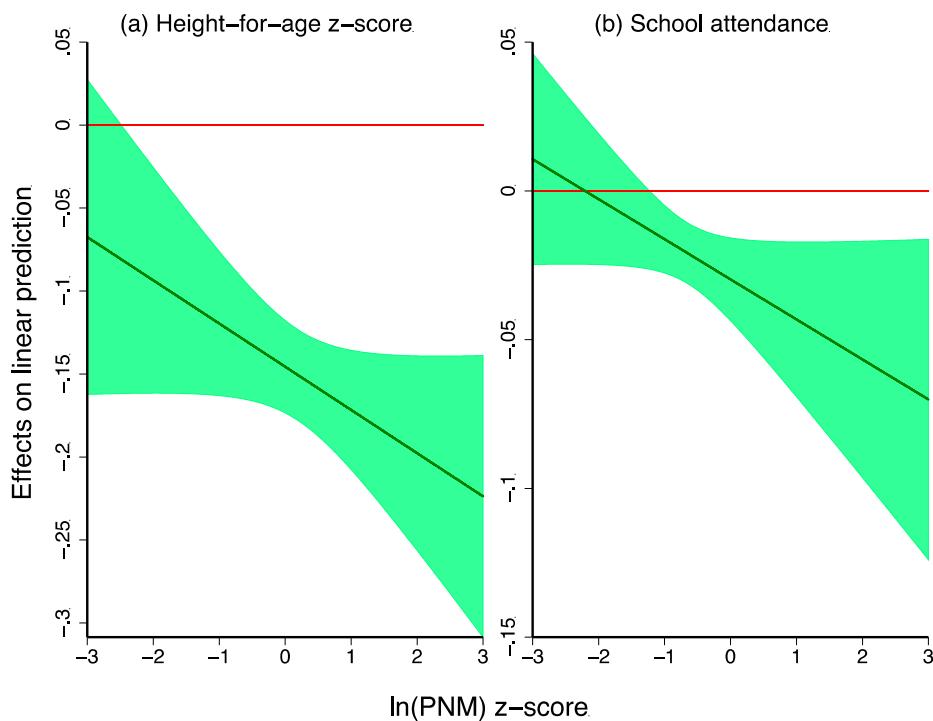


Figure 4. Average marginal effects of PNM across the distribution of PNM using survey fixed effects (95% CI)

Figure 4 shows the marginal effects of PNM across the PNM distribution using survey fixed effects. It is clear that when using temporal as well as spatial variations in adverse environment in infancy, the association with physical growth is strong and increases at higher levels of PNM. At the average PNM (0 z-score), a PNM z-score increase of one decreases the height-for-age by 0.146 z-score, which is about 8% of the mean deficit in height-for-age in the sample. At very high levels of adversity, such as at a z-score of two above the mean, the decrease is 0.198, or

about 11% of the sample mean. For school attendance, the marginal effect is negative, except at very low PNM. The marginal effect at the mean is about a .03 decrease in the linear probability of attending school for a z-score increase of one in PNM, or about 4% of the sample mean for school attendance.

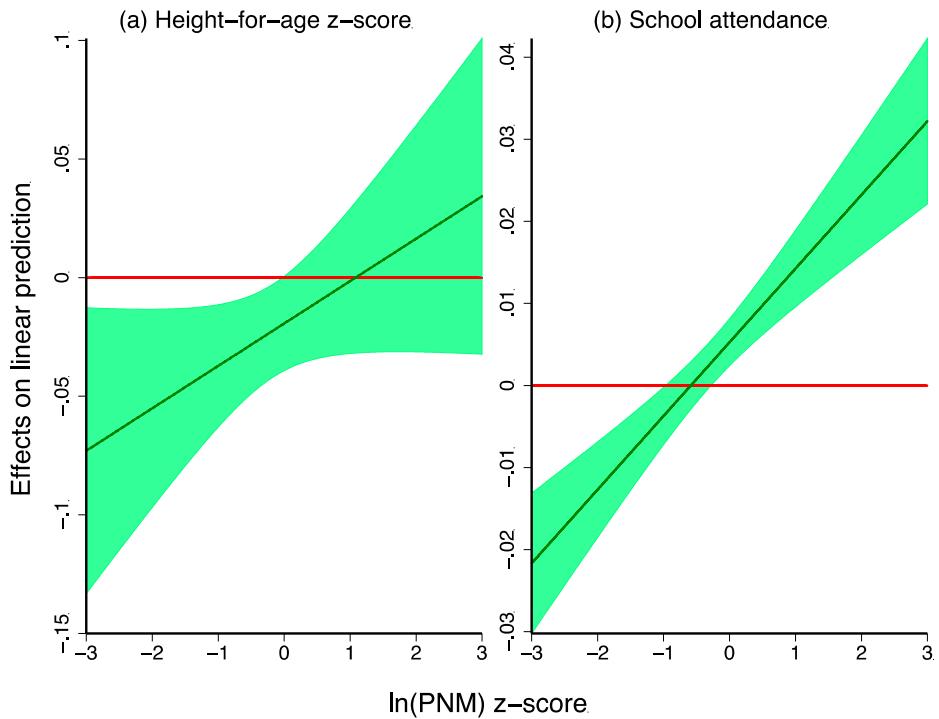


Figure 5. Average marginal effects of PNM at across the distribution of PNM using community fixed effects (95% CI)

Figure 5 shows the marginal effect of PNM across the PNM distribution using community fixed effects. For height-for-age (see Figure 5a), the marginal effect at very low levels of adversity, such as a z-score of -3, is about a 0.073 lower height-for-age z-score for a PNM z-score increase of one, which is about 4% of the sample mean. The effects decline sharply, and at a half a z-score below the mean of PNM, it goes down to being a 0.028 decrease in height-for-age z-score, which is about 1.5% of the sample mean. It is not statistically significant at the mean of and above. For schooling, in Figure 5b, at very low levels of PNM, such as at a -3 z-score, there is a 0.022 decrease in linear probability, which is around 3% of the sample mean. The marginal effects decline sharply as PNM increases. At the mean

level of adversity, there is a 0.0053 increase in the linear probability of attending school for a single standard deviation increase in adversity. At very high levels of adversity, such as at 2 z-scores above the mean, there is a 0.023 increase in linear probability, which is about 3% of the sample mean.

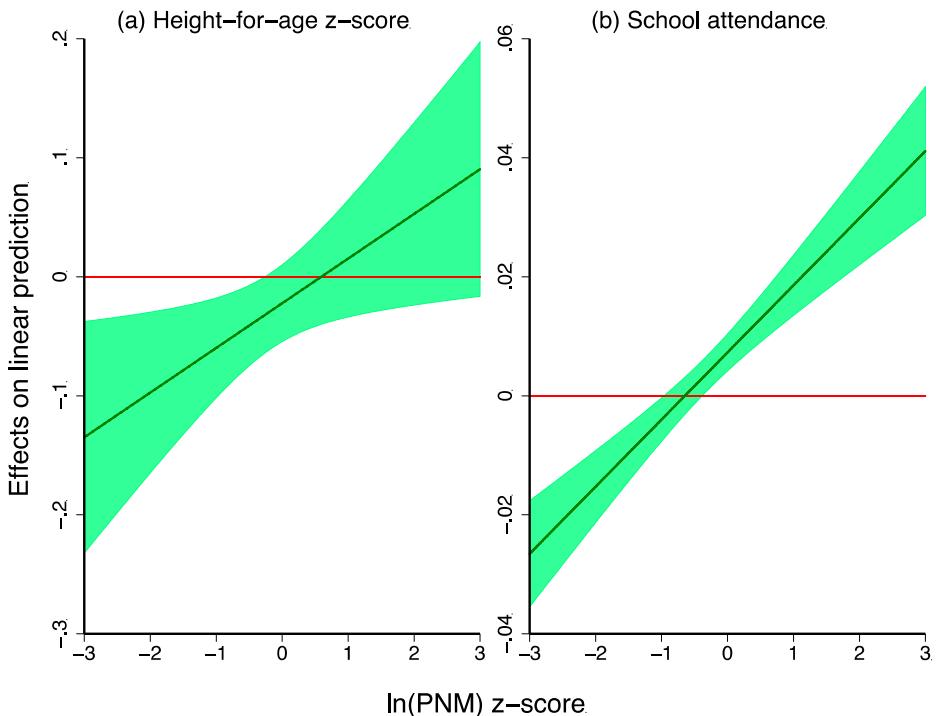


Figure 6. Average marginal effects of PNM at across the distribution of PNM using mother fixed effects (95% CI)

Figure 6 shows the marginal effects of PNM across the PNM distribution using mother fixed effects. The pattern is similar to the model using community fixed effects, but the negative effect at very low levels of PNM, such as 3 z-score below the mean, is substantially greater in magnitude: it is a 0.135 z-score decrease in height-for-age for a single standard deviation increase in PNM. As with the community fixed effects, the marginal effects decline sharply and are not statistically significant around the mean of PNM. For school attendance, the marginal effects look the same as when using community fixed effects.

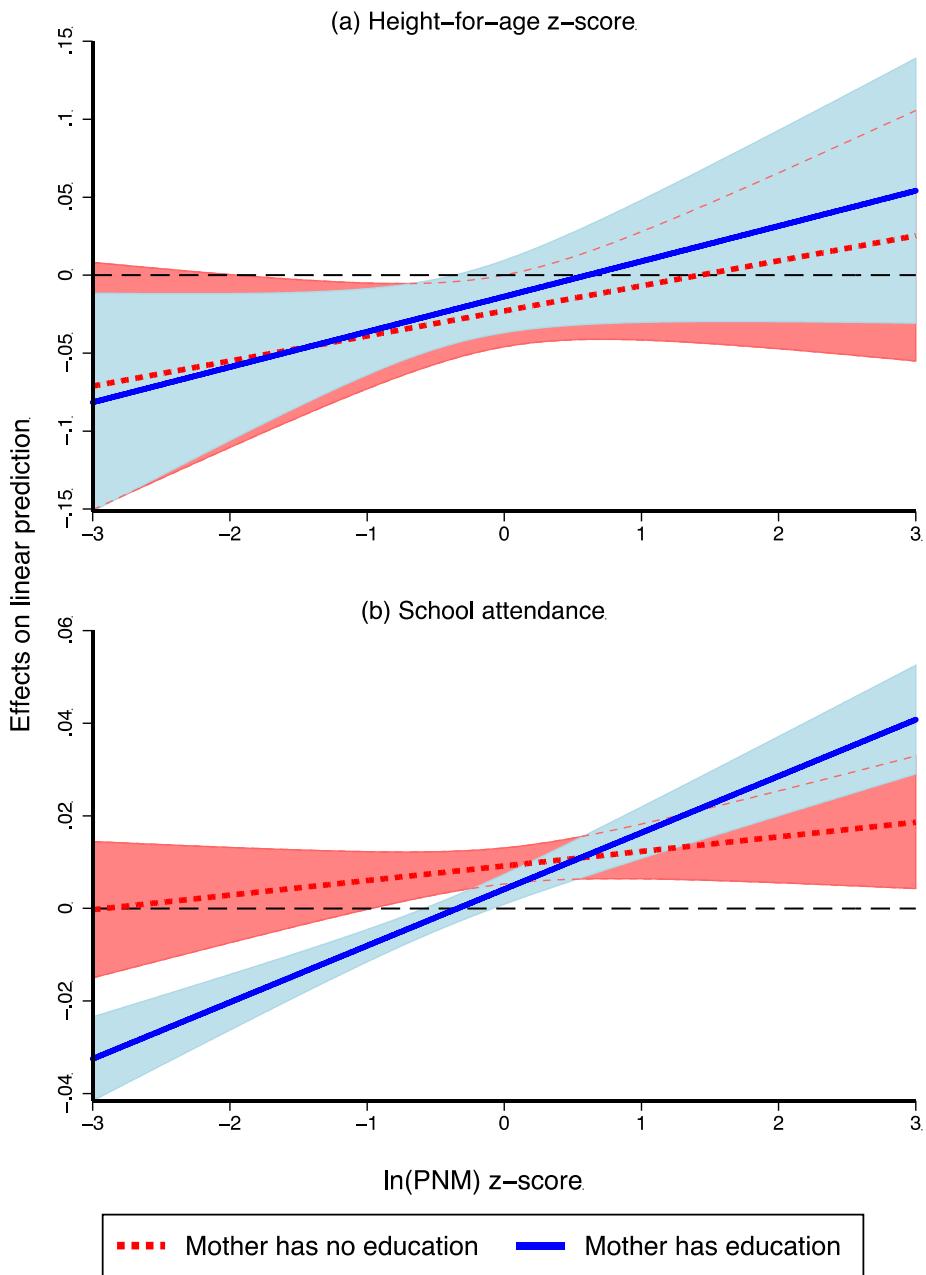


Figure 7. Average marginal effects of PNM at across the distribution of PNM by maternal education using community fixed effects (95% CI)

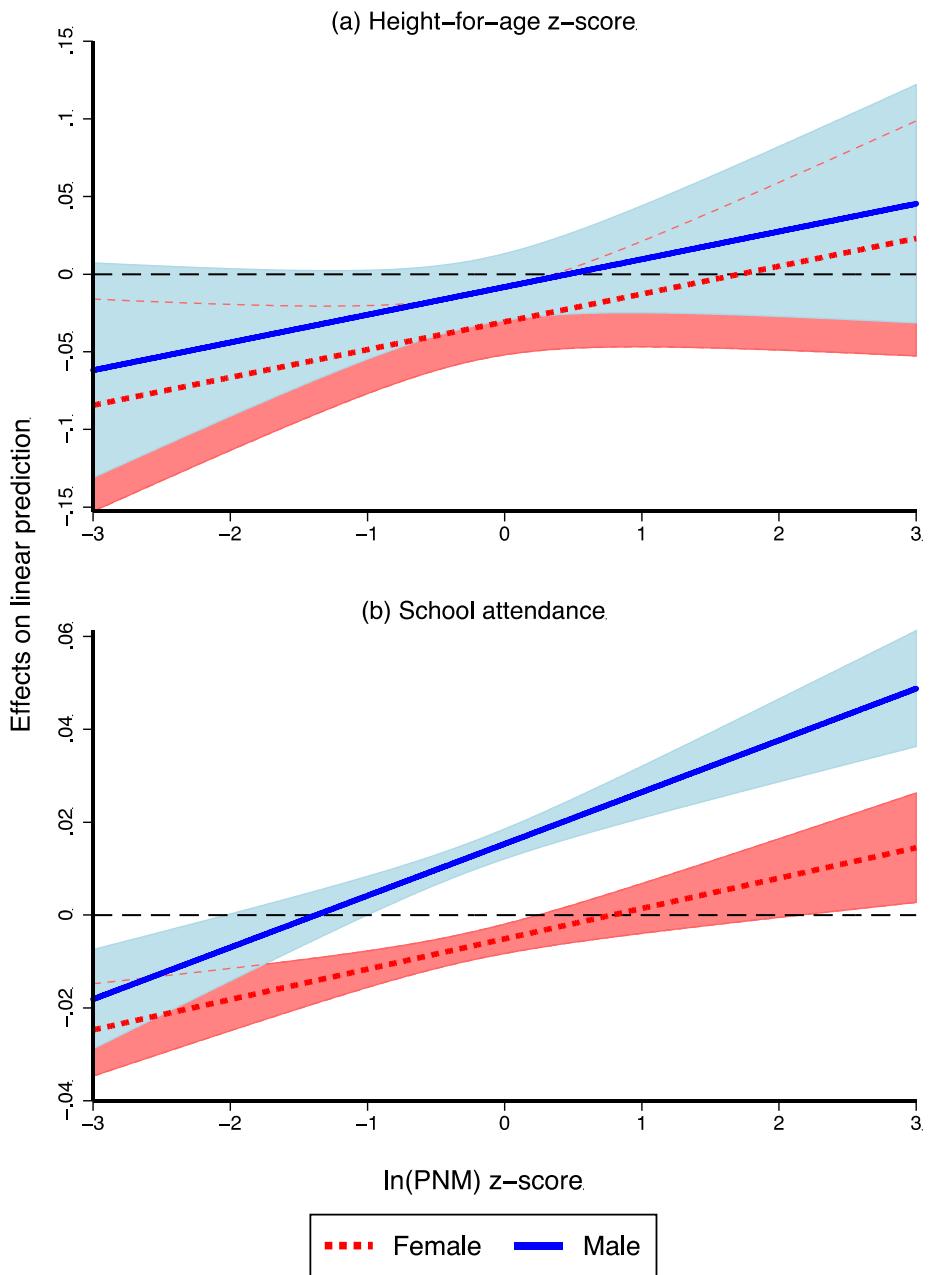


Figure 8 Average marginal effects of PNM at across the distribution of PNM by child's sex using community fixed effects (95% CI)

Figure 7 shows the results for the interactions between maternal education and PNM. Figure 7a shows that, regarding height-for-age, there is no difference in the effect of PNM on children born to mothers with education and on those born to mothers without education. The effect on school attendance, as shown in Figure 7b, shows a stronger negative effect at low levels of PNM for children born to educated mothers than for children born to mothers without education. The confidence intervals overlap in this case, at levels of adversity above the mean.

Figure 8 shows the results for the interaction between sex and PNM. Figure 8a shows that males and females have similar marginal effects of PNM on height-for-age although it is slightly greater for females. The confidence intervals do, however, overlap. Figure 8b shows that the marginal effects of PNM on school attendance are similar for males and females at very low levels of PNM and the confidence intervals overlap. The marginal effect attenuates more sharply for males, for whom PNM has a substantial positive marginal effect on school attendance at higher levels of PNM.

Discussion and Conclusion

Many infants in SSA face adversity in terms of undernutrition, and exposure to infectious diseases. These forms of adversity can have negative consequences for their development, such as impaired physical growth and school absence. This paper explores the effects of adverse environment in infancy on outcomes in childhood and adolescence using the postneonatal mortality incidence rate (PNM) as a proxy for adversity. When using both temporal and spatial variation in adversity, the marginal effects on child outcomes are negative and increase in magnitude at higher levels of adversity. When using community and mother fixed effects, there is only a negative effect—indicating scarring—at very low levels of adversity for both height-for-age and school attendance. For the mother fixed-effects models, the effect sizes are similar for school attendance but almost twice the size for height-for-age. The greater association of height-for-age with mother fixed effects undoubtedly reflects the sample restriction to women who have had two children over a four-year period. The different outcomes from models using spatial and temporal variation (survey fixed effects) and models using only temporal variation (community and mother fixed effects) are consistent with correlated environments, which has been suggested as a mechanism that biases the negative effects of exposures to adversity upwards (Preston et al., 1998).

The absence of association with height-for-age at higher levels of PNM is consistent with the selection mechanism increasing in importance at high levels of adversity (Bozzoli et al., 2009; Deaton, 2007; Gørgens et al., 2012). In this study, it appears that selection completely cancels out scarring effects at relatively low

levels of adversity (around the mean PNM level). A study on cohorts born earlier than those included in this study, based in an environment with considerably greater adversity, found a positive association between adversity in infancy and adult height, indicating that selection dominated over scarring (Deaton, 2007). In this study, a positive association is not observed for height-for-age at any level of adversity. This could indicate that the relative importance of selection has decreased for cohorts born more recently and into more favorable environments. Since this paper looks at anthropometric outcomes in childhood rather than adulthood, there may also be some further selection or catch-up in stature before these children reach adulthood (Preston et al., 1998; Steckel and Ziebarth, 2016).

A positive effect was found on school attendance at higher levels of adversity, especially for males. In addition to the selection mechanism and acquired immunity, smaller cohort sizes and increased investments in surviving children may be mechanisms underlying the higher school attendance rates of those exposed to high PNM levels in infancy. This paper did, however, find indications that the relationship between PNM and school attendance is highly non-linear, which may not be adequately captured by the models used.

This paper tests whether household socioeconomic status modifies the observed effect of PNM in infancy. Better-off households could have invested more in their children's health before, during, and following exposure to adversity, thus reducing negative impacts. Finally, the measure of adversity, PNM, may induce heterogeneity in the observed ITT effects since the actual exposure to adversity may differ by household socioeconomic status. For height-for-age, there is practically no difference in effects of adversity between children born to educated and non-educated mothers. For school attendance, at low PNM levels, there is a greater negative effect of PNM on school attendance for children born to educated mothers than for children born to non-educated mothers. There is a positive association between PNM and school attendance at high levels of PNM for both groups of children. Due to greater biological frailty among males, causing both higher mortality and stunting (Rutstein, 2005; Sawyer, 2012; Stinson, 1985; Wamani et al., 2007), the expectation was that possible selection or scarring may be greater for males than females. The interaction models for sex show that, for height-for-age, there is only a small difference in the effects of adversity in infancy, and the confidence intervals for males and females overlap. The positive effect of PNM on school attendance at high levels of adversity is much greater for males than for females.

A limitation of this paper is the use of a general measure of adversity that captures a host of infectious diseases in interaction with undernutrition, as well as other exposures such as indoor air pollution. Determining specific exposures, such as undernutrition, malaria, diarrhea, or pneumonia, would give more informative

estimates of the particular type of adversity that is detrimental for child development. This paper does, however, establish the relationship between general adversity and the two variables of nutrition and education. Calculating a relatively high-resolution estimate, such as the PNM in this paper, requires a sufficient number of deaths to acquire a reliable estimate. Therefore, these estimates are not nationally representative, as more extensively surveyed and more densely populated areas are more likely to be included in the analysis. Finally, although the exposure used in this paper is exogenous with respect to child health, it is strictly not a causal effect. Not all children are exposed to adversity in periods when post-neonatal mortality is high, and some children are exposed to adversity when post-neonatal mortality is low so the estimates represent ITT effects, which generally underestimate of the true causal effects (Van den Berg et al., 2016).

To conclude, this paper finds that in SSA, being exposed to adversity in infancy negatively affects physical growth and school attendance, possibly through scarring effects. The negative relationship is, however, small and only observed at low levels of adversity, which indicates that selective mortality cancels out scarring effects at higher levels of adversity.

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Appendix

Appendix A1. Calculating the postneonatal mortality incidence rate

D is a symmetric distance matrix

$$\mathbf{D} = \underbrace{\begin{bmatrix} d_{11} & \cdots & d_{c1} \\ \vdots & \ddots & \vdots \\ d_{1K} & \cdots & d_{CK} \end{bmatrix}}_{C \times K}$$

where $d_{ck} = 1$ when PSU c is within 50 km of PSU k and $d_{ck} = 0$ otherwise. $c = k$ and $d_{cc} = 1$ on the diagonal. The distance between c and k is calculated using the haversine formula. The elements in **D** may also contain some form of weighting by distance rather than a sharp cut-off point at 50 km.

Matrix **E** contains the number of children 1 – 12 months old in PSU c that are alive in month m . Each row represents a PSU and each column represents a month.

$$\mathbf{E} = \underbrace{\begin{bmatrix} e_{11} & \cdots & e_{1M} \\ \vdots & \ddots & \vdots \\ e_{c1} & \cdots & e_{CM} \end{bmatrix}}_{C \times M}$$

As a result of multiplying **D** by **E**

$$\mathbf{E}^{50} = \mathbf{D} \times \mathbf{E} = \underbrace{\begin{bmatrix} e_{11}^{50} & \cdots & e_{1M}^{50} \\ \vdots & \ddots & \vdots \\ e_{c1}^{50} & \cdots & e_{CM}^{50} \end{bmatrix}}_{C \times M}$$

\mathbf{E}^{50} contains children (1 – 12 months old) exposed in month m within a 50 km radius from PSU c . In the same way as **E** and \mathbf{E}^{50} were constructed, a matrix **P** consists of the number of post-neonatal deaths in each PSU c , and each month m , and \mathbf{P}^{50} contains the number of deaths in month m in a 50 km radius from PSU c .

The elements in \mathbf{E}^{50} are summed up as

$$e_{cb}^{50,12} = e_{cm}^{50} + e_{cm+1}^{50} + \cdots + e_{cm+11}^{50}$$

giving a $C \times B$ matrix $\mathbf{E}^{50,12}$, where b denotes a month of birth and number of columns $B = M - 11$.

$$\mathbf{E}^{50,12} = \underbrace{\begin{bmatrix} \sum_{m=1}^{12} e_{1m}^{50} & \cdots & \sum_{m=M-11}^M e_{1m}^{50} \\ \vdots & \ddots & \vdots \\ \sum_{m=1}^{12} e_{Cm}^{50} & \cdots & \sum_{m=M-11}^M e_{Cm}^{50} \end{bmatrix}}_{C \times M-11} = \underbrace{\begin{bmatrix} e_{11}^{50,12} & \cdots & e_{1B}^{50,12} \\ \vdots & \ddots & \vdots \\ e_{C1}^{50,12} & \cdots & e_{CB}^{50,12} \end{bmatrix}}_{C \times B}$$

The elements, $e_{cb}^{50,12}$, from $\mathbf{E}^{50,12}$ are the person-time-of exposures in a 50 km radius from PSU c and over a 12 month period starting at month b . A matrix $\mathbf{P}^{50,12}$ containing the number of deaths in a 50 km radius from PSU c occurring over a 12 month period starting at month b is constructed in the same way. The pnm are then calculated using an element wise division of $\mathbf{P}^{50,12}$ by $\mathbf{E}^{50,12}$ for all $e_{cb}^{50,12} \neq 0$.

$$\mathbf{PNM} = \underbrace{\begin{bmatrix} pnm_{11} & \cdots & pnm_{1B} \\ \vdots & \ddots & \vdots \\ pnm_{C1} & \cdots & pnm_{CB} \end{bmatrix}}_{C \times B} = \underbrace{\begin{bmatrix} p_{11}^{50,12}/e_{11}^{50,12} & \cdots & p_{1B}^{50,12}/e_{1B}^{50,12} \\ \vdots & \ddots & \vdots \\ p_{C1}^{50,12}/e_{C1}^{50,12} & \cdots & p_{CB}^{50,12}/e_{CB}^{50,12} \end{bmatrix}}_{C \times B}$$

The elements in **PNM** are posneonatal mortality incidence rate in a 50 km radius from of PSU c which a child born in PSU c in month b is exposed to over the period of infancy.

Table A1. Models from EQ1, shown in figures 3 – 6

Fixed effects (FE): VARIABLES	Survey Height-for-age z-score	PSU	Mother	Survey School attendance	PSU	Mother
In(PNM) z-score	-0.15*** (0.01)	-0.02* (0.01)	-0.02 (0.02)	-0.03*** (0.01)	0.01*** (0.00)	0.01*** (0.00)
In(PNM) z-score ²	-0.01* (0.01)	0.01* (0.01)	0.02** (0.01)	-0.01* (0.00)	0.00*** (0.00)	0.01*** (0.00)
In(Child's age+1)	-0.30*** (0.04)	-0.34*** (0.01)	-1.76*** (0.05)	0.30*** (0.03)	0.22*** (0.00)	1.37*** (0.02)
Number of siblings	0.21*** (0.03)	0.25*** (0.01)		-0.02*** (0.00)	-0.01*** (0.00)	
Birth Order	-0.28*** (0.03)	-0.28*** (0.01)	-0.87*** (0.04)	0.01*** (0.00)	-0.00*** (0.00)	-0.01*** (0.00)
Birth interval	0.00*** (0.00)	0.00*** (0.00)	0.01*** (0.00)	0.00*** (0.00)	0.00*** (0.00)	0.00*** (0.00)
First born	0.27*** (0.02)	0.22*** (0.02)	0.32*** (0.04)	0.01*** (0.00)	0.00 (0.00)	-0.01*** (0.00)
Maternal age at birth	0.08*** (0.01)	0.05*** (0.01)	-0.26*** (0.03)	0.01*** (0.00)	0.00 (0.00)	0.11*** (0.00)
Mat. age at birth ²	-0.00*** (0.00)	-0.00*** (0.00)	-0.00*** (0.00)	-0.00*** (0.00)	0.00 (0.00)	0.00*** (0.00)
Mother has education	0.46*** (0.05)	0.17*** (0.01)		0.19*** (0.03)	0.07*** (0.00)	
Female	0.18*** (0.01)	0.17*** (0.01)	0.17*** (0.02)	-0.02** (0.01)	-0.02*** (0.00)	-0.02*** (0.00)
First born twin	-0.36*** (0.05)	-0.42*** (0.04)	-0.05 (0.08)	0.04*** (0.01)	0.02*** (0.01)	0.03*** (0.01)
Later born twin	-0.45** (0.04)	-0.51*** (0.03)	-0.23*** (0.08)	0.03*** (0.01)	0.01** (0.01)	0.02*** (0.01)
Constant	-2.67*** (0.19)	-1.88*** (0.08)	15.05*** (0.76)	-0.80*** (0.17)	-0.30*** (0.02)	-8.71*** (0.16)
Observations	157,894	157,894	45,018	299,941	299,941	216,827
R-squared	0.05	0.02	0.09	0.09	0.03	0.07
Number of FE	86	19,576	22,050	58	19,465	86,639

Robust standard errors adjusted for clustering at the level of the respective fixed effects in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table A2. Models from EQ2 shown in figure 7

Fixed effects (FE): VARIABLES	Survey Height-for-age z-score	PSU	Mother	Survey School attendance	PSU	Mother
Mother educated						
* ln(PNM) z-score	0.04*** (0.01)	0.01 (0.01)	0.01 (0.03)	0.03** (0.01)	-0.01** (0.00)	-0.03*** (0.00)
Mother educated						
* ln(PNM) ² z-score	-0.03* (0.02)	0.00 (0.01)	-0.00 (0.02)	0.01 (0.00)	0.00*** (0.00)	0.00 (0.00)
ln(PNM) z-score	-0.17*** (0.02)	-0.02* (0.01)	-0.03 (0.02)	-0.04*** (0.01)	0.01*** (0.00)	0.02*** (0.00)
ln(PNM) ² z-score	0.00 (0.01)	0.01 (0.01)	0.02* (0.01)	-0.01* (0.00)	0.00 (0.00)	0.00** (0.00)
ln(Child's age+1)	-0.29*** (0.04)	-0.34*** (0.01)	-1.76*** (0.05)	0.30*** (0.04)	0.22*** (0.00)	1.37*** (0.02)
Birth Order	-0.28*** (0.03)	-0.28*** (0.01)	-0.87*** (0.04)	0.01*** (0.00)	-0.00*** (0.00)	-0.01*** (0.00)
Birth interval	0.00*** (0.00)	0.00*** (0.00)	0.01*** (0.00)	0.00*** (0.00)	0.00*** (0.00)	0.00*** (0.00)
First born	0.27*** (0.02)	0.22*** (0.02)	0.32*** (0.04)	0.01*** (0.00)	0.00 (0.00)	-0.01*** (0.00)
Number of siblings	0.21*** (0.03)	0.25*** (0.01)		-0.02*** (0.00)	-0.01*** (0.00)	
Maternal age	0.08*** (0.01)	0.05*** (0.01)	-0.26*** (0.03)	0.01*** (0.00)	0.00 (0.00)	0.11*** (0.00)
Maternal age ²	-0.00*** (0.00)	-0.00*** (0.00)	-0.00*** (0.00)	-0.00*** (0.00)	0.00 (0.00)	0.00*** (0.00)
Mother educated	0.48*** (0.06)	0.16*** (0.01)		0.18*** (0.03)	0.07*** (0.00)	
Female	0.18*** (0.01)	0.17*** (0.01)	0.17*** (0.02)	-0.02** (0.01)	-0.02*** (0.00)	-0.02*** (0.00)
First born twin	-0.36*** (0.05)	-0.42*** (0.04)	-0.05 (0.08)	0.04*** (0.01)	0.02*** (0.01)	0.03*** (0.01)
Later born twin	-0.45*** (0.04)	-0.51*** (0.03)	-0.23*** (0.08)	0.03*** (0.01)	0.01** (0.01)	0.02*** (0.01)
Constant	-2.69*** (0.19)	-1.88*** (0.08)	15.05*** (0.76)	-0.80*** (0.17)	-0.29*** (0.02)	-8.68*** (0.16)
Observations	157,894	157,894	45,018	299,941	299,941	216,827
R-squared	0.05	0.02	0.09	0.09	0.03	0.07
Number of FE	86	19,576	22,050	58	19,465	86,639

Robust standard errors adjusted for clustering at the level of the respective fixed effects in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table A3. Models from EQ2 shown in figure 8

Fixed effects (FE): VARIABLES	Survey Height-for-age z-score	PSU	Mother	Survey School attendance	PSU	Mother
Female *						
In(PNM) z-score	-0.03*** (0.01)	-0.02*** (0.01)	-0.01 (0.02)	-0.02*** (0.00)	-0.02*** (0.00)	-0.02*** (0.00)
Female *						
In(PNM)2 z-score	-0.01 (0.01)	-0.01 (0.01)	-0.01 (0.01)	-0.00 (0.00)	-0.00*** (0.00)	-0.01*** (0.00)
In(PNM) z-score	-0.14*** (0.02)	-0.01 (0.01)	-0.02 (0.02)	-0.02*** (0.01)	0.02*** (0.00)	0.02*** (0.00)
In(PNM) ² z-score	-0.01 (0.01)	0.01 (0.01)	0.02** (0.01)	-0.01** (0.00)	0.00*** (0.00)	0.01*** (0.00)
In(Child's age+1)	-0.24*** (0.03)	-0.29*** (0.01)	-1.69*** (0.06)	0.28*** (0.03)	0.20*** (0.00)	1.37*** (0.02)
Birth Order	-0.27*** (0.03)	-0.26*** (0.01)	-0.84*** (0.04)	0.01*** (0.00)	-0.00* (0.00)	-0.01*** (0.00)
Birth interval	0.00*** (0.00)	0.00*** (0.00)	0.01*** (0.00)	0.00** (0.00)	0.00*** (0.00)	0.00*** (0.00)
Number of siblings	0.20*** (0.03)	0.23*** (0.01)		-0.02*** (0.00)	-0.01*** (0.00)	
First born	0.27*** (0.02)	0.22*** (0.02)	0.32*** (0.04)	0.01*** (0.00)	0.00* (0.00)	-0.01*** (0.00)
Maternal age	0.08*** (0.01)	0.05*** (0.01)	-0.29*** (0.04)	0.01*** (0.00)	0.00 (0.00)	0.11*** (0.00)
Maternal age ²	-0.00*** (0.00)	-0.00*** (0.00)	-0.00 (0.00)	-0.00*** (0.00)	0.00 (0.00)	0.00*** (0.00)
Mother has education	0.47*** (0.06)	0.16*** (0.01)		0.20*** (0.03)	0.07*** (0.00)	
Female	0.19*** (0.01)	0.18*** (0.01)	0.19*** (0.02)	-0.01 (0.01)	-0.01*** (0.00)	-0.02*** (0.00)
First born twin	-0.35*** (0.06)	-0.43*** (0.04)	-0.04 (0.09)	0.04*** (0.01)	0.01** (0.01)	0.03*** (0.01)
Later born twin	-0.44*** (0.05)	-0.52*** (0.04)	-0.24*** (0.08)	0.04*** (0.01)	0.01** (0.01)	0.03*** (0.01)
Constant	-2.90*** (0.19)	-2.09*** (0.09)	14.99*** (0.82)	-0.75*** (0.16)	-0.24*** (0.02)	-8.79*** (0.17)
Observations	130,440	130,440	37,717	244,811	244,811	177,299
R-squared	0.05	0.02	0.09	0.09	0.03	0.06
Number of FE	70	16,366	18,483	48	16,097	70,789

Robust standard errors adjusted for clustering at the level of the respective fixed effects in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Paper II

Paper II

Maternal Height and Child Development in Sub-Saharan Africa: Underlying Mechanisms and the Role of Public Spending on Health¹

Abstract

Maternal height is associated with child health outcomes in many low-and-middle-income countries highlighting a strong intergenerational transmission of poor health. This study examines how it influences under-5 mortality and education in sub-Saharan Africa, which has the highest under-5 mortality, lowest education level, and lowest health expenditures in the world. We explore potential mechanisms and estimate whether public spending on health can mitigate this intergenerational transmission. We find a consistent association between maternal height and under-5 mortality while the association with education is weak and mostly explained by household level SES, community-level factors, and maternal education. Community-level factors, fertility, and SES explain much of the association for mortality, especially after the neonatal period, but there remains a sizeable residual association. Government health expenditures substantially weaken the association between maternal height and under-5 mortality, which indicates that intergenerational transmission of poor health, can be reduced by government interventions.

¹ Paper II was co-authored with Martin Dribe. Both authors contributed to the study concept and design, the interpretation of findings, and the drafting of the manuscript. The author of this dissertation acquired the data and conducted the statistical analysis.

Introduction

Intergenerational transmission of poor health from parents to children has received much attention in the social sciences. Identifying these links is important as they suggest targets for interventions for breaking negative intergenerational cycles and improving population health. It is particularly transmissions related to poverty and disadvantage that appeal to a sense of justice and equal opportunities. Children growing up in sub-Saharan Africa (SSA) face adverse living conditions due to rampant diseases and vulnerability to food insecurity which is aggravated by poor parental health, lack of resources, poor public health, and limited access to health care and other services. SSA has 11 percent of the world's population, 24 percent of the disease burden and 50 percent of under-5 deaths, but only 1 percent of the global health expenditures (WHO, 2006). Stunted physical growth and short adult stature of many women reflects early-life infections and undernutrition which have negative consequences for future health and related outcomes (Moradi, 2010; Silventoinen, 2003; Steckel, 1995a). Children are at risk of inheriting poor health from their mothers, especially when disadvantaged mothers cluster in disadvantaged areas with poor public health and little access to health care.

Maternal height has been used to study intergenerational transmission of health from mother to child and has been found to determine under-5 mortality in a large number of low- and middle-income countries (Bhalotra and Rawlings, 2011; Monden and Smits, 2009; Özaltin et al., 2010). The mechanisms behind the association are not entirely understood but suggested pathways include physiological factors and various consequences of harmful early-life exposures influencing human development, and consequently income and education (Currie and Vogl, 2012). Hence, early-life exposures reflected in maternal height can influence child outcomes through a variety of factors such as income, education, as well as accumulated health. Physiology in itself can also have direct consequences for the growth of the fetus during pregnancy, as well as for risks while giving birth.

An apparent intervention aimed at reducing the intergenerational transmission of disadvantage is to ensure a good early-early life environment, with sufficient nutrition and protection against infectious diseases. Improved nutrition and fewer infections early in life increased the stature of European men by 11 cm between 1870 – 1970 (Hatton and Bray, 2010). While improved early-life conditions is a long-term solution to promote population health and human capital formation, public interventions can reduce the harmful consequences of poor maternal health for child outcomes also in the shorter term by aiding disadvantaged women in childbirth and help to care for their children.

Previous papers studied interactions between maternal height and modifiable factors at the household-, district- and national level. The association was found to be lower for educated mothers. However, since education indicates not only present living standards but also past living standards, and ability, it may be determined simultaneously to height (Bhalotra and Rawlings, 2013; Monden and Smits, 2009). Therefore studies have explored whether factors beyond the household have implications for the observed association. These studies have found mixed results. Monden & Smits (2009) found no influence of district-level health care use and economic status, and national level GDP on the observed association between maternal height and under-5 mortality. On the other hand, Bhalotra & Rawlings (2013) found an interaction with changes in aggregate national level factors at the time of birth and find improved female education, vaccination rates, and GDP to attenuate the transmission.

This study aims at furthering our understanding of the intergenerational transmission of disadvantage and has two primary goals. The first is to estimate the association between maternal height and different child outcomes and quantify the extent to which the association is impacted by covariates likely to overlap with height, such as maternal education, household socioeconomic status (SES), fertility, and community and father characteristics. Our second goal is to test whether government expenditure on health can modify the association between maternal height and child outcomes. We extend previous research in several ways. First, we focus on the association between maternal height and child outcomes in SSA, the most deprived region in the world with the highest under-5 mortality and widespread stunting, the lowest health expenditures, and a high number of disadvantaged households. Secondly, by looking at child education as an outcome, we study longer-term consequences of maternal height for human development in a broader sense. Thirdly, by studying the extent to which various covariates impact the association. Finally, by studying the effectiveness of public spending on health in aiding disadvantaged households and reducing the association between maternal height and child outcomes. Our primary research questions are: Is maternal height associated with survival and school attendance in SSA? How much do covariates for maternal education, household SES, fertility, community-level factors, and household and father characteristic impact the association between maternal height and child outcomes? Do government expenditures on health lower the association?

We use individual-, household-, and community-level data from multiple Demographic and Health Surveys (DHS) from 35 countries in sub-Saharan Africa, conducted 1992-2016. We supplement these data with annual government expenditures on health from the WHO Global Health Expenditure Database. In line with previous research, we find a consistent relationship between maternal height and under-5 mortality, which is statistically significant in all but five countries. The association with neonatal mortality appears to be more direct than

the association with mortality later in childhood. Maternal height is also associated with school attendance, although the impact is weak, more variable between countries, and appears to operate mostly through community-level factors, maternal education, and SES. In line with the findings of Bhalotra and Rawlings (2013), we also find that improvements in aggregate national level factors, in our study government expenditures on health, substantially weaken the association between maternal height and mortality of children.

The sub-Saharan African context

Sub-Saharan Africa accounts for approximately 25 percent of global births but 50 percent of under-5 deaths, which is projected to increase to 33 percent and 60 percent, respectively in 2030. Also, many children are underweight and stunted (Liu et al., 2015; UN IGME, 2015; UNICEF-WHO-The World Bank, 2015). Under-5 mortality has declined in most of the region, especially after overcoming the setbacks in the 1990s related to HIV. Neonatal mortality has not declined as fast as mortality later in childhood, and today it accounts for 58 percent of under-5 deaths while it was 26 percent in 1990 (Rajaratnam et al., 2010; You et al., 2016). The prevalence of stunting has decreased from 41 percent in 1985 to 38 percent in 2011 (Stevens et al., 2016, p. 87). Trends in adult heights since 1945 differ slightly across countries, but there has been little change in the heights of adult women (Moradi, 2010). Enrollment in primary education has increased substantially over the past four decades, especially following international efforts such as the Millennium Development Goals and education for all after 2000. Even so, the proportion of children not attending school is higher than in any other part of the world (Lewin, 2009; UNESCO, 2011).

Even though SSA has 11 percent of the world's population, it carries 24 percent of the disease burden but has only 1 percent of the global health expenditure (WHO, 2006). Out of 49 countries with available data, only 20 had reached the spending of USD 44 per capita in 2014, which is the amount WHO estimates is needed to provide minimal, essential life-saving services. The median total health expenditure was USD 51 in 2014, up from USD 30 in 1995. Private health expenditure is a relatively large share of overall health expenditure in SSA, and between 1995 and 2014, 44 percent of health expenditure was private, thereof about 26 percent from private insurance, and 59 percent out-of-pocket expenditures (WHO, 2017a). In this study, we focus only on government expenditures as they are more likely to improve the health of disadvantaged households because they have little to spend on private health care provision. Although still insufficient, government spending on health has increased markedly in most SSA countries following the Abuja declaration in 2001 (WHO, 2011). While those with the worst health outcomes, e.g., poor rural households, tend to

have lower access to health care, there have been efforts to increase pro-poor public spending on health in developing countries (Gupta et al., 2002). Spending on health, especially public health expenditure, appears to have been effective in improving child health and has decreased infant mortality rates and increased life expectancy (Novignon et al., 2012). Public health care has also been found to perform better in providing essential care for newborn children (Wagner et al., 2014).

Theoretical background

Harmful environment and inadequate care and investments have negative consequences for survival, as well as child development more generally. Investments include nutrition, immunization, health care and education, and care relates to parental knowledge of feeding, and the ability to prevent, and respond to, diseases. Exposure to diseases and malnutrition early in life can have permanent effects on survival as well as on health, and cognitive development and consequently, education and income (Barker, 1997; Case and Paxson, 2006; Currie, 2008; Currie and Moretti, 2007; Gluckman et al., 2010; Steckel, 2008). Pregnancy and birth complications can similarly have immediate and long-term consequences for health and related outcomes (Van Handel et al., 2007). Maternal health and abilities are of particular importance, as they determine child health in utero, during birth, and after birth, as women are the primary caretakers of children in SSA (FAO, 2014).

It is not only harmful health exposures during pregnancy and child-rearing that can affect outcomes of children but also detrimental exposures the mother has suffered in her early life and the negative consequences these exposures have on her health, reflected in shorter stature (Bhalotra and Rawlings, 2011; Moradi, 2010). We expect a positive association between maternal height and human development regarding survival and primary school attendance in SSA. The association between maternal height and child outcomes should be explained by covariates for maternal education, adult living standards, fertility, community, and the characteristics of the father to some extent. We do, however, expect a residual association, net of these factors. The association should also diminish as resources and living standards beyond the household increase, specifically national-level government health expenditure around the time of birth.

Suggested mechanisms behind the effect of maternal height and child health include physiological, biological, genetic, and psychosocial mechanisms. One biological determinant is the growth of the fetus during pregnancy. There are indications that mothers with shorter stature have lower weight gain and a higher risk of developing hypertension during pregnancy, which puts their children at

risk. It also leads women to have a smaller uterus and ovaries, which restricts the growth of the fetus, leading to lower birth weight. A narrower pelvis can also cause birth complications (Perkins et al., 2016).

Other influences may develop after birth. Maternal height is associated with accumulated health which could influence her resources for taking care of children through feeding and preventing disease (Steckel, 1995b). Less-healthy mothers may have reduced energy and could be more easily fatigued, stressed or distracted. Environmental impact on adult height, which occurs primarily in early life, can simultaneously determine education attainment of women, which in turn has been shown to be related to child outcomes through various pathways (Caldwell, 1979; Miguel and Kremer, 2004). Maternal education, accumulated health, and other factors reflected in height can finally affect household living standards, and thus material resources and hygiene. Fertility-related characteristics, such as age at birth, and birth interval, are strongly related to child health outcomes, and may indirectly confound the relationship with height (Breierova and Duflo, 2004; LeGrand and Phillips, 1996). Father characteristics also influence child outcomes but are likely to be correlated with maternal factors since mating is positively assortative concerning factors such as height (Silventoinen et al., 2003). Despite substantial internal migration and general improvements in living standards, the early-life environment of mothers and their children are likely to be highly correlated with regards to disease environment, food security, and availability of services such as health and education. Correlated environments could cause spatial confounding of the relationship between education and health, and maternal height.

These different mechanisms are likely to be operating to a different extent depending on the outcome studied. The leading causes of under-5 mortalities are infectious diseases while preterm birth complications, pneumonia, and intrapartum-related complications are major causes of neonatal deaths. Infant- and especially child mortalities, result from a process with chronic conditions and multiple exposures to diseases and undernutrition, as well as pregnancy and birth complications, rather than being an acute phenomenon. Neonatal mortality, on the other hand, is more likely to directly relate to pregnancy and perinatal complications, such as preterm birth complications (Liu et al., 2017; Mosley and Chen, 1984; WHO, 2017b). Therefore we expect the association between maternal height and neonatal mortality to be more directly related to pregnancy- and perinatal related factors. The association with infant-, and especially child mortality, is likely to be more confounded or mediated by factors correlated with height, such as maternal education, adult living standards, fertility, community, and father characteristics. We expect the associations with school attendance to be related to accumulated health in childhood, as healthier children are more likely to attend school (Alderman et al., 2001; Heckman, 2007).

Government expenditure on health is likely to be particularly beneficial for disadvantaged households. Its main policy objectives relating to health are to increase efficiency by producing external benefits and public goods, as well as equity since access to health care cannot be provided universally solely through the market (Castro et al., 2000). This is especially true for preventive measures, such as control of infectious diseases, water, and food sanitation, which are mostly public goods, or at least interventions with high positive externalities. Government health spending is also better at funding curative as well as preventive health care at low cost to the end-consumer, which improves access for the poor (Musgrave, 1996). Increased government expenditures should improve access to health care and improve public health. If these benefits are universal, they can compensate for household disadvantages, reducing the association between maternal height and child outcomes. Isolating a specific effect of increased government health expenditure is difficult, because it relates to many aspects of population health, and development more generally. For example, giving birth in a health care facility and receiving pre- and antenatal care can significantly reduce the risks associated with giving birth for disadvantaged women. After birth, parents are supported in preventing and responding to diseases and improved infectious disease control will have benefits regardless of access. For these reasons, more public spending on health should mitigate the intergenerational transmission of disadvantage.

Data and variables

We use data from Demographic and Health Surveys (DHS) for 35 SSA countries surveyed 1992-2016. The sample sizes and countries included depend on the outcome studied and the estimation strategy used. The DHS aims to provide nationally representative and reliable data using standardized household surveys. The sampling procedure is a stratified two-stage sampling process, most commonly arranged by geographic or administrative regions crossed with the type of residence (urban/rural). Clusters (census enumeration areas) are randomly selected from each stratum, and then households are sampled from these clusters. Women, typically aged 15-49, from these households, are interviewed, and their birth histories gathered as well as information on the households and its members (Corsi et al., 2012; The DHS Program, 2016). We exclude children born to women less than 20 years old and that are not residents at the household of interview. We further only include singleton births. Table A0 shows the surveys included.

Outcome variables

The primary outcomes are binary variables for whether the child died before the age of five (Under-5 mortality) and whether school-age children and adolescents

attended school in the year of the survey (School Attendance). We also look at age-specific mortality, distinguishing between mortality in the first month of life (neonatal mortality), 1-12 months (postneonatal mortality), and 13-60 months (child mortality). Looking at age-specific mortality gives us an idea of different mechanisms since they are determined by somewhat different, although overlapping, factors. When looking at postneonatal mortality, we exclude children that died in their first month of life or were less than one month old at the time of the survey. When looking at child mortality, we exclude children that died before age 12 months or were below 12 months at the time of the survey. Education is derived from the household questionnaire, which records basic characteristics of all individuals present in the household at the time of the survey. Our analysis of education only includes children and adolescents aged 7-16 at the time of the survey. All outcomes are linked to the birth histories and only include biological children of respondents.

Maternal height

We calculate maternal height z-scores using the mean and standard deviation from our complete DHS sample. We exclude women under age 20, so most women in our sample have reached final adult height.

Government Health Expenditures

We use total government expenditures on health per capita in constant 2010 USD, generally available between 1995-2014, obtained from WHO's Global Health Expenditure Database (WHO, 2017a). Government expenditures on health include all resources channeled through the government, in addition to expenditures by compulsory health insurance, extrabudgetary entities, and parastatals. It includes expenditures by governmental entities regardless of source, including donor contributions flowing through them. It also includes governmental transfer payments to households intended to cover the cost of medical care and extrabudgetary funds for financing health services and health-related goods. It includes both current- and capital expenditures. We show government health expenditure graphically in Figure A1.

Control variables

The purpose of our control variables is to account for and estimate the statistical impact of potential confounders or mediators. Our basic control variables are; survey (i.e., country and year of survey), sex of the child, and months since birth (i.e., child's age if alive). Other covariates relate to SES and material living standards of the household. Maternal education, similarly to maternal height, signals past living standards, as well as acquired skills and capabilities. A household wealth factor scores, provided by the DHS and derived from a principal

component analysis using ownership of various assets and household materials, captures household living standards. We standardize the factor scores for each survey and calculate survey-specific z-scores of wealth. Finally, we use paternal education specified as a continuous measure of years of schooling. We adjust for missing information on paternal education using dummy variable adjustment.

Further covariates relate to birth and fertility. Mother's age at birth is associated with child outcomes through both SES and biological factors (Aldous and Edmonson, 1993; Fraser et al., 1995; Schmidt et al., 2011; Thurnham, 2013). A short birth interval has been found to have harmful effects on children and appears to be more common in households with lower SES (Curtis et al., 1993; Rutstein, 2005). We adjust birth interval for first-born children using dummy variable adjustment. Birth order, independent of mother's age, may influence the allocation of resources to children within the household as well as pregnancy-related aspects (Cabrera, 1980; Horton, 1988). Number of siblings (ever born) addresses strains on household resources as well as other factors.

In addition to these individual level controls, we also control for private health expenditure per capita and GDP per capita when looking at the interaction between maternal height and government health expenditure. As government health expenditure, we obtain these variables from the WHO's Global Health Expenditure Database (WHO, 2017a).

Methods

We start by estimating the relationship between maternal height and child outcomes and decompose the impact of covariates. We estimate linear probability models (LPM) for each child outcome y .

$$y = \alpha^{\text{basic}} + mz\beta^{\text{basic}} + \mathbf{x}'\boldsymbol{\gamma}^{\text{basic}} + \varepsilon^{\text{basic}} \quad (\text{Eq.1})$$

where mz is maternal height z-score. Our basic model includes controls in \mathbf{x}' , which are months since birth, child's sex, and survey. To test the influence of maternal education, SES, fertility, and community, we add four sets of additional covariates: 1) mother's education ($mEdu$); 2) household wealth index and father's education (SES); 3) variables related to birth and maternal fertility: being first born, birth order, mother's age at birth, mother's age at first birth, birth interval, and number of siblings (Fertility); 4) community-level-means of included observations for all independent variables (Community). Adding community-level-means of all independent variables (so-called Mundlak fixed effects) is parametrically identical to a fixed effects models controlling for unobserved community-level characteristics (or adding a dummy coded variable for

community) (Antonakis et al., 2010; Mundlak, 1978). Communities are identified with sampling clusters, which generally consist of an area such as a village or a neighborhood in an urban area (Measure DHS, 2012).

$$y = \alpha^{\text{full}} + mz\beta^{\text{full}} + \mathbf{x}'\gamma^{\text{full}} + \mathbf{SES}'\rho_1 + m\text{EDUP}_2 + \mathbf{Birth}'\rho_{3b} + \\ \mathbf{Community}'\rho_4 + \varepsilon^{\text{full}} \quad (\text{Eq.2})$$

We quantify the influence of each set of covariates on the association of maternal height and child outcomes using Gelbach decomposition. The total difference between the maternal height estimates from equations (1) and (2), i.e., $\delta = \beta^{\text{basic}} - \beta^{\text{full}}$, is the total amount of association between maternal height and child outcome explained by covariates for maternal education, SES, fertility, and community-level factors. δ is then decomposed into four components quantifying the impact of each set of covariates separately (see Gelbach, 2016).

We also estimate the influence of father's (strictly speaking, the husband of the respondent) characteristics (father fixed effects) on the estimate of maternal height in a similar way. The sample is restricted to polygynous households where at least two wives with at least one child each were interviewed. We identify polygynous households through several questions in the DHS. Women are identified as being in a polygynous marriage if they answer that they have one or more co-wives and are married to the household head. At least two co-wives have to be interviewed and have non-missing values on all variables used. We exclude the few cases where the husbands of the different cowives do not have the same reported education level. The basic model is the same as in equation (1), but the full model adds terms for father-specific means of included observations for all independent variables to equation (2), and excludes the terms for SES, as they do not vary for the same father. Using father fixed effects allows estimating whether father characteristics influenced the association between maternal height and child outcomes. Polygynous households, however, are unlikely to be representative of the whole population in many regards. Nonetheless, as maternal height appears to be consistently associated with under-5 mortality in different populations, estimates from this subsample should indicate whether fathers (or households) influence the relationship. The decomposition of the impact of covariates relating to maternal education, SES, and fertility are unlikely to be generalizable from the polygynous households, as they depend more on behavioral, social and cultural factors.

We then estimate the interactions between maternal height and government health expenditures. For this analysis, we restrict the sample to children with available health expenditure data for the year of birth and the year before birth.

$$y_{yob} = \alpha^I + (P_{yob} \times mz)\theta_0^I + (P_{yob-1} \times mz)\theta_1^I + P_{yob}\rho_0^I + P_{yob-1}\rho_1^I + mz\beta^I + z'_I \gamma_I^I + \varepsilon^I \quad (3)$$

In each model, we interact maternal height with median-centered government health expenditures, P , indexed to year of birth, yob , as well as the year before birth, $yob-1$. We center government expenditures on a median of available years 1995-2014 for all of SSA, with available data in the WHO Global Health Expenditure Database. We index government health expenditure to both year of birth and year before birth since they are both likely to be important for development and since it is unclear when precisely government health expenditure would have an effect on child health or moderate the relationship with maternal height. We present coefficients for health expenditures both in the year of birth and the year before birth, as well as the coefficients for their interaction with maternal height. There is, however, a high correlation between expenditure at the year of birth and year before birth, so the standard errors are inflated (variance inflation factor of around 40-50). Therefore we also index government health expenditure (and other macro variables) to single years in our sensitivity analysis, which reduces the variance inflation factor to around 10 but gives similar estimates. We present a linear combination of the two coefficients as our main estimates.

The control variables include SES, maternal education, fertility, community, and basic controls, as well as a country-specific year of birth trends. We also control for median-centered GDP and private health expenditure and their interaction with maternal height, both in the year of birth and year before birth. Finally, we control for interaction between mean-centered maternal age at birth and maternal height. It appears that an interaction between maternal age and maternal height bias the estimates upwards since mothers giving birth in later years, with more healthcare expenditure, are on average older. This is primarily a problem for models using mother fixed effects (see below), as estimates are considerably larger than for the other models when not taking the interaction between maternal age and maternal height into account. Centering interacted variables on their mean or median makes interpretation of other terms involving maternal height easier as the terms equate to zero when the interacted variable is at its median or mean. β^I is the baseline association for maternal height, i.e., when all macro variables are at their median and children are born to average aged mothers. We also show our results stratified by household wealth tercile and residency (urban/rural).

Finally, we explore our interaction models using different levels of fixed effects. Firstly we estimate mother fixed effects, adding a mother-specific mean for all independent variables, and secondly a country- and year-of-birth fixed effects, adding a ‘Country x Year of Birth’-specific mean for all independent variables. This is similar to the approach in Bhalotra and Rawlings (2013). The mother fixed

effects do not allow us to estimate the main association between maternal height and child outcomes (as it does not vary between siblings). ‘Country x Year of Birth’ fixed effects do not allow us to estimate the main association of government health expenditures on child outcomes (as we use annual country-level data which does not vary within a country and a year). Therefore, our preferred specifications are the community fixed-effects models. It is also unlikely that different levels of fixed effects, below the country level, have any great influence on the interaction between maternal height and government health expenditure as government health expenditure are measured at the national level. We present results comparing the three levels of fixed effects graphically where we categorize government health expenditures into septiles, where septile 4 is the omitted reference category.

Estimates are obtained using sampling weight. We adjust the sampling weights to add up to one for each survey, so larger sample sizes do not drive our results. An exception is estimates obtained from the polygamy sample which are unweighted. Standard errors are adjusted for clustering at the community level (PSU).

Empirical results

The map in Figure 1 shows the study area, which covers 35 of the 48 countries in SSA. For surveys with available GPS data the map shows locations of each community and indicates whether they have any polygynous households in the sample. Most polygynous households are in West Africa, but there are some polygynous households in most countries.

Table 1 shows descriptive statistics along with the variable specifications. We show descriptive statistics separately for observations from the full sample and observations included in the analysis of health expenditures only. The first row displays the mean of each outcome variable. Because individuals included in the analysis of government health expenditures are generally born later, they have on average a better outcomes, or 0.11 under-5 mortality compared to 0.14 in the full sample, and 0.76 attending school compared to 0.074. Table A1 (appendix) displays descriptive statistics for the samples used in alternative specifications (father fixed effects in polygynous households and women with at least two children for mother fixed effects).

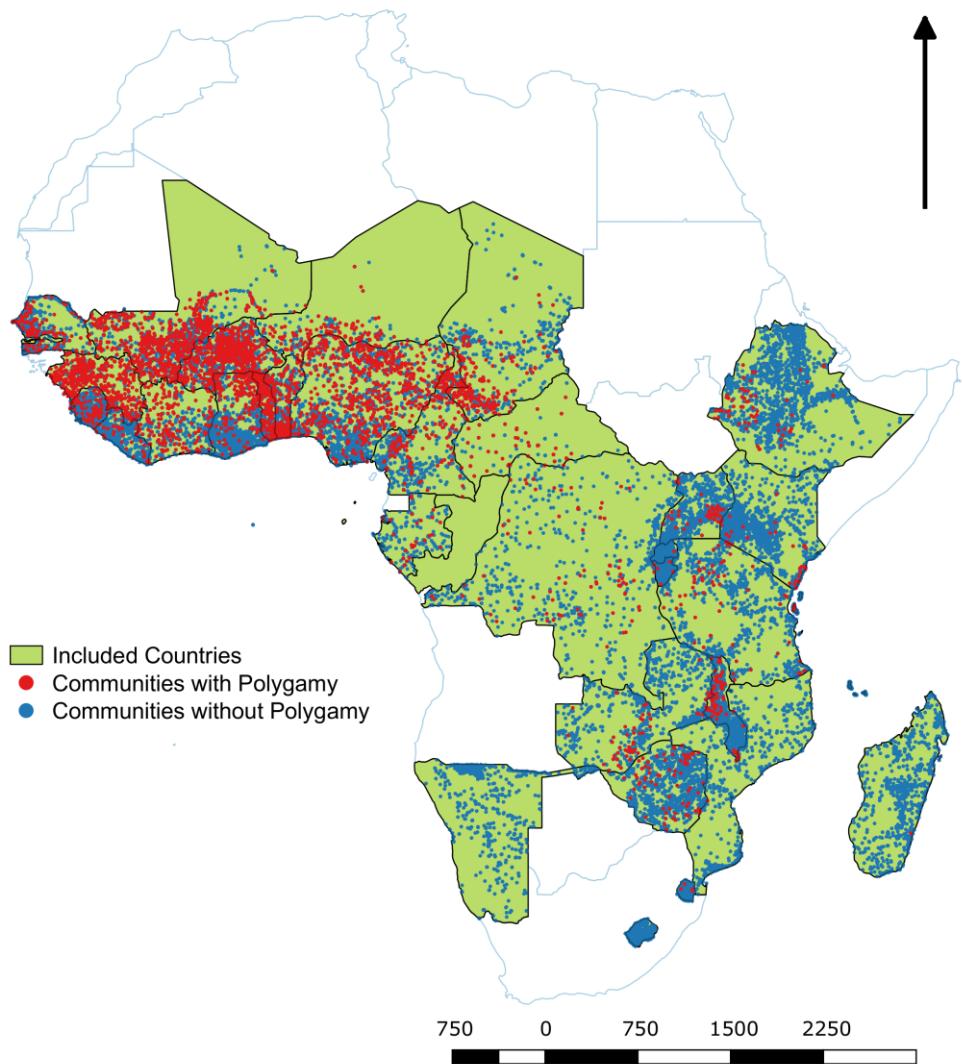


Figure 1. Map of the study area

Notes: Only communities with available GPS coordinates are marked with dots (More are included in the analysis). The map is for the mortality sample. Polygamy communities are marked above other communities. All countries have at least some polygamy, besides Comoros, Gambia, Lesotho, and Sao Tome & Principe for the mortality outcomes. Central African Republic, Comoros, and Gambia are excluded from the analysis of education on the full sample and additionally Namibia, and Swaziland on the polygamy sample.

Table 1. Descriptive statistics

	Full Sample		With Available Exp. Data		
	Under-5 Mort.	School.	Under-5 Mort.	School.	Form
Outcome ¹	0.14 [0.34]	0.74 [0.44]	0.11 [0.31]	0.76 [0.43]	Binary
Maternal Height Z-Score	0.01 [0.99]	-0.01 [0.99]	-0.01 [1.00]	-0.03 [0.99]	Linear
Year of Birth	1996.3 ² [9.23]	1997.3 ² [5.70]	2003.1 [4.79]	2001.2 [3.55]	Linear
Months since Birth	125.9 [88.76]	129.94 [33.54]	78.28 [53.69]	120.17 [30.84]	In
Female	0.49 [0.50]	0.49 [0.50]	0.49 [0.50]	0.49 [0.50]	Binary
Mat. Education (years)	3.14 [3.96]	3.59 [4.06]	3.37 [4.09]	3.74 [4.08]	Linear
Pat. Education (years)	4.39 [4.76]	4.93 [4.77]	4.65 [4.86]	5.15 [4.78]	Linear
Missing Pat. Educ.	0.07 [0.25]	0.06 [0.23]	0.06 [0.24]	0.06 [0.24]	Binary
Wealth Z-Score	-0.13 [0.91]	-0.11 [0.94]	-0.14 [0.91]	-0.14 [0.92]	Quadr.
Birth Interval (months)	34.68 [19.85]	35.98 [20.95]	36.55 [21.27]	36.14 [21.27]	Quadr.
Birth Order	3.35 [2.26]	3.49 [2.24]	3.65 [2.38]	3.49 [2.25]	Quadr.
First Born	0.24 [0.43]	0.22 [0.41]	0.2 [0.40]	0.22 [0.41]	Binary
Maternal Age at birth	24.66 [6.34]	25.42 [6.12]	26.05 [6.53]	25.54 [6.25]	Quadr.
Siblings Born	5.73 [2.73]	5.86 [2.43]	5.04 [2.56]	5.69 [2.40]	Linear
Mat. Age at 1st Birth	19.09 [4.02]	19.73 [4.28]	19.52 [4.18]	19.92 [4.38]	Quadr.
Gov. Exp. at Birth			16.48 [18.36]	15.56 [18.81]	In
Private Exp. Birth			23.73 [24.19]	21.32 [26.22]	In
GDP at Birth			904.86	844.24	In

			[1,178.04]	[1,278.27]	
Communities	42,333	27,140	37,561	21,526	
Mothers	508,718	194,861	408,754	128,858	
Countries	35	32	32	29	
Observations	2,060,693	385,255	1,088,191	219,412	

Notes: Means are adjusted using sample weights. Standard deviations in brackets. ¹Outcomes shown are Under-5 Mortality and School Attendance. Means for Neonatal- (.04 and .03), Postneonatal- (.06 and .05), and Child Mortality (.06 and .04) not shown. ²Variables not included in statistical models. Form indicates how the variables are included in statistical models. Abbreviations: Maternal (Mat); Paternal (Pat); Governmental (Gov.); Expenditure (Exp.); Mortality (Mort.); School Attendance (School.); Quadratic (Quadr.); Natural Logarithm (ln). Source: DHS (2016); WHO, (2017)

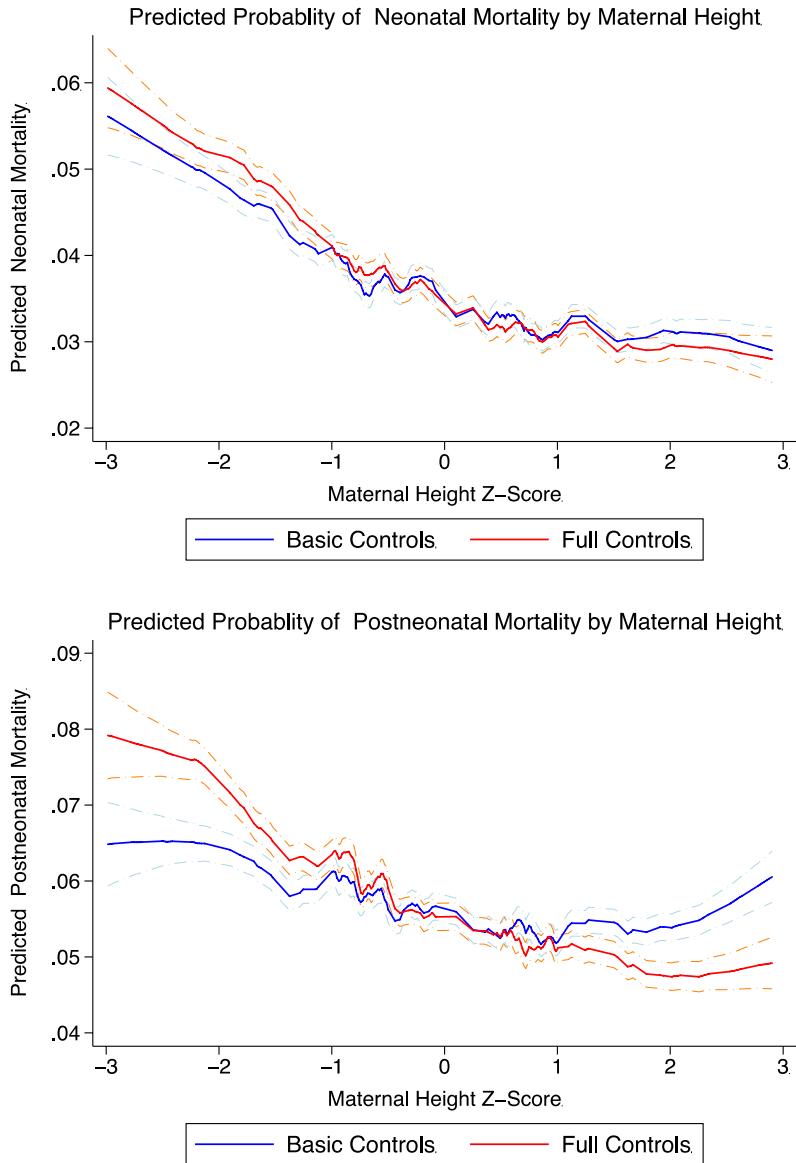
The main association between maternal height and child outcomes and decomposition of covariates

Figure 2 shows predicted probabilities of the different outcomes using fitted lines from a non-parametric nearest-neighbor local linear smoother (Royston and Cox, 2005), both adjusted for our basic controls and including a full set of covariates. The relationship appears to be reasonably linear between -2 and +2 standard deviations in height (z-scores). The gradient is more apparent for the basic models than the fully adjusted ones, for under-5 mortality, child mortality, and education. There is not as clear a difference in gradients for infant mortality and especially not for neonatal mortality.

Table 2 displays the results of our analysis on the association between maternal height and the different outcomes, as well as the decomposition of the impact of our covariates. The two columns titled Models show coefficients for maternal height from our basic model and our full model with added covariates for maternal education, household SES, fertility, and community-level fixed effects. All coefficients are statistically significant and in the expected direction in the full sample. For under-5 mortality, the different models show associations with maternal height, ranging from a .013 decrease in the linear probability of dying before age 5 for a single standard deviation increase in maternal height in the basic model to -.0087 in the full model. Considering a sample mean under-5 mortality of .14, the magnitudes of these estimates are considerable.

Looking at age-specific mortality, it is clear that the association is similar for neonatal- and postneonatal mortality in the basic models, -0.0053 and -0.0051, but when all controls are added the estimate for postneonatal mortality is considerably lower. The association with child mortality is lower than for the other two outcomes. Maternal height appears to be most important for neonatal mortality, especially considering that the sample mean for neonatal mortality is lower than for postneonatal and child mortality. The coefficient of maternal height for education is positive, as expected, but the magnitudes differ greatly from .017 in

the basic model to .0038 in the full model, indicating that mother's education, wealth, and father's education explain a large part of the raw association. Considering that the baseline probability of child education is .77, these estimates are quite small.



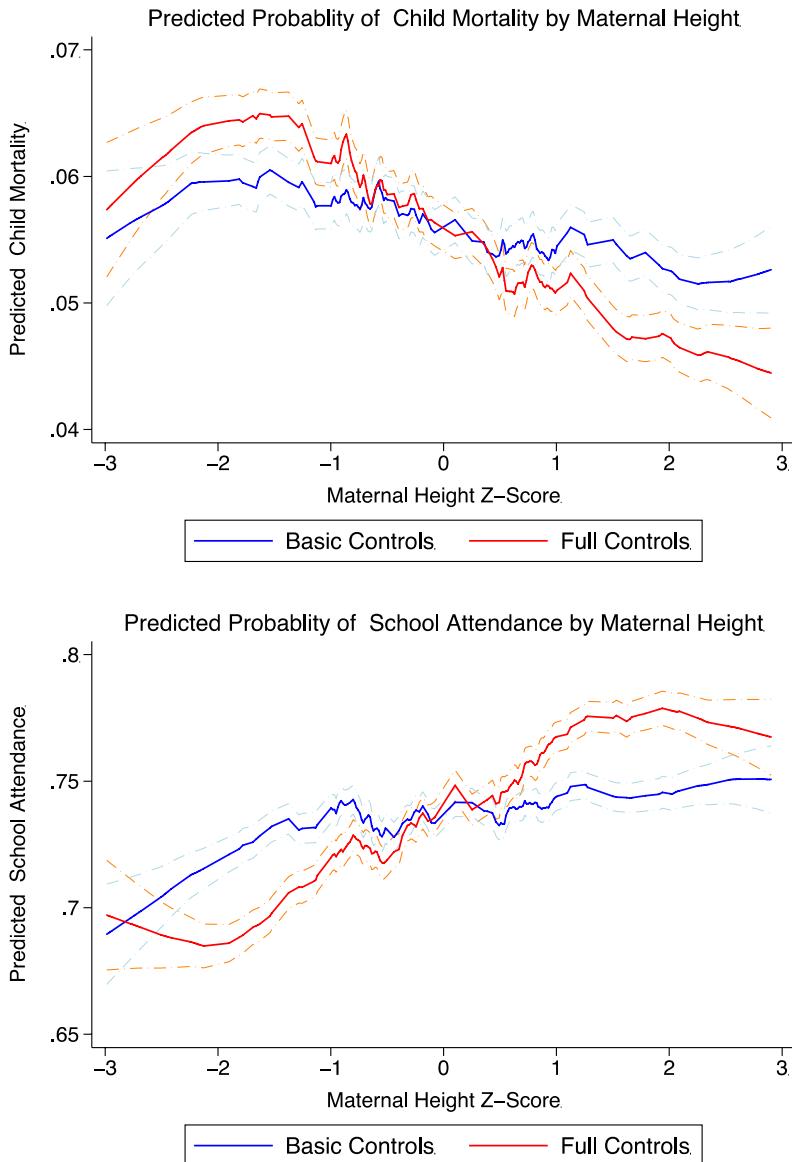


Figure 2. Predicated outcomes by maternal height (adjusted) using fitted lines from a non-parametric nearest-neighbor local linear smoother

Notes: Basic Controls: Survey (i.e., country and survey year); Months since birth; Sex of child; A constant term. Full Controls: Basic Controls; Mother's education; Mother's age at birth; Mother's age at first birth; Birth order, Birth interval; Number of siblings born; Being firstborn; Father's education and Household wealth; Community specific means of included observations for all independent variables (Community fixed effects). The fitted lines are obtained using a non-parametric a nearest-neighbor local linear smoother (weighted least squares). Estimation is run on 50 percent of the full sample, stratified by survey. For visualization purposes, maternal height z-scores above 3 and below -3 are omitted from the graph. Obtained using STATA mrunning command. Standard errors are underestimated as they are not adjusted for correlation with control variables (Royston and Cox, 2005).

Table 2. Association between maternal height and child outcomes and decomposition of covariates

Outcome	Models	Gelbach Decomposition of Covariates						Community	Total
		Basic (eq1)	Full (eq2)	Mat. Educ.	HH SES	Fertility	Community		
<i>Full Sample</i>									
Under-5 Mortality (N = 2,060,693)	-0.0130*** (0.0004)	-0.0087*** (0.0004)	-0.0003*** (0.0001)	-0.0012*** (0.0001)	-0.0011*** (0.0001)	-0.0016*** (0.0003)	-0.0016*** (0.0003)	-0.0042*** (0.0003)	
Neonatal Mortality (N = 2,060,693)	-0.0053*** (0.0002)	-0.0044*** (0.0002)	-0.0001** (0.0000)	-0.0000 (0.0000)	-0.0004*** (0.0000)	-0.0005*** (0.0000)	-0.0005*** (0.0001)	-0.0009*** (0.0001)	
Infant Mortality (N = 1,982,257)	-0.0051*** (0.0003)	-0.0030*** (0.0003)	-0.0001*** (0.0000)	-0.0005*** (0.0001)	-0.0005*** (0.0000)	-0.0009*** (0.0000)	-0.0009*** (0.0002)	-0.0021*** (0.0002)	
Child Mortality (N = 1,758,514)	-0.0044*** (0.0003)	-0.0025*** (0.0003)	-0.0001*** (0.0000)	-0.0008*** (0.0001)	-0.0003*** (0.0000)	-0.0005*** (0.0000)	-0.0005*** (0.0002)	-0.0018*** (0.0002)	
School Attendance (N = 385,255)	0.0170*** (0.0013)	0.0038*** (0.0010)	0.0029*** (0.0002)	0.0069*** (0.0003)	0.0004*** (0.0001)	0.0031*** (0.0009)	0.0031*** (0.0009)	0.0132*** (0.0010)	
<i>Polygamy Sample</i>									
Under-5 Mortality (N = 143,344)	-0.0139*** (0.0014)	-0.0081*** (0.0019)	-0.0001 (0.0001)	-0.0039** (0.0019)	-0.0004** (0.0002)	-0.0014 (0.0011)	-0.0014 (0.0011)	-0.0056*** (0.0016)	
Neonatal Mortality (N = 143,344)	-0.0068*** (0.0008)	-0.0058*** (0.0011)	-0.0000 (0.0001)	-0.0011 (0.0010)	-0.0000 (0.0001)	-0.0000 (0.0001)	-0.0001 (0.0006)	-0.0010 (0.0008)	
Infant Mortality (N = 135,535)	-0.0061*** (0.0010)	-0.0048*** (0.0013)	-0.0000 (0.0001)	-0.0003 (0.0013)	-0.0002** (0.0013)	-0.0008 (0.0001)	-0.0008 (0.0006)	-0.0013 (0.0010)	
Child Mortality (N = 115,678)	-0.0043*** (0.0011)	0.0003 (0.0015)	-0.0001 (0.0001)	-0.0035** (0.0001)	-0.0001 (0.0001)	-0.0009 (0.0009)	-0.0009 (0.0009)	-0.0046*** (0.0013)	
School Attendance	0.0130**	0.0057	0.0000	0.0055	0.0003	0.0015	0.0015	0.0073	

(N = 18,469)	(0.0053)	(0.0041)	(0.0002)	(0.0075)	(0.0004)	(0.0060)	(0.0056)
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Notes: Estimates from the Full Sample are adjusted using sampling weights, and standard errors in parentheses are adjusted for clustering at the community (Full Sample) or father level (Polygyny). All coefficients from models are for maternal height. Basic Controls: Survey (i.e., country and survey year); Months since birth; Sex of child; A constant term. Full Models: Basic Controls; Mother's age at birth; Mother's age at first birth; Birth order; Birth interval; Number of siblings born; Being first born (Fertility); Community specific means of included observations for all independent variables (Community); Father's education and Household wealth (HH SES) in models on Full Sample and Father specific means of included observations for all independent variables for Polygamy sample (HH/Father). The Gelbach decomposition quantifies the part of the association, between maternal height and child outcomes, attributable to four sets of controls added to the full model; maternal education (Mat. Educ.); Household wealth index and paternal education (HH SES) or unobserved father characteristics (HH/Father); fertility (Fertility), and unobserved community-level factors (Community). *p < .10; **p < .05; ***p < .01. Source: DHS, 2016.

The columns labeled Gelbach decomposition in Table 2 show the impact of each set of covariates on the association of maternal height with mortality and school attendance. The total contribution of all covariates added to the full models is -0.0042 (32 percent of the association in the basic model) for under-5 mortality, -0.0009 (17 percent) for neonatal mortality, -0.0021 (41 percent) for postneonatal mortality, -0.0018 (41 percent) for child mortality, and .0134 (78 percent) of the association with being in school. For under-5 mortality, community-level characteristics have the greatest influence, followed by fertility, household SES, and maternal education. For neonatal mortality, community and fertility explain the most, but maternal education also has an influence. For postneonatal mortality, community explains the most, followed by fertility, household SES, and maternal education. The results are somewhat different for child mortality and education. For child mortality, household SES explains the most, followed by community, fertility, and maternal education. For being in school, household SES explains most of the association, followed by community and maternal education. Our education outcome is different from the mortality outcomes as fertility explains very little of the association while maternal education explains a substantial part of the association.

The second panel of Table 2 shows the results for the polygyny sample where the full models are adjusted for unobserved paternal characteristics instead of household living standards and paternal education. The basic models look reasonably similar to the models on the full sample, although the estimates for neonatal and postneonatal mortality are greater. Adding a full set of controls, including the father fixed effects, shows that child mortality and education are not statistically significantly associated with maternal height, although the association with education is greater in magnitude than in the full sample. The association with under-5-, neonatal-, and postneonatal mortality are attenuated but still substantial, statistically significant, and of similar magnitudes as the estimates from the full sample. The father fixed effects explain most of the association between maternal height and child mortality. It also explains the attenuation in the association for education, but it is not statistically significant.

Figure A2 presents estimations of separate models for each country. The models include community fixed effects and a full set of control variables and show a consistent relationship between mother's height and under-5 mortality, which is always in the expected direction and is statistically significant in all but five countries. For child education, the associations are statistically significant in only eleven of the countries. The association between the mother's height and mortality is robust and is present in different contexts while context seems to matter more for the association between mother's height and child education.

Table 3. Interactions between government expenditure on health and maternal height using community fixed effects

Outcome	At Birth	Before Birth	Sum	Government Health Expenditure x Height			Height Sum
				At Birth	Before Birth	Sum	
Under-5 Mortality	-0.0086*** (0.0019)	-0.0059*** (0.0019)	-0.0145*** (0.0017)	0.0007 (0.0017)	0.0023 (0.0017)	0.0030*** (0.0010)	-0.0079*** (0.0005)
Neonatal Mortality	-0.0005 (0.0011)	-0.0026** (0.0012)	-0.0031*** (0.0010)	-0.0013 (0.0010)	0.0018* (0.0010)	0.0005 (0.0005)	-0.0039*** (0.0003)
Postneonatal Mortality	-0.0027** (0.0013)	-0.0038*** (0.0014)	-0.0065*** (0.0012)	0.0025* (0.0013)	0.0001 (0.0013)	0.0025*** (0.0013)	-0.0028*** (0.0004)
N = 1,050,028							
Child Mortality	-0.0062*** (0.0012)	0.0004 (0.0011)	-0.0058*** (0.0012)	-0.0003 (0.0010)	0.0007 (0.0010)	0.0004 (0.0005)	-0.0020*** (0.0003)
N = 907,078							
School Attendance	0.0487*** (0.0056)	0.0118* (0.0053)	0.0606*** (0.0067)	-0.0040 (0.0038)	0.0043 (0.0039)	0.0003 (0.0020)	0.0052*** (0.0014)
N = 219,409							

Notes: Estimates are weighted, and standard errors in parentheses are adjusted for clustering at the community level. Coefficients show the main association of government health expenditure. Interactions coefficients are differences in the slope for maternal height for children exposed to a log point increase in government health expenditure. 'At Birth' shows coefficients for government health expenditure (and its interactions with maternal height) linked to year of birth and 'Before Birth' shows coefficients for government health expenditure indexed to year before birth (and its interactions with maternal height). 'Sum' shows linear combination of the coefficients for government health expenditure linked to year of birth and year before birth (or their interaction with height). Controls: Months since birth; Sex of child; A constant term; Maternal age at birth; Maternal age at first birth; Birth order; Birth interval; Number of siblings born; Being firstborn; Paternal education; Household wealth; Community specific means of included observations for all independent variables (Community Fixed Effects). Other controls: GDP indexed to year of birth and year before birth; Maternal height interacted with GDP indexed to year of birth and year before birth; Private health expenditure indexed to year of birth and year before birth; Maternal height interacted with private health expenditure indexed to year of birth and year before birth; An interaction between maternal height and maternal age at birth; An interaction between child's year of birth (linear) and country; GDP, and government- and private health expenditure are median-centered (for all included countries and available data 1995 – 2014), so the interaction terms are 0 when they are at their median. Similarly, maternal age at birth is mean-centered when interacted with maternal height. If the main association of height is negative, a positive interaction coefficient means that an increase in government health expenditure decreases that association of maternal height with child outcome (and vice versa). * p < .10; ** p < .05; *** p < .01. Source: DHS, 2016; WHO, 2017.

Changing context: Interaction between government spending on health and maternal height

In Table 3 we turn to the estimates from the interaction models, where maternal height is interacted with government health expenditure at birth and year before birth separately. Government health expenditures are associated with all outcomes in the expected direction. The individual interaction coefficients do not show statistically significant associations for any of our outcomes. The linear combinations of the two interaction terms show that a ten percent increase in government health expenditures the year before birth and the year of birth lower the association of maternal height with postneonatal mortality by 0.00026 z-score ($\ln(1.1) \times 0.0027$). The main association of GDP and private health expenditures and its interaction with maternal height are generally not statistically significant (See Table A2).

Figure 3 shows the results graphically and makes comparisons with alternative fixed-effects models. It is clear that our results for under-5 mortality are highly similar across the different models. The baseline association of maternal height (septile 4) is -0.0069 in both the community- and ‘country x year of birth’ fixed-effects models. An increase in government health expenditures decreases the association of maternal height up to the median (septile 4). The maternal height associations for septiles 5 and 6 are not statistically significantly different from that in septile 4 in any model, but the highest septile has a weaker association, although it is not statistically significant when using mother fixed effects. Being born into the lowest septile (less than USD 6 per capita) compared to the median septile (USD 13-18 per capita) shows around twice the estimated association for maternal height or around 0.01 z-score. Being born into the highest expenditure (USD 119-762 per capita) also decreases the estimated association by about 0.01 z-score reducing it to practically 0. Only two countries in our sample reach this level of expenditure over the period studied. Figure 3 also shows that there is very little association between maternal height and education, and it is not statistically significant at the baseline (septile 4).

Table A3 displays results from the interaction models stratified by wealth terciles and residency (urban/rural), respectively. The main association between government health expenditures and under-5 mortality is strongest for the poorest terciles and rural residence, and weakest for the richest tercile. The interaction with maternal height, however, is only statistically significant for the middle tercile, and a 10 percent increase in expenditures decreases the association of maternal height by 0.0005 z-score ($\ln(1.1) \times 0.0052$). For education, the main association of government health expenditures is also the strongest for the poorest tercile and weakest for the richest tercile, as well as being stronger for rural residents.

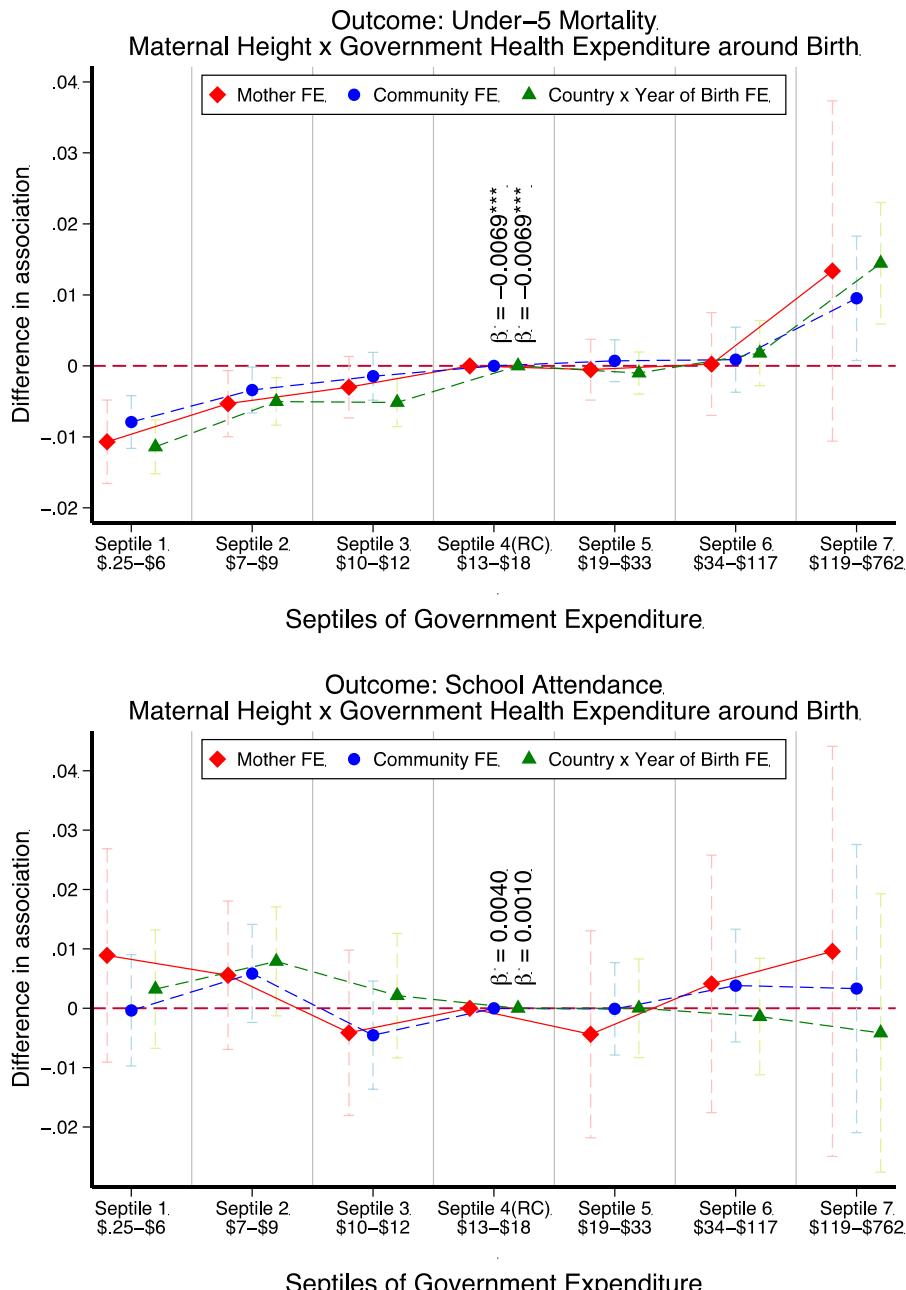


Figure 3. Interaction between septiles of government expenditure on health and maternal height.

Notes: Confidence intervals are 95 percent and are adjusted for clustering at the level of Mother (Mother FE) or Community (Community FE and Country x Year of birth FE). Estimates are weighted. The estimates are differences in

the slope for maternal height for children exposed to different septiles of expenditure compared to a reference category (fourth septile) for child outcomes. Each estimate is a linear combination of the interaction coefficient for maternal height and government health expenditure septile indexed to year before birth and year of birth. The baseline coefficient for maternal height and its p-value are shown above the fourth decile when using community, and country x year of birth fixed effects. Since height does not vary between siblings it is not possible to estimate the baseline coefficient when using mother fixed effects, only the difference. Controls: Months since birth; Sex of child; A constant term; Maternal education; Maternal age at birth; Maternal age at first birth; Birth order, Birth interval; Number of siblings born; Being firstborn; Paternal education; Household wealth; Either Community-, maternal-, or country x year of birth specific means of included observations for all independent variables (Community-, Mother-, or Country x Year of Birth Fixed Effects). Other controls: GDP indexed to year of birth and year before birth (except Country x Year of Birth FE); Maternal height interacted with GDP indexed to year of birth and year before birth; Private health expenditure indexed to year of birth and year before birth (except Country x Year of Birth FE); Maternal height interacted with private health expenditure indexed to year of birth and year before birth; Dummy coded septiles of government health expenditure (except Country x Year of Birth FE); An interaction between maternal height and maternal age at birth; An interaction between child's year of birth (linear) and country. GDP and private health expenditure are median-centered (for all included countries and available data 1995 – 2014), so the interaction terms are 0 when they are at their median. Similarly, maternal age at birth is mean-centered when interacted with maternal height. If the main association of height is negative, a positive interaction coefficient means that an increase in government health expenditure decreases that association of maternal height with child outcome (and vice versa). *p < .10; ** p < .05; *** p < .01. Source: DHS (2016); WHO (2017).

The interaction term, however, is only statistically significant for urban residents, and a 10-percent increase in expenditures decreases the association of maternal height by $0.00053 \text{ z-score} (\ln(1.1) \times 0.0056)$. The direct association of maternal height is not statistically significant for urban residents.

In table A4 we index government health expenditures to different years with respect to year of birth. Due to autocorrelation in government health expenditures, and uncertainty about when and for how long health expenditures in a single year is fully utilized to improve health or reduce the association of maternal height, these results cannot be interpreted as showing exactly when government health expenditures are most effective. They do, however, indicate that the time around birth is particularly crucial for mortality and its association with maternal height. The main association for government health expenditures is strongest around the time of birth for under-5 mortality. The coefficients are statistically significant from three years before birth until three years after birth. The interaction coefficients for under-5 mortality are statistically significant three years before birth and up to four years after birth but are strongest around the year of birth. For education, the association with government health expenditures is negative from 6 to 4 years before birth. Then it is positive from the year before birth until two years after birth, when it becomes negative again. The interaction coefficients are statistically significant when indexing government health expenditure to year 3-6 but it appears to increase the association with maternal height.

Discussion and Conclusion

We find a negative association between maternal height and under-5 mortality, in particular in the neonatal period and in infancy, which is highly robust to different model specifications. In particular, the associations do not depend on whether we

constrain identification within countries, communities or families (fathers). The association is present in almost all countries but varies in magnitude. Although maternal education and maternal height both relate to past living standards, education appears to explain little of the association between maternal height and under-5 mortality. The overlap between height and adult living standards, spatial clustering and fertility appear to account for much more of the association, especially for postneonatal and child mortality. Overall, our covariates explain 17 percent of the association between maternal height and neonatal mortality, and 42 percent of the association with postneonatal and child mortality, respectively, indicating that the association with neonatal mortality is direct to a much greater extent. The association is also greater for neonatal mortality relative to the mean than mortality later in childhood. Father and household characteristics explain most of the association with child mortality, but little of the association with neonatal and postneonatal mortality. The persistence and directness of the association with neonatal mortality indicate that maternal height relates to child outcomes through birth- and pregnancy-related complications. The height of the mother has a positive impact on the education of her children, although the strength of the relationship is weak and varies greatly between countries. Maternal education, household SES, and community level characteristics explain most of the association or around 78 percent. The weak association between maternal height and education indicates that maternal height does not influence child health in a way that also influences basic schooling to any great extent.

We find that government health expenditures around the time of birth substantially reduce the association between maternal height and under-5 mortality. However, it appears that such expenditures reduce the association for postneonatal and child mortality more than for neonatal mortality. Moreover, public spending on health does not modify the association between maternal height and child education. Although we find the main association with health spending to be more related to outcomes in poorer households, we only find a reduction in the maternal height association for the middle- and the highest tercile. This indicates that although government health expenditures have successfully reached and improved health outcomes of the poorer segments of the population, they do not seem to have lowered the intergenerational transmission of health from mother to child in this group. This could relate to differences in the type of health services benefiting the poorer and the better off. Increasing health expenditures can increase the health care utilization by the poor from nothing to basic lifesaving interventions in a way that increases the survival rate of their children, but does not prevent maternal disadvantages from being transmitted.

Our results confirm previous findings and show a consistent association between maternal height and under-5 mortality (Bhalotra and Rawlings, 2011; Monden and Smits, 2009; Özaltın et al., 2010). We further provide evidence that the observed

impact of maternal height is relatively direct for neonatal mortality but impacted by measures of fertility, community, and household living standards to a greater extent for mortality later in childhood. We also find a weak association between maternal height and schooling, which is mostly explained by household- and community level factors. Our results further corroborate a previous study which shows that improvements in aggregate national level factors, government health expenditures in our study, can substantially reduce, and even eliminate, the negative association between maternal height and under-5 mortality (Bhalotra and Rawlings, 2013).

Improvements in early life conditions between 1950 and 1980 in Europe brought about 3 cm increases in height for women (Garcia and Quintana-Domeque, 2007). Similar improvements in height would lead to almost half a standard deviation increase in the height of SSA women which would decrease under-5 deaths by about 0.4 percentage point, from about 10 percent to about 9.6 percent. Increasing adult heights by improving early-life conditions is a long-term solution. Public spending on health can have more immediate effects by reducing the intergenerational transmission of disadvantages as well as promoting child survival directly. For example, increasing government spending on health from under USD 6 to USD13-18 per capita is estimated to decrease the under-5 deaths attributable to maternal height by well over half. When government health expenditure is more than USD 119 per capita around the time of birth the estimated association between maternal height and under-5 mortality is practically zero.

To conclude, all over sub-Saharan Africa, poor health is transmitted from mothers to children. In the long term improved early-life conditions for women can eliminate this reproduction of poor health in the region. In the short-term, public spending on health can contribute significantly to lower the impact of poor maternal health and promote child survival.

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Appendix

Table A0. Countries and surveys included

Country	Survey years			
Benin	1996	2001	2006	2011
Burkina Faso	1992	1998	2003	2010
Burundi	2010			
Cameroon	1998	2004	2011	
Cent. Afr. Rep.	1994			
Chad	1996	2004	2014	
Comoros	1996	2012		
Congo	2005	2011		
Cote d'Ivoire	1994	1998	2011	
DR of the Congo	2007	2013		
Ethiopia	2000	2005	2011	2016
Gabon	2000	2012		
Gambia	2013			
Ghana	1993	1998	2003	2008
Guinea	1999	2005	2012	
Kenya	1993	1998	2003	2008
Lesotho	2004	2009	2014	
Liberia	2006	2013		
Madagascar	1997	2003	2008	
Malawi	1992	2000	2004	2010
Mali	1995	2001	2006	2012
Mozambique	1997	2003	2011	
Namibia	1992	2006	2013	
Niger	1998	2006	2012	
Nigeria	2003	2008	2013	
Rwanda	2005	2010	2014	
Sao Tome & Pr.	2008			
Senegal	2005	2010		
Sierra Leone	2008	2013		
Swaziland	2006			
Tanzania	1996	2004	2009	2015
Togo	1998	2013		
Uganda	1995	2000	2006	2011
Zambia	1996	2001	2007	2013

Zimbabwe 1994 1999 2005 2010 2015

Notes: If surveys were conducted on two calendar years the earlier year is shown

Table A1. Descriptive statistics. Polygynous households and mothers that have had at least two children.

	Polygyny		Mothers w. 2 children		Form
	Under-5 Mort.	School Att.	Under-5 Mort.	School Att.	
Outcome ¹	0.2 [0.40]	0.5 [0.50]	0.11 [0.32]	0.77 [0.42]	Binary
Mat. Height Z-Score	0.17 [0.96]	0.17 [0.97]	-0.01 [1.00]	-0.01 [0.99]	Linear
Year of Birth	1993.4 ² [8.48]	1995.4 ² [4.99]	2003.3 [4.68]	2001.7 [3.39]	Linear
Months Since Birth	126.89 [85.88]	129.89 [33.01]	82.23 [53.34]	126.17 [31.32]	In
Sex	0.49 [0.50]	0.47 [0.50]	0.49 [0.50]	0.49 [0.50]	Bin.
Maternal Education	0.87 [2.25]	0.98 [2.40]	3.2 [3.98]	3.49 [3.91]	Linear
Paternal Education	1.68 ² [3.52]	1.84 ² [3.75]	4.53 [4.80]	4.96 [4.68]	Linear
Missing Pat Education	0.02 ² [0.15]	0.01 ² [0.11]	0.05 [0.22]	0.06 [0.23]	Bin.
Wealth Z-Score	-0.31 ² [0.73]	-0.28 ² [0.77]	-0.18 [0.88]	-0.19 [0.88]	Quadr
Birth Interval	32.48 [17.10]	34.18 [19.12]	35.02 [18.88]	33.07 [16.95]	Quadr
Birth Order	3.74 [2.40]	3.99 [2.34]	3.74 [2.34]	3.71 [2.19]	Quadr
First Born	0.2 [0.40]	0.16 [0.36]	0.17 [0.37]	0.15 [0.35]	Bin.
Maternal Age at birth	24.91 [6.53]	25.97 [6.19]	26.06 [6.47]	25.79 [6.02]	Quadr
Nr. of Sib. Ever Born	6.52 [2.63]	6.68 [2.36]	5.3 [2.44]	6.21 [2.25]	Linear
Mat. Age at First Birth	18.14 [3.45]	18.39 [3.58]	19.39 [4.12]	19.99 [4.43]	Quadr
Gov. Exp. at Birth			16.42 [17.84]	15.67 [19.19]	In
Private Exp. at Birth			23.74 [23.59]	21.37 [27.41]	In
GDP at Birth			906.01 [1,152.06]	839.62 [1,325.80]	In
Communities	6,187	2,634	34,745	16,665	
Mothers	28,784	9,138	288,548	59,713	

Fathers	13,584	4,363	50,606	9,189
Countries	31	28	32	27
Observations	143,344	18,469	968,508	150,963

Notes: Means are adjusted using sample weights. Standard deviation adjusted for clustering in brackets. ¹Means for Neonatal- (.05 and .03), Postneonatal- (.08 and .05), and Child Mortality (.10 and .04) not shown. Only includes children born after 1995, when health expenditure data is available. Columns labeled Mother show mean for a subsample of mother's that had at least two children in the sample. ²Variable not included in statistical models. Form indicated the form the variables are entered in the statistical models. Source: DHS, 2017; WHO, 2017

Table A2. Interactions between macro-level variables and maternal height using community fixed effects.
Showing GDP and private health expenditure as well as government expenditure on health.

	Levels			Interactions		
	At Birth	Before Birth	Sum	At Birth	Before Birth	Sum
Under-5 Mortality (N = 1,088,191 $\beta^l = -0.0079^{***}(0.0005)$)						
Public	-0.0086***	-0.0059***	-0.0145***	0.0007	0.0023	0.0030***
Private	0.0009	0.0117***	0.0126***	-0.0035	0.0026	-0.0009
GDP	-0.0041	0.0037	-0.0004	0.0006	-0.0012	-0.0006
Neonatal Mortality (N = 1,088,191 $\beta^l = -0.0039^{***}(0.0003)$)						
Public	-0.0005	-0.0026**	-0.0031***	-0.0013	0.0018*	0.0005
Private	-0.0008	0.0022	0.0014	0.0002	-0.0001	0
GDP	-0.0078**	0.003	-0.0047	-0.0003	0.0006	0.0003
Postneonatal Mortality (N = 1,050,077 $\beta^l = -0.0028^{***}(0.0004)$)						
Public	-0.0027**	-0.0038***	-0.0065***	0.0025*	0.0001	0.0025***
Private	-0.0032	0.0128***	0.0095***	-0.0025	0.0015	-0.001
GDP	-0.0027	-0.003	-0.0057	-0.0031	0.0023	-0.0009
Child Mortality (N = 907,117 $\beta^l = -0.0020^{***}(0.0003)$)						
Public	-0.0062***	0.0004	-0.0058***	-0.0003	0.0007	0.0004
Private	0.0056***	-0.0027	0.003	-0.0014	0.0012	-0.0002
GDP	0.0057	0.0043	0.0101**	0.0042	-0.0041	0.0001
School Attendance (N = 219,412 $\beta^l = 0.0052^{***}(0.0014)$)						
Public	0.0487***	0.0118**	0.0606***	-0.004	0.0043	0.0003
Private	0.0221***	-0.0385***	-0.0164*	-0.006	0.0068	0.0008
GDP	-0.0289**	0.0495***	0.0206	0.0278***	-0.0301***	-0.0022

Notes: Estimates are weighted, and standard errors in parentheses are adjusted for clustering at the community level. Coefficients show the main association of government health expenditure. Interactions coefficients are differences in the slope for maternal height for children exposed to a log point increase in government health expenditure. At birth shows coefficients for government health expenditure (and its interactions with maternal height) linked to year of birth and Before birth shows coefficients for government health expenditure indexed to year before birth (and its interactions with maternal height). Sum shows a linear combination of the coefficients for government health expenditure linked to year of birth and year before birth (or their interaction with height). Controls: Months since birth; Sex of child; A constant term; Maternal education; Maternal age at birth; Maternal age at first birth; Birth order, Birth interval; Number of siblings born; Being firstborn; Paternal education; Household wealth; Community specific means of included observations for all independent variables (Community Fixed Effects). Other controls: An interaction between maternal height and maternal age at birth; An interaction between the child's year of birth (linear) and country. GDP, and government- and private health expenditure are median-centered (for all included countries and available data 1995 – 2014), so the interaction terms are 0 when they are at their median. Similarly, maternal age at birth is mean-centered when interacted with maternal height. If the main association of height is negative, a positive interaction coefficient means that an increase in government health expenditure decreases that association of maternal height with child outcome (and vice versa). β^l shows the main association of maternal height with child outcomes.*p < .10; ** p < .05; *** p < .01. Source: DHS (2016); WHO (2017).

Table A3. Interactions between government expenditure on health and maternal height stratified by wealth tertiles and residency (urban or rural)

Strata	Government Health Expenditure			Government Health Expenditure x Height			Height
	At Birth	Before Birth	Sum	At Birth	Before Birth	Sum	
Outcome: Under-5 Mortality							
Poorest <i>n</i> = 362,508	-0.0117*** (0.0028)	-0.0034 (0.0027)	-0.0151*** (0.0025)	-0.0012 (0.0025)	0.0028 (0.0025)	0.0016 (0.0013)	-0.0084*** (0.0008)
Middle <i>n</i> = 336,172	-0.0059** (0.0029)	-0.0061** (0.0028)	-0.0120*** (0.0026)	0.0027 (0.0026)	0.0025 (0.0025)	0.0052*** (0.0013)	-0.0077*** (0.0009)
Richest <i>n</i> = 354,549	-0.0091*** (0.0023)	-0.0015 (0.0024)	-0.0108*** (0.0021)	-0.0015 (0.0022)	0.0046** (0.0021)	0.0031*** (0.0011)	-0.0057*** (0.0006)
Urban <i>n</i> = 292,298	-0.0047* (0.0026)	-0.0046* (0.0027)	-0.0092*** (0.0024)	-0.0012 (0.0024)	0.0050** (0.0024)	0.0038*** (0.0012)	-0.0056*** (0.0007)
Rural <i>n</i> = 795,893	-0.0102*** (0.0018)	-0.0041** (0.0018)	-0.0143*** (0.0016)	0.0009 (0.0016)	0.0023 (0.0016)	0.0032*** (0.0009)	-0.0079*** (0.0005)
Outcome: School Attendance							
Poorest <i>n</i> = 66,949	0.1017*** (0.0098)	0.0187* (0.0097)	0.1204*** (0.0114)	-0.0041 (0.0067)	0.0027 (0.0069)	-0.0013 (0.0030)	0.0052** (0.0023)
Middle <i>n</i> = 67,148	0.0548*** (0.0076)	0.0183*** (0.0069)	0.0731*** (0.0094)	-0.0085* (0.0049)	0.0085* (0.0047)	0.0000 (0.0024)	0.0080*** (0.0027)
Richest <i>n</i> = 67,815	0.0274*** (0.0059)	0.0138** (0.0055)	0.0411*** (0.0068)	0.0020 (0.0044)	-0.0053 (0.0042)	-0.0034* (0.0018)	0.0018 (0.0016)
Urban <i>n</i> = 55,388	0.0346*** (0.0068)	0.0128** (0.0063)	0.0474*** (0.0078)	-0.0032 (0.0050)	-0.0007 (0.0049)	-0.0039** (0.0020)	0.0005 (0.0018)
Rural	0.0692***	0.0227***	0.0919***	-0.0036	0.0035	-0.0001	0.0058***

n = 164,024

(0.0054) (0.0050) (0.0067)

(0.0036) (0.0036) (0.0036)

(0.0017) (0.0017) (0.0014)

Notes: Estimates are weighted, and standard errors in parentheses are adjusted for clustering at the community level. Coefficients show the main association of government health expenditure. Interactions coefficients are differences in the slope for maternal height for children exposed to a log point increase in government health expenditure. At birth shows coefficients for government health expenditure (and its interactions with maternal height) linked to year of birth and Before birth shows coefficients for government health expenditure indexed to year before birth (and its interactions with maternal height). Sum shows a linear combination of the coefficients for government health expenditure linked to year of birth and year before birth (or their interaction with height). Controls: Months since birth; Sex of child; A constant term; Maternal education; Maternal age at birth; Maternal age at first birth; Birth order; Birth interval; Number of siblings born; Being firstborn; Paternal education; Household wealth; Community specific means of included observations for all independent variables (Community Fixed Effects). Other controls: GDP indexed to year of birth and year before birth; Maternal height interacted with private health expenditure indexed to year of birth and year before birth; Private health expenditure indexed to year of birth and year before birth; An interaction between child's year of birth (linear) and country; GDP, and government- and private health expenditure are median-centered (for all included countries and available data 1995 – 2014), so the interaction terms are 0 when they are at their median. Similarly, maternal age at birth is mean-centered when interacted with maternal height. If the main association of height is negative, a positive interaction coefficient means that an increase in government health expenditure decreases that association of maternal height with child outcome (and vice versa). **p* < .10; ***p* < .05; ****p* < .01. Source: DHS (2016); WHO (2017). Poorest is the lowest tertile, middle is the second tertile, and the richest is the third tertile.

Table A4. Government expenditure on health indexed to different years with respect to year of birth. Only indexed to a single year in each model

Index with r.t. birth year	Under-5 Mortality		Height	Observations	Exp.	Exp. x Height	Exp. x Height	Height	Observations
	Exp.	Community Fixed Effects							
-6	0.0036*	0.0013	-0.0075***	692,647	-0.0359***	0.0049*	0.0047**	111,137	
-5	0.0025	0.0011	-0.0075***	778,812	-0.0709***	0.0034	0.0047**	133,726	
-4	-0.001	0.001	-0.0079***	854,572	-0.0490***	0.0017	0.0043**	153,383	
-3	-0.0034**	0.0019**	-0.0078***	933,693	-0.0124*	0.0014	0.0050***	175,063	
-2	-0.0085***	0.0020**	-0.0078***	1,012,803	-0.0021	0.0018	0.0055***	195,525	
-1	-0.0118***	0.0028***	-0.0079***	1,093,261	0.0358***	0.0008	0.0056***	219,412	
0 (Birth year)	-0.0118***	0.0029***	-0.0081***	1,166,711	0.0511***	-0.0008	0.0047***	241,687	
1	-0.0060***	0.0028***	-0.0079***	1,235,877	0.0399***	-0.0011	0.0045***	263,619	
2	-0.0038***	0.0021**	-0.0081***	1,292,625	0.0186***	-0.0004	0.0046***	286,020	
3	-0.0041***	0.0022**	-0.0081***	1,338,897	-0.0145***	0.0033*	0.0044***	306,200	
4	-0.0007	0.0018**	-0.0082***	1,367,866	-0.0142***	0.0047**	0.0040***	322,387	
5	0.0021	0.0022**	-0.0083***	1,391,869	-0.0227***	0.0070***	0.0039***	337,543	
6	0.002	0.0017*	-0.0082***	1,402,987	-0.0235***	0.0064***	0.0034***	346,403	

Notes: Estimates are weighted, and standard errors in parentheses are adjusted for clustering at the community level. Coefficients show the main association of government health expenditure. Interactions coefficients are differences in the slope for maternal height for children exposed to a log point increase in government health expenditure. Controls: Months since birth; Sex of child; A constant term; Maternal education; Maternal age at birth; Birth order; Birth interval; Number of siblings born; Being firstborn; Paternal education; Household wealth; Community specific means of included observations for all independent variables (Community Fixed Effects). Other controls: GDP indexed to year; Maternal height interacted with private health expenditure indexed to year; Private health expenditure indexed to year; Maternal age at birth; An interaction between child's year of birth (linear) and country; GDP, and government- and private health expenditure are median-centered (for all included countries and available data 1995 – 2014), so the interaction terms are 0 when they are at their median. Similarly, maternal age at birth is mean-centered when interacted with maternal height. If the main association of height is negative, a positive interaction coefficient means that an increase in government health expenditure decreases that association of maternal height with child outcome (and vice versa). *p < .10; ** p < .05; *** p < .01. Source: DHS (2016); WHO (2017).

Table A5. Full outputs from EQ1 and EQ2 using community fixed effects

VARIABLES	EQ1	EQ2	EQ1	EQ2
	Under-5 Mortality	Under-5 Mortality	School Attendance	School Attendance
Maternal Height Z-Score	-0.0130*** (0.000434)	-0.009*** (0.000)	0.0170*** (0.00126)	0.004*** (0.001)
Female	-0.0152*** (0.000618)	-0.015*** (0.001)	-0.0157*** (0.00180)	-0.018*** (0.002)
In(Child's Age+1)	0.0365*** (0.000355)	0.011*** (0.000)	0.173*** (0.00476)	0.160*** (0.005)
Maternal Education		-0.001*** (0.000)	0.007*** (0.000)	
Maternal Age at birth		-0.011*** (0.001)	-0.000 (0.002)	-0.000 (0.002)
Maternal Age at Birth^2		0.000*** (0.000)	0.000*** (0.000)	0.000*** (0.000)
Maternal Age at First Birth		-0.007*** (0.001)	0.007*** (0.002)	0.007*** (0.002)
Maternal Age at First Birth^2		0.000*** (0.000)	-0.000*** (0.000)	-0.000*** (0.000)
Birth Interval		-0.004*** (0.000)	0.001*** (0.000)	0.001*** (0.000)
Birth Interval^2		0.000*** (0.000)	-0.000*** (0.000)	-0.000*** (0.000)
Birth Order		-0.010*** (0.001)	-0.017*** (0.003)	
Birth Order^2		-0.000** (0.000)	0.001*** (0.001)	0.001*** (0.001)

Number of Siblings Ever Born		(0.000)						
	0.013***		-0.002**					
First Born		(0.000)	(0.001)					
	-0.091***		0.003					
	(0.002)		(0.004)					
Wealth Z-Score		-0.010***		0.007***				
		(0.001)		(0.002)				
Wealth Z-Score^2		0.000		-0.015***				
		(0.000)		(0.001)				
Paternal Education		-0.001***		0.006***				
		(0.000)		(0.000)				
Missing Paternal Education		0.004***		0.032***				
		(0.002)		(0.005)				
Constant	1.643***		0.583***		-14.46***		-7.265***	
	(0.0512)		(0.074)		(0.452)		(0.419)	
Survey Means								
Female	-2.131***	0.054	5.888***	2.288***				
	(0.0890)	(0.097)	(0.139)	(0.172)				
In(Child's Age+1)	-0.138***	-0.001	2.377***	1.020***				
	(0.00437)	(0.005)	(0.0937)	(0.087)				
Maternal Height Z-Score	0.00569***	0.003	-0.0542***	-0.099***				
	(0.00212)	(0.002)	(0.00320)	(0.008)				
Community Means								
Female	0.010	0.004						
	(0.006)	(0.010)						
In(Child's Age+1)	-0.009***	0.112***						
	(0.003)	(0.023)						
Maternal Height Z-Score	-0.004***	-0.006						

Maternal Education	(0.001)	-0.006***
	(0.000)	0.007***
Wealth Z-Score	(0.001)	-0.017***
	(0.001)	-0.006***
Wealth Z-Score^2	(0.001)	-0.003***
	(0.001)	0.017***
Paternal Education	(0.001)	-0.002***
	(0.000)	0.023***
Missing Paternal Education	(0.000)	-0.030***
	(0.005)	0.254***
Maternal Age at birth	(0.005)	-0.010***
	(0.000)	0.007
Maternal Age at Birth^2	(0.004)	0.000*
	(0.000)	-0.000
Maternal Age at First Birth	(0.003)	0.008***
	(0.000)	-0.000***
Maternal Age at First Birth^2	(0.003)	-0.000***
	(0.000)	0.044***
Birth Interval	(0.000)	-0.003***
	(0.000)	-0.001*
Birth Order	(0.000)	-0.011
	(0.011)	0.036***
Birth Order^2	(0.001)	0.002**
	(0.001)	-0.001
Number of Siblings Ever Born	(0.002)	-0.029***

First Born		(0.004)	(0.003)
	-0.072**	-0.073*	
	(0.024)	(0.031)	
Observations	2,060,693	2,060,693	385,255
R-squared	0.016	0.045	0.065
			0.206

Robust standard errors in parentheses

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

Table A6. Full outputs from EQ1 and EQ2 using father fixed effects and polygyny sample

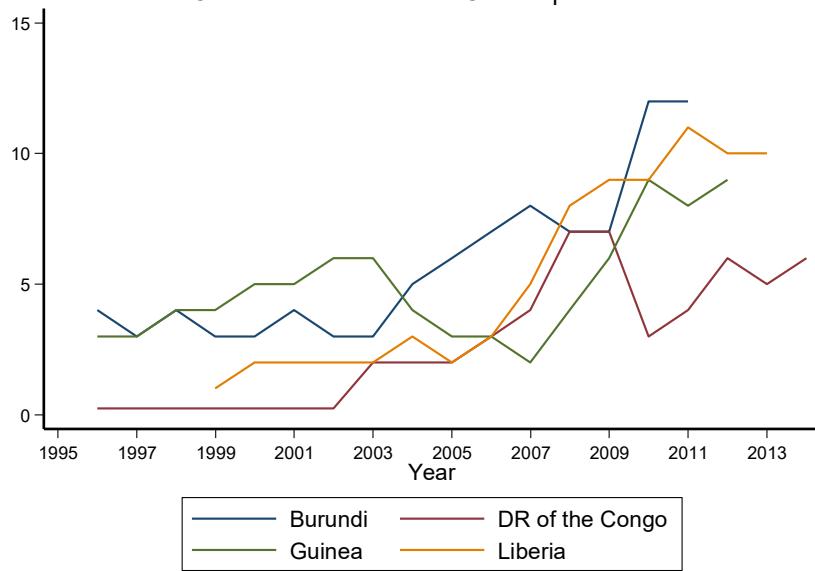
VARIABLES	EQ1 Under-5 Mortality	EQ2 Under-5 Mortality	EQ1 School Attendance	EQ2 School Attendance
Maternal Height Z-Score	-0.0139*** (0.00144)	-0.008*** (0.002)	0.0130** (0.00525)	0.006 (0.004)
Female	-0.0173*** (0.00213)	-0.018*** (0.002)	-0.0833*** (0.00743)	-0.087*** (0.007)
In(Child's Age+1)	0.0574*** (0.00105)	0.030*** (0.002)	0.158*** (0.0144)	0.145*** (0.017)
Maternal Education		-0.001 (0.001)	0.000 (0.002)	0.000 (0.002)
Maternal Age at birth		-0.010*** (0.002)	-0.001 (0.006)	-0.001 (0.006)
Maternal Age at Birth^2		0.000*** (0.000)	0.000 (0.000)	0.000 (0.000)
Maternal Age at First Birth		-0.011*** (0.003)	0.016** (0.007)	0.016** (0.007)
Maternal Age at First Birth^2		0.000* (0.000)	-0.000*** (0.000)	-0.000*** (0.000)
Birth Interval		-0.005*** (0.000)	0.000 (0.000)	0.000 (0.000)
Birth Interval^2		0.000*** (0.000)	-0.000 (0.000)	-0.000 (0.000)
Birth Order		-0.019*** (0.004)	-0.010 (0.010)	-0.010 (0.010)
Birth Order^2		-0.000 (0.000)	0.000 (0.000)	0.000 (0.000)

Number of Siblings Ever Born	(0.000)	(0.001)	(0.003)	(0.005)	(0.007)	(0.009)	(0.011)	(0.013)	(0.015)	(0.017)	(0.019)	(0.021)	(0.023)	(0.025)	(0.027)	(0.029)	(0.031)	(0.033)	(0.035)
First Born	0.010***	-0.003	(0.001)	(0.003)	(0.005)	(0.007)	(0.009)	(0.011)	(0.013)	(0.015)	(0.017)	(0.019)	(0.021)	(0.023)	(0.025)	(0.027)	(0.029)	(0.031)	(0.033)
Constant	-0.125***	-0.011	(0.006)	(0.008)	(0.010)	(0.012)	(0.014)	(0.016)	(0.018)	(0.020)	(0.022)	(0.024)	(0.026)	(0.028)	(0.030)	(0.032)	(0.034)	(0.036)	(0.038)
Survey Means																			
Female	0.313***	0.780***	(0.0768)	(0.127)	(1.243)	(1.360)	(1.478)	(1.596)	(1.714)	(1.832)	(1.950)	(2.068)	(2.186)	(2.304)	(2.422)	(2.540)	(2.658)	(2.776)	(2.894)
In(Child's Age+1)	-0.0474***	-0.013	(0.0103)	(0.011)	(0.0118)	(0.0127)	(0.0136)	(0.0145)	(0.0154)	(0.0163)	(0.0172)	(0.0181)	(0.0190)	(0.0199)	(0.0208)	(0.0217)	(0.0226)	(0.0235)	(0.0244)
Maternal Height Z-Score	0.0274***	0.006	(0.00536)	(0.006)	(0.0065)	(0.0074)	(0.0083)	(0.0092)	(0.0101)	(0.0110)	(0.0119)	(0.0128)	(0.0137)	(0.0146)	(0.0155)	(0.0164)	(0.0173)	(0.0182)	(0.0191)
Community Means																			
Female	0.018	-0.004	(0.016)	(0.016)	(0.0168)	(0.0177)	(0.0186)	(0.0195)	(0.0204)	(0.0213)	(0.0222)	(0.0231)	(0.0240)	(0.0249)	(0.0258)	(0.0267)	(0.0276)	(0.0285)	(0.0294)
In(Child's Age+1)	-0.0474***	0.152*	(0.008)	(0.008)	(0.0088)	(0.0097)	(0.0106)	(0.0115)	(0.0124)	(0.0133)	(0.0142)	(0.0151)	(0.0160)	(0.0169)	(0.0178)	(0.0187)	(0.0196)	(0.0205)	(0.0214)
Maternal Education	-0.001	-0.001	(0.002)	(0.002)	(0.0022)	(0.0021)	(0.0020)	(0.0019)	(0.0018)	(0.0017)	(0.0016)	(0.0015)	(0.0014)	(0.0013)	(0.0012)	(0.0011)	(0.0010)	(0.0009)	(0.0008)
Wealth Z-Score	-0.016***	0.092***	(0.003)	(0.003)	(0.0033)	(0.0032)	(0.0031)	(0.0030)	(0.0029)	(0.0028)	(0.0027)	(0.0026)	(0.0025)	(0.0024)	(0.0023)	(0.0022)	(0.0021)	(0.0020)	(0.0019)
Wealth Z-Score^2	-0.003***	-0.005	(0.001)	(0.001)	(0.0011)	(0.0010)	(0.0009)	(0.0008)	(0.0007)	(0.0006)	(0.0005)	(0.0004)	(0.0003)	(0.0002)	(0.0001)	(0.0000)	(0.0000)	(0.0000)	(0.0000)
Paternal Education	-0.004***	0.034***	(0.006)	(0.006)	(0.0063)	(0.0062)	(0.0061)	(0.0060)	(0.0059)	(0.0058)	(0.0057)	(0.0056)	(0.0055)	(0.0054)	(0.0053)	(0.0052)	(0.0051)	(0.0050)	(0.0049)

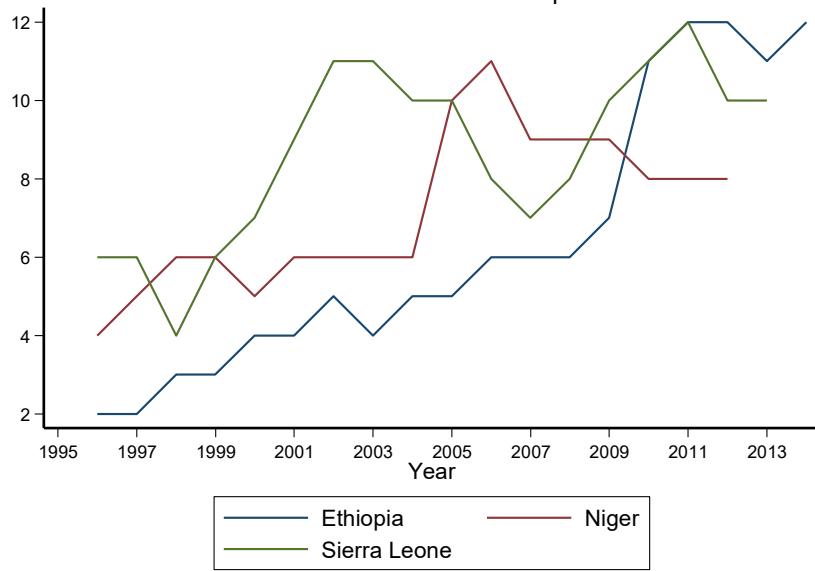
Missing Paternal Education	(0.001)	-0.031***	(0.009)	0.323***	(0.048)
Maternal Age at birth				0.034	(0.025)
Maternal Age at Birth^2				-0.002	(0.011)
Maternal Age at First Birth				0.000	(0.000)
Maternal Age at First Birth^2				0.013	(0.029)
Birth Interval				-0.000**	(0.009)
Birth Interval^2				-0.002	(0.000)
Birth Order				-0.037	(0.001)
Birth Order^2				0.002	(0.030)
Number of Siblings Ever Born				0.007	(0.009)
First Born				-0.057	(0.079)
Household Means					
Female				0.014	(0.010)
In(Child's Age+1)				0.009	(0.006)

Maternal Height Z-Score	-0.005*	(0.003)
Maternal Education	-0.003**	(0.001)
Maternal Age at birth	-0.017**	(0.007)
Maternal Age at Birth^2	-0.000	(0.000)
Maternal Age at First Birth	0.015**	(0.007)
Maternal Age at First Birth^2	-0.000	(0.000)
Birth Interval	-0.006**	(0.001)
Birth Interval^2	0.000***	(0.000)
Birth Order	0.025	(0.022)
Birth Order^2	0.000	(0.002)
Number of Siblings Ever Born	0.004	(0.006)
First Born	-0.219**	(0.055)
Observations	143,344	18,469
R-squared	0.021	0.056
Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1		0.198

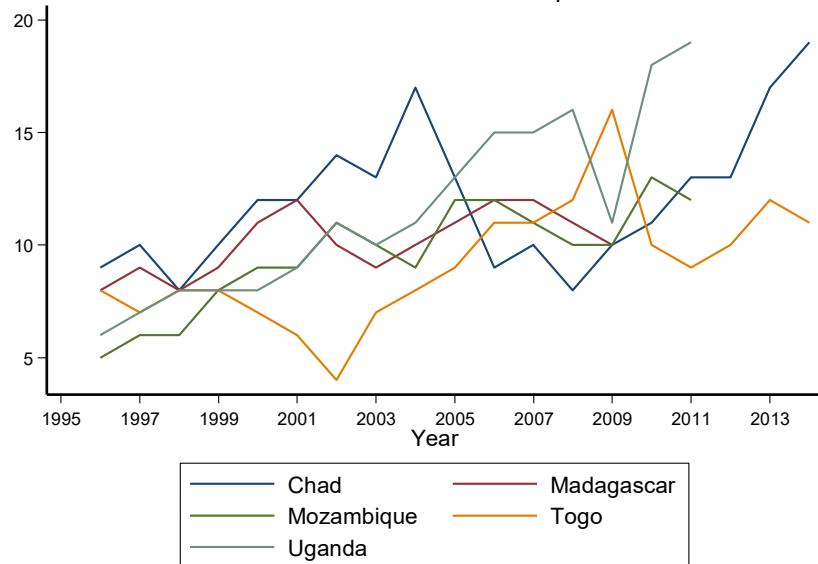
Octile 1 of Public Health Care Expenditures



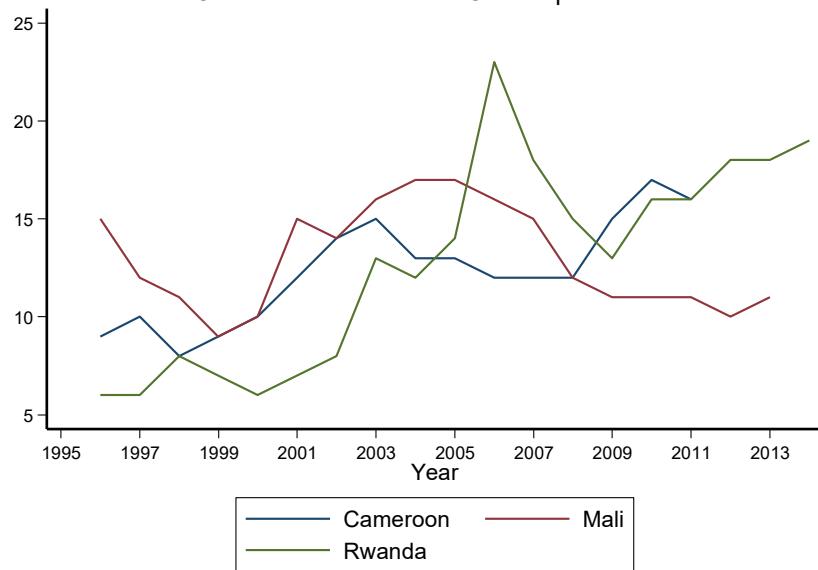
Octile 2 of Public Health Care Expenditures



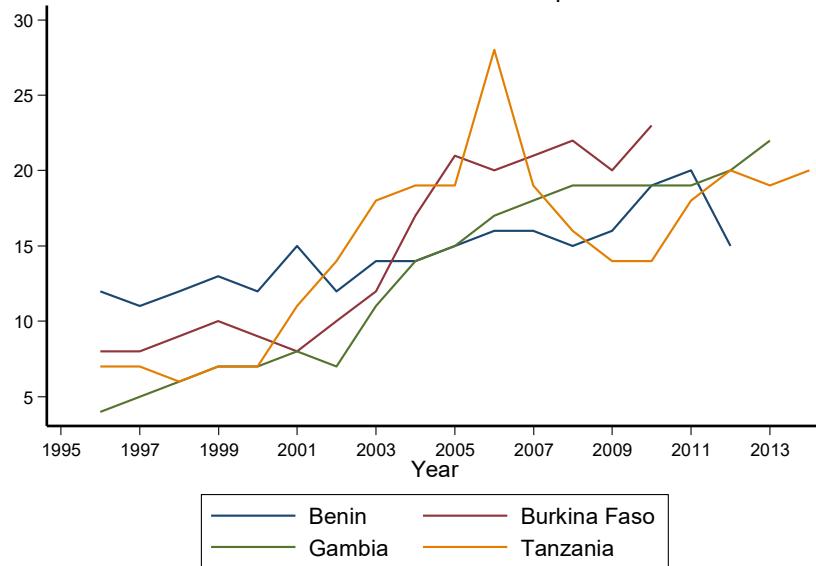
Octile 3 of Public Health Care Expenditures



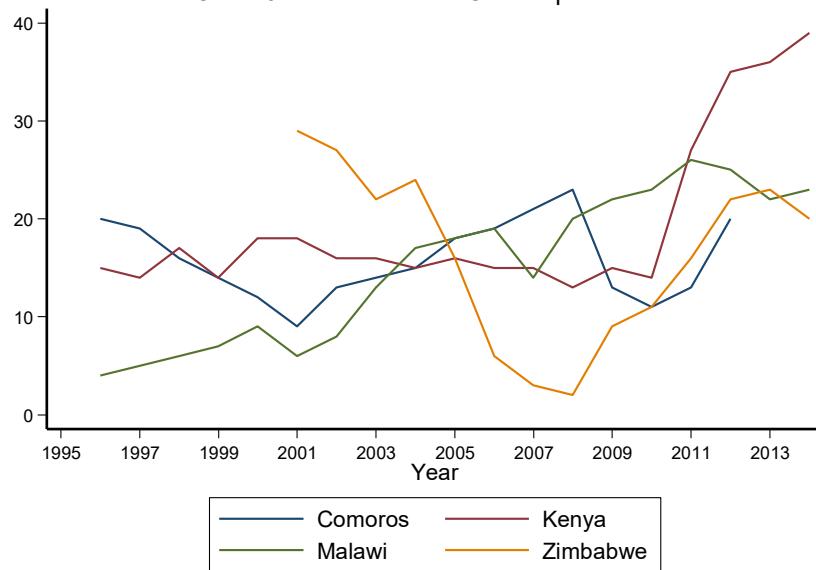
Octile 4 of Public Health Care Expenditures



Octile 5 of Public Health Care Expenditures



Octile 6 of Public Health Care Expenditures



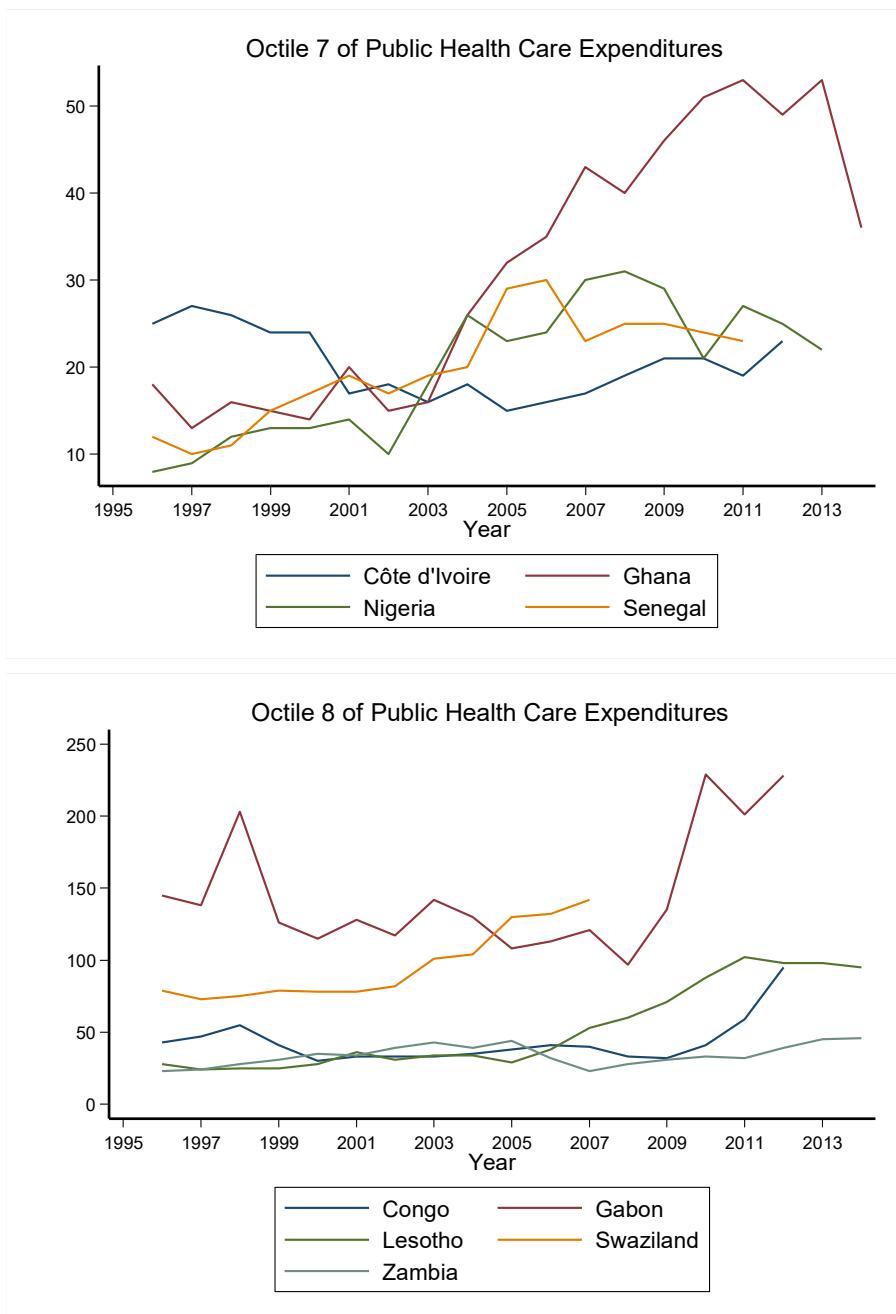
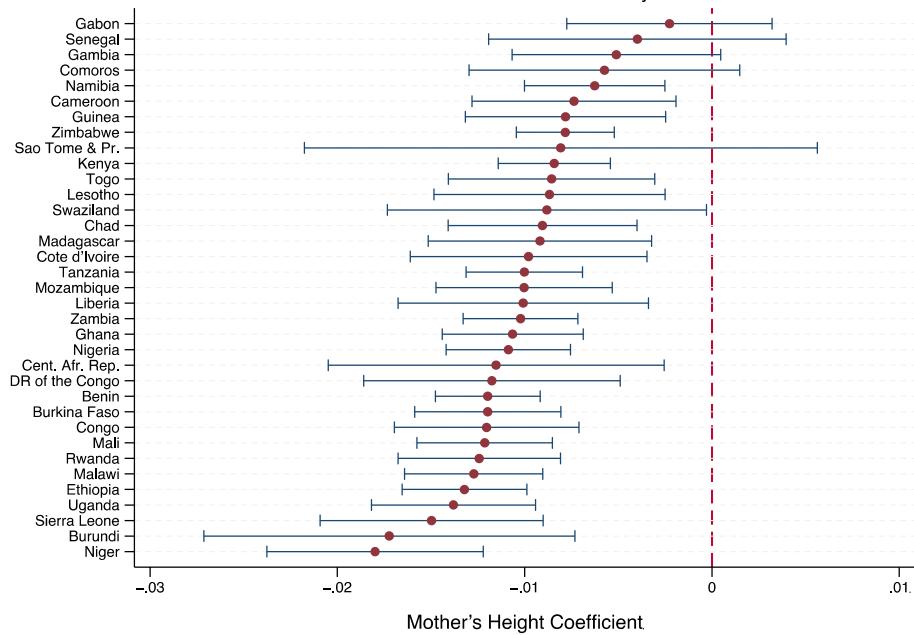


Figure A1. Government health expenditure over time. Countries grouped into octiles according to average annual expenditure

Outcome: Under-5 Mortality.



Outcome: School Attendance.

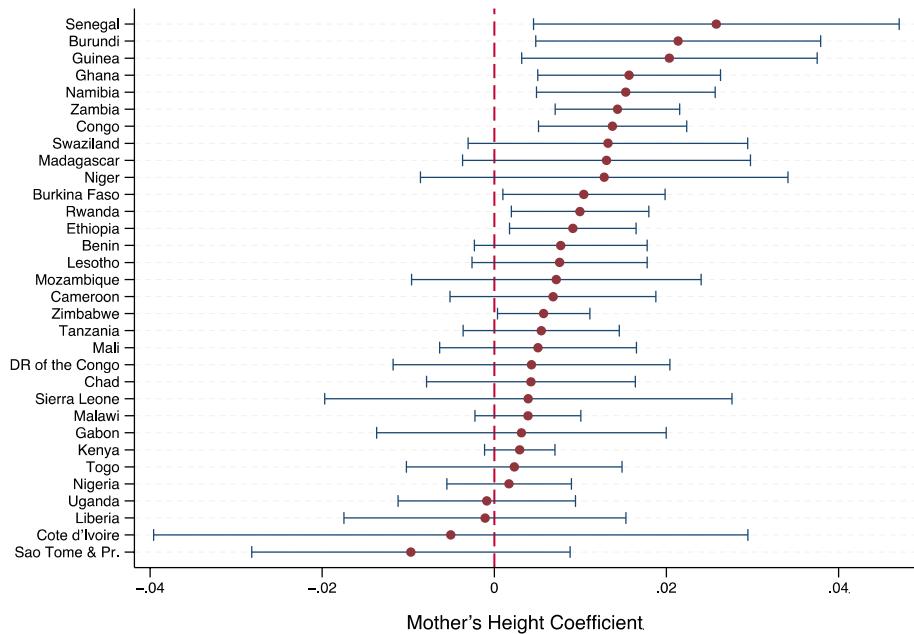


Figure A2. Coefficients for maternal height from models with a full set of controls and community fixed effects for each country

Notes: Estimates adjusted using sampling weights and 95 confidence intervals are adjusted for clustering at the community level. All coefficients are for maternal height from fully adjusted community fixed effects models. Controls: Months since birth; Sex of child; A constant term; Mother's education; Mother's age at birth; Mother's age at first birth; Birth order, Birth interval; Number of siblings born; Being firstborn; Father's education and Household wealth; Community specific means of included observations for all independent variables (Community Fixed Effects). Source: DHS (2016).

Paper III

Paper III

Weakening association of parental education: analysis of child health outcomes in 43 low- and middle-income countries¹

Abstract

Parental education has been suggested to be an effective instrument for improving child health in low- and middle-income countries. Both education and child health have improved, however, as well as related factors. These changes may have implications for the observed association. We used Demographic and Health Surveys conducted in 43 countries at two points in time, between 1991 and 2016, to test if the association of parental education with child health has changed over time. We explored how changes relate to commonly cited confounders and pathways, including fertility, household living standards, health care use, urbanicity and geographical clustering. We used linear probability models, Gelbach decomposition, and assessed a range of sensitivity specifications. The point estimate for an additional year of maternal education has attenuated by 0.27% points (56%) for under-5 mortality, 0.34% points (15%) for child stunting, 0.42% points (30%) for child underweight and 0.09% points (24%) for child wasting. The point estimate for paternal education has attenuated by 0.20% points (53%) for under-5 mortality, 0.15% points (8%) for child stunting, 0.28% points (24%) for underweight and 0.06% points (19%) for wasting. Changes in confounding and mediation by fertility, household living standards and urban-rural differences explain to a large extent the attenuations. Geospatial clustering increasingly drives the association of parental education with child health. The role of parental education in child health has attenuated considerably over time in

¹ Paper III was co-authored with Jan-Walter De Neve and S.V. Subramanian. All of the authors contributed to the study concept and design, the interpretation of findings, and the drafting of the manuscript. The author of this dissertation acquired the data and performed the statistical analysis. The paper has been published in the International Journal of Epidemiology (Karlsson et al., 2018).

low-resource settings. Decision makers should take into account this weakening association when designing policies aimed at improving child health.

Introduction

Improving health among children aged younger than 5 years has received attention among population health researchers and policy makers for several decades. In the 1970s, Caldwell's (1979) influential paper showed a strong protective association between maternal education and child mortality in a low-resource setting, above other measures of socioeconomic status (SES), such as paternal education and occupation. Many studies (Grepin and Bharadwaj, 2015; Harttgen et al., 2013; Headey, 2013; Lakshman et al., 2013; Makoka and Masibo, 2015; Murphy et al., 2009; Thomas et al., 1990) have reiterated this finding, and the United Nations and the World Bank underline increasing maternal education as a cost-effective instrument for improving child health in low- and middle-income countries (LMICs) (Jamison et al., 1993; United Nations, 2014, 1994; Veneman, 2007). Hypothesized pathways include increased health care use (Elo, 1992; Onsomu et al., 2015), knowledge, skills, SES (Cleland and Van Ginneken, 1988), information use (Handa, 1999; Thomas et al., 1991), assortative mating (Breierova and Duflo, 2004) and reduced fertility (Grepin and Bharadwaj, 2015). The association of maternal education has been attributed directly to proximate determinants of child health, such as illness control and nutrition, whereas that of paternal education has been attributed mostly to increased earnings (Mosley and Chen, 1984).

More recent literature has moved away from relying exclusively on maternal education as an SES determinant of particular relevance for child health, and pointed out the need to better understand underlying pathways and recognizing country heterogeneity in the association (Subramanian and De Neve, 2017). A recent large-scale study found associations of similar magnitude for maternal and paternal education with child nutrition, suggesting that education of both parents is an important marker for child nutrition (Vollmer et al., 2017a). The observed association is far from universal; there is vast country heterogeneity, and in many countries, no significant relationship remains after accounting for living standards and geographical clustering, suggesting that maternal education may not be an SES determinant of particular importance (Desai and Alva, 1998; Jeong et al., 2018). One empirical concern with many previous studies is that a range of unobserved variables, such as innate ability (Card, 2001) and other endowments (Behrman and Wolfe, 1987) that are associated with both education and child health, may make observational studies subject to confounding bias. In the absence of randomized trials assessing formal parental education, quasi-experimental studies have exploited education policy reforms as 'natural experiments' and have found conflicting results (Currie and Moretti, 2003; De Neve and Subramanian,

2017; Grepin and Bharadwaj, 2015; Güneş, 2015; Lindeboom et al., 2009; McCrary and Royer, 2011). The current literature indicates important confounding and between-context variation in the relationship of parental education with child health and mortality.

Since the context in LMICs is changing with improvements in education and child health as well as related factors and economic development (Lomazzi et al., 2014), the relationship between parental education and child health is also likely to change over time. Caldwell (1979) already observed that the association between maternal education and child survival attenuated over time in the 1970s. A study of several sub-Saharan African countries found lower disparities in under-5 mortality by maternal education in more recent birth cohorts of children born 1990–2010 (Bado and Sathiya Susuman, 2016). A large-scale study using pooled Demographic and Health Surveys (DHS) found a modest decline in differences in predicted child undernutrition by maternal education between surveys conducted before and after 2000 (Vollmer et al., 2017b).

In Table 1, we show possible reasons why the association between parental education and child health may have changed over time in LMICs. We define three broad categories: (i) ‘artefact of the measure’, which relates to changes in education as an exposure (e.g., regarding quality (Nhundu, 1992) or content of the formal school curriculum); (2) ‘confounding change’, which relates in large part to who obtains education (e.g., their innate ability, living standards (Datar et al., 2010), as well as geographical clustering); and, finally, (iii) ‘mediator and moderator change’, which relates to changes in causal pathways such as income, fertility (Martin, 1995), health care use (Barrera, 1990; Strauss, 1990; Thomas et al., 1990), health outcomes (Cutler and Lleras-Muney, 2006), as well as the broader epidemiological, economic and social environment (Antonovsky, 1967; Bengtsson and van Poppel, 2011) (see Supplementary Appendix A1, , for additional details on these categories). A wide range of reasons why the relationship of parental education with child health and mortality may change are identified. To our knowledge, however, no studies have assessed changes in the relationship between schooling of both parents and child health and mortality, over two points in time for multiple LMICs, and explored the underlying drivers of change.

Table 1. Possible reasons for a weakening or strengthening association of parental education with child health in developing countries

Weaker association	Stronger association	Underlying mechanism
Artefact of the measure		
Decline in quality of education (e.g. because higher enrolment is not met with sufficient increase in funding)	Increase in quality of education	Skills and knowledge
Change in curriculum: education less relevant for child-rearing	Change in curriculum: education more relevant for child-rearing	Child-rearing capabilities
Diminishing returns to education regarding child health	Increasing returns to education regarding child health	Functional form of the relationship, nonlinearity
Confounding change		
Educated parents are a less selected group regarding early life living standards and innate ability	Those who remain uneducated, despite increased enrolment, are an increasingly marginalized group	Parental early life living standards, innate ability, discrimination
Returns to education, regarding child health, lower for those affected by expansion of education (e.g. barriers to entering education are lower while early life conditions affecting health and cognition remain the same)	Returns to education are greater for those affected by expansion of education (e.g. for minorities, which have been under-represented in education)	Parental early life living standards, innate ability, discrimination, self-productivity of skills, living standards
Decreased geographical clustering regarding parental education and other factors favourable to child health, e.g. health care and other infrastructure	Increased geographical clustering: educated parents are increasingly clustered in more favourable areas	Geographical clustering
Mediators and moderators change		
Structural changes: more educated workforce migrates to urban areas, living in less ideal conditions	Structural changes: demand for educated labour increases economic opportunities and returns to education in urban areas	Living standards, fertility, urban-rural differences
Diffusion of attitudes, behaviours and knowledge promoting child health from educated to less educated	Increased cultural segregation of educated and uneducated	Behaviours, attitudes, fertility
Decreased assortative mating	Increased assortative mating	Living standards, assortative mating
Externalities of education improve social context for uneducated	Educated emigrate, reducing community-level education	Institutions, geographical clustering
Environmental improvements (e.g. lower risk of disease exposures which educated parents could more effectively shelter their children from)	Environmental deterioration/shocks (e.g. educated parents are more likely to be capable of sheltering children from famines and disease outbreaks)	Self-reliance, living standards
Provision of services increases, equally available for all and a substitute for parental skills (e.g. parents are not on their own in insuring the health of their children)	Provision of services decrease, more available to educated, or a compliment (e.g. better-educated groups use health care more, increasing their advantage)	Health care access and use, self-reliance
	Postponed first birth resulting from longer duration of education	Fertility

The items in the column labelled 'Weaker association' provide examples for why the association between parental education and child health may attenuate over time in developing countries. The items in the column labelled 'Stronger association' provide examples for why the association between parental education and child health may increase over time. The column labelled 'Underlying mechanism' summarizes key factors linking parental education and child health which are most relevant for each example.

To address this gap in the literature, we use the largest available, nationally representative and mutually comparable repeated cross-sectional datasets from LMICs to examine the associations of parental education with under-5 mortality, stunting, wasting and underweight, over time. We explore the influence of fertility, household living standards, health care use, assortative mating, maternal early life conditions, urban-rural differences, nonlinearity, as well as geographical clustering. Finally, we identify country heterogeneity in changes in the association between parental education and child health over time.

Data and Methods

Data source

Our data came from the DHS, which are mutually comparable, nationally representative household surveys, using a multi-staged stratified sampling design, administered by ICF International. Primary sampling units (PSU), consisting of neighborhoods or villages, were randomly sampled from a stratum of regions and type of residence (urban or rural) with a probability proportional to population size. In the second stage of sampling, households were randomly sampled from the selected PSUs. Women 15–49 years old were interviewed and their birth histories and information on the household and its members recorded (Corsi et al., 2012; Measure DHS, 2012).

Our inclusion criterion was any country with at least two available DHS surveys conducted after 1990 and which included all variables used in the current study. We used the newest and the oldest survey for each country. We excluded respondents who did not have a partner (i.e. not married or cohabiting), had had more than one partner in their lifetime, and those who were not residents at the place of interview. Our final analytical sample sizes were 495 597 children for under-5 mortality and 406 416 children for anthropometric outcomes. In Supplementary Table A1, we show countries, sample sizes and survey years used in the study. Supplementary Table A2 shows sample deduction. Missing values were a potential concern for anthropometric measures and paternal education (Supplementary Table A3), which we addressed in our sensitivity analyses.

Outcomes

Our outcomes were under-5 mortality and three nutritional indicators for surviving children: stunting (low height-for-age, an indicator of chronic malnutrition), underweight (low weight-for-age, a composite indicator of acute and chronic malnutrition) and wasting (low weight-for-height, an indicator of acute malnutrition). All outcomes refer to children born 0–59 months before the survey.

Exposure

Our exposure variable was number of completed years of schooling, separately for mothers and fathers (or partner of the mother).

Covariates

Covariates in our basic models were maternal age at the time of survey, months since birth (i.e. child's age if alive), child's sex and year of birth. Covariates added in our full models were education of the other parent, DHS-provided wealth index (a measure of relative household living standards)(DHS, 2016), maternal age at first birth, maternal age at birth, number of siblings ever born, birth order, birth interval (dummy variable adjustment for firstborns), maternal height (a widely used indicator for accumulated health and early life living standards [Bozzoli et al., 2009]), whether the child was born in a health facility or at home, and whether the child received any immunizations (only available for surviving children). Additional details are provided in Supplementary Appendix A2.

Statistical analysis

We used linear probability models (LPM). All independent variables were demeaned from a survey-specific means of included observations. Our basic models were specified as:

$$y = \alpha^{basic} + \beta^{basic} E + \delta_e^{basic} (S2 \times E) + \gamma^{basic'} x + \delta_x^{basic'} (S2 \times x) + \varepsilon^{basic}$$

(Eq1.)

y is one of four outcomes. E is years of education completed by a parent and $S2$ is an indicator for being in the newest surveys. x is a vector of basic controls: child's year of birth controls for linear trends in child health outcomes and parental education; child's age and child's sex relate to health and therefore improve fit and reduce standard errors; and maternal age accounts for the wide age range of women interviewed in the DHS. β^{basic} shows the main association between parent's education and child outcomes in the oldest surveys, and δ_e^{basic} , our coefficient of primary interest, shows the change in the association of parent's education between the oldest and the newest surveys. This specification is parametrically identical to running two separate regressions for oldest and newest surveys as:

$$y = \alpha + \beta^{basic} E + \gamma^{basic'} x + \varepsilon_1$$

for the oldest surveys, and

$$y = \pi + (\beta^{basic} + \delta_e^{basic}) E + (\gamma^{basic} + \delta_x^{basic})' x + \varepsilon_2$$

for the newest surveys.

After obtaining δ_e^{basic} we added a vector of covariates z interacted with being in the newest surveys, as well as a squared term for parent's education:

$$y = \alpha^{full} + \beta^{full}E + \delta_e^{full}(S2 \times E) + \gamma^{full}x + \delta_x^{full}(S2 \times x) + \vartheta'z + \delta_z'(S2 \times z) + \rho E^2 + \varepsilon^{full} \quad (\text{Eq2.})$$

Here z contains variables capturing a broad range of possible confounders and mediators. z also includes PSU-specific means of included observations for each independent variable (except being rural, since PSUs are either rural or urban), which is parametrically identical to adding an indicator for each PSU capturing geographical clustering (Mundlak Fixed Effects) (Antonakis et al., 2010; Mundlak, 1978).

We explored the role of underlying mechanisms behind the changing association by quantifying the contribution of eight sets of covariates from z to the overall change in association, δ_e^{basic} , using Gelbach decomposition (Gelbach, 2016). The total impact of the added covariates on the change parameter is $\tau = \delta_e^{basic} - \delta_e^{full}$. τ was decomposed into eight components showing the independent impact of sets of covariates separately. These sets were: (i) education of the other parent; (ii) household living standards; (iii) fertility; (iv) health care use; (v) maternal height (indicating early life living standards); (vi) urban-rural differences; (vii) geographical clustering; and, finally, (viii) nonlinearity (the squared term for parent's education). A ninth set, (ix) non-interacted 'baseline covariates' ($\vartheta'z$), was also quantified. All sets except (viii) and (ix) included variables interacted with being in the newest surveys (i.e. from $\delta_z'(S2 \times z)$). The main interest was to quantify how much change in confounding or mediation by the respective sets of covariates influence δ_e^{basic} . The impact of the covariates may be in either direction, decreasing the change coefficient if the impact of covariates decreased, or increasing the change coefficient if the impact increased. Similarly, covariates can suppress the association to a different extent (MacKinnon et al., 2000). We also show decomposition of the main association of parental education, in both the oldest (β) and the newest surveys ($\beta + \delta_e$), where we quantified the impact of non-interacted covariates on the main association between parental education and child health (additional details in Supplementary Appendix A3).

Table 2 shows the functional form of our variables. For the education of the other parent, we included a linear and squared term as well as an interaction between mean-centered education of each parent. We also assessed how generalizable our findings are across countries by graphically showing estimates from Eq1 from each country. We adjusted the DHS-provided sampling weights to sum up to one for each survey so that surveys with large sample sizes do not drive our main findings. Significance testing and standard errors were adjusted for clustering at the PSU level. All analyses were done using STATA v14.1.

Sensitivity analyses

We conducted sensitivity analyses to assess the robustness of our findings. First, we present estimates when categorizing parental education as no education, primary education and secondary or more (as opposed to years of schooling completed). Second, we present estimates when defining our nutrition indicators as continuous variables. Third, we present estimates from logit models as opposed to from LPM. Fourth, we present estimates when using alternative sampling weights (Deaton, 1997; Rutstein and Rojas, 2006). Fifth, we present estimates corrected for missing values using multinomial selection models, and by including children with missing values for covariates. Sixth, we present estimates when including single mothers and mothers that have been married more than once. Supplementary Appendix A4 includes additional details on sensitivity analyses.

Ethical clearance

This analysis was reviewed by the Harvard T.H. Chan School of Public Health Institutional Review Board and was considered exempt from full review because the study was based on an anonymous public use dataset with no identifiable information on the survey participants.

Results

Table 2 shows descriptive statistics of study participants. Under-5 mortality decreased from 0.08 to 0.04, stunting from 0.38 to 0.28, underweight from 0.19 to 0.13 and wasting from 0.07 to 0.06, between the oldest and newest surveys. Maternal education increased by 1.5 years and paternal education by 1.1 years. Childbearing decreased, and fewer children were born at home. The average time span between the oldest and the newest survey was 13 years.

Figure 1 shows the locally weighted scatterplot smoothing (LOWESS) smoothed fit of the relationship between parental education and child health in the pooled sample (Royston and Cox, 2005). As can be seen from the figure, the education gradient is lower in the newest surveys. There are also indications that the relationships are nonlinear.

Table 2. Descriptive statistics of the study sample

	Mortality		Anthropometry		Functional form
	Oldest	Change	Oldest	Change	
Died	0.081 (0.001)	-0.036*** (0.001)			Binary
Stunted			0.377 (0.002)	-0.097*** (0.003)	Binary
Underweight			0.192 (0.002)	-0.060*** (0.003)	Binary
Wasted			0.075 (0.001)	-0.009*** (0.001)	Binary
Maternal education (years)	4.078 (0.040)	1.540*** (0.057)	4.272 (0.042)	1.426*** (0.056)	Linear/Quadr.
Paternal education (years)	5.417 (0.042)	1.077*** (0.056)	5.628 (0.043)	0.947*** (0.055)	Linear/Quadr.
Received any immunization	0.880 (0.002)	0.055*** (0.002)	0.899 (0.002)	0.043*** (0.002)	Binary
Year of birth	1996.1 (0.048)	13.42*** (0.061)	1996.1 (0.048)	13.31*** (0.062)	Linear
Months since birth	29.112 (0.042)	-0.186*** (0.064)	28.720 (0.049)	-1.184*** (0.079)	Natural log
Household wealth	-0.152 (0.008)	0.070*** (0.011)	-0.131 (0.008)	0.056*** (0.011)	Quadratic
Maternal age at birth	26.019 (0.029)	0.199*** (0.039)	26.106 (0.030)	0.186*** (0.040)	Quadratic
Maternal age at first birth	19.359 (0.021)	0.663*** (0.031)	19.445 (0.022)	0.622*** (0.031)	Linear
Birth interval (months)	36.179 (0.091)	6.231*** (0.158)	36.875 (0.098)	6.206*** (0.167)	Linear
Birth order	3.579 (0.012)	-0.459*** (0.017)	3.562 (0.013)	-0.449*** (0.017)	Linear
Number of siblings	1.780 (0.004)	-0.162*** (0.005)	1.742 (0.004)	-0.150*** (0.005)	Linear
First born	0.230 (0.001)	0.039*** (0.002)	0.227 (0.002)	0.040*** (0.002)	Binary
Delivered at home	0.534 (0.004)	-0.219*** (0.005)	0.520 (0.004)	-0.215*** (0.005)	Binary
Maternal height (cm)	156.35 (0.049)	0.734*** (0.064)	156.25 (0.050)	0.619*** (0.066)	Linear
Rural	0.679 (0.005)	-0.067*** (0.007)	0.666 (0.005)	-0.062*** (0.007)	Binary
Maternal age at survey	28.410 (0.030)	0.197*** (0.040)	28.463 (0.031)	0.100** (0.041)	Linear
Year of survey	1998.3 (0.047)	13.389*** (0.060)	1998.2 (0.047)	13.198*** (0.060)	Linear

PSUs	20,729	59,397	20,729	59,397
Observations	222,385	495,597	181,317	406,416

Notes: *P < .10; ** p < .05; *** p < .01. 2 sided. All estimates are weighted and standard errors in parentheses adjusted for clustering at the PSU level. Functional form refers to how variables are entered into statistical models. Parental education is entered as quadratic except in the basic models, where it is linear. Change refers to the difference in means in the newest surveys compared with the oldest. Observations below the change columns refer to the complete sample from both newest and oldest surveys. All independent variables were demeaned from a survey-specific mean of included observations when entered into linear statistical models. PSUs were used to estimate the impact of geographic clustering.

Tables 3 and 4 show our main results (see Supplementary Tables A14–A16 for detailed outputs). There is a negative association between parental education and all child health outcomes in both the oldest and newest surveys. As expected, after fully adjusting for covariates, the association is reduced for all outcomes. Out of the covariates considered in this study, fertility, household living standards, urban-rural differences, as well as geographical clustering, account most for the association between parental education and child health. The impact of some of our included covariates differs between the oldest and the newest surveys—both in absolute terms and as a proportion of the association. All covariates appear to confound or mediate the association, except urban-rural differences which appear to suppress the education coefficients in the newest surveys. The impact of covariates is similar for maternal and paternal education.

Our primary results, changes in the association between the oldest and the newest surveys, are displayed in columns labelled ‘Change.’ Table 3 shows results for mortality and stunting. For mortality, the point estimate for an additional year of maternal education attenuated by 0.0027 (95% confidence interval [CI] 0.0022 – 0.0032; P<0.0001) (56% of the association in the oldest surveys) and 0.0020 (95% CI 0.0016 – 0.0025; P<0.0001) (53%) for paternal education. The decomposition of covariates shows that changing impact of urban-rural differences accounts for 0.0003 (95% CI 0.0001– 0.0006; P=0.0201) (11% of the change coefficient from the basic model) for maternal education and 0.0003 (95% CI 0.0000–0.0005; P=0.0192) (15%) for paternal education. Changing impact of fertility accounts for 0.0002 (95% CI 0.0000–0.0004; P=0.027) (10%) for paternal education.

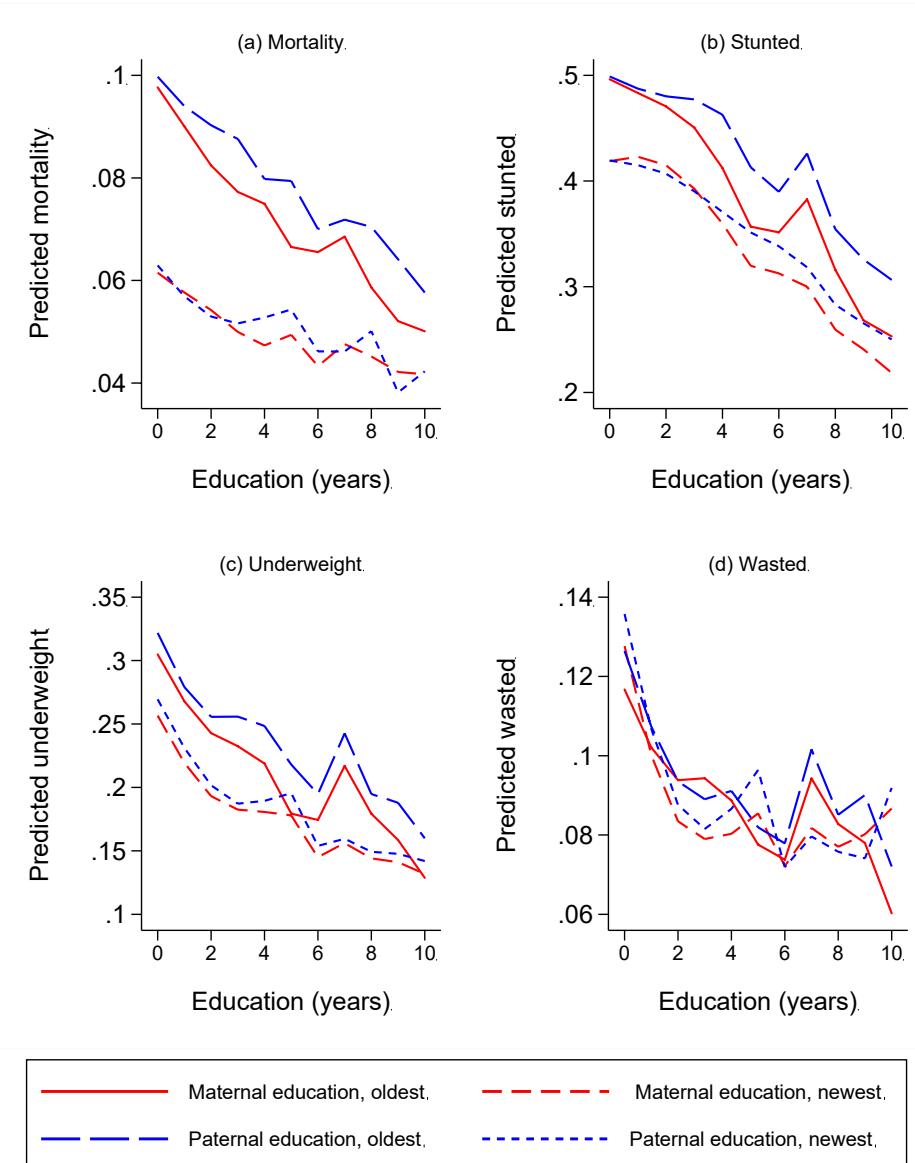


Figure 1. Functional form of the relationship between parental education and child health

Functional form of the relationship between parental education and child health. Locally weighted scatterplot smoothing (LOWESS) fit, adjusted as in Eq1., showing the functional form of the relationship between parental education and (a) mortality, (b) stunting, (c) underweight and (d) wasting. Each line is obtained separately for each of the four outcome variables. Covariates include: survey; months since birth; child's sex; child's year of birth; maternal age at survey and a constant term. All estimates are weighted. Results were obtained using STATA's mrunning command (Royston and Cox, 2005). For visualization purposes, years of education above 10 were excluded from the figure (less than 10% of the sample).

Table 3. Linear regression and decomposition of covariates results: mortality and stunting

	Maternal education			Paternal education		
	Oldest	Newest	Change	Oldest	Newest	Change
Outcome: mortality (n = 495,597)						
Coefficient: basic model	-0.0048***	-0.0021***	0.0027***	-0.0038***	-0.0018***	0.0020***
Coefficient: full model	-0.0003	0.0004	0.0008*	-0.0004	0.0004	0.0008**
Decomposition: covariates' impact on basic coefficient						
Total impact	-0.0045***	-0.0026***	0.0019***	-0.0034***	-0.0021***	0.0012***
Nonlinearity	-0.0007*	-0.0008*	-0.0001*	-0.0005	-0.0005	-0.0000
Education of other parent	-0.0001	0.0001	0.0001	-0.0003*	0.0000	0.0002
Household living standards	-0.0006**	-0.0001	0.0005	-0.0005**	-0.0000	0.0004*
Fertility	-0.0017***	-0.0010***	0.0002	-0.0013***	-0.0009***	0.0002*
Health care use	0.0001	-0.0000	-0.0001	0.0001	-0.0000	-0.0001
Urban/rural	-0.0002	0.0002**	0.0003**	-0.0001	0.0001**	0.0003**
Geographic clustering	-0.0012***	-0.0009***	0.0003	-0.0006***	-0.0007***	-0.0003
Maternal height	-0.0002***	-0.0001***	0.0001*	-0.0001***	-0.0001***	0.0001*
Baseline covariates			0.0006***		0.0005***	
Outcome: stunted (n = 406,416)						
Coefficient: basic model	-0.0230***	-0.0196***	0.0034***	-0.0177***	-0.0162***	0.0015***
Coefficient: full model	-0.0060***	-0.0050***	0.0010	-0.0038***	-0.0044***	-0.0005
Decomposition: covariates' impact on basic coefficient						
Total impact	-0.0171***	-0.0146***	0.0025***	-0.0138***	-0.0118***	0.0020***
Nonlinearity	0.0022**	0.0023**	0.0002**	0.0018*	0.0018*	0.0001*
Education of other parent	-0.0013***	-0.0015***	-0.0004	-0.0020***	-0.0014***	0.0005
Household living standards	-0.0079***	-0.0063***	0.0015**	-0.0065***	-0.0054***	0.0013***
Fertility	-0.0021***	-0.0014***	0.0007**	-0.0015***	-0.0011***	0.0005*
Health care use	-0.0013***	-0.0008***	0.0002	-0.0010***	-0.0007***	0.0002
Urban/rural	-0.0001	0.0011***	0.0012***	-0.0001	0.0009***	0.0010***
Geographic clustering	-0.0036***	-0.0054***	-0.0018**	-0.0023***	-0.0040***	-0.0020***

Maternal height	-0.0029***	-0.0026***	0.0003***	-0.0022***	-0.0020***	0.0002***
Baseline covariates		0.0006*				0.0002

Notes: *P < .10; ** p < .05; *** p < .01. 2 sided. All estimates are weighted and P-values adjusted for clustering at the PSU level. All coefficients are for an increase in the linear probability of experiencing the outcome to a single year increase in parental education. Basic model covariates: months since birth; child's sex; child's year of birth; maternal age at survey; a constant term. Full model covariates: household living standards (wealth index z-score); fertility (maternal age at birth, number of siblings ever born, birth interval [dummy variable adjustment for firstborn], birth order, maternal age at first birth); health care use (vaccinated [surviving children only], born at home); urban-rural difference (rural resident); maternal height (indicating maternal early life living standards); geographical clustering (PSU-specific means of all independent variables, except for rural residents); education of other parent (other parent's years of education, interaction between mean-centred years of education of both parents), nonlinearity (squared term for parents' education). All independent variables were demeaned from a survey-specific mean of included observations. The 'baseline covariates' set contains the terms for the main associations of covariates when decomposing the 'change coefficient'. When decomposing the 'change coefficient' all sets (except 'nonlinearity' and 'baseline covariates') contain covariates interacted with being in the newest surveys. Coefficients are from Eq1, and Eq2.; oldest is β ; newest is β^+ ; change is $\delta\beta$; and change is $\delta\beta^+$. The decomposition of the 'change coefficients' quantifies the changing impact of the respective sets of covariates (except 'nonlinearity' and 'baseline covariates') on the coefficients from the basic models. The decomposition of main coefficients from the oldest and newest surveys shows the impact of each set of covariates on the main association. Full outputs, exact P-values, 95% confidence intervals and standard errors are shown in Tables A14 - A16 in the Appendix.

Table 4. Linear regression and decomposition of covariates results: underweight and wasted

	Maternal education			Paternal education			
	Oldest	Newest	Change	Oldest	Newest	Change	
Outcome: underweight (n = 406,416)							
Coefficient: basic model	-0.0142***	-0.0101***	0.0042***	-0.0118***	-0.0090***	0.0028***	
Coefficient: full model	-0.0044***	-0.0029***	0.0015**	-0.0045***	-0.0042**	0.0002	
Decomposition: covariates' impact on basic coefficient							
Total impact	-0.0099***	-0.0072***	0.0027***	-0.0074***	-0.0048***	0.0026***	
Nonlinearity	0.0016**	0.0018**	0.0001**	0.0017**	0.0017**	0.0001**	
Education of other parent	-0.0013***	-0.0012***	-0.0001	-0.0011***	-0.0002	0.0007**	
Household living standards	-0.0040***	-0.0030***	0.0010**	-0.0033***	-0.0026***	0.0009**	
Fertility	-0.0009***	-0.0004***	0.0004**	-0.0007***	-0.0003***	0.0003**	
Health care use	-0.0008***	-0.0004***	0.0002	-0.0006***	-0.0004***	0.0001	
Urban/rural	-0.0003	0.0006***	0.0009***	-0.0002	0.0005**	0.0007***	
Geographic clustering	-0.0026***	-0.0033***	-0.0007	-0.0018***	-0.0025***	-0.0008	
Maternal height	-0.0016***	-0.0013***	0.0004***	-0.0013***	-0.0010**	0.0003**	
Baseline covariates			0.0005**			0.0003	
Outcome: wasted (n = 406,416)							
Coefficient: basic model	-0.0038***	-0.0028***	0.0009**	-0.0031***	-0.0025***	0.0006**	
Coefficient: full model	-0.0015**	-0.0010*	0.0005	-0.0015**	-0.0013**	0.0001	
Decomposition: covariates' impact on basic coefficient							
Total impact	-0.0023***	-0.0018***	0.0004	-0.0017***	-0.0012**	0.0005	
Nonlinearity	0.0004	0.0005	0.0000	0.0005	0.0005	0.0000	
Education of other parent	-0.0005**	-0.0003	0.0001	-0.0005**	-0.0001	0.0004	
Household living standards	-0.0012***	-0.0004*	0.0008*	-0.0010***	-0.0003	0.0007**	
Fertility	-0.0001	0.0000	0.0001	-0.0001	-0.0000	0.0001	
Health care use	-0.0002**	-0.0002**	0.0000	-0.0002**	-0.0001*	0.0000	
Urban/rural difference	0.0000	0.0001	0.0000	0.0000	0.0000	0.0000	
Geographic clustering	-0.0006*	-0.0014***	-0.0008*	-0.0004	-0.0011**	-0.0007*	

Maternal height	-0.0001***	-0.0002***	-0.0000	-0.0001***	-0.0001***	-0.0000
Baseline covariates			0.0001			0.0000

Notes: *P < .10; ** p < .05; *** p < .01. 2 sided. All estimates are weighted and P-values adjusted for clustering at the PSU level. All coefficients are for an increase in the linear probability of experiencing the outcome to a single year increase in parental education. Basic model covariates: months since birth; child's sex; child's year of birth; maternal age at survey; a constant term. Full model covariates: basic covariates; household living standards (wealth index z-score); fertility (maternal age at birth, number of siblings ever born, birth interval [dummy variable adjustment for firstborn], birth order, maternal age at first birth); health care use (vaccinated [surviving children only], born at home); urban-rural difference (rural resident); maternal height (indicating maternal early life living standards); geographical clustering (PSU-specific means of all independent variables, except for rural residents); education of other parent (other parent's years of education, interaction between mean-centred years of education, interaction between mean of included observations of other parent's years of education, interaction between mean of included observations of other parent's education). All independent variables were demeaned from a survey-specific mean of included observations. The 'baseline covariates' set contains the terms for the main associations of covariates when decomposing the 'change coefficient'. When decomposing the 'change coefficient' all sets (except 'nonlinearity' and 'baseline covariates') contain covariates interacted with being in the newest surveys. Coefficients are from Eq1, and Eq2.; oldest is β ; newest is β^* ; change is $\delta\beta$. The decomposition of the 'change coefficients' quantifies the changing impact of the respective sets of covariates (except 'nonlinearity' and 'baseline covariates') on the coefficients from the basic models. The decomposition of main coefficients from the oldest and newest surveys shows the impact of each set of covariates on the main association. Full outputs, exact P-values, 95% confidence intervals and standard errors are shown in Tables A14 - A16 in the Appendix.

For stunting, the association with maternal education attenuated by 0.0034 (95% CI 0.0023–0.0046; P<0.0001) (15%) and 0.0015 (95% CI 0.0004–0.0025; P=0.0059) (8%) for paternal education. The decomposition of covariates shows that for maternal education, changing impact of household living standards accounts for 0.0015 (95% CI 0.0002–0.0027; P=0.0187) (44%), urban-rural differences for 0.0012 (95% CI 0.0004–0.0020; P=0.0031) (35%), fertility for 0.0007 (95% CI 0.0001–0.0012; P=0.0133) (21%), maternal height for 0.0003 (95% CI 0.0001–0.0005; P=0.0013) (9%), and nonlinearity for 0.0002 (95% CI 0.0000–0.0004; P=0.0199) (6%), of the change coefficient. For paternal education, changing impact of household living standards account for 0.0013 (95% CI 0.0002–0.0023; P =0.0013) (87%), urban-rural differences for 0.001 (95% CI 0.0003–0.0017; P=0.0029) (67%), maternal height for 0.0002 (95% CI 0.0001–0.0004; P=0.0013) (13%), and fertility for 0.0005 (95% CI 0.0001–0.0025; P=0.0082) (33%), of the change coefficient. Changes in geographical clustering enhances the change coefficient by 0.0018 (95% CI -0.00034– -0.0001; P =0.0338) (53%) for maternal education, and 0.0020 (95% CI -0.0035– -0.0006; P=0.0053) (133%) for paternal education.

In Table 4, we show results for the relationship between parental education and the probability of being wasted and underweight. For underweight, the association attenuated by 0.0042 (95% CI 0.0033–0.0051; P<0.0001) (30%) for maternal education and 0.0028 (95% CI 0.0020–0.0036;P<0.0001) (24%) for paternal education. Decomposing the impact of covariates on the change coefficients shows that for maternal education, changing impact of household living standards accounts for 0.0010 (95% CI 0.0001–0.0019; P=0.0348) (24%), urban-rural differences for 0.0009 (95% CI 0.0003–0.0015; P=0.0031) (21%), maternal height for 0.0004 (95% CI 0.0002–0.0005;P<0.0001) (10%), fertility for 0.0004 (95% CI 0.0000–0.0009; P=0.0389) (10%), and nonlinearity for 0.0001(95% CI 0.0000–0.0003; P=0.0156) (2%). For paternal education, changing impact of household living standards accounts for 0.0009 (95% CI 0.0001–0.0017; P=0.0275)(32%), urban-rural differences for 0.0007 (95% CI 0.0002–0.0012; P=0.0032) (25%), maternal education for 0.0007 (95% CI 0.0000–0.0014; P=0.0443) (25%), fertility for 0.0003 (95% CI 0.0000–0.0006; P=0.0235)(11%), maternal height for 0.0003 (95% CI 0.0002–0.0004; P<0.0001) (11%), and nonlinearity for 0.0001(95% CI 0.0000–0.0001; P=0.0262) (4%).

Similarly, we find that the association with wasting has attenuated by 0.0009 (95% CI 0.0003–0.0015; P=0.0022) (24%) for maternal education and 0.0006 (95% CI 0.0001–0.0012; P=0.0209) (19%) for paternal education. Decomposing the impact of covariates on the change coefficients shows that changing impact of household living standards accounts for 0.0008 (95% CI 0.0001–0.0015; P=0.0183)(89%) of the attenuation for maternal education and 0.0007 (95% CI 0.0001–0.0025; P=0.0151) (117%) for paternal education.

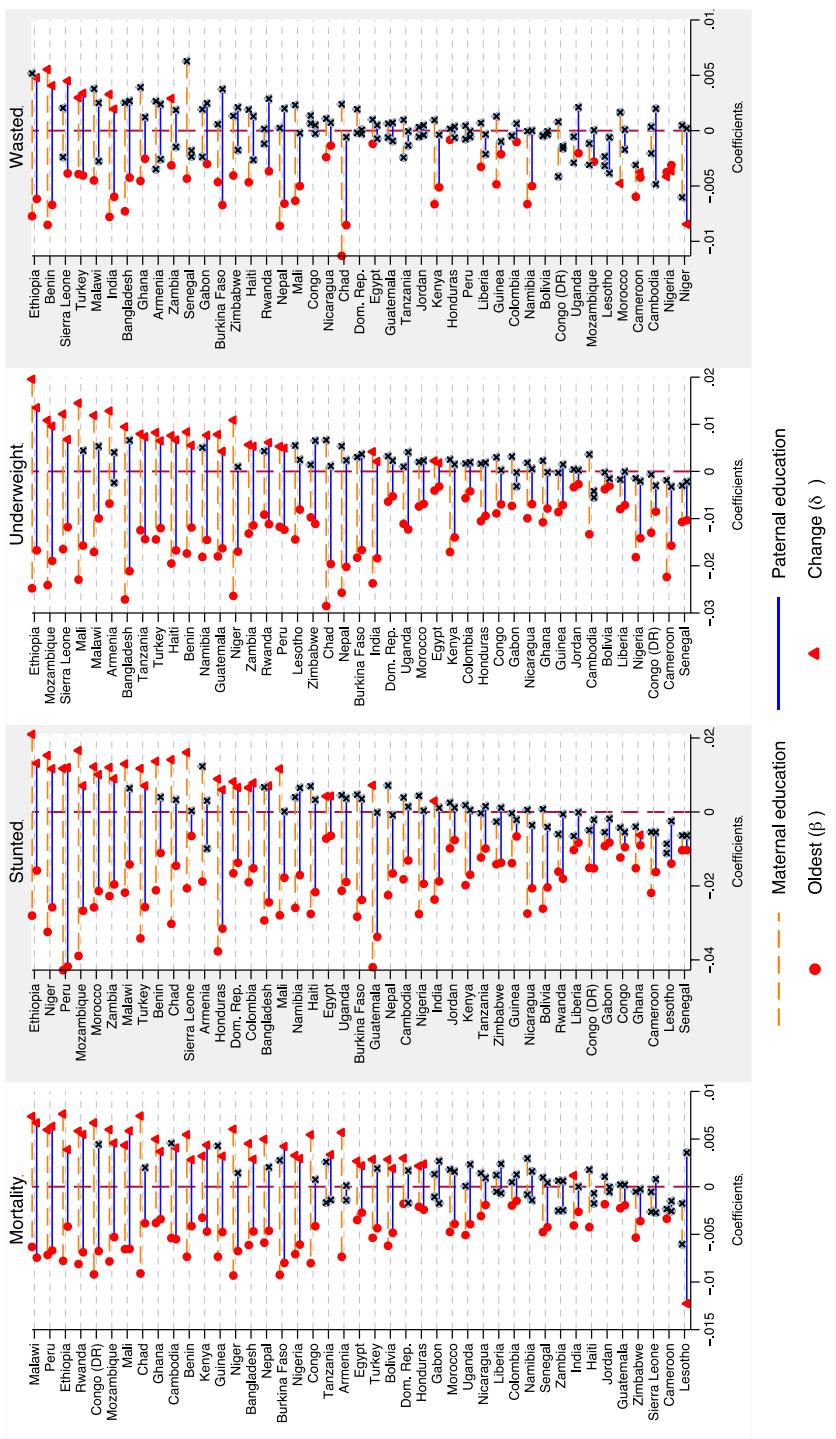


Figure 2. Coefficients for the association of parental education in the oldest survey and change in association, by child outcome and country

Coefficients reflect an increase in the linear probability of experiencing the outcome resulting from an additional year in parental education, separately for maternal and paternal education. Countries are ordered according to the average magnitude of change in the association of parental education for each outcome, between the oldest and newest surveys. Basic model covariates include: months since birth; child's sex; child's year of birth; maternal age at survey; a constant term. All independent variables were demeaned from a survey-specific mean of included observations. Estimates with P-values greater than 0.05 are marked with X. All estimates are weighted and P-values adjusted for clustering at the PSU level. Abbreviations: Dom. Rep., Dominican Republic; DR, Democratic Republic.

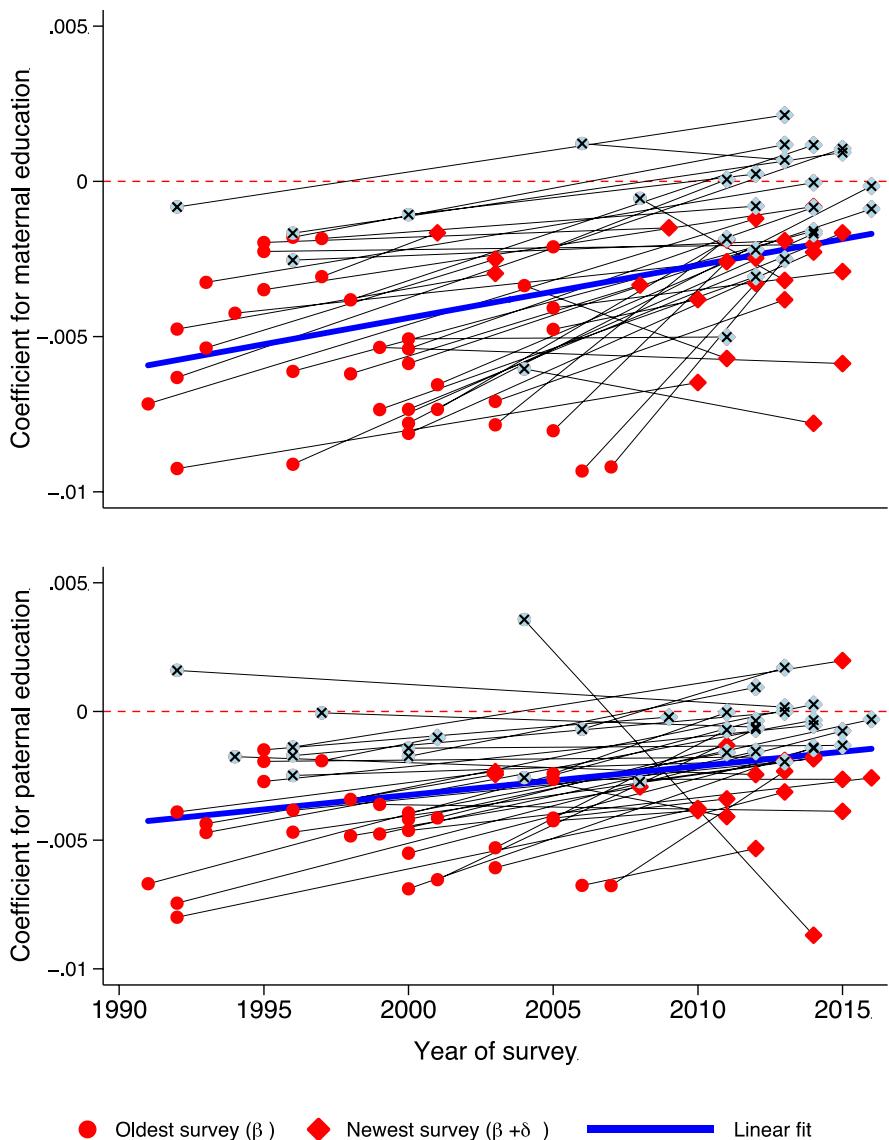


Figure 3. Coefficients for association of parental education with mortality, by survey year

Coefficients reflect an increase in the linear probability of under-five mortality resulting from an additional year in parental education, separately for maternal and paternal education. Basic model covariates include: months since birth; child's sex; child's year of birth; maternal age at survey; and a constant term. All independent variables were demeaned from a survey-specific mean of included observations. Coefficients are for the main association in oldest and newest surveys, connected with a line for each country. Estimates with P-values greater than 0.05 are marked with X. All estimates are weighted and P-values adjusted for clustering at the PSU level.

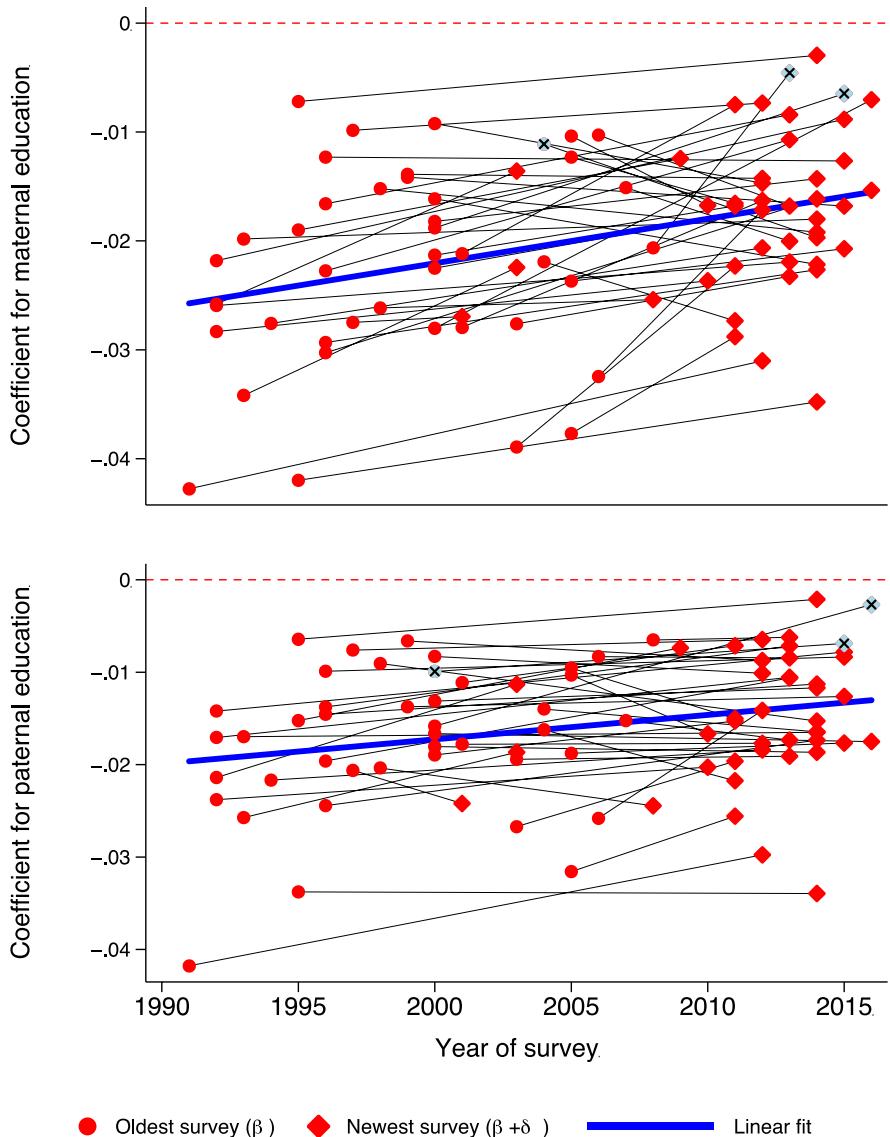


Figure 4. Coefficients for association of parental education with stunting, by survey year

Coefficients reflect an increase in the linear probability of a child being stunted resulting from an additional year in parental education, separately for maternal and paternal education. Basic model covariates include: months since birth; child's sex; child's year of birth; maternal age at survey; and a constant term. All independent variables were demeaned from a survey-specific mean of included observations. Coefficients are for the main association in oldest and newest surveys, connected with a line for each country. Estimates with P-values greater than 0.05 are marked with X. All estimates are weighted and P-values adjusted for clustering at the PSU level.

Results from sensitivity analyses are shown in Supplementary Tables A4–A13. Our conclusions for maternal education are robust to all sensitivity analyses. Our conclusions for paternal education and nutrition outcomes are not robust to using categorized education (Supplementary Tables A4 and A5), continuous outcomes (Supplementary Table A6), logit (Supplementary Table A7) and multinomial selection models (Supplementary Table A11). Although there is attenuation, the coefficient is small and not statistically significant ($P>0.05$). Results for paternal education, however, are robust to all sensitivity analyses for mortality.

Figure 2 shows results for each country. There is country heterogeneity in the main association as well as attenuation in association over time. Countries with the greatest association in the oldest surveys generally have greater attenuation. Figures 3 and 4 show coefficients for parental education in the oldest and newest surveys, plotted against year of surveys, for mortality and stunting. We find attenuation over time overall. Attenuation is statistically significant ($P<0.05$) in 23, 18, 17 and 4 countries for respectively mortality, stunting, underweight and wasting for maternal education, and 18, 12, 14, 5 countries, respectively, for paternal education. Supplementary Figures A1–A23 show additional country specific results. Most notably, Supplementary Figures A20– A23 show a positive correlation between the magnitude of attenuation and economic growth.

Discussion

We analyzed 86 DHS surveys conducted at two points in time in 43 low- and middle-income countries between 1991 and 2016. The association between parental education and child health weakened over time for under-5 mortality, chronic malnutrition (stunting), a composite indicator for acute and chronic malnutrition (underweight), and acute malnutrition (wasting). The magnitude of the changes in proportion to the association in the oldest surveys is substantial, especially for under-5 mortality. In the oldest surveys, 1 more year of maternal education was associated with a 0.48% point (0.0048×100) lower linear probability for mortality, 2.3 for stunting, 1.4 for underweight, and 0.4 for wasting, but this relationship had attenuated by 56% for mortality, 15% for stunting, 30% for underweight and 24% for wasting on average 13 years later. A 1-year increase in paternal education was associated with a 0.4% point lower linear probability for mortality, 1.8 for stunting, 1.2 for underweight and 0.3 for wasting, but attenuated by 53% for mortality, 8% for stunting, 24% for underweight and 19% for wasting. Despite this decline, there remains a significant association in the newest surveys in our pooled sample for all outcomes and education of both parents, indicating that parental education may still be an important determinant of child health.

In Table 1 in the Introduction, we described possible reasons why the association between parental education and child health may weaken or strengthen, and identified likely underlying mechanisms. Our analysis indicates a reduced impact of household living standards, fertility and early life living standards (as proxied by maternal height) on the association between parental schooling and child health. In contrast, geographical clustering increasingly explains the association with child health, in particular for stunting. In the newest surveys, the association between parental education and child nutrition reflects geographical clustering to a greater extent than in the oldest surveys.

Furthermore, changes in urban-rural differences have interesting implications for the change in the association, and in the newest surveys, these appear to suppress the association. Indeed, better-educated parents more commonly reside in urban areas, and residing in an urban area is associated with adverse child health outcomes, independently of other covariates, in the newest surveys. This finding suggests an ‘urban penalty’ on child health, which has been hypothesized to emerge when increasing urbanization is not met with sufficient improvements in urban environments (Gould, 1998).

Our results support earlier findings on declining differences in under-5 mortality by maternal education in more recent birth cohorts in several sub-Saharan African countries (Bado and Sathiya Susuman, 2016). Our findings are also similar to those of a decline in differences in the predicted prevalence of undernutrition by maternal education in LMICs, between DHS conducted before and after 2000 (Vollmer et al., 2017b).

Limitations

This analysis has several limitations. First, this study does not determine causality. Nevertheless, we explore the influence of known confounders, most importantly unobserved geographical clustering. Second, we acknowledge the potential of measurement error in our variables. However, this concern mostly has implications for our conclusions to the extent that measurement error is different between the oldest and the newest surveys. We have no reason to believe that this is the case, as the DHS has maintained rigorous standards across survey rounds (Corsi et al., 2012). Third, our outcomes indicate severe deficiencies in health, and patterns may differ when looking at more subtle health differences. These are, however, standard outcomes when looking at child health in developing countries and are a part of the Sustainable Development Goals. Our sensitivity analysis on continuous nutrition measures further indicates that our conclusions apply to more subtle differences in child health as well. Fourth, data were not available in the DHS datasets on a number of factors listed in Table 1, to examine other potential

reasons for a weakening association of parental education with child health, including changes in education quality and the formal school curriculum.

Conclusion

To our knowledge, this study is the first to comprehensively assess changes in the association of parental education with child health and mortality over time and to determine possible underlying mechanisms behind these changes. The role of parental education, at the individual level, appears to attenuate over time and reflects geographical clustering to a greater extent. Urban-rural differences are likely to suppress the association, indicating the potential emergence of an ‘urban penalty’ on child health. Since parental education is a widely cited instrument for improving child health, these findings have implications for policy efforts to reduce child mortality and improve health. More broadly, this paper underlines that social determinants of child health are dynamic, and policy makers should take note of changing contexts to further improve child health in the post-2015 development agenda.

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Supplements

Appendix A1. Mechanisms behind changing association between parental education and child health in developing countries

Drivers of changes in the association between parental education and child health in developing countries can relate to the evolution in education itself, benefits of education in a changing epidemiological, economic and social environment, and the extent to which education determines underlying pathways and how pathways determine child health. How education reflects the underlying confounders may also have changed. Table 1 shows an overview of the potential causes of the changing association between parental education and child health outcomes.

‘Artifact of the measure’ captures anything related to the measure of education and what it means as an exposure. It can relate to changes in the quality of education, e.g., if funding increases insufficiently, as enrollment increases. The curriculum may change and be of different relevance for child health. Returns to education, regarding child health, may diminish at higher levels of education.

‘Confounding change’: Broadly, education may signal living standards less as it becomes more available, or, conversely, those who remain uneducated despite improved access may represent a more marginalized group. Lower living standards in early childhood reduce accumulated health stock at schooling age, reducing benefits of education (Heckman, 2007). Similarly, if education is scarce, more innately able children may be more likely to be sent to school (Datar et al., 2010), then being educated will reflect the innate ability to a lower extent, lowering the association with child health. Conversely, education may be a way out of poverty for children from deprived backgrounds and, if these children benefit more from education, the association may increase while education keeps expanding to poorer segments of the population.

‘Mediator and moderator change’: For example, fertility is a potential mediator between parental education and child health (Grepin and Bharadwaj, 2015). If education determines fertility to a lower extent (Martin, 1995) - or fertility determines child health to a lower extent, the association will decrease. The relationship between education and health (Cutler and Lleras-Muney, 2006) could also change over time, changing the association with child health.

Moderators can also change. If parental education interacts with health care availability, either as a complement or a substitute (Barrera, 1990; Strauss, 1990; Thomas et al., 1990), and health care becomes increasingly available over time, the association may increase if it is complementary, or decrease if it is a substitute. The underlying epidemiological environment can also interact with parental education. When highly virulent communicable diseases are the leading cause of death, there tend to be no socio-economic status (SES) differences in mortality. Public health improvements may benefit higher classes disproportionately, and SES differences emerge but can also decline as improvements reach the whole population. An SES gap may emerge again when new medical knowledge and

technology increasingly become utilized by better off segments of the population (Antonovsky, 1967; Bengtsson and van Poppel, 2011).

Appendix A2. Variables used in the study

Children under five years old were measured with an accuracy of 0.1 cm and 0.1 kg. We calculated height-for-age Z-scores (HAZ), weight-for-age Z-scores (WAZ) and weight-for-height Z-scores (WHZ) based on the WHO 2006 sex-specific growth standards (WHO, 2006). Children were coded as stunted, underweight or wasted respectively if their HAZ, WAZ or WHZ fell below -2 z-scores of the reference median. Recumbent length was assumed for children under 24 months old. Children with implausible values ($|HAZ| > 6$; $|WHZ| > 5$; and $WAZ < -6$ or > 5) were excluded.

All independent variables were demeaned using survey specific mean of all valid observations, i.e., observations that were included in the final analysis of the respective outcomes. This procedure is equivalent to adding dummy coded variables for survey and accounts for differences in outcomes and parental education between survey years and countries, which are at different stages of development.

As our basic control variables, we included maternal age at the time of the survey, child's age, child's sex and child's year of birth. Controlling for child's year of birth captures linear trends in child health outcomes and parental education. Child's age and child's sex are unlikely to be mediating or confounding the relationship or the change in the relationship between parental education and child health, but relate to health and therefore improve fit and reduce standard errors. Maternal age controls for the wide age range of women interviewed in the Demographic and Health Surveys (DHS).

We avoided controls that may capture pathways between parental education and child health in our basic controls. We then explored the impact of household living standards, fertility, and health care use, which are likely to be on the causal pathway between parental education and child health, as discussed in the main text. Additionally, the impact of maternal height (an indicator for early life living standards), urban-rural differences, as well as geographic clustering is explored. At the individual level, these covariates are more likely to act as confounders of the relationship between child health and parental education.

Household living standard was measured using a wealth index provided by the DHS and derived from a principal component analysis using various indicators of living standards such as ownership of certain assets, source of drinking water, and quality of dwelling (DHS, 2016). The index is specific to a survey. We calculated a wealth index z-score using the mean and standard deviation of the factor scores for each survey.

We used several variables to capture the impact of fertility on the relationship between parental education and child health; maternal age at birth, maternal age at first birth, birth interval, birth order, and number of siblings ever born. Mother's

age at birth relates to child outcomes in various ways. Firstly, children of young mothers may have worse outcomes due to lower capabilities for childrearing, physiological immaturity, and SES disadvantages (Fraser et al., 1995). Older mothers may have more experience and have more resources, but biological factors may also cause worse outcomes for children (Schmidt et al., 2011). Therefore, mother's age is specified as a quadratic polynomial. Mother's age at first birth can influence child health for similar reasons. If a mother gives birth at a very young age, before she reaches adult height, it may lead to shorter adult stature and worse pregnancy outcomes (Özaltin et al., 2010; Thurnham, 2013). However, if a mother has her first child very late, it may also lead to negative consequences for the health of her children (Aldous and Edmonson, 1993). Schooling can both delay the age at first birth (Gupta and Mahy, 2003), and first childbirth can interfere with schooling (Waite and Moore, 1978). Short birth interval has been found to have harmful effects on children and appears to be more common in households with lower SES (Curtis et al., 1993; Rutstein, 2005). We adjusted the birth interval for first-born children using dummy variable adjustment. Birth order, independent of mother's age, may influence the allocation of resources to children within the household as well as pregnancy-related aspects (Cabrera, 1980; Horton, 1988). Number of siblings ever born addresses strains on household resources as well as other factors.

Maternal height is a widely used indicator for early-life living standards, which have long-term consequences for health and related outcomes, such as education, as well as the health outcomes of her children (Case and Paxson, 2008; Özaltin et al., 2010). It is unlikely to be a pathway between parental education and child health, but early-life living standards may determine educational attainment through self-productivity of skills, as healthier children are more likely to attend school (Heckman, 2007; Miguel and Kremer, 2004; Mukudi, 2003). It may also capture early-life and childhood living standards more generally, such as with regards to availability of schooling. Unfortunately, the DHS generally does not record height of fathers.

Urban-rural differences in child health have been studied where children in rural areas are generally disadvantaged regarding health. There is, however, the risk that an 'urban penalty' emerges as more impoverished people from rural areas migrate into urban slums (Fotso, 2007; Gould, 1998).

The impact of nonlinearity is estimated by adding a squared term for parental education. Its impact on the main association of parental education is the difference in the linear association and the association at very low levels of education (strictly speaking going from 0 years of education to one year of education).

Appendix A3. Gelbach decomposition

Gelbach decomposition was used to estimate the impact of covariates on the relationship of interest (Gelbach, 2016). We briefly explain the procedure here using a hypothetical example. Multiple regression models are frequently estimated in the social sciences, starting with a simple model and then stepwise adding control variables, to account for the impact of different covariates on the relationship of interest. One goal of this approach is to determine how much of cross-category difference in a specific outcome is attributable to category-level heterogeneity in the covariates. For example, how much of the difference in the height between children born to educated and non-educated mothers is due to differences in fertility, SES, and health care use between the two groups? One issue when using such sequential addition, in order to comment on the impact of each of the added covariates on the relationship of interest, is that the results are sensitive to the order in which variables are entered into the model. Gelbach proposes a decomposition to avoid this problem.

In practice, the decomposition is done by first running a restricted regression with the outcome y , the exposure variable of interest E and a vector \mathbf{x} containing any number of basic control variables. Say the outcome y is child's height, the exposure E is maternal education, and \mathbf{x} contains the control variables child's age and child's sex.

$$y = \alpha^{basic} + E \beta^{basic} + \mathbf{x}' \rho^{basic} + \varepsilon^{basic}$$

Then another regression is run including an additional set of controls

$$y = \alpha^{full} + E \beta^{full} + \mathbf{x}' \rho^{full} + \mathbf{z}'_f \gamma_f + \varepsilon^{full}$$

where \mathbf{z} contains F number of variables, e.g., income, number of siblings, and number of vaccines received. The interest is in estimating the independent impact of each of the additional covariates, from \mathbf{z} , which appears in the unrestricted regression only, on the parameter of interest β^{basic} . The Gelbach decomposition uses the formula for omitted variable bias to decompose the influence of \mathbf{z} on β^{basic} .

The difference between the two estimates is

$$\tau = \beta^{basic} - \beta^{full}$$

where τ is the total impact of the all the covariates in \mathbf{z} on the association between maternal education and child height, adjusted for \mathbf{x} . In this example \mathbf{z} has 3 variables, income, vaccines and siblings, so $\mathbf{z}_f = z_1, z_2, z_3$. Three auxiliary regressions can be ran with the first one being

$$z_1 = \alpha_1 + \alpha_{1E} E + \alpha_{1x}' \mathbf{x} + \omega_1$$

and the same for two and three. α_{IE} is the association between maternal income and maternal education, independent of \mathbf{x} . The total impact on β^{basic} attributable to all three covariates is

$$\tau = \beta^{basic} - \beta^{full} = \sum_{f=1}^3 \gamma_f \alpha_{fE}$$

The independent impact of income is obtained as

$$\tau_1 = \gamma_1 \alpha_{1E}$$

In this application, the impact of income is determined by (1) strength of the association between income and child height independent of maternal education, vaccinations, number of siblings, age and sex (y_1), and (2) strength of the association of maternal education with income, independent of age and sex (α_{IE}). The same goes for the other two variables in \mathbf{z} , vaccines and siblings.

The decomposition allows for any number of basic controls in \mathbf{x} and any number of covariates in \mathbf{z} . The impact can easily be summed up of over a set of covariates, relating to similar mechanisms. For example, rather than only using number of siblings to capture fertility, a set of variables can be used such as number of siblings, maternal age, and birth interval. Then the total impact of fertility is the sum of the estimated impact of each of the variables relating to fertility.

This paper used fixed effects which control for group-level heterogeneity at the level of the community (PSU). In order to be able to decompose the impact of the community using the Gelbach decomposition, we use so-called Mundlak fixed effects, by adding community level (PSU) means of all valid observations, for all independent variables (Antonakis et al., 2010; Mundlak, 1978). This procedure provides community-level parameters which can be used to decompose the impact of between-community heterogeneity for the relationship of interest. An alternative way to control for community-level heterogeneity would be to add a dummy coded indicator for each community. However, since the number of communities in this paper is almost 60,000, this is not computationally feasible. A third common approach to adjust for a large number of groups would be to subtract a group level mean of valid observations from each independent variable. However, this does not provide estimates for group levels, which are needed to estimate the impact of between- community heterogeneity on the relationship of interest when using Gelbach decomposition. All of the above-described methods for controlling for group level heterogeneity give parametrically identical

estimates for variables measured below the group level. The impact obtained from the Gelbach decomposition using Mundlak fixed effects is parametrically identical to decomposing a set of group level dummy coded variables.

Appendix A4. Sensitivity analyses

We performed a range of sensitivity analyses to assess whether model specification, sampling weights, variable definition, or missing values, influenced our findings.

First, there may be “threshold” levels for parental education to affect child health (Makoka and Masibo, 2015) – i.e., parental education still matters, but higher levels are now required to see an effect of parental education on child health (i.e., secondary education or more). Also, since education is completed in stages, it might be reasonable to look at associations for each stage. We, therefore, present estimates of parental education categorized as no education, primary education and secondary or more. We present estimates for the difference in outcomes for children born to parents with primary compared to no education in Table A4 and secondary or more education compared to no education in Table A5.

Second, although stunting, wasting and underweight are accepted thresholds indicating the prevalence of undernutrition, harmful environmental exposures can shift the whole distribution of height and weight downwards, decreasing the mean. We, therefore, redid our analysis using continuous measures of child nutrition which are presented in Table A6.

Third, linear probability models (LPM) are increasingly accepted and preferred for their simplicity and flexibility and are often preferable when estimating a large number of fixed effects. However, nonlinear models have been recommended when estimating binary outcomes (Verbeek, 2012, p. 418). Therefore we redid our analysis using logit models and present estimates as average marginal effects in Table A7.

Fourth, since our analysis used pooled DHS surveys, there are several ways in which to adjust the provided sampling weights. We rescaled the DHS sampling weights, to sum up to population size in Table A8 and show results without sample weights in Table A9. The former gives globally representative estimates (for the included countries). To rescale the sampling weights to be globally representative, we obtained estimates of the population under five years old for each country and survey year from the United Nations (DESA, 2010).

Fifth, many observations are excluded because of missing values for covariates, paternal education and anthropometric information (Table A2). We also find indications that these are not missing at random (Table A3). We explore the implication of missing observation in a couple of ways. We redo our basic models including observations with missing covariates (Table A10). To explore the implications of missing nutrition information, we redo our analysis using multinomial selection models, to account for selection into the sample (Tables A11 and A12). Our selection models are done in two steps. First, a multinomial logit model is estimated on several categories indicating whether and why

anthropometric data was missing. Then the predicted probability of being observed and included in the sample is obtained. These predicted probabilities are then used in the second step to adjust for selection into the sample when estimating the association between parental education and child health. Specifically, we use a multinomial selection model first developed by Dubin and McFadden (Dubin and McFadden, 1984) and extended by Bourguignon, Fournier, and Gurgand (Bourguignon et al., 2007). These are an extension of Heckman's selection model (Heckman, 1979). Although the selection equation is formally identified using the same independent variables in both steps, this has been shown to be inefficient and lead to biased estimates (Brandt and Schneider, 2007). It is recommended to include some variables in the first step which are not in the second step, which determines selection but is unrelated to the outcome in the second step.

Several papers have used selection models to account for missing outcomes in the DHS when using the anthropometric data (Desai, 1995), as well as HIV testing (Bärnighausen et al., 2011; Clark and Houle, 2014; McGovern et al., 2015). We similarly model our selection. We model three different reasons for selection: mortality, a flagged measure (due to inconsistency), and a missing measure. We specify the first step of the selection model as using the same independent variables as in our main models, in addition to variables related to interviews and interviewers, and under-5 deaths of siblings. Interview related variables are likely to capture selection due to missing or flagged data and are weekday of interview, number of visits, and how many days into the fieldwork a household was interviewed. Interview related variables are unlikely to relate to nutrition outcomes but appear to correlate highly with whether the observation was missing, somewhat with observation being flagged, but not mortality. Since under-5 deaths cluster within households, we include a variable for number of siblings that died before the age of five, over the last five years, to adjust for selection due to mortality. However, it is unlikely that there is any variable that relates to mortality but not child nutrition outcomes so these results should be interpreted with caution. All selection variables are interacted with being in oldest or newest survey. Therefore we redo our models excluding selection due to mortality, as well as the sibling death selection predictor. We present estimates from these models in Tables A11 and A12.

There are some notable differences between our main results compared to our sensitivity analyses. When using the categorical specification for educational attainment, the attenuation in the association of paternal education (but not for maternal education) with nutrition outcomes is not statistically significant. Another implication from categorizing education is that fertility appears to be of more importance than household living standards when comparing the change in association for mothers with primary education compared to those without education (Table A4 and A5). Using continuous nutrition measures shows similar

patterns to our main results for maternal education but, although there is attenuation for paternal education and stunting, it did not reach conventional benchmarks for statistical significance (Table A6). Using logit models in lieu of LPMs, the attenuation appears to be smaller but remains statistically significant for maternal education, while the attenuation is not statistically significant for paternal education and nutrition outcomes (Table A7). When sampling weights are rescaled to be globally representative or when not using sampling weights, we obtain similar results, although the attenuation differs in magnitude (Table A8 and A9). When including those respondents with missing covariates, we find similar results (Table A10). The results from the selection models (Table A11), we find that the attenuation is not statistically significant for paternal education and stunting.

Although there are some differences in statistical significance for the different models, the estimates show broadly similar results, attenuation in the association between parental education and child health, of similar magnitudes. The attenuation in estimates appears to be more consistent for maternal education than for paternal education.

Table A1. Countries, survey years and number of observations included in the study

Country	Oldest survey	Newest survey	Difference	Obs. (mortality)	Obs. (nutrition)
Armenia	2000	2015	15	3,087	2,239
Bangladesh	1996	2014	18	9,147	7,624
Benin	2001	2011	10	14,196	8,584
Bolivia	1998	2008	10	12,405	11,050
Burkina Faso	1992	2010	18	10,691	8,563
Cambodia	2000	2014	14	7,970	6,827
Cameroon	2004	2011	7	6,504	5,419
Chad	1996	2014	18	15,075	12,131
Colombia	1995	2009	14	11,114	10,487
Congo	2005	2011	6	5,177	4,509
Congo (DR)	2007	2013	6	9,784	7,913
Dom. Rep.	1996	2013	17	4,171	3,715
Egypt	1995	2014	19	24,827	21,378
Ethiopia	2000	2016	16	15,210	9,947
Gabon	2000	2012	12	3,790	3,176
Ghana	1998	2014	16	4,086	3,566
Guatemala	1995	2014	19	16,788	15,443
Guinea	1999	2012	13	7,201	4,658
Haiti	1994	2012	18	4,617	3,884
Honduras	2005	2011	6	13,776	12,556
India	2005	2015	10	84,401	71,961
Jordan	1997	2012	15	12,029	10,887
Kenya	1993	2014	21	12,046	10,671
Lesotho	2004	2014	10	2,223	1,728
Liberia	2006	2013	7	5,071	4,108
Malawi	1992	2015	23	6,749	4,204
Mali	2001	2012	11	15,035	11,387
Morocco	1992	2003	11	9,779	8,549
Mozambique	2003	2011	8	13,432	11,318
Namibia	1992	2013	21	2,520	1,851
Nepal	2000	2016	16	8,128	6,536
Nicaragua	1997	2001	4	8,741	7,550
Niger	2006	2012	6	8,123	6,549
Nigeria	2003	2013	10	29,971	23,350
Peru	1991	2012	21	13,837	12,691
Rwanda	2000	2014	14	8,278	6,885
Senegal	2005	2010	5	5,683	4,570
Sierra Leone	2008	2013	5	5,729	4,030
Tanzania	1996	2015	19	11,027	7,374
Turkey	1993	2003	10	7,370	6,568
Uganda	2000	2011	11	6,141	4,921

Zambia	1996	2013	17	13,269	11,146
Zimbabwe	1999	2015	16	6,399	3,913

Abbreviations: Dom. Rep., Dominican Republic; DR, Democratic Republic, Obs. Observations.

Table A2. Sample deduction

Reason for exclusion	n=338,916 Oldest	n=701,950 Newest	Missing from complete	Missing from remaining	Missing from remaining	Sample remaining
Mother not resident in household	12,469	12,469	23,200	23,200	1,005,197	
Mother not sampled for height measure	3,68%	3,68%	3,31%	3,31%	96,57%	
Mother not in union	11,598	11,442	99,362	97,562	896,193	
Mother had more than one union	3,42%	3,51%	14,16%	14,37%	86,10%	
Missing union information	30,273	28,041	55,323	42,195	825,957	
Maternal education missing	223	134	135	74	746,895	
Missing covariates	20,757	5,990	260,929	201,829	538,871	
Died	29,822	17,425	37,202	13,689	464,483	
Child not sampled for height/weight measure	24,051	2,213	112,061	4,584	457,686	
No immunization info	26,453	136	56,274	9,457	448,093	
	7.81%	0.07%	8.02%	3.71%	43,05%	

Missing stunted	97,143	20,732	195,761	19,484	407,877
	28.66%	10.23%	27.89%	7.94%	39.19%
Missing underweight	96,444	57	194,266	50	407,770
	28.46%	0.03%	27.68%	0.02%	39.18%
Missing wasted	97,068	505	195,889	849	406,416
	28.64%	0.28%	27.91%	0.38%	39.05%
Any missing anthropometry	97,895	197,578			406,416
	28.88%	28.15%			39.05%

Notes: Children over 59 months old are excluded. Only observations with all non-missing anthropometry outcomes are included in the anthropometry analysis. The disproportionate number of missing paternal education and covariates in the newest surveys are all because of a large number of missing values in the India 2015-16 survey.

Table A3. Sample deduction: means and differences in means between children with and without missing values

	Oldest	Newest	Non missing	Difference	Difference	Difference - difference
Missing vs. nonmissing covariates or paternal education						
Died	0.081	0.035***	0.045	0.018***	-0.017***	
Stunted	0.377	-0.013	0.280	0.079***	0.093***	
Underweight	0.194	-0.000	0.134	0.132***	0.133***	
Wasted	0.075	0.008*	0.065	0.063***	0.055***	
Maternal education	4.078	-0.354***	5.618	-0.449***	-0.095	
Wealth index z-score	-0.152	0.103***	-0.083	0.017	-0.086***	
Missing vs. nonmissing anthropometric because of mortality						
Maternal education	4.171	-1.462***	5.712	-1.754***	-0.293***	
Paternal education	5.538	-1.512***	6.570	-1.592***	-0.080	
Wealth index z-score	-0.127	-0.202***	-0.073	-0.210***	-0.008	
Missing vs. nonmissing anthropometric because of missing covariates						
Maternal education	4.077	-0.266***	5.671	-0.611***	-0.345***	
Paternal education	5.460	-0.415***	6.534	-0.579***	-0.164	
Wealth index z-score	-0.147	0.022**	-0.091	0.093***	0.071**	

Notes: *P < .10; **P < .05; ***P < .01. 2 sided. Children over 59 months old, women that are not in a union, women that were not sampled for height measurements, and women that are not residents at the place of interview are excluded. When looking at anthropometric outcomes children that were not sampled for anthropometric outcomes are excluded. We explore the implication of missing observations in Appendix A4 below.

Table A4. Sensitivity analysis: linear regressions with a categorized education variable, primary education compared to no education

	Maternal Education						Paternal Education					
	Oldest	Newest	Change	Oldest	Newest	Change	Oldest	Newest	Change	Oldest	Newest	Change
Outcome: Mortality (n = 495,597)												
Coefficient: Basic Model	-0.0205***	-0.0095***	0.0109***	-0.0127***	-0.0044**	0.0078**						
Coefficient: Full Model	-0.0018	0.0028	0.0046	0.0019	0.0030	0.0011						
Decomposition: Covariates' Impact on Basic Coefficient												
Total Impact	-0.0187***	-0.0123***	0.0064***	-0.0146***	-0.0074***	0.0067**						
Nonlinearity	-0.0013*	-0.0011*	0.0001	-0.0020***	0.0004	0.0024***						
Education of Other Parent	-0.0030***	-0.0008	0.0024*	-0.0048***	-0.0004	0.0049***						
HH Living Standards	-0.0018***	-0.0001	0.0016*	-0.0011**	-0.0002	0.0010						
Fertility	-0.0050***	-0.0048***	0.0013	-0.0032***	-0.0030***	0.0008						
Health Care Use	0.0005	-0.0002	-0.0008	0.0004	-0.0002	-0.0006						
Urban/Rural	-0.0002	0.0001	0.0010**	-0.0001	0.0000	0.0007**						
Geographic Clustering	-0.0075***	-0.0049***	0.0024	-0.0037**	-0.0040**	-0.0001						
Maternal Height	-0.0005***	-0.0003***	0.0002*	-0.0001**	-0.0000	0.0000						
Baseline Covariates												
Outcome: Stunted (n = 406,416)												
Coefficient: Basic Model	-0.0924***	-0.0785***	0.0132**	-0.0484***	-0.0603***	-0.0129*						
Coefficient: Full Model	-0.0139***	-0.0025	0.0114	-0.0024	-0.0102*	-0.0077						
Decomposition: Covariates' Impact on Basic Coefficient												
Total Impact	-0.0785***	-0.0760***	0.0018	-0.0460***	-0.0501***	-0.0052						
Nonlinearity	-0.0021	-0.0030**	-0.0009	0.0001	-0.0027**	-0.0028*						
Education of Other Parent	-0.0071***	-0.0095***	-0.0024	-0.0073***	-0.0062***	0.0028						
HH Living Standards	-0.0245***	-0.0199***	0.0036*	-0.0167***	-0.0160***	0.0031*						
Fertility	-0.0070***	-0.0074***	0.0051***	-0.0045***	-0.0054***	0.0038**						
Health Care Use	-0.0062***	-0.0050**	0.0013	-0.0043***	-0.0041***	0.0011						
Urban/Rural	0.0007	0.0024***	0.0036***	0.0004	0.0018***	0.0027***						
Geographic Clustering	-0.0243***	-0.0271***	-0.0054	-0.0113***	-0.0164***	-0.0071						
Maternal Height	-0.0081***	-0.0064**	0.0008**	-0.0024***	-0.0010	0.0001						

Baseline Covariates		Outcome: Underweight (n = 406,416)		Outcome: Wasted (n = 406,416)	
Coefficient: Basic Model	-0.0793***	Coefficient: Full Model	-0.0133***	Coefficient: Basic Model	-0.0038
Decomposition: Covariates' Impact on Basic Coefficient					
Total Impact	-0.0659***	-0.0549***	0.0105**	-0.0484***	-0.0043*
Nonlinearity	-0.0014	-0.0006	0.0008	-0.0019**	-0.0019**
Education of Other Parent	-0.0102***	-0.0109***	-0.0002	-0.0092***	-0.0082***
HH Living Standards	-0.0128***	-0.0095***	0.0028**	-0.0086***	-0.0075***
Fertility	-0.0034***	-0.0028**	0.0029**	-0.0023***	-0.0020***
Health Care Use	-0.0037***	-0.0027***	0.0010	-0.0026***	-0.0022***
Urban/Rural	-0.0004	0.0012**	0.0027***	-0.0002	0.0009**
Geographic Clustering	-0.0294***	-0.0263***	0.0025	-0.0223***	-0.0220***
Maternal Height	-0.0046***	-0.0031***	0.0009***	-0.0014***	0.0014
Baseline Covariates			-0.0030*		-0.0071***
Decomposition: Covariates' Impact on Basic Coefficient					
Total Impact	-0.0173***	-0.0201***	-0.0029	-0.0205***	-0.0139***
Nonlinearity	-0.0013*	0.0003	0.0016	-0.0019***	0.0004
Education of Other Parent	-0.0023**	-0.0049***	-0.0024	-0.0035***	-0.0028*
HH Living Standards	-0.0041***	-0.0013*	0.0027***	-0.0027***	-0.0010*
Fertility	-0.0008*	0.0001	0.0016*	-0.0004	0.0002
Health Care Use	-0.0012**	-0.0008	0.0004	-0.0008**	-0.0005
Urban/Rural	0.0001	-0.0000	0.0001	0.0000	0.0001
Geographic Clustering	-0.0074***	-0.0131***	-0.0052	-0.0110***	0.0017
Maternal Height	-0.0003***	-0.0004***	-0.0001	-0.0001*	-0.0000
Baseline Covariates			-0.0014***		-0.0029***

Notes: See Tables 3 and 4 in the manuscript.

Table A5. Sensitivity analysis: linear regressions with a categorized education variable, secondary education or more compared to no education

	Maternal Education			Paternal Education		
	Oldest	Newest	Change	Oldest	Newest	Change
Outcome: Mortality (n = 495,597)						
Coefficient: Basic Model	-0.0518***	-0.0202***	0.0298***	-0.0395***	-0.0177***	0.0203***
Coefficient: Full Model	-0.0059	0.0044	0.0102*	0.0032	-0.0035	-0.0067
Decomposition: Covariates' Impact on Basic Coefficient						
Total Impact	-0.0459***	-0.0245***	0.0195***	-0.0426***	-0.0142***	0.0269***
Nonlinearity	-0.0063*	-0.0059*	0.0004	-0.0103***	0.0023	0.0125***
Education of Other Parent	-0.0008	0.0003	0.0009	-0.0053***	0.0002	0.0041**
HH Living Standards	-0.0066***	-0.0003	0.0057*	-0.0048**	-0.0008	0.0039
Fertility	-0.0173***	-0.0116***	0.0028*	-0.0128***	-0.0094***	0.0024**
Health Care Use	0.0013	-0.0005	-0.0016	0.0010	-0.0004	-0.0014
Urban/Rural	-0.0005	0.0002	0.0034**	-0.0004	0.0002	0.0029**
Geographic Clustering	-0.0137***	-0.0058*	0.0031	-0.0088***	-0.0055**	-0.0007
Maternal Height	-0.0018***	-0.0010***	0.0008*	-0.0013***	-0.0007***	0.0005*
Baseline Covariates			0.0040***			0.0026***
Outcome: Stunted (n = 406,416)						
Coefficient: Basic Model	-0.2427***	-0.2034***	0.0330***	-0.1892***	-0.1743***	0.0101
Coefficient: Full Model	-0.0328***	-0.0126*	0.0202*	-0.0279***	-0.0174**	0.0105
Decomposition: Covariates' Impact on Basic Coefficient						
Total Impact	-0.2098***	-0.1909***	0.0127	-0.1613***	-0.1569***	-0.0004
Nonlinearity	-0.0106	-0.0160*	-0.0054	0.0004	-0.0145*	-0.0149*
Education of Other Parent	-0.0138***	-0.0126**	0.0004	-0.0193***	-0.0110***	0.0087*
HH Living Standards	-0.0807***	-0.0650***	0.0131**	-0.0658***	-0.0567***	0.0119**
Fertility	-0.0213***	-0.0157***	0.0080***	-0.0157***	-0.0132***	0.0064***
Health Care Use	-0.0142***	-0.0098***	0.0024	-0.0114***	-0.0084***	0.0022
Urban/Rural	0.0025	0.0081***	0.0123***	0.0020	0.0069***	0.0104**
Geographic Clustering	-0.0443***	-0.0544***	-0.0256***	-0.0306***	-0.0420***	-0.0231***
Maternal Height	-0.0274***	-0.0254***	0.0030***	-0.0208***	-0.0178***	0.0021***

Baseline Covariates		Outcome: Underweight (n = 406,416)				Outcome: Wasted (n = 406,416)			
Coefficient: Basic Model	Coefficient: Full Model	-0.1575***	-0.1174***	0.0362***	-0.1400***	-0.1107***	0.0263***	0.0040	-0.0040
Decomposition: Covariates' Impact on Basic Coefficient									
Total Impact									
Nonlinearity	-0.0071	-0.0030	0.0041	-0.0100**	-0.0105**	-0.0213***	0.0032		
Education of Other Parent	-0.0151***	-0.0125***	0.0021	-0.0145***	-0.0078***	0.0054			
HH Living Standards	-0.0420***	-0.0311***	0.0097**	-0.0336***	-0.0266***	0.0086**			
Fertility	-0.0098***	-0.0052**	0.0050**	-0.0075***	-0.0044***	0.0042**			
Health Care Use	-0.0084***	-0.0053***	0.0019	-0.0067***	-0.0045***	0.0017			
Urban/Rural	-0.0012	0.0041**	0.0094***	-0.0009	0.0034**	0.0078***			
Geographic Clustering	-0.0342***	-0.0364***	-0.0133*	-0.0306***	-0.0304***	-0.0079			
Maternal Height	-0.0155***	-0.0124***	0.0037***	-0.0118***	-0.0087***	0.0026***			
Baseline Covariates		0.0050***	0.0050***	0.0013	0.0013				
Outcome: Wasted (n = 406,416)									
Total Impact									
Nonlinearity	-0.0415***	-0.0364***	0.0046	-0.0378***	-0.0350***	0.0024	-0.0149***	0.0024	-0.0173***
Education of Other Parent	-0.0066	-0.0094**	-0.0029	0.0024	-0.0201***				
HH Living Standards	-0.0350***	-0.0269***	0.0075	-0.0402***	-0.0197***				
Fertility	-0.0064*	0.0015	0.0079	-0.0101***	0.0021				
Health Care Use	-0.0020	-0.0054**	-0.0035	-0.0063***	-0.0040**				
Urban/Rural	-0.0136***	-0.0040*	0.0092***	-0.0105***	-0.0035*				
Geographic Clustering	-0.0014	0.0001	0.0020	-0.0013	0.0000				
Maternal Height	-0.0026**	-0.0019*	0.0003	-0.0021**	-0.0015*				
Baseline Covariates		0.0002	0.0003	0.0002	-0.0000				

Notes: See Tables 3 and 4 in the manuscript.

Table A6. Sensitivity analysis: linear regressions with continuous nutrition outcomes

	Maternal education			Paternal education			
	Oldest	Newest	Change	Oldest	Newest	Change	
Outcome: height-for-age Z-score (n = 406,416)							
Coefficient: basic model	0.0839***	0.0748***	-0.0091***	0.0651***	0.0616***	-0.0036*	
Coefficient: full model	0.0095***	0.0050	-0.0045	0.0104***	0.0125***	0.0021	
Total impact	0.0744***	0.0698***	-0.0046	0.0547***	0.0490***	-0.0057**	
Nonlinearity	0.0032	0.0035	0.0003	-0.0028	-0.0029	-0.0001	
Education of Other Parent	0.0050***	0.0061***	0.0015	0.0067***	0.004***	-0.0024	
HH living standards	0.0265***	0.0225***	-0.0034	0.0216***	0.0194***	-0.0030	
Fertility	0.0080***	0.0065***	-0.0010	0.0057***	0.0050***	-0.0009	
Health care use	0.0047***	0.0020***	-0.0017**	0.0036***	0.0016***	-0.0014**	
Urban/rural	0.0015	-0.0048***	-0.0063***	0.0012	-0.0041***	-0.0053***	
Geographic clustering	0.0142***	0.0229***	0.0082***	0.0098***	0.0168***	0.0070***	
Maternal height	0.0113***	0.0112***	-0.0005	0.0089***	0.0087***	-0.0004	
Baseline covariates			-0.0016			0.0007	
Outcome: weight-for-age Z-score (n = 406,416)							
Coefficient: basic model	0.0695***	0.0597***	-0.0098***	0.0554***	0.0490***	-0.0064***	
Coefficient: full model	0.0075***	0.0033	-0.0041*	0.0124***	0.0116***	-0.0008	
Total impact	0.0621***	0.0564***	-0.0057**	0.0429***	0.0373***	-0.0056***	
Nonlinearity	0.0042	0.0046	0.0004	-0.0038	-0.0040	-0.0001	
Education of Other Parent	0.0056***	0.0050***	-0.0000	0.0062***	0.0045***	-0.0021	
HH living standards	0.0214***	0.0178***	-0.0032*	0.0175***	0.0153***	-0.0028*	
Fertility	0.0048***	0.0034***	-0.0010	0.0036***	0.0027***	-0.0009	
Health care use	0.0043***	0.0026***	-0.0008	0.0033***	0.0021***	-0.0007	
Urban/rural	0.0017	-0.0033***	-0.0049***	0.0013	-0.0028***	-0.0041***	
Geographic clustering	0.0125***	0.0188***	0.0054**	0.0090***	0.0136***	0.0036	
Maternal height	0.0075***	-0.0002	0.0059***		0.0058***	-0.0002	

Baseline covariates	Outcome: weight-for-height Z-score (n = 406,416)			
				-0.0012
Coefficient: basic model	0.0284***	0.0229***	-0.0055***	0.0240***
Coefficient: full model	0.0021	-0.0002	-0.0023	0.0087***
Decomposition: Covariates' Impact on Basic Coefficient				0.0063**
Total impact	0.0263***	0.0232***	-0.0032	0.0153***
Nonlinearity	0.0034	0.0037	0.0003	-0.0030
Education of Other Parent	0.0038***	0.0025*	-0.0009	0.0029***
HH living standards	0.0085***	0.0071***	-0.0012	0.0070***
Fertility	0.0004	-0.0003	-0.0005	0.0004
Health care use	0.0021***	0.0020***	0.0003	0.0016***
Urban/rural	0.0011	-0.0008	-0.0018	0.0009
Geographic clustering	0.0058***	0.0075***	0.0007	0.0046***
Maternal height	0.0012***	0.0015***	0.0002	0.0010***
Baseline covariates			-0.0001	0.0021***

Notes: See Tables 3 and 4 in the manuscript.

Table A7. Sensitivity analysis: average marginal effects from logit regressions

Outcome	Oldest	Change
Maternal education		
Mortality	-0.0044*** (0.0002)	0.0017*** (0.0003)
Stunted	-0.0220*** (0.0004)	0.0020*** (0.0006)
Underweight	-0.0135*** (0.0004)	0.0018*** (0.0005)
Wasted	-0.0033*** (0.0002)	0.0007** (0.0003)
Paternal education		
Mortality	-0.0031*** (0.0002)	0.0010*** (0.0003)
Stunted	-0.0163*** (0.0004)	0.0002 (0.0005)
Underweight	-0.0103*** (0.0003)	0.0005 (0.0004)
Wasted	-0.0026*** (0.0002)	0.0005* (0.0003)

Notes: *P < .10; ** p < .05; *** p < .01. 2 sided. All estimates are weighted and standard errors in parentheses adjusted for clustering at the PSU level. Average marginal effects from logit models. All coefficients are for an increase in the probability of experiencing an outcome to a single year increase in parental education. Controls: months since birth; sex, year of birth; maternal age at survey; survey. Note that independent variables were not demeaned as in linear models, but include a dummy coded survey controls.

Table A8. Sensitivity analysis: linear regressions, globally representative sampling weights

	Maternal education			Paternal education			
	Oldest	Newest	Change	Oldest	Newest	Change	Change
Outcome: mortality (n = 495,597)							
Coefficient: basic model	-0.0043***	-0.0025***	0.0018***	-0.0033***	-0.0021***	0.0011***	
Coefficient: full model	-0.0018**	-0.0005	0.0013**	0.0002	0.0005	0.0004	
Total impact	-0.0025***	-0.0020***	0.0005	-0.0034***	-0.0027***	0.0008**	
Nonlinearity	0.0002	0.0002	0.0000	-0.0010	-0.0010	-0.0000	
Education of Other Parent	-0.0002	-0.0003	-0.0001	-0.0008***	-0.0002	0.0005*	
HH living standards	-0.0006*	0.0000	0.0006	-0.0005*	0.0000	0.0006*	
Fertility	-0.0012***	-0.0008***	0.0002	-0.0009***	-0.0007***	0.0001	
Health care use	0.0006***	-0.0000	-0.0005***	0.0004***	-0.0000	-0.0004***	
Urban/rural	-0.0001	0.0001	0.0002	-0.0001	0.0001	0.0002	
Geographic clustering	-0.0010***	-0.0010***	-0.0001	-0.0005*	-0.0008***	-0.0004	
Maternal height	-0.0002***	-0.0001***	0.0001	-0.0001***	-0.0001***	0.0000	
Baseline covariates			0.0002			0.0002	
Outcome: stunted (n = 406,416)							
Coefficient: basic model	-0.0250***	-0.0190***	0.0060***	-0.0194***	-0.0157***	0.0037***	
Coefficient: full model	-0.0048***	-0.0045***	0.0003	-0.0028*	-0.0014	0.0014	
Total impact	-0.0202***	-0.0145***	0.0057***	-0.0166***	-0.0143***	0.0023***	
Nonlinearity	0.0022*	0.0024*	0.0002*	-0.0010	-0.0010	-0.0000	
Education of Other Parent	-0.0032***	-0.0018**	0.0016*	-0.0016***	-0.0012***	0.0006	
HH living standards	-0.0092***	-0.0064***	0.0024***	-0.0076***	-0.0056***	0.0022***	
Fertility	-0.0015***	-0.0010***	0.0007**	-0.0012***	-0.0007***	0.0005**	
Health care use	-0.0013***	-0.0008***	0.0002	-0.0009***	-0.0007***	0.0002	
Urban/rural	0.0004	0.0013***	0.0009**	0.0003	0.0011***	0.0007**	
Geographic clustering	-0.0053***	-0.0061***	-0.0019*	-0.0026***	-0.0044***	-0.0020**	
Maternal height	-0.0024***	-0.0022***	0.0005***	-0.0021***	-0.0018***	0.0004***	

Baseline covariates		Outcome: underweight (n = 406,416)		0.0010**		-0.0002	
Coefficient: basic model	-0.0241***	-0.0143***	0.0098***	-0.0183***	-0.0120***	0.0063***	
Coefficient: full model	-0.0057***	-0.0037***	0.0020*	-0.0015	-0.0013	0.0001	
Decomposition: Covariates' Impact on Basic Coefficient							
Total impact	-0.0185***	-0.0106***	0.0078***	-0.0168***	-0.0107***	0.0061***	
Nonlinearity	0.0012	0.0014	0.0001	-0.0015	-0.0016	-0.0001	
Education of Other Parent	-0.0019***	-0.0015***	0.0004	-0.0024***	-0.0010***	0.0014**	
HH living standards	-0.0076***	-0.0041***	0.0032***	-0.0064***	-0.0035***	0.0029***	
Fertility	-0.0008***	-0.0005***	0.0006*	-0.0007***	-0.0004***	0.0004*	
Health care use	-0.0010***	-0.0003	0.0005*	-0.0007***	-0.0002	0.0004	
Urban/rural	0.0002	0.0020***	0.0018***	0.0002	0.0017***	0.0015***	
Geographic clustering	-0.0066***	-0.0063***	0.0002	-0.0037***	-0.0046***	-0.0002	
Maternal height	-0.0019***	-0.0014***	0.0008***	-0.0016***	-0.0011***	0.0006***	
Baseline covariates		0.0003	0.0003	-0.0008	-0.0008	-0.0008	
Outcome: wasted (n = 406,416)							
Coefficient: basic model	-0.0080***	-0.0046***	0.0035***	-0.0058***	-0.0038***	0.0020***	
Coefficient: full model	-0.0022*	-0.0012	0.0010	0.0010	0.0012	0.0002	
Decomposition: Covariates' Impact on Basic Coefficient							
Total impact	-0.0058***	-0.0033***	0.0025***	-0.0068***	-0.0050***	0.0018***	
Nonlinearity	-0.0003	-0.0003	-0.0000	-0.0016	-0.0016	-0.0001	
Education of Other Parent	-0.0001	-0.0001	0.0000	-0.0013***	-0.0007**	0.0006	
HH living standards	-0.0029***	-0.0011***	0.0017***	-0.0025***	-0.0009***	0.0016**	
Fertility	0.0002	0.0000	0.0000	0.0001	-0.0000	-0.0001	
Health care use	-0.0001	-0.0001	-0.0000	-0.0001	-0.0001	-0.0000	
Urban/rural	0.0001	0.0011***	0.0009***	0.0001	0.0009***	0.0008***	
Geographic clustering	-0.0024***	-0.0028***	-0.0002	-0.0014***	-0.0024***	-0.0004	
Maternal height	-0.0003***	-0.0002***	0.0002*	-0.0003***	-0.0001***	0.0001*	
Baseline covariates		-0.0001	-0.0001	-0.0006*	-0.0006*	-0.0006*	

Notes: See Tables 3 and 4 in the manuscript.

Table A9. Sensitivity analysis: linear regressions, without sampling weights

	Maternal education			Paternal education		
	Oldest	Newest	Change	Oldest	Newest	Change
Outcome: mortality (n = 495,597)						
Coefficient: basic model	-0.0042***	-0.0022***	0.0020***	-0.0034***	-0.0019***	0.0015***
Coefficient: full model	-0.0007**	0.0001	0.0009***	-0.0003	0.0001	0.0004*
Decomposition: covariates' impact on basic coefficient						
Total impact	-0.0035***	-0.0023***	0.0012***	-0.0031***	-0.0020***	0.0011***
Nonlinearity	-0.0003	-0.0004	-0.0000	-0.0005*	-0.0006*	-0.0000*
Education of other parent	-0.0003	-0.0002	-0.0000	-0.0004***	-0.0001	0.0003*
HH living standards	-0.0005***	-0.0001	0.0003	-0.0004***	-0.0001	0.0003
Fertility	-0.0014***	-0.0010***	0.0001	-0.0011***	-0.0008***	0.0001*
Health care use	0.0002**	0.0000	-0.0001*	0.0001**	0.0000	-0.0001*
Urban/rural	0.0001*	0.0000	-0.0001	0.0001*	0.0000	-0.0001
Geographic clustering	-0.0011***	-0.0006***	0.0006**	-0.0007***	-0.0004***	0.0001
Maternal height	-0.0002***	-0.0001***	0.0001*	-0.0002***	-0.0001***	0.0000*
Baseline covariates			0.0004***			0.0000***
Outcome: stunted (n = 406,416)						
Coefficient: basic model	-0.0227***	-0.0188***	0.0039***	-0.0183***	-0.0155***	0.0028***
Coefficient: full model	-0.0046***	-0.0043***	0.0003	-0.0036***	-0.0033***	0.0003
Decomposition: covariates' impact on basic coefficient						
Total impact	-0.0181***	-0.0145***	0.0036***	-0.0147***	-0.0122***	0.0025***
Nonlinearity	0.0011*	0.0012*	0.0001*	0.0009	0.0010	0.0000
Education of other parent	-0.0020***	-0.0015***	0.0004	-0.0020***	-0.0018***	0.0002
HH living standards	-0.0079***	-0.0058***	0.0018***	-0.0067***	-0.0050***	0.0016***
Fertility	-0.0018***	-0.0012***	0.0004**	-0.0014***	-0.0009***	0.0003***
Health care use	-0.0015***	-0.0005***	0.0006***	-0.0012***	-0.0005***	0.0005***
Urban/rural	0.0004*	0.0005**	0.0001	0.0003*	0.0004***	0.0001
Geographic clustering	-0.0036***	-0.0045***	-0.0011*	-0.0023***	-0.0033***	-0.0012**
Maternal height	-0.0028***	-0.0026***	0.0004***	-0.0024***	-0.0021***	0.0003***

Baseline covariates		Outcome: underweight (n = 406,416)		0.009***		0.0006***	
Coefficient: basic model	-0.0158***	-0.0126***	0.0032***	-0.0132***	-0.0109***	0.0024***	
Coefficient: full model	-0.0045***	-0.0037***	0.0007	-0.0042***	-0.0038***	0.0004	
Decomposition: covariates' impact on basic coefficient							
Total impact	-0.0113***	-0.0088***	0.0025***	-0.0090***	-0.0071***	0.0019***	
Nonlinearity	0.0015***	0.0016***	0.0011***	0.0015***	0.0016***	0.0000***	
Education of other parent	-0.0018***	-0.0014***	0.0003	-0.0016***	-0.0012***	0.0004	
HH living standards	-0.0053***	-0.0038***	0.0014***	-0.0045***	-0.0033***	0.0012***	
Fertility	-0.0009***	-0.0005***	0.0003*	-0.0007***	-0.0004***	0.0002*	
Health care use	-0.0011***	-0.0004***	0.0004***	-0.0009***	-0.0004***	0.0004***	
Urban/rural	0.0005**	0.0009***	0.0004**	0.0004**	0.0007***	0.0004**	
Geographic clustering	-0.0024***	-0.0037***	-0.0010*	-0.0017***	-0.0029***	-0.0009*	
Maternal height	-0.0018***	-0.0015***	0.0004***	-0.0015***	-0.0012***	0.0003***	
Baseline covariates		0.0002			-0.0000		
Outcome: wasted (n = 406,416)							
Coefficient: basic model	-0.0042***	-0.0036***	0.0006**	-0.0034***	-0.0033***	0.0001	
Coefficient: full model	-0.0016***	-0.0011***	0.0005	-0.0014***	-0.0013***	0.0000	
Decomposition: covariates' impact on basic coefficient							
Total impact	-0.0026***	-0.0024***	0.0002	-0.0021***	-0.0020***	0.0001	
Nonlinearity	0.0002	0.0003	0.0000	0.0006	0.0007	0.0000	
Education of other parent	-0.0004**	-0.0003*	0.0001	-0.0007***	-0.0004***	0.0003	
HH living standards	-0.0017***	-0.0011***	0.0005*	-0.0015***	-0.0010***	0.0005*	
Fertility	0.0000	-0.0000	-0.0001	-0.0000	-0.0001	-0.0001	
Health care use	-0.0002**	-0.0001	0.0001	-0.0002*	-0.0001	0.0001	
Urban/rural	0.0004***	0.0005**	0.0002	0.0003***	0.0004***	0.0001	
Geographic clustering	-0.0006*	-0.0014***	-0.0006	-0.0004*	-0.0014***	-0.0007*	
Maternal height	-0.0002***	-0.0002***	0.0000	-0.0002***	-0.0002***	0.0000	
Baseline covariates		-0.0001			-0.0002*		

Notes: See Tables 3 and 4 in the manuscript.

Table A10. Sensitivity analysis: linear regressions, including children with missing covariates

Outcome	Oldest	Change
Maternal education		
Mortality	-0.0047***	0.0027***
n = 719,866	(0.0002)	(0.0002)
Stunted	-0.0219***	0.0036***
n = 603,379	(0.0002)	(0.0005)
Underweight	-0.0129***	0.0031***
n = 605,205	(0.0003)	(0.0004)
Wasted	-0.0030***	0.0007***
n = 603,316	(0.0002)	(0.0003)
Paternal education		
Mortality	-0.0038***	0.0021***
n = 515,348	(0.0002)	(0.0002)
Stunted	-0.0169***	0.0016***
n = 425,695	(0.0004)	(0.0005)
Underweight	-0.0109***	0.0024***
n = 426,977	(0.0003)	(0.0004)
Wasted	-0.0027***	0.0006**
n = 425,656	(0.0002)	(0.0003)

Notes: *P < .10; ** p < .05; *** p < .01. 2 sided. All estimates are weighted and standard errors in parentheses adjusted for clustering at the PSU level. All coefficients are for an increase in the linear probability of experiencing an outcome to a single year increase in parental education. Controls: months since birth; sex, year of birth; maternal age at survey; survey. All independent variables were demeaned from a survey-specific mean of included observations.

Table A11. Sensitivity analysis: multinomial selection models

Outcome	Oldest	Change
Maternal education		
Stunted	-0.0199*** (0.0005)	0.0024*** (0.0005)
n = 598,159		
Underweight	-0.0119*** (0.0004)	0.0024*** (0.0004)
n = 599,995		
Wasted	-0.0028*** (0.0002)	0.0007** (0.0003)
n = 598,093		
Height-for-age z-score	0.0755*** (0.0017)	-0.0075*** (0.0020)
n = 598,159		
Weight-for-height z-score	0.0610*** (0.0014)	-0.0068*** (0.0017)
n = 599,995		
Weight-for-height z-score	0.0237*** (0.0014)	-0.0030* (0.0017)
n = 598,093		
Paternal education		
Stunted	-0.0152*** (0.0004)	0.0007 (0.0005)
n = 420,057		
Underweight	-0.0101*** (0.0003)	0.0018*** (0.0004)
n = 421,347		
Wasted	-0.0025*** (0.0002)	0.0005** (0.0003)
n = 420,012		
Height-for-age z-score	0.0585*** (0.0016)	-0.0026 (0.0020)
n = 420,057		
Weight-for-height z-score	0.0489*** (0.0013)	-0.0042*** (0.0015)
n = 421,347		
Weight-for-height z-score	0.0201*** (0.0013)	-0.0031** (0.0015)
n = 420,012		

Notes: *P < .10; ** p < .05; *** p < .01. 2 sided. All estimates are weighted and standard errors in parentheses adjusted for clustering at the PSU level. Average marginal effects from logit models. All coefficients are for an increase in the probability of experiencing an outcome to a single year increase in parental education. Controls: months since birth; sex, year of birth; maternal age at survey; survey. Note that independent variables were NOT demeaned as in linear models, but include a dummy coded survey controls. First step selection models; multinomial models to obtain the predicted probability of being measured and included in the sample. Three categories of selection out of the sample are used; mortality, flagged because of inconsistency, and missing for other reasons. It uses the same independent variables as in the main model in addition to weekday of survey, day of field work the household was interviewed, number of visits, and sibling deaths. Standard errors should be interpreted with caution as they have not been adjusted for two-step procedure.

Table A12. Sensitivity analysis: multinomial selection models excluding mortality

Outcome	Oldest	Change
	Maternal education	
Stunted	-0.0228***	0.0038***
n = 598,159	(0.0004)	(0.0005)
Underweight	-0.0132***	0.0030***
n = 599,995	(0.0003)	(0.0004)
Wasted	-0.0026***	0.0006**
n = 598,093	(0.0002)	(0.0003)
Height-for-age z-score	0.0847***	-0.0121***
n = 598,159	(0.0016)	(0.0020)
Weight-for-height z-score	0.0653***	-0.0088***
n = 599,995	(0.0013)	(0.0017)
Weight-for-height z-score	0.0219***	-0.0023
n = 598,093	(0.0013)	(0.0017)
Paternal education		
Stunted	-0.0177***	0.0020***
n = 420,057	(0.0004)	(0.0005)
Underweight	-0.0112***	0.0023***
n = 421,347	(0.0003)	(0.0004)
Wasted	-0.0024***	0.0005*
n = 420,012	(0.0002)	(0.0003)
Height-for-age z-score	0.0667***	-0.0068***
n = 420,057	(0.0016)	(0.0020)
Weight-for-height z-score	0.0529***	-0.0062***
n = 421,347	(0.0012)	(0.0016)
Weight-for-height z-score	0.0192***	-0.0028*
n = 420,012	(0.0012)	(0.0016)

Notes: *P < .10; ** p < .05; *** p < .01. 2 sided. All estimates are weighted and standard errors in parentheses adjusted for clustering at the PSU level. Average marginal effects from logit models. All coefficients are for an increase in the probability of experiencing an outcome to a single year increase in parental education. Controls: months since birth; sex, year of birth; maternal age at survey; survey. Note that independent variables were NOT demeaned as in linear models, but include a dummy coded survey controls. First step selection models; multinomial models to obtain the predicted probability of being measured and included in the sample. Two categories of selection out of the sample are used; flagged because of inconsistency, and missing for other reasons. Those who died are excluded. It uses the same independent variables as in the main model in addition to weekday of survey, day of field work the household was interviewed, and number of visits. Standard errors should be interpreted with caution as they have not been adjusted for two-step procedure.

Table A13. Sensitivity analysis: linear regressions for maternal education, including single mothers and mothers married more than once

		Maternal Education					
		Oldest	Newest	Change	Oldest	Newest	Change
		Outcome: Mortality (n = 835,353)			Outcome: Underweight (n = 687,420)		
Coefficient: Basic Model		-0.0048*** (0.0002)	-0.0023*** (0.0001)	0.0025*** (0.0002)	-0.0139*** (0.0003)	-0.0103*** (0.0002)	0.0036*** (0.0004)
Coefficient: Full Model		-0.0008* (0.0004)	-0.0002 (0.0004)	0.0006 (0.0004)	-0.0045*** (0.0007)	-0.0035*** (0.0006)	0.0009 (0.0006)
		Decomposition: Covariates' Impact on Basic Coefficient					
Total Impact		-0.0041*** (0.0004)	-0.0022*** (0.0004)	0.0019*** (0.0003)	-0.0094*** (0.0006)	-0.0067*** (0.0006)	0.0027*** (0.0005)
Nonlinearity		-0.0004 (0.0003)	-0.0005 (0.0004)	-0.0000 (0.0000)	0.0011** (0.0005)	0.0012** (0.0006)	0.00011** (0.0001)
Education of Other Parent		-0.0003 (0.0002)	0.0002* (0.0001)	0.0005* (0.0002)	-0.0005 (0.0003)	-0.0001 (0.0002)	0.0002 (0.0003)
HH Living Standards		-0.0004** (0.0002)	-0.0000 (0.0002)	0.0003 (0.0003)	-0.0041*** (0.0003)	-0.0032*** (0.0003)	0.0009** (0.0004)
Fertility		-0.0015*** (0.0001)	-0.0010*** (0.0001)	0.0001 (0.0001)	-0.0010*** (0.0001)	-0.0006*** (0.0001)	0.0003* (0.0002)
Health Care Use		0.0000 (0.0001)	-0.0001* (0.0001)	-0.0001 (0.0001)	-0.0008*** (0.0001)	-0.0004*** (0.0002)	0.0002 (0.0001)
Urban/Rural		-0.0002* (0.0001)	0.0002*** (0.0001)	0.0004*** (0.0001)	-0.0000 (0.0001)	0.0007*** (0.0002)	0.0007*** (0.0003)
Geographic Clustering		-0.0012*** (0.0002)	-0.0009*** (0.0002)	0.0003 (0.0003)	-0.0027*** (0.0004)	-0.0031*** (0.0003)	-0.0004 (0.0005)
Maternal Height		-0.0002*** (0.0001)	-0.0001*** (0.0001)	0.0001 (0.0001)	-0.0016*** (0.0001)	-0.0013*** (0.0003)	0.0003*** (0.0003)

	Outcome: Stunted (n = 687,420)		Outcome: Wasted (n = 687,420)	
Baseline Covariates	(0.0000)	(0.0000)	(0.0001)	(0.0001)
Marital Status	0.0001*** (0.0000)	0.0000** (0.0000)	-0.0000 (0.0000)	0.0001** (0.0000)
			0.0000 (0.0000)	-0.0000 (0.0000)
			0.0005*** (0.0001)	0.0005*** (0.0002)
Coefficient: Basic Model	-0.0228*** (0.0004)	-0.0196*** (0.0003)	0.0032*** (0.0005)	-0.0036*** (0.0002)
				0.0009*** (0.0003)
Coefficient: Full Model	-0.0056*** (0.0008)	-0.0043*** (0.0008)	0.0013* (0.0007)	-0.0015*** (0.0005)
				-0.0009* (0.0004)
				0.0007 (0.0004)
Decomposition: Covariates' Impact on Basic Coefficient				
Total Impact	-0.0173*** (0.0008)	-0.0154*** (0.0008)	0.0019*** (0.0007)	-0.0021*** (0.0004)
				-0.0019*** (0.0004)
Nonlinearity	0.0006	0.0007	0.0001	0.0002
				0.0002
Education of Other Parent	-0.0008** (0.0007)	-0.0006* (0.0008)	0.0000 (0.0001)	-0.0001 (0.0004)
				-0.0001 (0.0004)
HH Living Standards	-0.0075*** (0.0004)	-0.0062*** (0.0004)	0.0013** (0.0005)	-0.0012*** (0.0002)
				-0.0005*** (0.0003)
Fertility	-0.0022*** (0.0002)	-0.0016*** (0.0002)	0.0004* (0.0002)	-0.0002* (0.0002)
				-0.0000 (0.0003)
Health Care Use	-0.0013*** (0.0002)	-0.0008** (0.0001)	0.0003 (0.0002)	-0.0002** (0.0001)
				-0.0001** (0.0001)
Urban/Rural	0.0000	0.0010*** (0.0002)	0.0000 (0.0004)	0.0001 (0.0001)
				0.0001 (0.0002)
Geographic Clustering	-0.0034*** (0.0002)	-0.0052*** (0.0001)	-0.0017** (0.0005)	-0.0014*** (0.0008)
				-0.0008** (0.0004)

Maternal Height	(0.0005)	(0.0005)	(0.0007)	(0.0003)	(0.0002)
	-0.0029***	-0.0027***	0.0002***	-0.0001***	-0.0001***
	(0.0001)	(0.0001)	(0.0001)	(0.0000)	(0.0000)
Marital Status	0.0001***	0.0000	-0.0001*	0.0000	0.0000
	(0.0000)	(0.0000)	(0.0001)	(0.0000)	(0.0000)
Baseline Covariates		0.0004*		0.0001**	
			(0.0003)	(0.0001)	(0.0001)

Notes: Missing paternal education is adjusted for using dummy variable adjustment. *P < .10; ** p < .05; *** p < .01. 2 sided. All estimates are weighted and p-values adjusted for clustering at the PSU level. All coefficients are for an increase in the linear probability of experiencing the outcome to a single year increase in parental education. Basic model covariates: months since birth; sex; year of birth; maternal age at survey; a constant term. Full model covariates: basic covariates; marital status; household living standards (wealth index z-score); fertility (maternal age at birth, number of siblings ever born, birth interval, being first born, birth order, maternal age at first birth); health care use (vaccinated (surviving children only), born at home); urban-rural difference (rural resident); maternal height (indicating maternal early-life living standards); geographic clustering (PSU specific means of all independent variables, except for rural resident); education of partner (other parents years of education, interaction between mean-centered years of education of both parents); nonlinearity (squared term for parents' education). All independent variables were demeaned from a survey-specific mean of included observations. The "baseline covariates" set contains the terms for the main associations of covariates when decomposing the "change coefficient". When decomposing the "change coefficient" all sets (except "nonlinearity" and "baseline covariates") contain covariates interacted with being in the newest surveys. Parameters are from Eq1 and Eq2; newest is $\beta + \delta_b$; and change is δ_b . The decomposition of the "change coefficients" quantifies the changing impact of the respective sets of covariates (except "nonlinearity" and "baseline covariates") on the coefficients from the basic models. The decomposition of main coefficients from the oldest and newest surveys shows the impact of each set of covariates on the main association.

Table A14. Main results, including standard errors

	Maternal education			Paternal education			
	Oldest	Newest	Change	Oldest	Newest	Change	
Outcome: mortality (n = 495,597)							
Coefficient: basic model	-0.0048*** (0.0002)	-0.0021*** (0.0002)	0.0027*** (0.0002)	-0.0038*** (0.0001)	-0.0018*** (0.0001)	0.0020*** (0.0002)	
Coefficient: full model	-0.0003 (0.0005)	0.0004 (0.0005)	0.0008* (0.0004)	-0.0004 (0.0005)	0.0004 (0.0005)	0.0008** (0.0003)	
Decomposition: covariates' impact on basic coefficient							
Total impact	-0.0045*** (0.0005)	-0.0026*** (0.0004)	0.0019*** (0.0004)	-0.0034*** (0.0005)	-0.0021*** (0.0005)	0.0012** (0.0003)	
Nonlinearity	-0.0007* (0.0004)	-0.0008* (0.0005)	-0.001* (0.0005)	-0.0005 (0.0004)	-0.0005 (0.0005)	-0.0005 (0.0005)	-0.0000 (0.0000)
Education of other parent	-0.0001 (0.0002)	0.0001 (0.0002)	0.0001 (0.0003)	-0.0003* (0.0002)	0.0000 (0.0001)	0.0000 (0.0001)	0.0002 (0.0002)
HH living standards	-0.0006** (0.0002)	-0.0001 (0.0002)	0.0005 (0.0003)	-0.0005** (0.0002)	-0.0000 (0.0002)	-0.0000 (0.0002)	0.0004* (0.0003)
Fertility	-0.0017*** (0.0001)	-0.0010*** (0.0001)	0.0002 (0.0001)	-0.0013*** (0.0001)	-0.0009*** (0.0001)	0.0002** (0.0001)	
Health care use	0.0001 (0.0001)	-0.0000 (0.0001)	-0.0001 (0.0001)	0.0001 (0.0001)	-0.0000 (0.0001)	-0.0000 (0.0001)	-0.0001 (0.0001)
Urban/rural	-0.0002 (0.0001)	0.0002** (0.0001)	0.0003** (0.0001)	-0.0001 (0.0001)	0.0001** (0.0001)	0.0003** (0.0001)	
Geographic clustering	-0.0012*** (0.0003)	-0.0009*** (0.0002)	0.0003 (0.0004)	-0.0006*** (0.0002)	-0.0007*** (0.0002)	-0.0003 (0.0003)	
Maternal height	-0.0002*** (0.0000)	-0.0001*** (0.0000)	0.0001* (0.0000)	-0.0001*** (0.0000)	-0.0001*** (0.0000)	0.0000* (0.0000)	
Baseline covariates				0.0006*** (0.0001)	0.0005*** (0.0001)	0.0005*** (0.0001)	
Outcome: stunted (n = 406,416)							

	Coefficient: basic model	-0.0230*** (0.0004)	-0.0196*** (0.0004)	0.0034*** (0.0006)	-0.0177*** (0.0004)	-0.0162*** (0.0004)	0.0015*** (0.0005)
	Coefficient: full model	-0.0060*** (0.0010)	-0.0050*** (0.0010)	0.0010 (0.0009)	-0.0038*** (0.0010)	-0.0044*** (0.0010)	-0.0005 (0.0007)
Decomposition: covariates' impact on basic coefficient							
Total impact		-0.0171*** (0.0010)	-0.0146*** (0.0010)	0.0025*** (0.0008)	-0.0138*** (0.0010)	-0.0118*** (0.0010)	0.0020*** (0.0006)
Nonlinearity		0.0022** (0.0009)	0.0023** (0.0010)	0.0002** (0.0001)	0.0018* (0.0009)	0.0018* (0.0009)	0.0001* (0.0000)
Education of other parent		-0.0013*** (0.0005)	-0.0015*** (0.0004)	-0.0004 (0.0006)	-0.0020*** (0.0003)	-0.0014*** (0.0003)	0.0005 (0.0005)
HH living standards		-0.0079*** (0.0005)	-0.0063*** (0.0004)	0.0015** (0.0006)	-0.0065*** (0.0004)	-0.0054*** (0.0004)	0.0013** (0.0005)
Fertility		-0.0021*** (0.0002)	-0.0014** (0.0002)	0.0007** (0.0003)	-0.0015** (0.0002)	-0.0011*** (0.0001)	0.0005*** (0.0002)
Health care use		-0.0013*** (0.0002)	-0.0008*** (0.0002)	0.0002 (0.0002)	-0.0010*** (0.0002)	-0.0007*** (0.0001)	0.0002 (0.0002)
Urban/rural		-0.0001 (0.0003)	0.0011** (0.0003)	0.0012*** (0.0004)	-0.0001 (0.0003)	0.0009*** (0.0002)	0.0010*** (0.0003)
Geographic clustering		-0.0036*** (0.0006)	-0.0054** (0.0005)	-0.0018** (0.0008)	-0.0023** (0.0005)	-0.0040*** (0.0005)	-0.0020*** (0.0007)
Maternal height		-0.0029*** (0.0001)	-0.0026*** (0.0001)	0.0003*** (0.0001)	-0.0022*** (0.0001)	-0.0020*** (0.0001)	0.0002*** (0.0001)
Baseline covariates				0.0006* (0.0003)	0.0006* (0.0003)	0.0002 (0.0003)	0.0002 (0.0003)
Outcome: underweight ($n = 406,416$)							
Coefficient: basic model		-0.0142*** (0.0003)	-0.0101** (0.0003)	0.0042*** (0.0004)	-0.0118*** (0.0003)	-0.0090*** (0.0003)	0.0028*** (0.0004)
Coefficient: full model		-0.0044*** (0.0008)	-0.0029*** (0.0008)	0.0015** (0.0007)	-0.0045*** (0.0008)	-0.0042*** (0.0008)	0.0002 (0.0005)

Decomposition: covariates' impact on basic coefficient						
Total impact	-0.0099*** (0.0008)	-0.0072** (0.0007)	0.0027*** (0.0006)	-0.0074*** (0.0008)	-0.0048*** (0.0008)	0.0026*** (0.0005)
Nonlinearity	0.0016** (0.0007)	0.0018** (0.0007)	0.0011** (0.0001)	0.0017** (0.0007)	0.0017** (0.0007)	0.0001** (0.0000)
Education of other parent	-0.0013*** (0.0003)	-0.0012*** (0.0003)	-0.0001 (0.0004)	-0.0011*** (0.0004)	-0.0002 (0.0002)	0.0007*** (0.0003)
HH living standards	-0.0040*** (0.0004)	-0.0030** (0.0003)	0.0010** (0.0005)	-0.0033*** (0.0003)	-0.0026*** (0.0003)	0.0009** (0.0004)
Fertility	-0.0009*** (0.0002)	-0.0004*** (0.0001)	0.0004** (0.0002)	-0.0007*** (0.0001)	-0.0003*** (0.0001)	0.0003*** (0.0002)
Health care use	-0.0008*** (0.0002)	-0.0004*** (0.0001)	0.0002 (0.0002)	-0.0006*** (0.0002)	-0.0004*** (0.0001)	0.0001 (0.0001)
Urban/rural	-0.0003 (0.0002)	0.0006** (0.0002)	0.0009*** (0.0003)	-0.0002 (0.0002)	0.0005*** (0.0002)	0.0007*** (0.0003)
Geographic clustering	-0.0026*** (0.0005)	-0.0033*** (0.0004)	-0.0007 (0.0006)	-0.0018*** (0.0004)	-0.0025*** (0.0004)	-0.0008 (0.0006)
Maternal height	-0.0016*** (0.0001)	-0.0013*** (0.0001)	0.0004*** (0.0001)	-0.0013*** (0.0001)	-0.0010*** (0.0001)	0.0003*** (0.0001)
Baseline covariates			0.0005* (0.0002)	0.0003 (0.0002)	0.0003 (0.0002)	0.0003 (0.0002)
Outcome: wasted (n = 406,416)						
Coefficient: basic model	-0.0038*** (0.0002)	-0.0028** (0.0002)	0.0009*** (0.0003)	-0.0031*** (0.0002)	-0.0025*** (0.0002)	0.0006** (0.0003)
Coefficient: full model	-0.0015** (0.0006)	-0.0010* (0.0006)	0.0005 (0.0005)	-0.0015** (0.0005)	-0.0013** (0.0006)	0.0001 (0.0004)
Total impact	-0.0023*** (0.0005)	-0.0018*** (0.0006)	0.0004 (0.0004)	-0.0017*** (0.0006)	-0.0012** (0.0006)	0.0005 (0.0003)
Nonlinearity	0.0004	0.0005	0.0000	0.0005	0.0005	0.0000

	(0.0005)	(0.0000)	(0.0005)	(0.0005)	(0.0005)
Education of other parent	-0.0005*	0.0001	-0.0005**	-0.0001	0.0004
	(0.0002)	(0.0002)	(0.0003)	(0.0002)	(0.0003)
HH living standards	-0.0012***	0.0008**	-0.0010***	-0.0003	0.0007***
	(0.0003)	(0.0002)	(0.0003)	(0.0002)	(0.0003)
Fertility	-0.0001	0.0000	0.0001	-0.0000	0.0001
	(0.0001)	(0.0001)	(0.0002)	(0.0001)	(0.0001)
Health care use	-0.0002**	0.0000	-0.0002**	-0.0001*	0.0000
	(0.0001)	(0.0001)	(0.0001)	(0.0001)	(0.0001)
Urban/rural	0.0000	0.0001	0.0000	0.0000	0.0000
	(0.0001)	(0.0001)	(0.0002)	(0.0001)	(0.0002)
Geographic clustering	-0.0006*	-0.0014***	-0.0008*	-0.0004	-0.0011***
	(0.0003)	(0.0003)	(0.0004)	(0.0003)	(0.0004)
Maternal height	-0.0001***	-0.0002**	-0.0000	-0.0001***	-0.0000
	(0.0000)	(0.0000)	(0.0001)	(0.0000)	(0.0000)
Baseline covariates		0.0001	0.0001	0.0000	0.0000
		(0.0001)	(0.0001)		(0.0001)

Notes: See Tables 3 and 4 in the manuscript.

Table A15a. Main results for maternal education, including exact p-values and 95% confidence intervals

	Maternal Education			Change			p	CI (95%)
	Oldest	Est.	p	Cl (95%)	Newest	Est.		
Outcome: Mortality (n=495,597)								
Coefficient: Basic Model	-0.0048	<0.0001	-0.0052, -0.0044	-0.0021	<0.0001	-0.0024, -0.0018	0.0027	<0.0001
Coefficient: Full Model	-0.0003	0.5083	-0.0014, 0.0007	0.0004	0.3368	-0.0005, 0.0014	0.0008	0.0522
Decomposition: covariates' impact on basic coefficient								
Total Impact	-0.0045	<0.0001	-0.0054, -0.0036	-0.0026	<0.0001	-0.0034, -0.0017	0.0019	<0.0001
Nonlinearity	-0.0007	0.0934	-0.0015, 0.0001	-0.0008	0.0934	-0.0017, 0.0001	-0.0001	0.0959
Education of Other Parent	-0.0001	0.5459	-0.0006, 0.0003	0.0001	0.3967	-0.0002, 0.0005	0.0001	0.6648
HH Living Standards	-0.0006	0.0146	-0.0010, -0.0001	-0.0001	0.7793	-0.0004, 0.0003	0.0005	0.1143
Fertility	-0.0017	<0.0001	-0.0019, -0.0015	-0.0001	<0.0001	-0.0012, -0.0008	0.0002	0.1125
Health Care Use	0.0001	0.248	-0.0001, 0.0003	0	0.53	-0.0002, 0.0001	-0.0001	0.1954
Urban/Rural	-0.0002	0.1508	-0.0004, 0.0001	0.0002	0.0464	0.0000, 0.0003	0.0003	0.0201
Geographic Clustering	-0.0012	<0.0001	-0.0017, -0.0006	-0.0009	0.0001	-0.0013, -0.0004	0.0003	0.3589
Maternal Height	-0.0002	<0.0001	-0.0003, -0.0001	-0.0001	0.0001	-0.0002, -0.0001	0.0001	0.0931
Baseline Covariates	0	0.5277	-0.0000, 0.0000	0	0.677	-0.0000, 0.0000	0.0006	<0.0001
Outcome: Stunted (n=406,416)								
Coefficient: Basic Model	-0.023	<0.0001	-0.0239, -0.0222	-0.0196	<0.0001	-0.0204, -0.0189	0.0034	<0.0001
Coefficient: Full Model	-0.006	<0.0001	-0.0080, -0.0040	-0.005	<0.0001	-0.0071, -0.0030	0.001	0.268
Decomposition: covariates' impact on basic coefficient								
Total Impact	-0.0171	<0.0001	-0.0190, -0.0151	-0.0146	<0.0001	-0.0165, -0.0126	0.0025	0.0016
Nonlinearity	0.0022	0.0188	0.0004, 0.0040	0.0023	0.0187	0.0004, 0.0043	0.0002	0.0199

Education of Other Parent	-0.0013	0.0054	-0.0021, -0.0004	-0.0015	0.0001	-0.0022, -0.0008	-0.0004	0.5205	-0.0015, 0.0007							
HH Living Standards	-0.0079	<0.0001	-0.0088, -0.0070	-0.0063	<0.0001	-0.0072, -0.0055	0.0015	0.0187	0.0002, 0.0027							
Fertility	-0.0021	<0.0001	-0.0025, -0.0017	-0.0014	<0.0001	-0.0017, -0.0010	0.0007	0.0133	0.0001, 0.0012							
Health Care Use	-0.0013	<0.0001	-0.0017, -0.0009	-0.0008	<0.0001	-0.0011, -0.0005	0.0002	0.3017	-0.0002, 0.0007							
Urban/Rural	-0.0001	0.7642	-0.0007, 0.0005	0.0011	<0.0001	0.0006, 0.0016	0.0012	0.0031	0.0004, 0.0020							
Geographic Clustering	-0.0036	<0.0001	-0.0048, -0.0025	-0.0054	<0.0001	-0.0065, -0.0043	-0.0018	0.0338	-0.0034, -0.0001							
Maternal Height	-0.0029	<0.0001	-0.0031, -0.0026	-0.0026	<0.0001	-0.0028, -0.0024	0.0003	0.0013	0.0001, 0.0005							
Baseline Covariates	0	0.2504	-0.0000, 0.0000	0	0.2233	-0.0000, 0.0000	0.0006	0.0565	-0.0000, 0.0011							
Outcome: Underweight(n=406,416)																
Coefficient: Basic Model	-0.0142	<0.0001	-0.0149, -0.0136	-0.0101	<0.0001	-0.0106, -0.0095	0.0042	<0.0001	0.0033, 0.0051							
Coefficient: Full Model	-0.0044	<0.0001	-0.0060, -0.0027	-0.0029	0.0003	-0.0044, -0.0013	0.0015	0.0313	0.0001, 0.0028							
Decomposition: covariates' impact on basic coefficient																
Total Impact	-0.0099	<0.0001	-0.0114, -0.0084	-0.0072	<0.0001	-0.0086, -0.0057	0.0027	<0.0001	0.0015, 0.0039							
Nonlinearity	0.0016	0.014	0.0003, 0.0029	0.0018	0.0141	0.0004, 0.0032	0.0001	0.0156	0.0000, 0.0003							
Education of Other Parent	-0.0013	<0.0001	-0.0020, -0.0007	-0.0012	<0.0001	-0.0017, -0.0006	-0.0001	0.8429	-0.0009, 0.0007							
HH Living Standards	-0.004	<0.0001	-0.0047, -0.0033	-0.003	<0.0001	-0.0036, -0.0024	0.001	0.0348	0.0001, 0.0019							
Fertility	-0.0009	<0.0001	-0.0013, -0.0006	-0.0004	0.0024	-0.0007, -0.0002	0.0004	0.0389	0.0000, 0.0009							
Health Care Use	-0.0008	<0.0001	-0.0011, -0.0004	-0.0004	0.0004	-0.0007, -0.0002	0.0002	0.3938	-0.0002, 0.0005							
Urban/Rural	-0.0003	0.2991	-0.0007, 0.0002	0.0006	0.0006	0.0003, 0.0010	0.0009	0.0031	0.0003, 0.0015							
Geographic Clustering	-0.0026	<0.0001	-0.0035, -0.0016	-0.0033	<0.0001	-0.0041, -0.0025	-0.0007	0.281	-0.0020, 0.0006							
Maternal Height	-0.0016	<0.0001	-0.0018, -0.0015	-0.0013	<0.0001	-0.0014, -0.0012	0.0004	<0.0001	0.0002, 0.0005							
Baseline Covariates	0	0.2501	-0.0000, 0.0000	0	0.6022	-0.0000, 0.0000	0.0005	0.0113	0.0001, 0.0009							
Outcome: Wasted(n=406,416)																
Coefficient: Basic Model	-0.0038	<0.0001	-0.0042, -0.0033	-0.0028	<0.0001	-0.0033, -0.0024	0.0009	0.0022	0.0003, 0.0015							
Coefficient: Full Model	-0.0015	0.01	-0.0027, -0.0004	-0.0001	0.0807	-0.0022, -0.0001	0.0005	0.3239	-0.0005, 0.0015							

Decomposition: covariates' impact on basic coefficient						
Total Impact	<0.0001	-0.0033, -0.0012	-0.0018	0.0009	-0.0029, -0.0007	0.0004
Nonlinearity	0.0004	0.3669	-0.0005, 0.0014	0.0005	0.367	-0.0006, 0.0015
Education of Other Parent	-0.0005	0.0434	-0.0009, -0.0000	-0.0003	0.2153	-0.0007, 0.0002
HH Living Standards	-0.0012	<0.0001	-0.0017, -0.0007	-0.0004	0.0878	-0.0009, 0.0001
Fertility	-0.0001	0.3034	-0.0003, 0.0001	0	0.9708	-0.0002, 0.0002
Health Care Use	-0.0002	0.0432	-0.0004, -0.0000	-0.0002	0.0497	-0.0003, -0.0000
Urban/Rural	0	0.8335	-0.0002, 0.0003	0.0001	0.6546	-0.0002, 0.0003
Geographic Clustering	-0.0006	0.0636	-0.0012, 0.0000	-0.0014	<0.0001	-0.0019, -0.0008
Maternal Height	-0.0001	0.0026	-0.0002, -0.0000	-0.0002	<0.0001	-0.0002, -0.0001
Baseline Covariates	0	0.2582	-0.0000, 0.0000	0	0.6889	-0.0000, 0.0000

Notes: P values are two sided. All estimates are weighted and p-values and confidence intervals adjusted for clustering at the PSU level. All coefficients are for an increase in the linear probability of experiencing the outcome to a single year increase in parental education. Basic model covariates: months since birth; sex, year of birth; maternal age at survey; a constant term. Full model covariates: basic covariates; household living standards (wealth index z-score); fertility (maternal age at birth, number of siblings ever born, birth interval, being first born, birth order, maternal age at first birth); health care use (vaccinated (surviving children only), born at home) urban-rural difference (rural resident); maternal height (indicating maternal early-life living standards); geographic clustering (PSU specific means of all independent variables, except for rural resident); education of other parent (other parents years of education, interaction between mean-centered years of education of both parents); nonlinearity (squared term for parents' education). All independent variables were demeaned from a survey-specific mean of included observations. The "baseline covariates" set contains the terms for the main associations of covariates when decomposing the "change coefficient". When decomposing the "change coefficient" all sets (except "nonlinearity" and "baseline covariates") contain covariates interacted with being in the newest surveys. The decomposition of the "change coefficients" quantifies the changing impact of the respective sets of covariates (except "nonlinearity" and "baseline covariates") on the coefficients from the basic models. The decomposition of main coefficients from the oldest and newest surveys shows the impact of each set of covariates on the main association.

Table A15b. Main results for paternal education including exact p-values and 95% confidence intervals

Paternal Education						Change		
	Oldest	Est.	p	Cl (95%)	Newest	Est.	p	Cl (95%)
Outcome: Mortality (n=495,597)								
Coefficient: Basic Model	-0.0038	<0.001	-0.0042, -0.0034	-0.0018	<0.0001	-0.0020, -0.0015	0.002	<0.0001
Coefficient: Full Model	-0.0004	0.4202	-0.0014, 0.0006	0.0004	0.4512	-0.0006, 0.0013	0.0008	0.0198
Decomposition: covariates' impact on basic coefficient								
Total Impact	-0.0034	<0.001	-0.0043, -0.0024	-0.0021	<0.0001	-0.0030, -0.0012	0.0012	<0.0001
Nonlinearity	-0.0005	0.2454	-0.0014, 0.0004	-0.0005	0.2453	-0.0014, 0.0004	0	0.2475
Education of Other Parent	-0.0003	0.0733	-0.0006, 0.0000	0	0.8491	-0.0002, 0.0003	0.0002	0.4442
HH Living Standards	-0.0005	0.0133	-0.0008, -0.0001	0	0.8386	-0.0004, 0.0003	0.0004	0.099
Fertility	-0.0013	<0.001	-0.0014, -0.0011	-0.0009	<0.0001	-0.0010, -0.0007	0.0002	0.027
Health Care Use	0.0001	0.2454	-0.0001, 0.0003	0	0.5188	-0.0001, 0.0001	-0.0001	0.19
Urban/Rural	-0.0001	0.1493	-0.0003, 0.0001	0.0001	0.044	0.0000, 0.0003	0.0003	0.0192
Geographic Clustering	-0.0006	0.004	-0.0011, -0.0002	-0.0007	0.0001	-0.0011, -0.0004	-0.0003	0.3639
Maternal Height	-0.0001	<0.001	-0.0002, -0.0001	-0.0001	0.0001	-0.0001, -0.0000	0.0001	0.0914
Baseline Covariates	0	0.2704	-0.0000, 0.0000	0	0.8249	-0.0000, 0.0000	0.0005	<0.0001
Outcome: Stunted(n=406,416)								
Coefficient: Basic Model	-0.0177	<0.001	-0.0184, -0.0169	-0.0162	<0.0001	-0.0169, -0.0155	0.0015	0.0059
Coefficient: Full Model	-0.0038	0.0002	-0.0059, -0.0018	-0.0044	<0.0001	-0.0063, -0.0024	-0.0005	0.4242
Decomposition: covariates' impact on basic coefficient								
Total Impact	-0.0138	<0.001	-0.0158, -0.0119	-0.0118	<0.0001	-0.0137, -0.0099	0.002	0.0012
Nonlinearity	0.0018	0.0551	-0.0000, 0.0035	0.0018	0.0551	-0.0000, 0.0037	0.0001	0.0636
Education of Other Parent	-0.0002	<0.001	-0.0026, -0.0013	-0.0014	<0.0001	-0.0020, -0.0008	0.0005	0.2551
HH Living Standards	-0.0065	<0.001	-0.0072, -0.0057	-0.0054	<0.0001	-0.0062, -0.0047	0.0013	0.0165

Fertility	-0.0015	<0.0001	-0.0018, -0.0012	-0.0011	<0.0001	-0.0009, -0.0004	0.0002	0.304	-0.0002, 0.0005
Health Care Use	-0.001	<0.0001	-0.0013, -0.0007	-0.0007	<0.0001	-0.0009, -0.0004	0.0002	0.0029	0.0003, 0.0017
Urban/Rural	-0.0001	0.7574	-0.0006, 0.0004	0.0009	<0.0001	0.0005, 0.0013	0.001	0.0053	-0.0035, -0.0006
Geographic Clustering	-0.0023	<0.0001	-0.0033, -0.0013	-0.004	<0.0001	-0.0049, -0.0030	-0.002	0.0013	0.0001, 0.0004
Maternal Height	-0.0022	<0.0001	-0.0024, -0.0021	-0.002	<0.0001	-0.0022, -0.0019	0.0002	0.0013	0.0001, 0.0004
Baseline Covariates	0	0.1489	-0.0000, 0.0000	0	0.5164	-0.0000, 0.0000	0.0002	0.4647	-0.0003, 0.0008
Outcome: Underweight (n=406, 416)									
Coefficient: Basic Model	-0.0118	<0.0001	-0.0125, -0.0112	-0.009	<0.0001	-0.0095, -0.0085	0.0028	<0.0001	0.0020, 0.0036
Coefficient: Full Model	-0.0045	<0.0001	-0.0061, -0.0028	-0.0042	<0.0001	-0.0058, -0.0027	0.0002	0.6829	-0.0008, 0.0013
Decomposition: covariates' impact on basic coefficient									
Total Impact	-0.0074	<0.0001	-0.0089, -0.0058	-0.0048	<0.0001	-0.0063, -0.0033	0.0026	<0.0001	0.0017, 0.0035
Nonlinearity	0.0017	0.019	0.0003, 0.0030	0.0017	0.019	0.0003, 0.0031	0.0001	0.0262	0.0000, 0.0001
Education of Other Parent	-0.0011	<0.0001	-0.0016, -0.0007	-0.0002	0.2856	-0.0007, 0.0002	0.0007	0.0443	0.0000, 0.0014
HH Living Standards	-0.0033	<0.0001	-0.0039, -0.0027	-0.0026	<0.0001	-0.0031, -0.0021	0.0009	0.0275	0.0001, 0.0017
Fertility	-0.0007	<0.0001	-0.0009, -0.0005	-0.0003	0.0005	-0.0005, -0.0002	0.0003	0.0235	0.0000, 0.0006
Health Care Use	-0.0006	<0.0001	-0.0008, -0.0003	-0.0004	0.0004	-0.0006, -0.0002	0.0001	0.4007	-0.0002, 0.0004
Urban/Rural	-0.0002	0.2986	-0.0006, 0.0002	0.0005	0.0006	0.0002, 0.0008	0.0007	0.0032	0.0002, 0.0012
Geographic Clustering	-0.0018	<0.0001	-0.0026, -0.0010	-0.0025	<0.0001	-0.0032, -0.0018	-0.0008	0.1616	-0.0019, 0.0003
Maternal Height	-0.0013	<0.0001	-0.0014, -0.0012	-0.001	<0.0001	-0.0011, -0.0009	0.0003	<0.0001	0.0002, 0.0004
Baseline Covariates	0	0.1496	-0.0000, 0.0000	0	0.8989	-0.0000, 0.0000	0.0003	0.193	-0.0001, 0.0007
Outcome: Wasted (n=406, 416)									
Coefficient: Basic Model	-0.0031	<0.0001	-0.0035, -0.0028	-0.0025	<0.0001	-0.0029, -0.0021	0.0006	0.0209	0.0001, 0.0012
Coefficient: Full Model	-0.0015	0.0132	-0.0026, -0.0003	-0.0013	0.0232	-0.0025, -0.0002	0.0001	0.7553	-0.0006, 0.0009
Decomposition: covariates' impact on basic coefficient									

Total Impact	-0.0017	0.0029	-0.0028, -0.0006	-0.0012	0.0355	-0.0023, -0.0001	0.0005	0.1264	-0.0001, 0.0011
Nonlinearity	0.0005	0.3099	-0.0005, 0.0016	0.0005	0.31	-0.0005, 0.0016	0	0.3162	-0.0000, 0.0001
Education of Other Parent	-0.0005	0.0061	-0.0008, -0.0001	-0.0001	0.5407	-0.0005, 0.0002	0.0004	0.1723	-0.0002, 0.0009
HH Living Standards	-0.001	<0.001	-0.0014, -0.0006	-0.0003	0.1019	-0.0007, 0.0001	0.0007	0.0151	0.0001, 0.0013
Fertility	-0.0001	0.2215	-0.0003, 0.0001	0	0.9083	-0.0002, 0.0001	0.0001	0.5029	-0.0001, 0.0003
Health Care Use	-0.0002	0.0407	-0.0003, -0.0000	-0.0001	0.0572	-0.0003, 0.0000	0	0.9441	-0.0002, 0.0002
Urban/Rural	0	0.8132	-0.0002, 0.0002	0	0.6697	-0.0002, 0.0002	0	0.9133	-0.0003, 0.0003
Geographic Clustering	-0.0004	0.1427	-0.0009, 0.0001	-0.0011	0.0001	-0.0016, -0.0005	-0.0007	0.0773	-0.0014, 0.0001
Maternal Height	-0.0001	0.0026	-0.0001, -0.0000	-0.0001	<0.0001	-0.0002, -0.0001	0	0.5253	-0.0001, 0.0001
Baseline Covariates	0	0.1571	-0.0000, 0.0000	0	0.7216	-0.0000, 0.0000	0	0.7965	-0.0002, 0.0002

Notes: P values are two sided. All estimates are weighted and p-values and confidence intervals adjusted for clustering at the PSU level. All coefficients are for an increase in the linear probability of experiencing the outcome to a single year increase in parental education. Basic model covariates: months since birth; sex, year of birth; maternal age at survey; a constant term. Full model covariates: basic covariates; household living standards (wealth index z-score); fertility (maternal age at birth, number of siblings ever born, birth interval, being first born, birth order, maternal age at first birth); health care use (vaccinated (surviving children only), born at home); urban-rural difference (rural resident); maternal height (indicating maternal early-life living standards); geographic clustering (PSU specific means of all independent variables, except for rural resident); education of other parent (other parents years of education, interaction between mean-centered years of education of both parents), nonlinearity (squared term for parents' education). All independent variables were demeaned from a survey-specific mean of included observations. The "baseline covariates" set contains the terms for the main associations of covariates when decomposing the "change coefficient". When decomposing the "change coefficient" all sets (except "nonlinearity" and "baseline covariates") contain covariates interacted with being in the newest surveys. The decomposition of the "change coefficients" quantifies the changing impact of the respective sets of covariates (except "nonlinearity" and "baseline covariates") on the coefficients from the basic models. The decomposition of main coefficients from the oldest and newest surveys shows the impact of each set of covariates on the main association.

Table A16. Full outputs for fully adjusted models

VARIABLES	Maternal education	Mortality	Stunted	Underweight	Wasted	Mortality	Paternal education	Stunted	Underweight	Wasted
Paternal education	5.86e-05 (0.000700)	-0.00383*** (0.00137)	-0.00357*** (0.00110)	-0.000547 (0.000756)	-0.000420 (0.000520)	-0.00383*** (0.00103)	-0.00446*** (0.000826)	-0.00146** (0.000590)		
Paternal education \times Newest	-0.000279 (0.000922)	-0.000434 (0.00187)	-0.00171 (0.00149)	-0.00199* (0.00110)	0.000782** (0.000336)	-0.000549 (0.000687)	0.000219 (0.000536)	0.000128 (0.000379)		
Maternal education	-0.000347 (0.000524)	-0.00599*** (0.00103)	-0.00435*** (0.000837)	-0.00151** (0.000586)	-0.000550 (0.000704)	-0.00667*** (0.00133)	-0.00494*** (0.00108)	-0.00140* (0.000739)		
Maternal education \times Newest	0.000792* (0.000408)	0.000953 (0.000680)	0.00147*** (0.000681)	0.000490 (0.000497)	0.00121 (0.000886)	0.00246 (0.00184)	0.00272* (0.00146)	0.000215 (0.00105)		
Maternal education 2	-5.66e-05* (3.37e-05)	0.000173* (7.36e-05)	0.000129** (5.27e-05)	3.55e-05 (3.94e-05)	-3.40e-05 (5.34e-05)	0.000238** (0.000107)	0.000191** (7.67e-05)	3.16e-05 (5.32e-05)		
Female	-0.00780*** (0.00154)	-0.0384*** (0.00284)	-0.0177*** (0.00229)	-0.00980*** (0.00161)	-0.00781*** (0.00154)	-0.0384*** (0.00284)	-0.0177*** (0.00229)	-0.00980*** (0.00161)		
Year of birth	-0.00497 (0.0147)	0.222*** (0.0414)	0.101*** (0.0288)	0.0602*** (0.0232)	-0.00505 (0.0147)	0.222*** (0.0414)	0.101*** (0.0268)	0.0602*** (0.0232)		
In(months since birth)	0.0281*** (0.00179)	0.227*** (0.00374)	0.0855*** (0.00301)	-0.0148*** (0.00269)	0.0281*** (0.00179)	0.227*** (0.00374)	0.0855*** (0.00301)	-0.0148*** (0.00269)		
Maternal age at survey	-0.00815 (0.0147)	0.155*** (0.0413)	0.0642** (0.0267)	0.0484** (0.0232)	-0.00824 (0.0147)	0.154*** (0.0413)	0.0640** (0.0267)	0.0485** (0.0232)		
Female \times Newest	0.000936 (0.00196)	0.000937 (0.00394)	-0.000291 (0.00308)	-0.00176 (0.00225)	0.000392 (0.00196)	0.000940 (0.00394)	-0.000302 (0.00308)	-0.00178 (0.00225)		
Year of birth \times Newest	0.00262 (0.0239)	-0.0997* (0.0512)	-0.0512 (0.0325)	-0.0706*** (0.0266)	0.00266 (0.0239)	-0.0984* (0.0512)	-0.0707*** (0.0325)	-0.0707*** (0.0266)		

In(months since birth)×Newest	-0.0126*** (0.00230)	-0.0639*** (0.00524)	-0.0247*** (0.00409)	-0.00567 (0.00371)	-0.0126*** (0.00230)	-0.0639*** (0.00524)	-0.0248*** (0.00409)	-0.00568 (0.00371)
Maternal age at survey×Newest	0.00381 (0.0238)	-0.0872* (0.0511)	-0.0403 (0.0325)	-0.0612** (0.0266)	0.00387 (0.0238)	-0.0869* (0.0511)	-0.0400 (0.0325)	-0.0613** (0.0266)
cMaternal.edu×cPaternal.edu	0.000230*** (5.89e-05)	4.96e-05 (0.00019)	0.000246*** (9.15e-05)	8.58e-05 (6.62e-05)	0.000190*** (5.91e-05)	1.18e-05 (0.000118)	0.000160* (9.01e-05)	3.64e-05 (6.50e-05)
Maternal education^2×Newest					-4.45e-05 (6.66e-05)	-0.000135 (0.000144)	-0.000125 (0.000103)	1.20e-05 (7.67e-05)
cMat.edu×cPaternal.edu×Newest	-0.000160** (6.76e-05)	4.39e-06 (0.000140)	-0.000125 (0.000106)	-4.49e-05 (8.06e-05)	-7.85e-05 (6.48e-05)	7.77e-05 (0.000137)	4.90e-05 (0.000102)	5.87e-05 (7.62e-05)
Paternal education^2×Newest	-7.14e-05 (4.60e-05)	0.000125 (9.15e-05)	5.35e-05 (7.06e-05)	-2.95e-05 (4.95e-05)	-3.69e-05 (3.17e-05)	0.000124* (6.45e-05)	0.000117* (4.97e-05)	3.74e-05 (3.68e-05)
Wealth index Z	-0.00321* (0.00171)	-0.0561*** (0.00334)	-0.0288*** (0.00258)	-0.00866*** (0.00175)	-0.00324* (0.00171)	-0.0561*** (0.00333)	-0.0289*** (0.00258)	-0.00872*** (0.00175)
Wealth index Z^2	-0.001112** (0.000570)	0.00187 (0.00120)	0.00138 (0.000884)	0.000325 (0.000710)	-0.00113* (0.000570)	0.00185 (0.00120)	0.00136 (0.000883)	0.000310 (0.000710)
Wealth index×Newest	0.00276 (0.000820)	0.00946** (0.00217)	0.00685** (0.00461)	0.00559** (0.00337)	0.00287 (0.00240)	0.00957** (0.00217)	0.00708** (0.00461)	0.00572** (0.00337)
Maternal age at birth	-0.00111 (0.0147)	-0.165*** (0.0414)	-0.0694*** (0.0268)	-0.0493** (0.0232)	-0.00104 (0.0147)	-0.164*** (0.0414)	-0.0692*** (0.0268)	-0.0493** (0.0232)
Number of siblings	0.0633*** (0.00168)	-0.0174*** (0.00259)	-0.00917*** (0.00213)	0.000350 (0.00147)	0.0633*** (0.00168)	-0.0174*** (0.00259)	-0.00918*** (0.00213)	0.000342 (0.00147)
Maternal age at first birth	-0.00181*** (0.000554)	0.00228** (0.000800)	0.000556 (0.000553)	0.000220 (0.000554)	-0.00181** (0.000944)	0.00226** (0.000944)	0.000548 (0.000800)	0.000229 (0.000553)
Maternal age at birth^2	0.000197*** (8.34e-05***)	7.30e-05*** (7.34e-05***)	1.82e-05 (0.000198***)		8.40e-05*** (0.000198***)	7.34e-05*** (1.82e-05)		

First born	(1.89e-05)	(3.22e-05)	(2.81e-05)	(2.05e-05)	(1.89e-05)	(3.22e-05)	(2.81e-05)	(2.05e-05)
	0.0304***	-0.0681***	-0.0342***	0.000973	0.0303***	-0.0682***	-0.0342***	0.000991
Birth interval	(0.00324)	(0.00587)	(0.00454)	(0.00329)	(0.00324)	(0.00587)	(0.00454)	(0.00329)
	-0.000141**	-0.00116***	-0.000754***	-7.19e-06	-0.000141**	-0.00116***	-0.000753***	-7.07e-06
Birth order	(5.75e-05)	(0.000105)	(8.12e-05)	(6.01e-05)	(5.76e-05)	(0.000105)	(8.12e-05)	(6.02e-05)
	-0.00257**	0.0127***	0.00444***	0.000407	-0.00257**	0.0127***	0.00446***	0.000409
Maternal age at birth×Newest	(0.00119)	(0.00201)	(0.00172)	(0.00115)	(0.00119)	(0.00201)	(0.00172)	(0.00115)
	0.0849*	0.0412	0.0613*	-0.000665	0.0847*	0.0410	0.0614*	0.00409
Number of siblings×Newest	(0.0239)	(0.0512)	(0.0326)	(0.0266)	(0.0239)	(0.0512)	(0.0326)	(0.0266)
	-0.0203***	0.0102***	0.00609**	0.000369	-0.0203***	0.0102***	0.00610*	0.000373
Maternal age at first birth×Newest	(0.00223)	(0.00389)	(0.00296)	(0.00205)	(0.00223)	(0.00369)	(0.00296)	(0.00205)
	0.000345	0.000455	0.000455	1.19e-05	0.000125*	0.000394	0.000493	-2.16e-07
Maternal age at first birth^2×Newest	(0.000686)	(0.00129)	(0.00106)	(0.000763)	(0.000697)	(0.00129)	(0.00106)	(0.000765)
	-7.88e-05***	6.01e-05	-1.59e-05	-4.39e-06	-7.92e-05***	5.88e-05	-1.73e-05	-4.21e-06
First born×Newest	(2.42e-05)	(4.53e-05)	(3.70e-05)	(2.73e-05)	(2.42e-05)	(4.53e-05)	(3.71e-05)	(2.73e-05)
	-0.00731*	0.00988	0.0111*	-0.000864	-0.00729*	0.00999	0.0112*	-0.000918
Birth interval×Newest	(0.00411)	(0.00797)	(0.00613)	(0.00450)	(0.00411)	(0.00797)	(0.00613)	(0.00450)
	0.000171**	0.000454***	0.000363***	-2.26e-05	0.000170**	0.000453***	0.000362***	-2.24e-05
Birth order×Newest	(7.17e-05)	(0.000136)	(0.000103)	(7.77e-05)	(7.17e-05)	(0.000136)	(0.000103)	(7.77e-05)
	0.00355**	-0.00459	-0.00172	0.000170	0.00355**	-0.00459	-0.00172	0.000167
Delivered at home	(0.00155)	(0.00286)	(0.00234)	(0.00165)	(0.00155)	(0.00286)	(0.00234)	(0.00165)
	-0.00286	0.0264***	0.0148**	0.00416*	-0.00287	0.0264***	0.0147***	0.00414*
Received immunization	(0.00247)	(0.00433)	(0.00355)	(0.00243)	(0.00247)	(0.00433)	(0.00355)	(0.00242)
	-0.0153***	-0.0115**	-0.00465	-0.00465	-0.0155***	-0.0114**	-0.00463	-0.00463
Delivered at home×Newest	(0.00576)	(0.00507)	(0.00385)	(0.00576)	(0.00576)	(0.00507)	(0.00385)	(0.00507)
	0.00409	-0.00545	-0.00320	0.00230	0.00414	-0.00537	-0.00306	0.00235
	(0.00316)	(0.00607)	(0.00497)	(0.00343)	(0.00316)	(0.00607)	(0.00497)	(0.00342)

Immunization × Newest	0.00497 (0.00873)	0.00640 (0.00758)	0.0135** (0.00583)	0.00496 (0.00873)	0.00636 (0.00758)	0.0135** (0.00583)
Rural	0.00408 (0.00284)	0.00218 (0.00728)	0.00569 (0.00548)	-0.000665 (0.00316)	0.00409 (0.00284)	0.00225 (0.00728)
Rural×Newest	-0.00815** (0.00350)	-0.0278*** (0.00938)	-0.0207*** (0.00698)	-0.000604 (0.00425)	-0.00821** (0.00350)	-0.0280*** (0.00938)
Maternal height	-0.000747*** (0.000140)	-0.0116*** (0.000280)	-0.00658*** (0.000227)	-0.000466*** (0.000154)	-0.000748*** (0.000140)	-0.0116*** (0.000280)
Maternal height×Newest	0.000299* (0.000178)	0.00125*** (0.000386)	0.00153*** (0.000304)	-0.000139 (0.000212)	0.000301* (0.000178)	0.00125*** (0.000386)
PSU Sex	-0.00303 (0.00530)	-0.00108 (0.0108)	-0.00233 (0.00890)	0.00154 (0.00551)	-0.00306 (0.00530)	-0.00116 (0.0108)
PSU year of birth	0.0237 (0.0153)	-0.175*** (0.0431)	-0.0588** (0.0287)	-0.0400* (0.0240)	0.0238 (0.0153)	-0.174*** (0.0431)
PSU ln(months since birth)	0.000815 (0.00652)	-0.0513*** (0.0148)	0.00148 (0.0123)	0.0199** (0.00782)	0.000774 (0.00652)	-0.0514*** (0.0148)
PSU maternal age at survey	0.0129 (0.0151)	-0.159*** (0.0426)	-0.0637** (0.0292)	-0.0441* (0.0238)	0.0130 (0.0151)	-0.158*** (0.0425)
PSU female×Newest	-0.000614 (0.00634)	0.0105 (0.0139)	0.00944 (0.0109)	0.00944 (0.00709)	-0.000574 (0.00634)	-0.000760 (0.0139)
PSU year of birth×Newest	-0.0130 (0.0244)	0.107** (0.0532)	0.0200 (0.0354)	0.0478* (0.0275)	-0.0131 (0.0244)	0.106** (0.0532)
PSU Ln(mon. since birth)×Newest	0.00684 (0.00784)	0.0101 (0.0189)	-0.0257* (0.0148)	-0.0140 (0.00984)	0.00686 (0.00784)	0.0101 (0.0189)
PSU Mat. age at survey×Newest	-0.0129 (0.0242)	0.0983* (0.0525)	0.0359 (0.0349)	0.0568** (0.0272)	-0.0130 (0.0242)	0.0962* (0.0525)
PSU Paternal education	0.00287* (0.00504)	-0.00542* (0.00289*)	-0.00382** (0.00359)	0.00259** (0.00359)	-0.00376* (0.00359)	-0.00119 (0.00359)

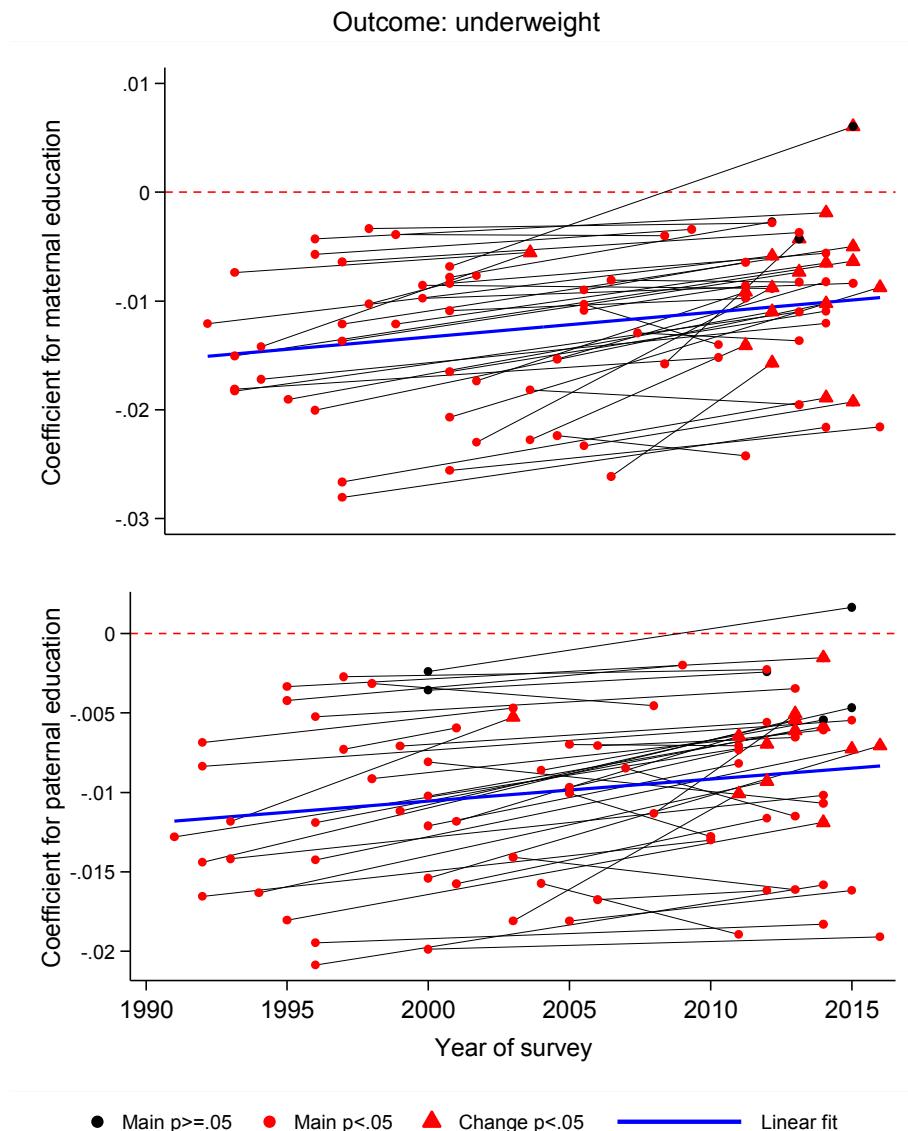
PSU Paternal education×Newest	(0.00171)	(0.00359)	(0.00318)	(0.00186)	(0.00121)	(0.00261)	(0.00223)	(0.00138)
-0.00176	-0.00277	0.00359	0.00548**	-0.00121*	0.000237	-3.75e-05	-3.79e-05	
(0.00222)	(0.00479)	(0.00394)	(0.00253)	(0.000732)	(0.00166)	(0.00139)	(0.000595)	
PSU Maternal education	-0.00469***	-0.0127***	-0.0102***	-0.00188	-0.00434***	-0.0133***	-0.0101***	-0.00247
(0.00123)	(0.00282)	(0.00216)	(0.00130)	(0.00167)	(0.00352)	(0.00352)	(0.00300)	(0.00176)
PSU Mat. education^2	0.000191**	0.000567***	0.000572***	7.79e-05	0.000163	0.000642**	0.000548*	-0.00107
(8.07e-05)	(0.000191)	(0.000141)	(8.85e-05)	(0.000129)	(0.000292)	(0.000232)	(0.000138)	
PSU cMaternal.edu×cPaternal.edu	9.40e-05	0.000380	0.000105	-5.77e-06	8.99e-05	0.000229	0.000213	0.000284*
PSU Maternal education×Newest	(0.000141)	(0.000298)	(0.000241)	(0.000150)	(0.000142)	(0.000302)	(0.000249)	(0.00153)
PSU Maternal education×Newest	0.00301***	0.00202	0.000141	-0.000776	0.00228	0.00300	-0.00133	-0.00392*
(0.000956)	(0.00215)	(0.00170)	(0.00108)	(0.00209)	(0.00475)	(0.00381)	(0.00236)	
PSU Maternal education^2×Newest					5.44e-05	-0.000125	5.65e-05	0.000340*
PSU cMat.edu×cPat..edu×Newest	-0.000198	-0.000304	1.71e-05	0.000248	(0.000161)	(0.000383)	(0.000286)	(0.00177)
(0.000157)	(0.000342)	(0.000267)	(0.000176)	(0.000150)	-0.000189	-1.44e-05	-0.000208	-0.000316*
PSU Paternal education^2	-0.000105	-0.000240	0.000446**	0.000289**	-8.35e-05	-0.000135	0.000326*	(0.000275)
(0.000115)	(0.000239)	(0.000202)	(0.000121)	(7.59e-05)	(0.000162)	(0.000129)	(8.43e-05)	(0.00171)
PSU Wealth index Z	0.00129	0.00249	-0.00248	0.00145	0.00131	0.00239	-0.00242	0.00165
(0.00272)	(0.00628)	(0.00485)	(0.00291)	(0.00272)	(0.00628)	(0.00485)	(0.00291)	
PSU Wealth index Z^2	-0.00160*	-0.000971	-0.00402**	0.000121	-0.00161*	-0.000975	-0.00396**	0.000152
(0.000914)	(0.00215)	(0.00181)	(0.00107)	(0.000915)	(0.00215)	(0.00180)	(0.00107)	
PSU Wealth index Z×Newest	-0.00731**	-0.0184**	-0.00524	-0.00612	-0.00736**	-0.0182**	-0.00548	-0.00667*
(0.00337)	(0.00836)	(0.00620)	(0.00394)	(0.00337)	(0.00836)	(0.00620)	(0.00393)	
PSU Wealth index Z^2×Newest	0.00196	0.00577*	0.00228	0.000430	0.00196	0.00584*	0.00210	0.000218
(0.00125)	(0.00320)	(0.00238)	(0.00158)	(0.00125)	(0.00319)	(0.00238)	(0.00158)	
PSU Maternal age at birth	-0.0128	0.157***	0.0651**	0.0470**	-0.0129	0.156***	0.0649**	0.0471**
(0.0153)	(0.0431)	(0.0297)	(0.0240)	(0.0153)	(0.0431)	(0.0297)	(0.0240)	

PSU Number of siblings	-0.0115** (0.00364)	0.0138* (0.00806)	0.00791 (0.00661)	0.00141 (0.00395)	-0.0115*** (0.00364)	0.0139* (0.00806)	0.00791 (0.00661)	0.00138 (0.00395)
PSU Maternal age at first birth	9.64e-05 (0.00141)	0.000473 (0.00300)	-0.000627 (0.00252)	-0.00220 (0.00157)	0.000109 (0.00141)	0.000435 (0.00300)	-0.000645 (0.00252)	-0.00214 (0.00157)
PSU Maternal age at birth*x2	-1.46e-05 (5.00e-05)	-7.02e-06 (0.000105)	-6.84e-05 (9.02e-05)	-4.54e-05 (5.51e-05)	-1.51e-05 (5.00e-05)	-7.15e-06 (0.000105)	-6.84e-05 (9.02e-05)	-4.60e-05 (5.51e-05)
PSU First born	-0.0223* (0.0114)	0.0127 (0.0237)	0.0116 (0.0193)	-0.000845 (0.0116)	-0.0222* (0.0114)	0.0127 (0.0237)	0.0115 (0.0193)	-0.000731 (0.0116)
PSU Birth interval	-0.000252 (0.000175)	0.000485 (0.000378)	0.000663** (0.000297)	6.98e-06 (0.000186)	-0.000252 (0.000175)	0.000483 (0.000175)	0.000662** (0.000297)	7.76e-06 (0.000186)
PSU Birth order	0.00117 (0.00307)	0.00642 (0.00646)	0.00447 (0.00553)	-0.000999 (0.00329)	0.001118 (0.00329)	0.00639 (0.00646)	0.00442 (0.00553)	-0.000990 (0.00553)
PSU Maternal age at birth*x>Newest	0.0126 (0.0243)	-0.0986* (0.0533)	-0.0439 (0.0356)	-0.0614** (0.0275)	0.0126 (0.0243)	-0.0984* (0.0533)	-0.0438 (0.0356)	-0.0615** (0.0275)
PSU Number of siblings*x>Newest	0.0106** (0.00460)	-0.0105 (0.0109)	-0.00173 (0.00846)	-0.00317 (0.00534)	0.0106** (0.00460)	-0.0105 (0.0109)	-0.00172 (0.00846)	-0.00326 (0.00846)
PSU Mat. age at first birth*x>Newest	0.00110 (0.00175)	-0.00329 (0.00310)	3.71e-05 (0.00204)	0.00204 (0.00204)	0.00105 (0.00175)	-0.00326 (0.00386)	6.49e-05 (0.00311)	0.00194 (0.00311)
PSU Mat. age at 1 st bir.*x2>Newest	-1.19e-05 (6.05e-05)	9.71e-05 (0.000137)	0.000154 (0.000113)	6.22e-05 (7.30e-05)	-1.05e-05 (6.06e-05)	9.67e-05 (0.000137)	0.000154 (0.000113)	6.48e-05 (7.31e-05)
PSU First born*x>Newest	0.00665 (0.0135)	-0.0147 (0.0293)	-0.00533 (0.0233)	-0.00381 (0.0151)	0.00638 (0.0135)	-0.0147 (0.0298)	-0.00505 (0.0234)	-0.00406 (0.0152)
PSU Birth interval*x>Newest	0.000274 (0.000207)	-0.000746 (0.000458)	-0.000459 (0.000351)	5.63e-05 (0.000230)	0.000272 (0.000207)	-0.000747 (0.000458)	-0.000456 (0.000351)	5.81e-05 (0.000230)
PSU Birth order*x>Newest	0.00448 (0.00395)	-0.000304 (0.00874)	0.000982 (0.00710)	0.00238 (0.00455)	0.00441 (0.00395)	-0.000335 (0.00874)	0.000109 (0.00710)	0.00240 (0.00455)
PSU Delivered at home	0.0184*** (0.0480***)	0.0169** (0.0184***)	0.0169** (0.0169***)	0.00361 (0.0184***)	0.0184*** (0.0169***)	0.0478*** (0.0169***)	0.0169** (0.0169***)	0.00391 (0.0169***)

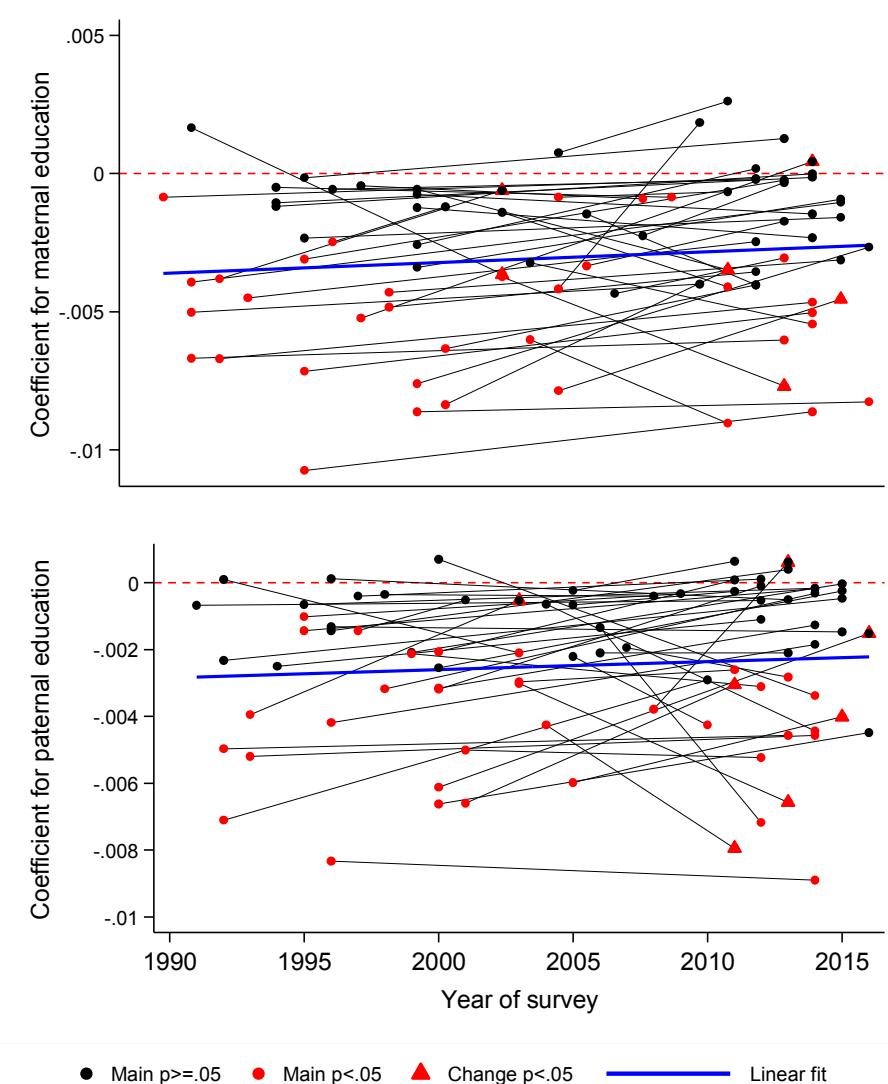
PSU Received immunization	(0.00492)	(0.0102)	(0.00859)	(0.00517)	(0.00493)	(0.00102)	(0.00860)	(0.00518)
	0.0101	-0.0330**	-0.0225**	-0.0225**		0.0102	-0.0330**	-0.0227**
	(0.0158)	(0.0160)	(0.00959)	(0.00959)		(0.0158)	(0.0160)	(0.00959)
PSU Delivered at home×Newest	-0.0162***	0.00590	0.00917	-0.00538	-0.0163***	0.00628	0.00915	-0.00605
	(0.00614)	(0.0136)	(0.0113)	(0.00730)	(0.00616)	(0.0136)	(0.0113)	(0.00731)
PSU Immunization×Newest	0.0151	0.00474	-0.0284*	-0.0284*		0.0149	0.00476	-0.0281*
	(0.0229)	(0.0220)	(0.0149)	(0.0149)		(0.0229)	(0.0220)	(0.0149)
PSU Maternal height	-0.000218	-0.00252***	0.000275	-0.000130	-0.000223	-0.00253***	0.000283	-0.000113
	(0.000320)	(0.000679)	(0.000573)	(0.000356)	(0.000319)	(0.000679)	(0.000572)	(0.000555)
PSU Maternal height×Newest	6.55e-05	-0.00211**	-0.00114	0.000713	7.37e-05	-0.00209*	-0.00115	0.000685
	(0.000412)	(0.000922)	(0.000729)	(0.000471)	(0.000411)	(0.000922)	(0.000729)	(0.000471)
Paternal education^2×Newest	7.82e-05	-8.30e-06	0.000142	0.000156**				
	(6.15e-05)	(0.000125)	(9.56e-05)	(7.18e-05)				
PSU Paternal education^2	3.93e-05	0.000226	-0.000263	-0.000412**				
	(0.000148)	(0.000319)	(0.000253)	(0.000166)				
Constant	0.0657***	0.339***	0.168***	0.0712***	0.0657***	0.338***	0.168***	0.0713***
	(0.000577)	(0.00147)	(0.00126)	(0.000716)	(0.000577)	(0.00147)	(0.00126)	(0.000717)
Observations	495,597	406,416	406,416	406,416	495,597	406,416	406,416	406,416
R-squared	0.033	0.122	0.048	0.015	0.033	0.122	0.048	0.015

Notes: *P < .10; ** P < .05; *** P < .01. 2 sided. Robust standard errors adjusted for clustering at PSU level in parentheses. PSU refers to PSU specific means of included observations for variables.

Figures A1 – A2. Country-specific regression results: coefficients for the main association of parental education with underweight and wasted, by year of survey

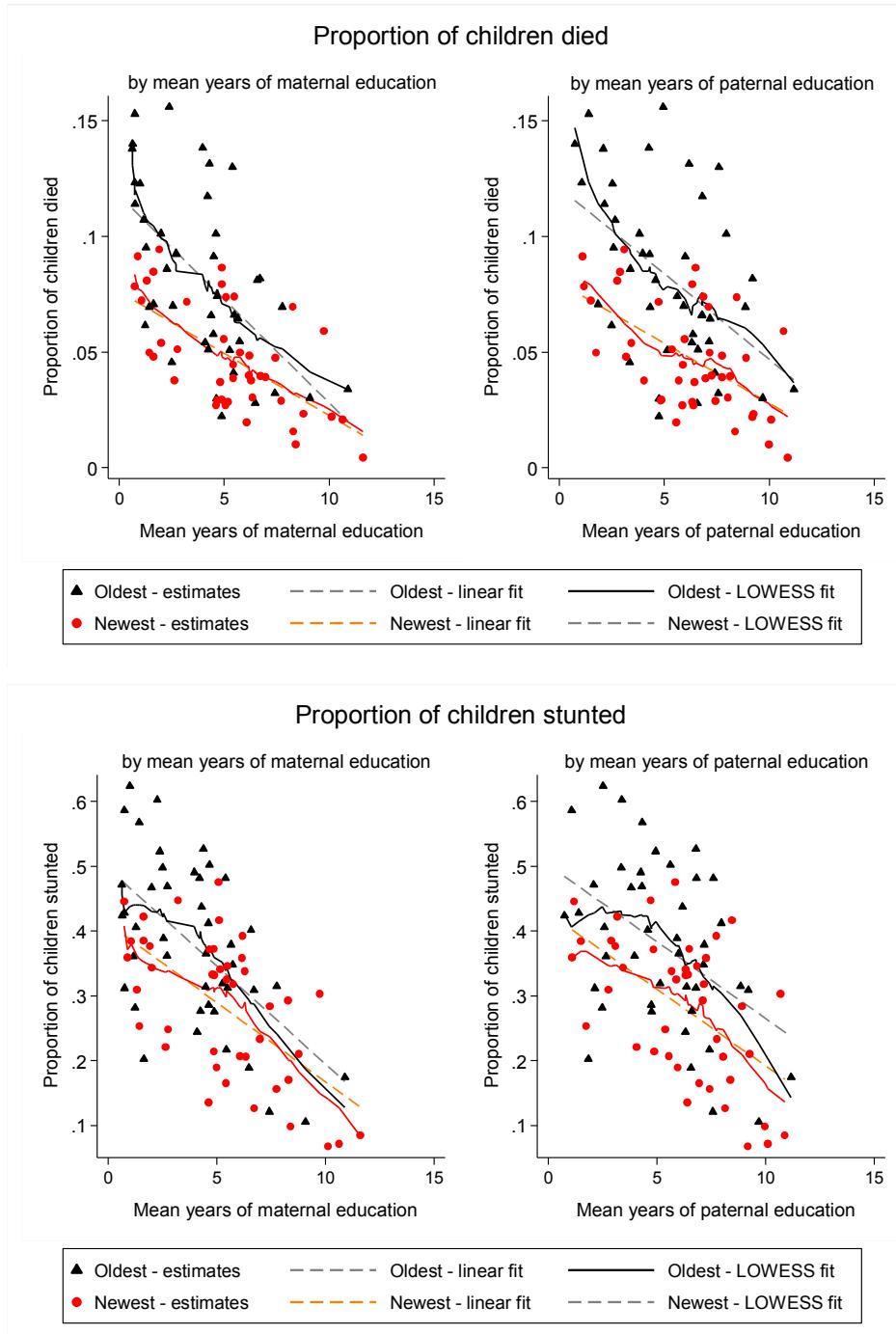


Outcome: wasted

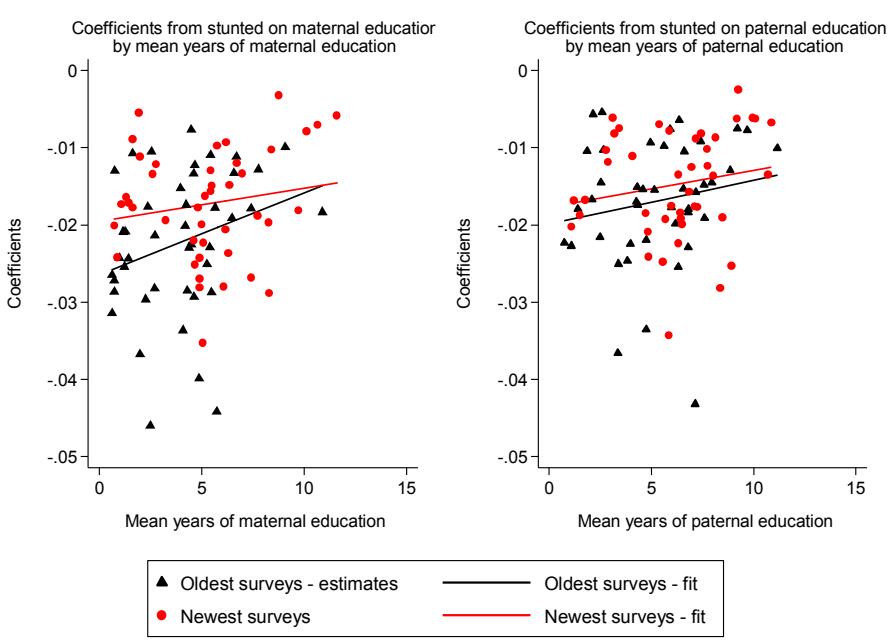
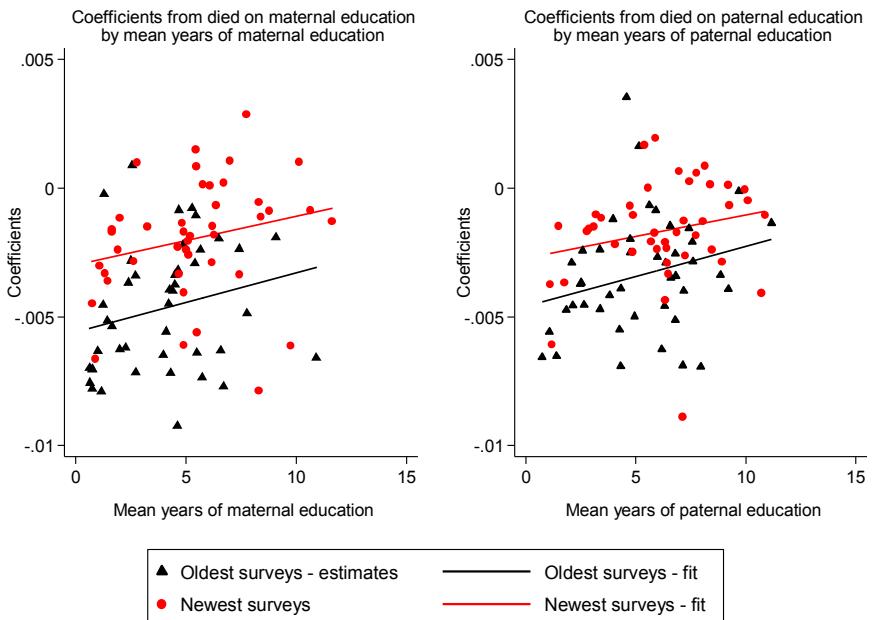


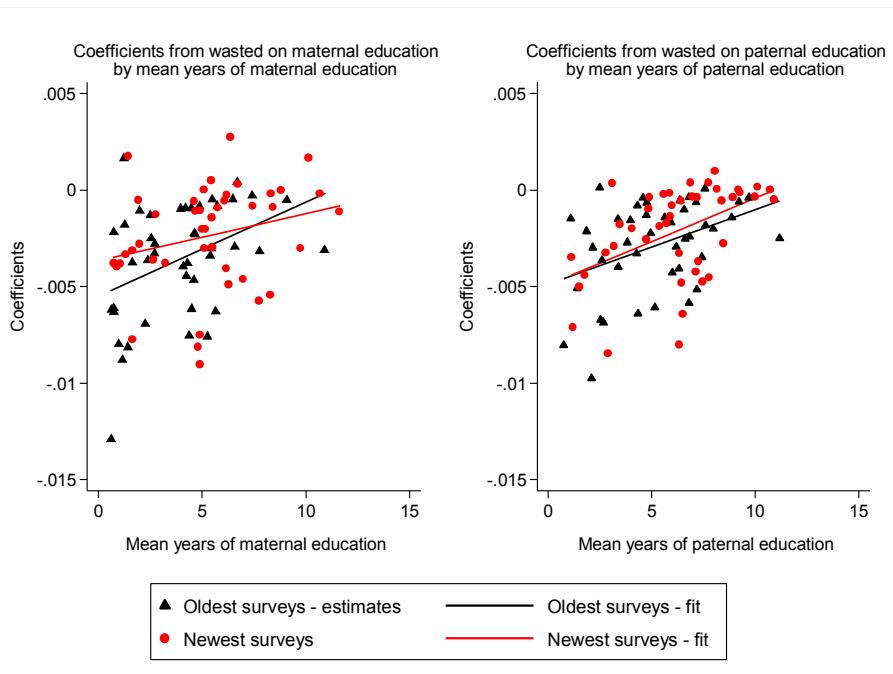
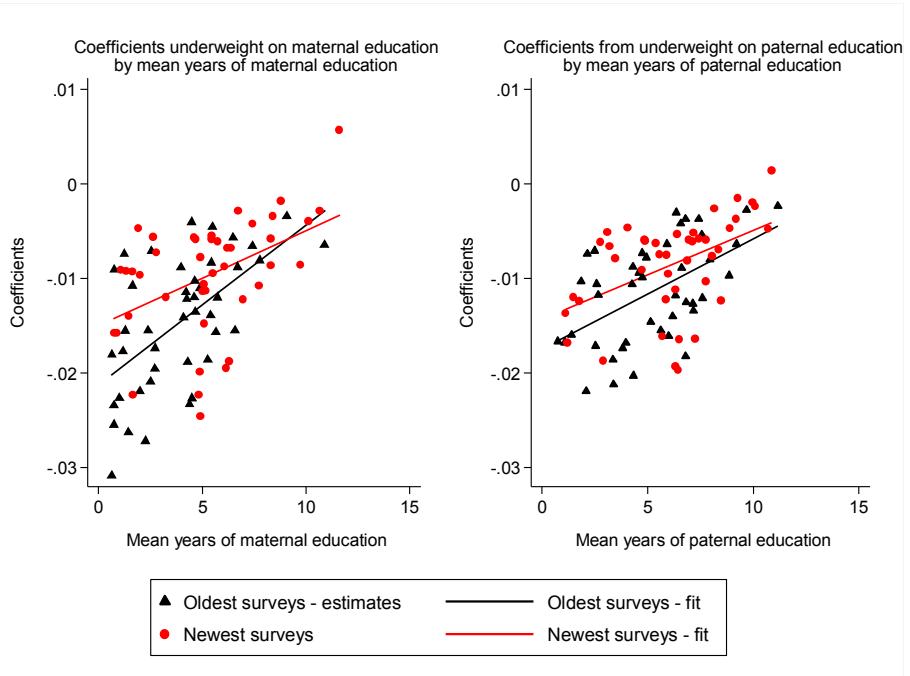
Notes: All estimates are weighted and p-values adjusted for clustering at the PSU level. All coefficients are for an increase in the linear probability of experiencing the outcome to a single year increase in parental education. Basic model covariates: months since birth; sex, year of birth; maternal age at survey; a constant term. All independent variables were demeaned from a survey-specific mean of included observations. Coefficients are for main association in oldest and newest surveys, connected with a line for each country. Main statistical significance refers of the coefficient for main association and change refers to a statistical significant change in the association between the oldest and the newest surveys.

Figures A3 – A6. Country-specific results: prevalence of child outcomes, by mean years of parental education

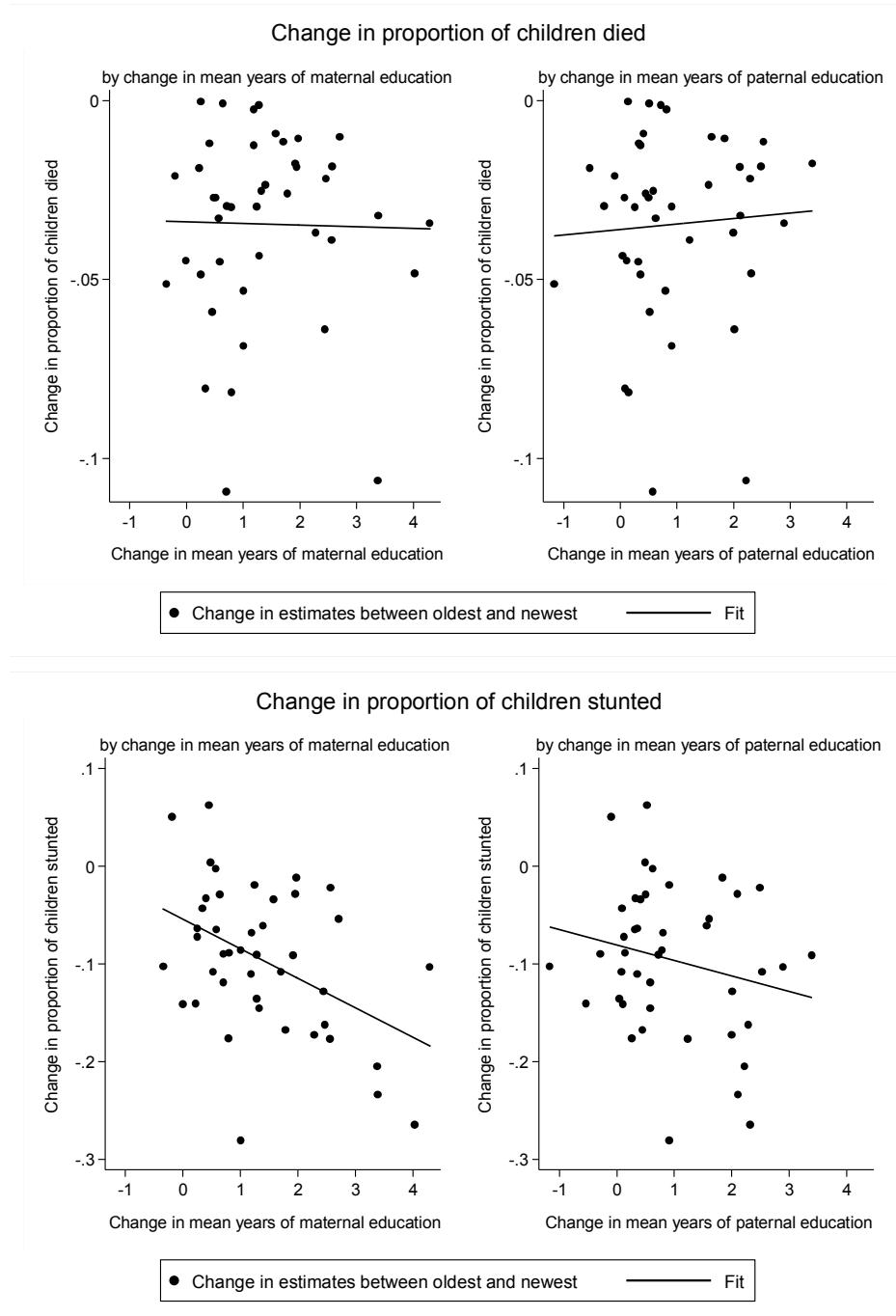


Figures A7 – A10. Country-specific results: strength of association by country mean years of parental education

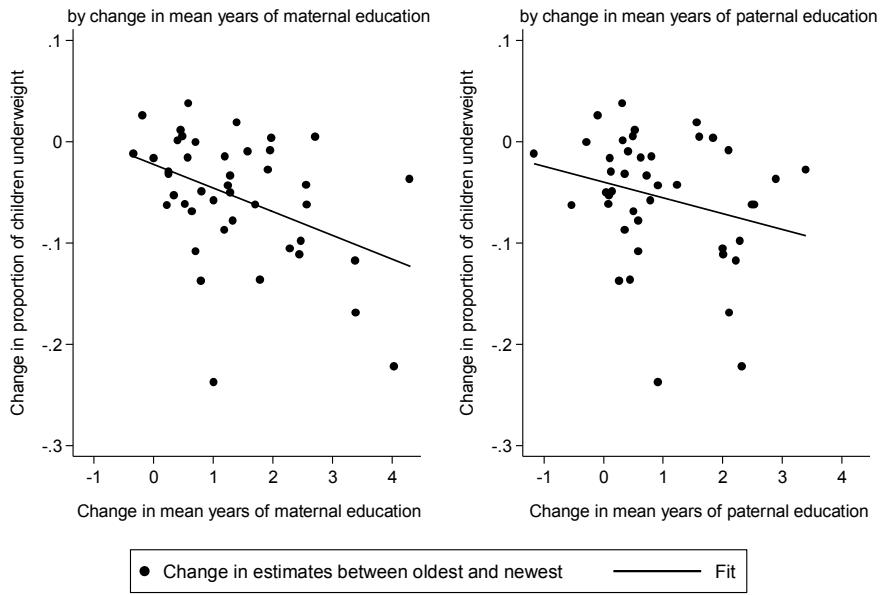




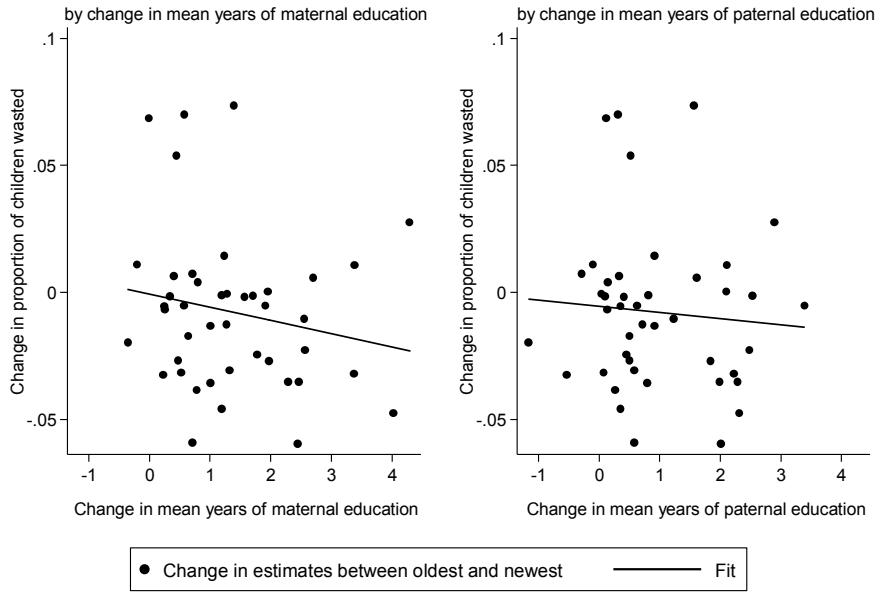
Figures A11 – A14. Country-specific results: country change in parental education by change in outcomes



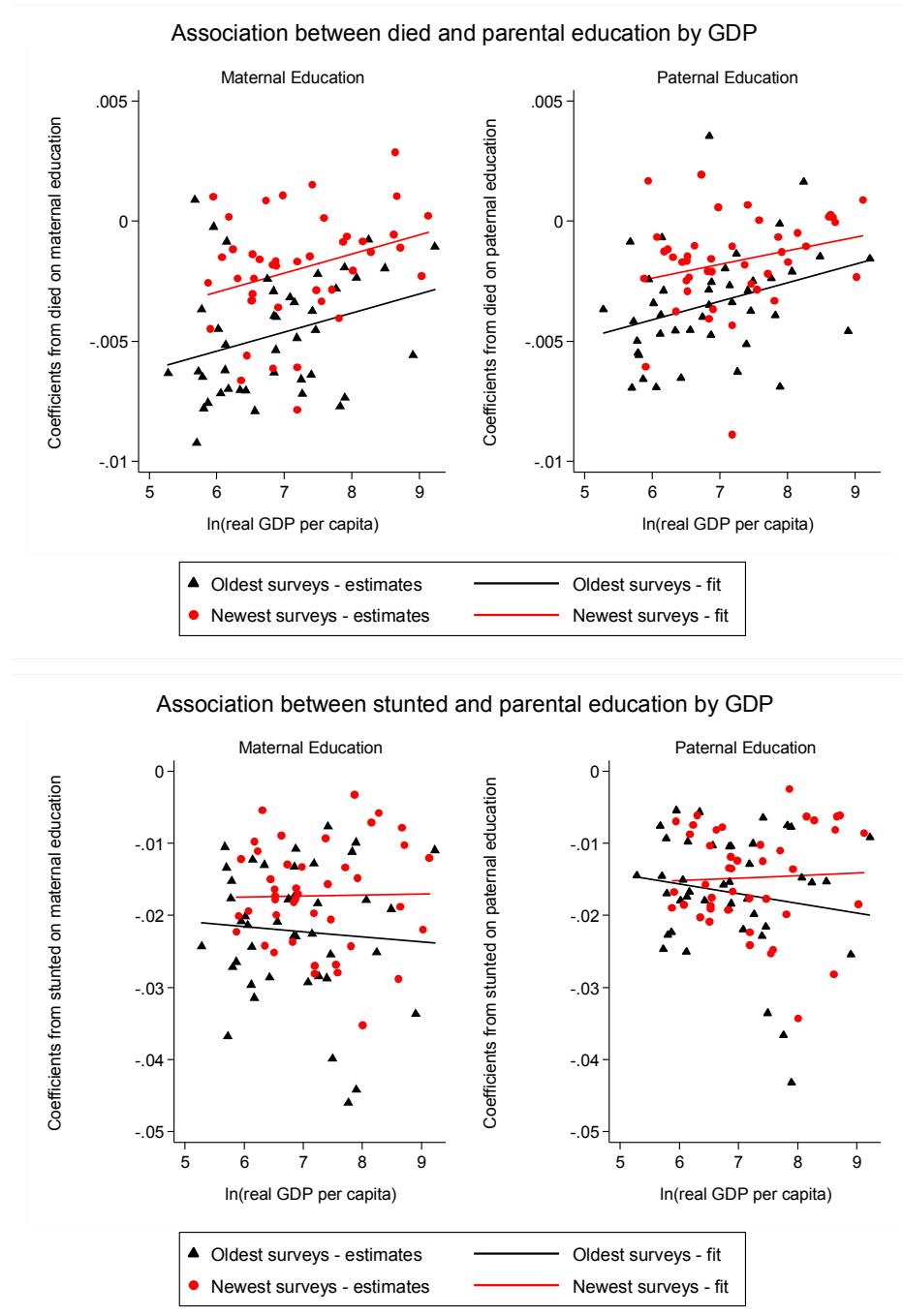
Change in proportion of children underweight



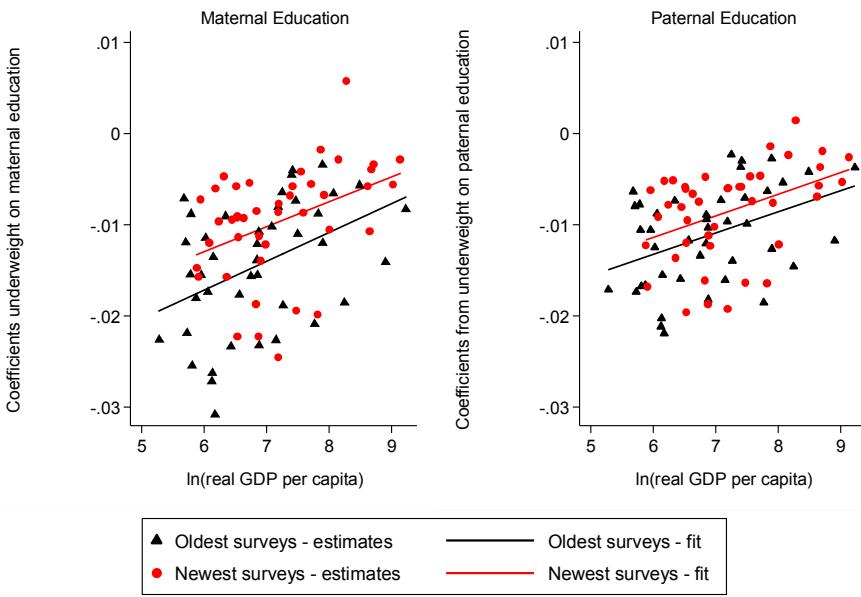
Change in proportion of children wasted



Figures A15 – A19. Country-specific results: association between parental education and child health, by GDP



Association between underweight and parental education by GDP



Association between wasted and parental education by GDP

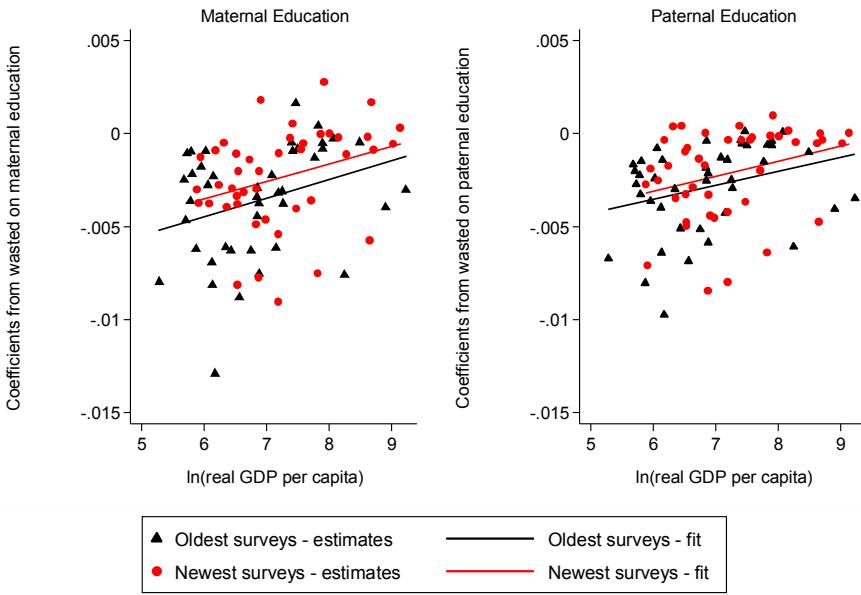
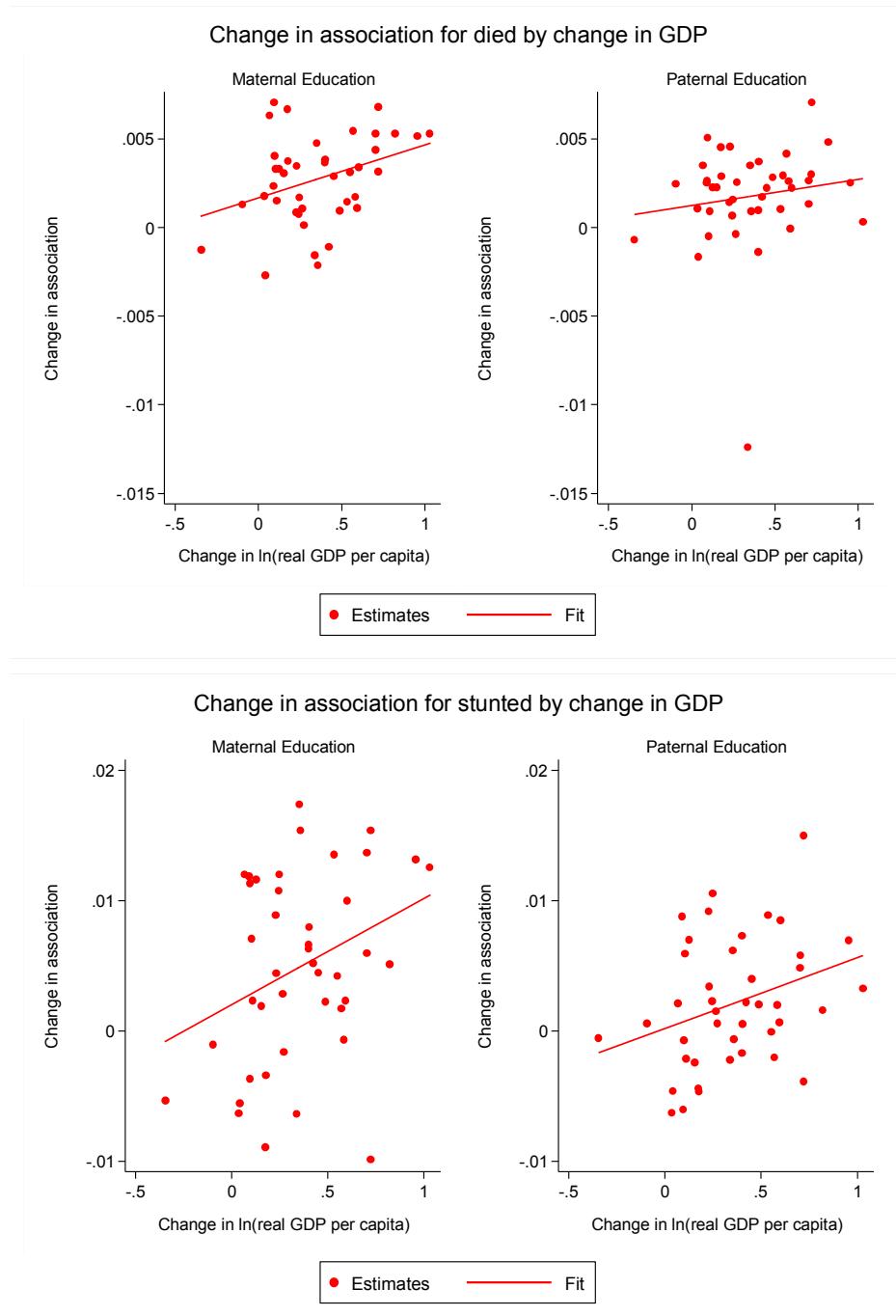
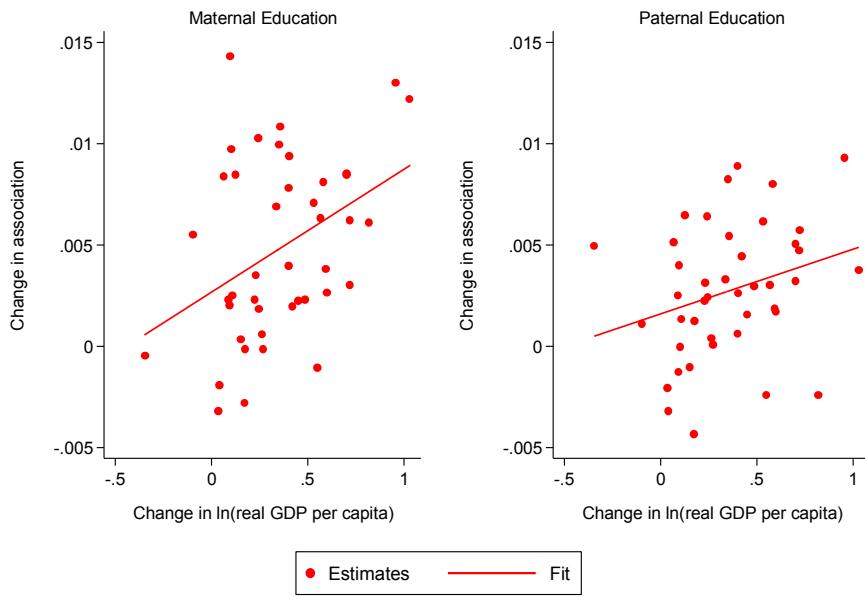


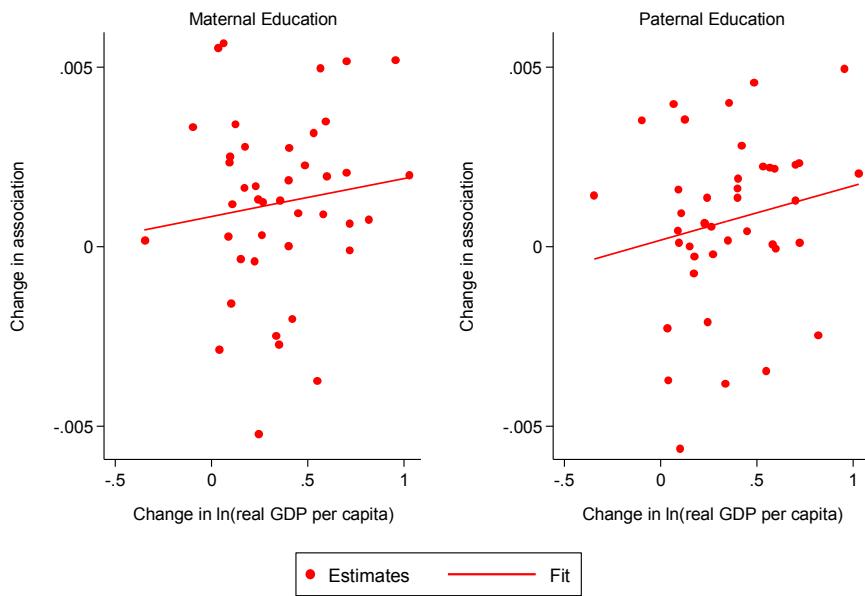
Figure A20 – A23. Country-specific results: change in association between parental education and child health, by GDP growth



Change in association for underweight by change in GDP



Change in association for wasted by change in GDP



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Paper IV

Paper IV

Child health disparities by religious affiliation in West and Central Africa

Abstract

Previous studies have suggested that a causal link exists between behaviors and attitudes prescribed by religious doctrine and child health. However, religious affiliation can also be a marker of differential access to resources and living standards. Studies have found that children born to Muslims in West Africa have poorer health outcomes compared with children born to Christians. Despite coexisting within national borders, communities affiliated with these two religions are highly clustered, geographically. This study investigates differences in child health among Christian and Muslim communities within 11 religiously heterogeneous West and Central African countries and explores the implications of geographic clustering and community-level religious composition for child health. The results of the study reveal substantial differences between Muslims and Christians living within religiously homogenous communities that can be attributed largely to living standards and health care use. Conversely, no differences in child health were found between Muslims and Christians coexisting within religiously heterogeneous communities.

Introduction

Safeguarding the health of children growing up in sub-Saharan Africa (SSA) remains a daunting task. Despite substantial improvements, approximately 8% of children in this region die before they reach the age of five, with West and Central Africa accounting for the highest mortality rates (You et al., 2016, 2015). Many of the children who survive suffer from chronic malnutrition, reflected in reduced physical growth, which has long-term consequences for their future health and development (Hoddinott et al., 2013; Liu et al., 2017; UNICEF-WHO-The World Bank, 2015; You et al., 2016). Household-level factors, such as parental education and material means, and contextual factors, such as the epidemiological environment and access to health care, are widely studied determinants of child health (Charmarbagwala et al., 2004; Mosley and Chen, 1984).

Moreover, the role of religion, which is a major social force in SSA, has been investigated as a determinant of child health and health investments, with several studies indicating that children born to Muslims have worse health outcomes than children born to Christians (Antai, 2009; Gyimah, 2007; Gyimah et al., 2006). However, these studies, which were conducted for individual West African countries, only focused on religious affiliations at the household level, largely discounting the implications of the geographic clustering of religious groups, which can significantly influence the results and interpretation of observed differences. Although large proportions of Muslims and Christians coexist within national borders, their living environments evidence dramatic geographical and even climatic variations. In West Africa, most Muslims live in and around the Sahelian and the Sudano-Sahelian climatic zones which stretch across the continent, whereas Christians mostly populate the coast of the Gulf of Guinea. Religiously diverse communities are found in between these zones and, in and at the peripheries of urban centers. Therefore religious affiliation may reflect underlying differences in living standards related to geographic clustering of religious groups within West and Central African countries.

This study examined differences in child health between Muslims and Christians in nine religiously heterogeneous West African countries and two adjacent countries in Central Africa. The rationale for focusing on West and Central Africa was that these regions are religiously the most diverse, with substantial shares of Muslims and Christians, as well as having the poorest child health outcomes. The primary aim was to explore the implications of the geographic clustering of religious groups in relation to observed differences in child health. First, children born to Muslims were compared with children born to Christians within mixed communities. Second, children within all-Muslim communities were compared with children within all-Christian communities. The expectation was that observed differences in child health outcomes would be primarily between children in all-

Muslim communities and children in all-Christian communities, given the substantial variations in their circumstances and environments. An important contribution of this study to the existing literature lies in the use of Gelbach decomposition method to quantify the impacts of various measures of living standards and demographic characteristics on observed differences in child health (Gelbach, 2016). The following dimensions were examined: ethnicity and region, urban-rural differences, mothers' migrant status, fertility, mothers' use of contraceptives, health care use, household wealth, paternal and maternal education, maternal marital status, and type of toilet facilities.

The study further explored the implications of community-level religious composition for the relationship for child health, which, in addition to reflecting living standards, may influence religiosity, social capital, experiences of discrimination and social isolation among religious minorities, and their adaptation to the behaviors of the majority. The findings of the study will contribute to a deeper understanding of differences in child health outcomes between religious groups in West and Central Africa, and to the identification of underlying factors that may link religious affiliation to child health.

Background

Over 90% of people in SSA consider religion to be central to their lives (Lugo and Cooperman, 2011, 2010). SSA is characterized by religious diversity, with many countries in this region having large proportions of Muslims and Christians of various denominations. Christianity has the most followers in this region (57%), followed by Islam (29%). West Africa has the largest proportion of Muslims (52%), whereas the percentage of Muslims in Southern Africa is the smallest (1%). The practice of both Islam and Christianity in SSA is a relatively recent phenomenon; in 1900, only 14% of the region's population practiced Christianity, and 9% practiced Islam. However, Islam has had an enduring presence in West Africa for centuries. The practice of both Christianity and Islam in SSA is unique, as active followers of both religions retain their beliefs in their local traditional religions, for example, reincarnation, and continue to engage in associated practices, such as witchcraft, spirit invocation, sacrifices, and healing. Studies have found that 30% of Muslims and 25% of Christians in SSA believe that sacrifices offered to spirits or ancestors protect them from harm (Hunziker 2005; Lugo and Cooperman 2010, 2011).

Two main hypotheses have been proposed for determining the relationship between child health and parental religious affiliation (Gyimah, 2007; Soura et al., 2013; van Poppel et al., 2002). The particularized theology hypothesis suggests that doctrinal teachings that determine behaviors (in areas such as education,

hygiene, health care, and contraceptive use) and attitudes (e.g., fatalistic attitudes and a preference for prayers over medical interventions) have causal effects on child health via various pathways. The selectivity hypothesis, on the other hand, suggests that differences in observed child health between religious groups can be attributed to differential access to various resources and variations in living standards. These two hypotheses are not mutually exclusive and can simultaneously explain differences in child health outcomes between religious groups.

It is difficult to assess whether observed differences support the particularized theology hypothesis or the selectivity hypothesis. For example, maternal education has been found to determine child health via various pathways (Caldwell, 1979). Different religious doctrines emphasize formal education to varying degrees, with some emphasizing literacy for the purpose of reading religious scriptures. From this perspective, parental education mediates between religion and child health. However, religious groups may have differential access to education as a result of geographic, socioeconomic, demographic, and other structural factors. From this perspective, parental education confounds the association between parental religion and child health. Similarly, religions may inculcate different attitudes toward reproductive behavior, medical interventions, hygiene, and work, which may affect child health as well as reflect social status and living standards.

After accounting for living standards, studies have found that there are residual differences in child health between religious groups. These findings support the particularized theology hypothesis, suggesting that lifestyle and behavioral differences that are rooted in theology can explain differences in child health. For example, in the nineteenth and early twentieth century, children born to Jewish parents in Europe and in the United States had better health outcomes than children born to Christians, independent of observed socioeconomic and demographic characteristics (Condran and Kramarow, 1991; Derosas, 2003; Preston et al., 1994; Preston and Haines, 1991; Reid, 1997; van Poppel et al., 2002). These differences may have been rooted in lifestyle differences, such as an emphasis on personal hygiene and handwashing before meals, within Judaism. Similar to Judaism, Islam prescribes explicit rules emphasizing hygiene related to body and hand washing, food preparation, and the use of toilet facilities. Whereas Muslims in India are at an overall economic and social disadvantage compared with Hindus, their child survival rates are better (Bhalotra et al., 2010). Studies have found that both Hindus and Muslims are more likely to survive in predominantly Muslim neighborhoods, a possible reason being the lower prevalence of open defecation amongst Muslims (Geruso and Spears, 2015).

Religion also influences attitudes toward marriage, procreation, and contraception and may therefore impact child health through fertility, with higher fertility

affecting child health negatively (LeGrand and Phillips, 1996). Although there is little information on contraceptive use documented in Christian scriptures, some Christian churches, such as the Roman Catholic Church, have upheld a negative view toward contraceptive use (Campbell, 1960). Restrictions on the use of contraceptives may be particularly detrimental in countries with high prevalence of HIV/AIDS. Polygamy is prevalent in West Africa, particularly among Muslims. Polygamy is condemned among some Christian denominations, such as the Roman Catholic Church. However, many local West African churches uphold varying values and norms relating to marriage, as a result of which polygamy is not uncommon amongst Christians in this region (Caldwell et al., 1991; Hayase and Liaw, 1997). Polygamy can be detrimental for child health as polygamous households may have diluted resources and crowding, which increases the chances of infections. However, polygamy can also signal a host of attributes such as more traditional values and various dimensions of living standards (Arthi and Fenske, 2018; Mott, 1982).

There are numerous examples illustrating possible linkages between religiously determined behaviors and attitudes, and child health. However, there are no obvious theological reasons why Christian children would have better health outcomes than Muslim children. A few studies have examined the relationship between religious affiliations at the household level, and child health and maternal health behaviors in West Africa. Two studies from Burkina Faso and Nigeria found that vaccination rates among children born to Muslims were lower than those among children born to Christians (Antai, 2009; Soura et al., 2013). Another study on Ghana found that the rate of the utilization of maternal health services by Muslim mothers was lower than that of Christian mothers (Gyimah et al., 2006). After controlling for socioeconomic status (SES), these studies found residual differences, indicating that the particularized theology hypothesis may offer an explanation. However, a second study from Ghana found that SES explained the lower survival rate of children born to Muslim women (Gyimah, 2007).

In most of these studies, however, the extent of the geographic clustering of religious groups was not fully accounted for when testing for differences between Muslims and Christians in relation to child health. These two religious groups are concentrated in clusters within the national borders of West and Central African countries, with Muslims predominantly residing in and around the Sahelian and the Sudano-Sahelian climatic zone as well as in urban centers within the region. The spread of these religions through trade, migration, conquest, and missionary work demonstrates a clear geographical association (Hunziker, 2005; Levzion and Pouwels, 2000). Moreover, geography encompasses resources, land endowments, disease environments, living standards, and other factors. Consequently, even though many countries have large proportions of both Muslims and Christians

within their populations, the extent to which these religious groups share a common living environment is limited.

The current study

In this paper, children born to Muslims and Christians coexisting in similar proportions within religiously mixed communities were first compared to assess groups sharing a common environment. The expectation was that there would be no differences in child health outcomes between the two religious groups within religiously mixed communities. Subsequently, exclusively Muslim and exclusively Christian communities were compared. The expectation in these cases was that there would be differences in child health outcomes between Muslim and Christian communities and that these differences could be explained by observable measures of living standards and demographic characteristics.

Additionally, the study explored the implications of community-level religious composition by testing differences in Christian and Muslim child health outcomes between communities comprising differing proportions of Christians and Muslims: all-Christian, predominantly Christian, majority Christian, mixed, majority Muslim, predominantly Muslim, and all-Muslim communities. Although this part of the analysis was exploratory, some hypotheses can be offered on the expected relationship. In addition to reflecting living standards, a community's religious composition may influence child health through religiosity, social capital, experiences of discrimination and social isolation amongst religious minorities, and their adaptation to behaviors of the majority. Hypotheses relating to each of these factors are described below.

Migration to more prosperous areas and urban centers may be the underlying reason why communities become religiously diverse and therefore signal living standards. Religiosity may be more prominent in religiously homogenous areas and may, thus, reinforce the effects of religion on health. Moreover, religion is practiced both privately and collectively within a community and Durkheim (1961) argued that the health benefits of religion are derived from its role as an integrative and regulative social institution, thereby providing social support and regulating health behaviors (Anson et al., 1991). Durkheim posited that the extent of intragroup interactions would determine the intensity of religious commitment and that increased interactions between members of religious groups, and their isolation from different ideologies, would lead to a heightened religiosity (Lamm and Myers, 1978, p. 163).

Some studies have found that social capital is positively related to health (Kawachi et al., 2008). However, it has been suggested that in the short term, ethnic diversity negatively affects social capital, which could have implications for religiously

diverse communities (Putnam, 2007). Religious or ethnic discrimination against minorities may result in adverse health outcomes among these groups (Antai et al., 2009; Williams et al., 1997). Conversely, social isolation of minorities may result in the improved health of members of these groups as a result of slower transmission of infectious diseases (van Poppel et al., 2002). Lower levels of social capital within diverse communities may also lead to lower levels of social interactions. Last, minority groups may adjust to the customs and behaviors of the majority group through adaptive behavior. This has been discussed primarily with regards to fertility (Johnson, 1993).

Data

Data were extracted from the Demographic and Health Surveys (DHS) for nine religiously mixed countries in West Africa and two adjacent counties in Central Africa. DHS surveys are nationally representative household surveys conducted in numerous low- and middle-income countries. Two-stage sampling is used in these surveys, generally stratified by geographic or administrative regions and type of residency (urban or rural). Primary sampling units are relatively small geographic areas commonly consisting of neighborhoods or villages and are usually sampled with a probability proportional to population size. Subsequently, households within these primary sampling units are listed and selected to yield a sample of approximately 30 women aged 15–49 years who are interviewed.

The selection criteria for this study were surveys from West and Central African countries which recorded all variables used and had some exclusively Muslim, exclusively Christian, and religiously heterogeneous communities. Where data for more than one survey were available in a country, the latest survey for that country was selected. After applying these criteria, surveys from 11 countries, conducted between 1995 and 2008, were included in the study. Table 1 shows the countries, survey years, response rates, and sample sizes. The full sample, totaling 111,595 observations, comprised children born to respondents who were residents of the interviewed households, and who were born less than 60 months prior to the month of the survey. Children born more than 59 months from month of survey were excluded as height-for-age, one of the outcomes in this paper, and health care use, which are important covariates, were generally only recorded for children less than 5 years old. For the mortality sample, 2,324 observations were excluded, mostly because of missing covariates. Of the surviving children, 27,411 were excluded from the height-for-age sample, primarily because of missing information on height (see Table A1 for the numbers of exclusions).

Table 1. Surveys, response rates, populations and samples

Country	Survey year	Response rate for households (%)	Response rate for women 15-49 (%)	Population of women 15-49	Sample of women 15-49	Full sample of children 0-60	Sample for mortality	Sample for height-for-age
Côte d'Ivoire	1994	97.2	97.9	3,050,53	8,099	6,365	6,161	3,020 ¹
Chad	1996	98.7	96.7	1,551,10	7,454	7,164	7,093	5,308
Togo	1998	96.6	95.6	1,093,57	8,569	6,806	6,723	3,294 ¹
Burkina Faso	2003	99.4	96.3	2,902,26	12,477	10,468	10,326	7,900
Cameroon	2004	97.6	94.3	3,917,42	10,656	7,650	7,532	2,933
Guinea	2005	99.2	97.2	2,192,44	7,954	6,165	6,055	2,476
Mali	2006	98.8	96.6	2,940,17	14,583	14,133	13,932	10,543
Benin	2006	99.1	94.4	1,899,58	17,794	15,835	15,039	11,380
Ghana	2008	98.9	96.5	5,853,55	4,916	2,968	2,944	2,341
Nigeria	2008	98.3	96.5	34,558,55	33,385	28,444	27,909	18,638
Sierra Leone	2008	97.6	94	1,460,12	7,374	5,597	5,557	2,019
				61,419,27	133,261	111,595	109,271	69,852

Notes: Population of women 15-49 years old (thousands) is used to rescale sampling weights. Population numbers are available from United Nations Population divisions at 5-year intervals. Intermittent years are interpolated. The full sample of children, 111,595 observations, consists of children born less than 60 months prior to month of survey to respondents that were residents at the household of interview. Response rates are available in final reports for each survey on the DHS website. Where the surveying period was more than one year the earlier year is shown. Only includes children 0 - 36 months old. Data source: DHS, 2018b; UN, 2017.

Outcomes

Two outcomes were studied: physical growth of a child and under-five mortality. Physical growth of children aged 0–59 months (0–35 months for Togo and Côte d'Ivoire) was measured as height-for-age z-scores (HAZ), which are standard deviations in height from a median growth trajectory of healthy children, based on the growth standards of the World Health Organization (WHO) released in 2006. Height-for-age is a commonly used nutritional indicator for net nutritional intake after accounting for tolls by infectious diseases (WHO, 2006). The under-five mortality is a binary outcome that is coded as one if a child, born 0–59 months before the implementation of a survey, has died by the time of the survey, and zero if the child has survived.

Religion

Respondents were divided into three categories based on their religions: Christians (42%, weighted), Muslims (51%), and other (7%). In this study, the results obtained for children born to mothers belonging to religions other than Islam and Christianity were omitted because they accounted for a small proportion of the population. Communities were identified from the primary sampling units of the DHS, which covered relatively small geographic areas such as a village or a neighborhood. The community-level religious composition was calculated as the percentage of Christian respondents within a community (excluding respondents of other religions from the denominator) within the entire sample, including all of the women interviewed, apart from those who were not residents of the interviewed households. Accordingly, a variable with a range of values from 0 (all-Muslim) to 100 (all-Christian) was obtained. Seven religious composition categories were constructed: all-Christian, predominantly Christian (90–99% Christian), majority Christian (65–90% Christian), mixed (35–65% of each group), majority Muslim (65–90% Muslim), predominantly Muslims (90–99% Muslim), and all-Muslim. Apart from the categories of communities that were exclusively Christian or Muslim, all of the other categories were separated by the religious affiliations of the mothers obtaining 12 categories, each indicating a maternal religious affiliation and community-level religious composition.

Covariates

Several sets of covariates were included in the models. Basic covariates used in all specifications comprised country, maternal age at the time of the survey, months since the birth, indicators for first- or second-born twin, sex of the child, the percentage of community members belonging to a religion other than Islam or Christianity, and an indicator for mother belonging to another religion. Sets of covariates that captured potential pathways and confounders were subsequently added. Levels of maternal and paternal education (with missing paternal education

adjusted for using dummy variable adjustment) were used as measures of parents' past and present living standards, child-rearing capabilities, and socioeconomic status. Household wealth, which was used to capture material living standards, was measured using a survey-specific z-score calculated using a DHS-provided wealth index factor score. The covariates used to capture fertility were birth intervals (adjusted for firstborns using dummy variable adjustment), birth order, maternal age at birth, maternal age at first birth, and the number of all siblings ever born. Covariates also included the mothers' contraceptive use (modern or traditional/folkloric). The measure applied for health care use was the place of delivery (i.e., home, public health facility, private health facility, another type of health facility, or place of birth unknown), and whether or not the child received any vaccinations. However, information on vaccines was only available for surviving children and was therefore excluded from the analysis of mortality. The types of toilet facilities used, which were indicative of the practice of open defecation which has a negative influence on child health, were divided into four categories: flush toilet, pit toilet, no toilet, and another type of toilet. The mother's migration was considered a binary indicator of whether she had ever migrated, thus controlling for higher proportions of migrants within more religiously heterogeneous communities. Additionally, residence within a rural community controlled for rural communities with lower degrees of religious heterogeneity. Lastly, a region and ethnicity Mundlak fixed effects were added to control for and estimate the statistical impacts of the overlap of ethnicity and region with religious affiliation (Antonakis et al., 2010; Mundlak, 1978).

Methods

Ordinary least-square regressions were used for both outcomes as follows:

$$y = \alpha^{\text{basic}} + \mathbf{cr}'\boldsymbol{\beta}^{\text{basic}} + \mathbf{x}'\boldsymbol{\gamma}^{\text{basic}} + \varepsilon^{\text{basic}} \quad (\text{eq. 1})$$

where \mathbf{cr} contained 11 indicators for religious affiliation and community-level religious composition with one omitted reference category (RC). Basic controls, denoted by \mathbf{x} , included covariates for country, maternal age at the time of the survey, the number of lapsed months following the birth, twin status, sex of the child, the proportion of community members not Muslim or Christian, and an indicator for being born to a woman whose religion was neither Islam nor Christianity.

Vectors \mathbf{z} and \mathbf{e} were added to a second regression equation. In equation 2 \mathbf{z} contained maternal and paternal education, household wealth, fertility, the mother's contraceptive use, migrant status and marital status, health care use, type of toilet facility, and an indicator for residing in a rural community. \mathbf{e} contained

ethnicity and region-specific means for all valid observations of all independent variables (Mundlak fixed effects) from equation 2 which is equivalent to adding a dummy coded variable for ethnicity and region (parametrically identical when using unweighted estimates).

$$y = \alpha^{\text{full}} + cr'\beta^{\text{full}} + x'y^{\text{full}} + [z'\rho + e'\vartheta] + \varepsilon^{\text{full}} \quad (\text{eq. 2})$$

Equation 2 was formulated with the aim of estimating the statistical impacts of the added covariates, shown in brackets, on observed health differences, denoted as β^{basic} . The total impact of the covariates in z , and e amounted to the difference in the β s obtained in the first and second equations, that is, $\delta = \beta^{\text{full}} - \beta^{\text{basic}}$. The impacts of the sets of covariates on the coefficients in β^{basic} were quantified using Gelbach's decomposition method (Gelbach, 2016). Accordingly, auxiliary regressions were run for each of the added covariates. Thus, for example, equation 3 regresses maternal education, denoted as z_1 , on an indicator for belonging to a specific religious group in a community with a specific religious composition, denoted as cr_2 (e.g., Muslims in all-Muslim communities) with cr_1 (e.g., Muslims in mixed communities) as an omitted RC (in this example ignoring other elements from cr for simplification).

$$z_1 = \tau_1 + cr_2\tau_{1cr} + x'\tau_{1x} + \omega_1 \quad (\text{eq. 3})$$

In the first auxiliary regression, maternal education was regressed on all of the independent variables from equation 1. The difference in maternal education for group cr_2 above the RC was expressed as τ_{1cr} . The statistical impact of maternal education on observed health differences was estimated using equation 4:

$$\delta_1 = \rho_1\tau_{1cr} \quad (\text{eq. 4})$$

where ρ_1 denotes the coefficient for maternal education obtained from equation 2. The magnitude of the statistical impact of maternal education, denoted by δ_1 , is determined by (1) the difference in maternal education between cr_2 and the RC, independent of basic covariates in x (τ_{1cr}), and (2) the association between maternal education and child health, independent of all other independent variables in equation 2 (ρ_1). The same process was applied for all of the covariates in z and e . The total statistical impact of a particular phenomenon was obtained by summing up the impact of covariates reflecting that phenomena. For example, the impact of covariates for number of siblings, birth interval, birth order, maternal age at birth, and maternal age at first birth were summed to quantify the statistical impact of fertility. Similarly, the statistical impact of ethnicity and region was quantified by summing up the impact of all the ethnicity and region-specific means (which is parametrically equivalent to quantifying the impact of a dummy coded variable for ethnicity and region but is computationally more efficient). The

main benefit of using the Gelbach decomposition method for analyzing the impact of covariates on observed differences in child health is that the results are not sensitive to the order in which the covariates are added, as is the case when sequentially adding covariates.

Standard errors were adjusted for clustering at the community level (primary sampling units). Estimates were weighted using sampling weights adjusted to add up to the size of the population of women aged 15–49 years at the time of each survey within the entire sample (i.e., all of the interviewed respondents). Robustness checks were conducted for the results obtained from equation 1 for each country to determine the degree of consistency of the results from the pooled analysis over different contexts. A second robustness check was conducted in which religious affiliation was divided into three categories, that is, Muslim, Catholic, and Protestant to explore the implications of denominational differences for community-level religious composition and child health. The community-level religious composition was then calculated as a percentage of each religion within a community (including respondents of other religions in the denominator). Separate regressions were run for each religious group that were equivalent to equation 1 in which children born to women within mixed communities were used as an RC.

Results

Figure 1 shows a map of the study area. The religious groups are distributed in geographical clusters. In West and Central Africa, most communities along the coast are Christian, whereas Muslim communities are located in the northern areas in and around the Sahel. The biomes, which describe vegetation and climate, in which most Christians reside are characterized by tropical and subtropical moist broadleaf forests (54% of geocoded all-Christian communities, unweighted), whereas the biomes in which Muslims most commonly reside are characterized by tropical and subtropical grassland, savanna, and shrubland (76% of geocoded all-Muslim communities). Mixed communities are predominantly interspersed between these areas, and in and around urban areas.

Table 2 shows descriptive statistics separated by community-level religious composition. Both mortality and height-for-age were the least favorable in all-Muslim communities, with mortality at 13% and the height-for-age at 1.84 z-score below the reference median. Outcomes were the most favorable in communities that were predominantly- or majority Christian, with 9% mortality and a 1.15 height-for-age z-score below the reference median. Many of the covariates also varied according to community-level religious composition. Notably, on average, maternal and paternal education increased in relation to an increasing proportion of Christians.

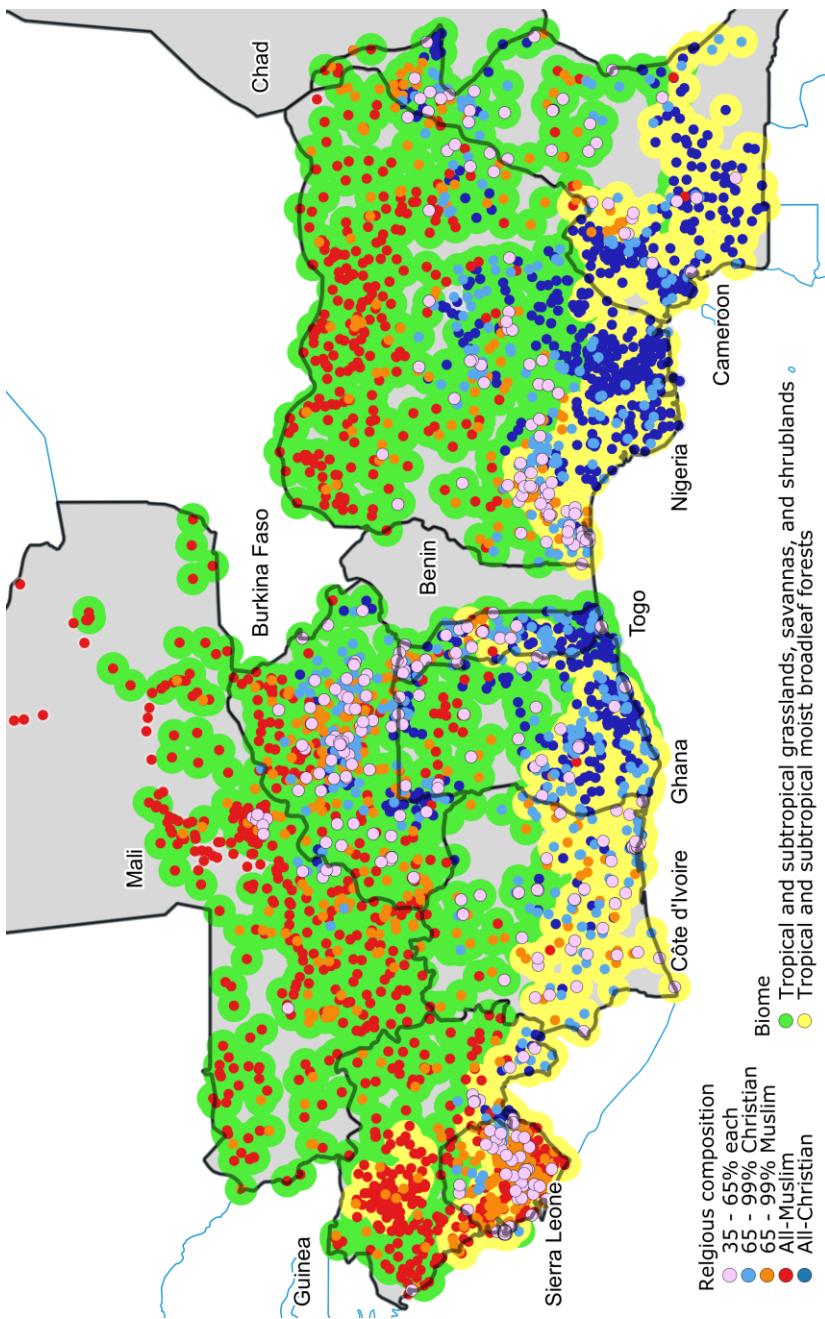


Figure 1. Map of study area, communities, and biomes
 Notes: Only communities with available GPS coordinates are shown. Where communities overlap mixed communities are displayed on top and all-Muslim and all-Christian communities on the bottom. Biome data was obtained from IPUMS. Data source: Boyle et al., 2018; DHS, 2018b.

Table 2. Descriptive statistics and variable specification

	All-Muslim	65 - 99% Muslim	35 - 65% Christian	65 - 99% Christian	All-Christian	Complete	Functional Form
Under-5 mortality	0.13 [0.34]	0.11 [0.31]	0.09 [0.28]	0.09 [0.28]	0.1 [0.30]	0.11 [0.31]	Binary
Height-for-age z-score	-1.84 [2.18]	-1.51 [2.03]	-1.31 [1.87]	-1.15 [1.94]	-1.19 [1.83]	-1.45 [2.02]	Linear
Received any vaccinations	0.58 [0.49]	0.77 [0.42]	0.84 [0.37]	0.87 [0.34]	0.85 [0.35]	0.75 [0.43]	Binary
Mother of other religion	0.01 [0.12]	0.05 [0.21]	0.1 [0.30]	0.1 [0.30]	0.12 [0.32]	0.07 [0.25]	Binary
Percentage other religion in community	1.59 [6.92]	4.76 [12.54]	8.9 [19.12]	9.57 [18.08]	10.73 [21.50]	6.37 [16.13]	Linear
Maternal age at interview (years)	28.73 [7.40]	28.93 [7.04]	29.3 [6.81]	29.7 [6.67]	29.56 [6.75]	29.18 [7.02]	Linear
Months since birth	28.6 [17.25]	28.74 [17.30]	29.23 [17.30]	28.78 [17.45]	28.68 [17.31]	28.72 [17.31]	Natural log
Female	0.49 [0.50]	0.5 [0.50]	0.49 [0.50]	0.49 [0.50]	0.49 [0.50]	0.49 [0.50]	Binary
Twin: Singleton birth	0.97 [0.17]	0.96 [0.19]	0.97 [0.18]	0.96 [0.20]	0.96 [0.20]	0.96 [0.19]	RC
First or multiple births	0.01 [0.12]	0.02 [0.14]	0.02 [0.12]	0.02 [0.14]	0.02 [0.14]	0.02 [0.13]	Binary
Second or higher of multiple births	0.01 [0.12]	0.02 [0.14]	0.02 [0.13]	0.02 [0.14]	0.02 [0.14]	0.02 [0.13]	Binary
Maternal education (years)	0.87 [2.48]	2.73 [4.20]	4.37 [4.93]	5.85 [5.16]	6.14 [4.67]	3.64 [4.70]	Quadratic
Paternal education (years)	2.1 [4.19]	4.17 [5.44]	5.65 [5.65]	7.38 [5.57]	7.56 [4.88]	4.96 [5.47]	Quadratic
Missing paternal education	0.03 [0.05]	0.05 [0.07]	0.06 [0.08]	0.07 [0.07]	0.07 [0.06]	0.07 [0.06]	Binary

		[0.23]	[0.26]	[0.27]	[0.23]
	Household wealth z-score	Quadratic	Quadratic	Quadratic	Quadratic
	-0.44	[0.22]	[0.25]	[0.27]	[0.23]
	0.08	0.28	0.35	0.35	-0.04
	[0.71]	[0.98]	[1.09]	[1.10]	[0.97]
Birth interval (months)	35.17	37.1	39.59	39.73	37.2
	[17.54]	[19.74]	[22.92]	[22.87]	[20.62]
Firsborn	0.16	0.18	0.21	0.23	0.19
	[2.77]	[2.55]	[2.36]	[2.35]	[2.56]
Birth order	4.34	3.96	3.57	3.47	3.6
	[0.37]	[0.38]	[0.41]	[0.42]	[0.40]
Maternal age at birth (years)	26.48	26.66	26.99	27.43	27.29
	[7.34]	[6.95]	[6.73]	[6.58]	[6.64]
Number of siblings ever born	4.78	4.36	3.92	3.83	4.01
	[2.78]	[2.58]	[2.39]	[2.38]	[2.58]
Maternal age at first birth (years)	18.1	18.87	19.78	20.51	20.19
	[3.46]	[3.70]	[4.08]	[4.36]	[4.25]
Place of birth: Home birth	0.85	0.53	0.4	0.35	0.42
	[0.36]	[0.50]	[0.49]	[0.48]	[0.50]
<i>Public health care birth</i>	0.13	0.34	0.36	0.36	0.33
	[0.34]	[0.47]	[0.48]	[0.48]	[0.47]
<i>Private health care birth</i>	0.01	0.07	0.15	0.19	0.21
	[0.08]	[0.26]	[0.36]	[0.39]	[0.40]
<i>Other health care birth</i>	0	0.01	0.02	0.03	0.02
	[0.04]	[0.11]	[0.15]	[0.16]	[0.14]
<i>Place of delivery unknown</i>	0.01	0.05	0.06	0.07	0.02
	[0.11]	[0.21]	[0.24]	[0.25]	[0.15]
Mother never migrated	0.38	0.33	0.26	0.28	0.33
	[0.49]	[0.47]	[0.44]	[0.45]	[0.47]
Rural community	0.84	0.59	0.54	0.56	0.72
	[0.36]	[0.49]	[0.50]	[0.50]	[0.45]
Toilet facility: Flush toilet	0.01	0.08	0.13	0.2	0.13
					0.09
					RC

<i>Pit toilet</i>	[0.11]	[0.26]	[0.34]	[0.40]	[0.34]	[0.45]	[0.39]	[0.29]
	0.69	0.59	0.45	0.39	0.45	0.45	0.39	0.55
	[0.46]	[0.49]	[0.50]	[0.49]	[0.50]	[0.50]	[0.50]	Binary
<i>No toilet</i>	0.28	0.32	0.41	0.39	0.38	0.38	0.34	Binary
	[0.45]	[0.47]	[0.49]	[0.49]	[0.49]	[0.49]	[0.47]	Binary
<i>Other toilet</i>	0.01	0.01	0.01	0.02	0.02	0.04	0.02	Binary
Mat. contrac use: Using modern method	0.03	[0.09]	[0.10]	[0.09]	[0.12]	[0.15]	0.14	0.02
	[0.17]	[0.29]	[0.29]	[0.35]	[0.36]	[0.34]	[0.29]	Binary
<i>Using traditional method</i>	0.01	0.04	0.06	0.1	0.1	0.1	0.06	Binary
	[0.10]	[0.19]	[0.24]	[0.30]	[0.30]	[0.30]	[0.23]	Binary
<i>Non-user - intends to use later</i>	0.18	0.3	0.35	0.37	0.37	0.37	0.29	Binary
	[0.38]	[0.46]	[0.48]	[0.48]	[0.48]	[0.48]	[0.46]	RC
<i>Does not intend to use</i>	0.78	0.57	0.45	0.37	0.39	0.39	0.55	RC
	[0.41]	[0.49]	[0.50]	[0.48]	[0.49]	[0.49]	[0.50]	Binary
Maternal marital status: Never in union	0	0.01	0.03	0.04	0.04	0.04	0.02	Binary
	[0.06]	[0.12]	[0.17]	[0.20]	[0.20]	[0.20]	[0.15]	Binary
<i>In union: No cowife</i>	0.54	0.57	0.65	0.71	0.71	0.71	0.62	Binary
	[0.50]	[0.50]	[0.48]	[0.46]	[0.46]	[0.46]	[0.48]	Binary
<i>In union: One cowife</i>	0.33	0.29	0.2	0.15	0.14	0.14	0.24	Binary
	[0.47]	[0.45]	[0.40]	[0.36]	[0.35]	[0.35]	[0.42]	Binary
<i>In union: More than one cowife</i>	0.11	0.1	0.09	0.06	0.05	0.05	0.08	Binary
	[0.31]	[0.30]	[0.29]	[0.23]	[0.23]	[0.23]	[0.27]	Binary
<i>Formerly in union</i>	0.02	0.03	0.03	0.04	0.05	0.05	0.03	Binary
Communities	1,105	833	452	941	1,393	4,724		
Ethnicities and regions	368	611	528	599	375	1,077		
Observations	34,676	20,849	9,265	18,728	25,753	109,271		

Notes: Estimates are weighted using adjusted sampling weights. Standard deviations are shown in brackets. Functional form refers to variables in statistical models. Estimates refer to mortality sample (except Received any vaccinations' and 'height-for-age' which refers to the height-for-age sample). Maternal contraceptive use abbreviated as Mat. contrac. use. RC stands for reference category. Data source: DHS, 2018b; UN, 2017.

The proportion of children born in health facilities was also low within all-Muslim communities, where 85% of the children were born at home. Mixed communities evidenced the lowest proportions of children within rural communities and of children born to mothers who had never migrated.

Communities that were predominantly or majority Christian had the highest z-scores for household wealth and evidenced lower fertility rates, indicated by the numbers of siblings. Most children were born within households that lacked flush toilets, but those with no toilet facilities at all were rarest in all-Muslim communities. All-Muslim communities had the highest share of children born to women who did not use and did not intend to use modern contraceptive methods. Most children were born to women who were married or cohabiting. Polygamy was most prevalent within all-Muslim communities, where 44% of children were born to mothers in polygamous households, whereas 19% of children within all-Christian communities were born to mothers living in polygamous households. Most of the surveyed communities were either all-Muslim (1,105) or all-Christian (1,393). There were 452 communities with similar proportions of Muslims and Christians.

Figure 2 shows differences in the height-for-age between religious groups, with Christians residing in mixed communities serving as the reference group. The top panel in Figure 2 shows differences in the height-for-age obtained from models that included only basic controls. The bottom panel in Figure 2 shows these differences obtained from models that included the complete set of controls. First, neither model revealed any differences between Christians and Muslims living in mixed communities. For Christians, height-for-age did not vary systematically in either the basic or the fully adjusted model in relation to community-level religious composition. Height-for-age for Muslims living in majority or predominantly Christian communities did not differ statistically significantly from those of Christians. Height-for-age of Muslim children appeared to deteriorate with an increasing share of Muslims within a community, with the height-for-age of Muslims living in all-Muslim communities being lower by 0.5 z-scores compared with those of Christians living in mixed communities. The lower panel depicted in Figure 3 shows the estimates that were obtained after adjusting for a complete set of covariates. The addition of covariates eliminated most of the observed differences in the height-for-age between groups. However, the height-for-age of Muslims living in all-Muslim communities was statistically significantly lower than those of Christians in mixed communities.

Outcome: Height-for-age z-score

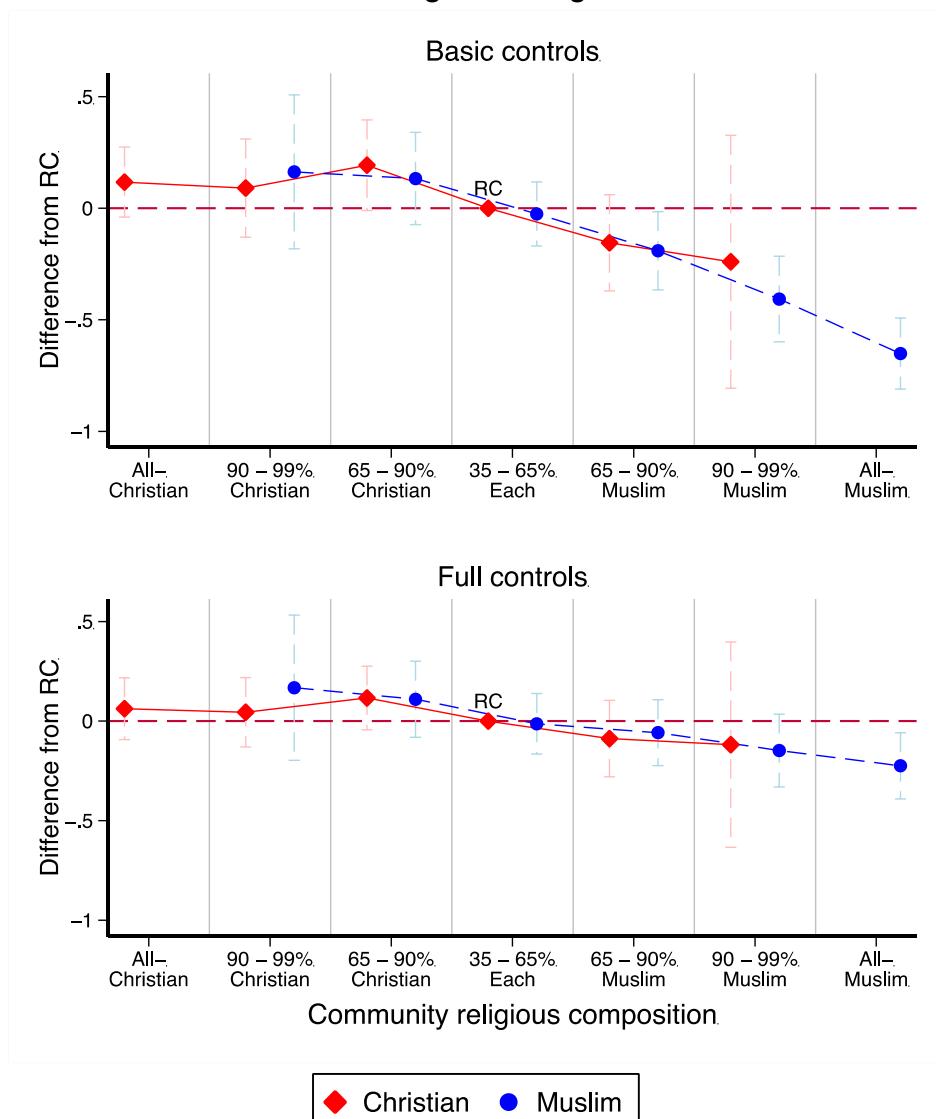


Figure 2. Differences in height-for-age z-score by religion and community level religious composition with Christians in mixed communities as a reference category (RC)

Notes: Estimates are weighted using adjusted sampling weights. 95% confidence intervals are adjusted for clustering at the community level. Basic controls: country; months since birth; female; other religion; percentage other religion in a community; twin; a constant term. Full controls: basic controls; rural; maternal education; paternal education; household wealth; maternal age at birth; maternal age at first birth; birth order, birth interval; number of siblings born; place of birth; received any vaccination (height-for-age-only); toilet facilities; maternal marital status; maternal contraceptive use; region and ethnicity. See Table 2 for details on variables. Data source: DHS, 2018b; UN, 2017.

Outcome: Under-5 mortality

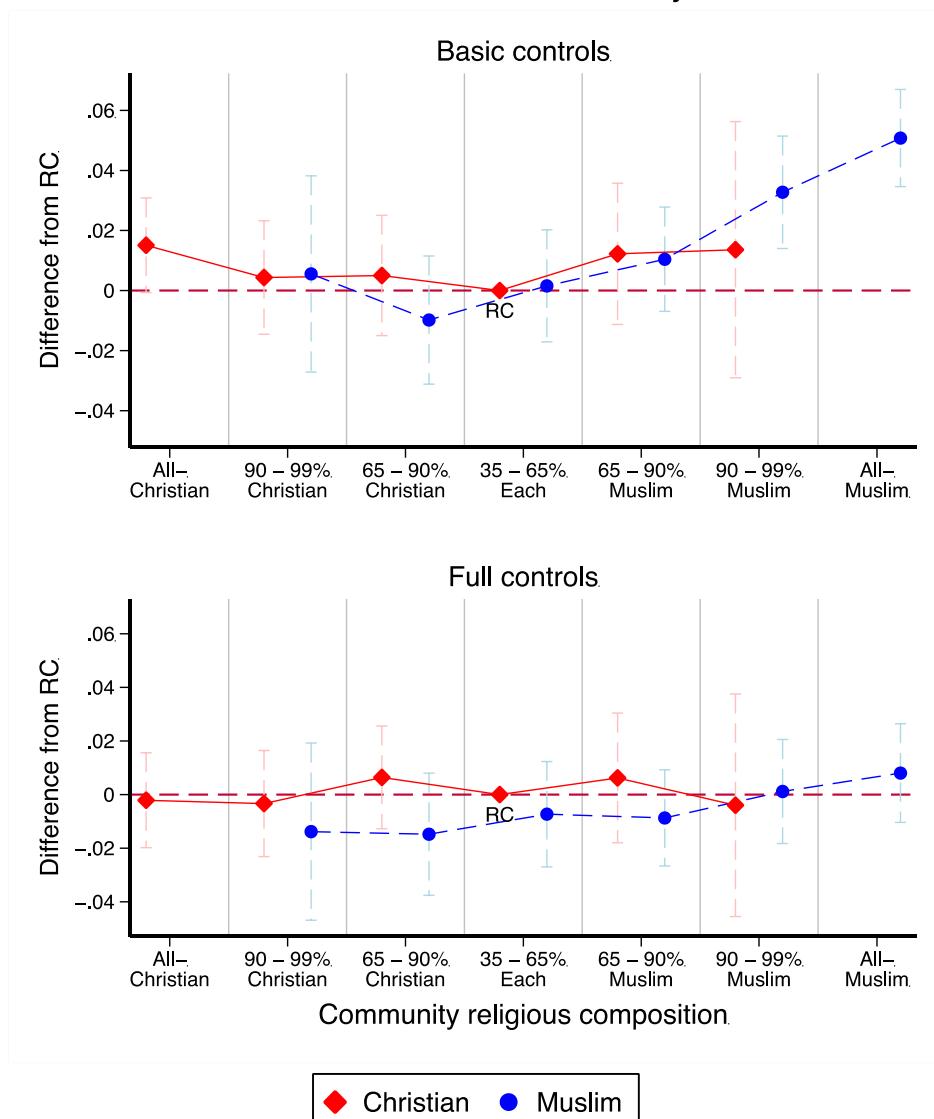


Figure 3. Differences in mortality by religion and community level religious composition with Christians in mixed communities as a reference category (RC)

Notes: Estimates are weighted using adjusted sampling weights. 95% confidence intervals are adjusted for clustering at the community level. Basic controls: country; months since birth; female; other religion; percentage other religion in a community; twin; a constant term. Full controls: basic controls; rural; maternal education; paternal education; household wealth; maternal age at birth; maternal age at first birth; birth order, birth interval; number of siblings born; place of birth; received any vaccination (height-for-age-only); toilet facilities; maternal marital status; maternal contraceptive use; region and ethnicity. See Table 2 for details on variables. Data source: DHS, 2018b; UN, 2017.

Figure 3 shows the results for mortality. As with height-for-age, mortality outcomes for Christians and Muslims within mixed communities did not differ. However, mortality outcomes for children born to Muslims appeared to decline with an increasing share of Muslims within a community, when only including basic covariates. Children born to Muslims living in all-Muslim and predominantly Muslim communities had worse health outcomes than children born to Christians living in mixed communities. However, the difference for Muslims living in majority Muslims communities was not statistically significant. The linear probability of mortality among Muslims living in all-Muslim communities exceeded that of Christians living in mixed communities by 0.05. Community-level religious composition did not appear to matter for Christians. However, the linear probability of mortality for children within all-Christian communities was greater than that of Christian children living in mixed communities by just under 0.02, although the difference was not statistically significant. When adding a complete set of covariates, as shown in the lower panel of Figure 3, the differences that were previously observed disappeared.

Table 3 shows differences in child health based on two different comparisons. The first comparison was between Muslims living in all-Muslim communities and Christians living in all-Christian communities, as shown in columns one and two, and the second comparison was between Muslims living in mixed communities and all-Muslim communities, as shown in columns three and four. The influence of covariates on differences in child health was quantified using Gelbach's decomposition method. The second row indicates the RC and the third row indicates the comparison group, (*cr*). Columns one and three show results for height-for-age and columns two and four show results for mortality.

The first columns of Table 3 show differences in the height-for-age between all-Christian communities and all-Muslim communities. The results of the basic model indicated that the height-for-age for children living in all-Christian communities exceeded the height-for-age for children living in all-Muslim communities by 0.768 z-scores. This difference was reduced substantially after a complete set of controls was added, but a difference of 0.287 z-scores remained. The ethnicity and region had the most explanatory power for the observed difference in the basic model or 0.214 (28% of the estimate from the basic model). Household wealth, maternal education, health care use, and rural residence further accounted for 0.124 (16%), 0.066 (9%), 0.063 (8%), and 0.015 (2%), respectively.

The second column of Table 3 shows difference in mortality for Christians in all-Christian communities compared with Muslims in all-Muslim communities. The linear probability of mortality of Christians in all-Christian communities was 0.036 lower than that of Muslims in all-Muslim communities in the basic model.

Table 3. Differences in child health by religion and community-level religious composition and decomposition of covariates

	N:	69,852	109,271	69,852	109,271
	Outcome:	HAZ	Mortality	HAZ	Mortality
Reference category (RC):		Muslims in all-Muslim communities		Muslims in all-Muslim communities	
cr:		Christians in all-Christian communities		Muslims in mixed communities	
Models:					
Basic model		0.768*** (0.053)	-0.036*** (0.005)	0.626*** (0.067)	-0.049*** (0.008)
Full model		0.287*** (0.081)	-0.010 (0.008)	0.211*** (0.079)	-0.015* (0.008)
Gelbach decomposition of covariates:					
Total difference		0.482*** (0.075)	-0.026*** (0.008)	0.415*** (0.060)	-0.034*** (0.006)
Maternal education		0.066** (0.027)	0.004 (0.003)	0.048** (0.019)	0.003 (0.002)
Paternal education		-0.039* (0.020)	-0.005* (0.003)	-0.020 (0.014)	-0.003* (0.002)
Household wealth		0.122*** (0.021)	-0.006*** (0.002)	0.170*** (0.030)	-0.008*** (0.003)
Maternal marital status		0.014 (0.010)	-0.002 (0.001)	0.009* (0.005)	-0.001 (0.001)
Fertility		0.016 (0.011)	-0.012*** (0.002)	-0.000 (0.013)	-0.018*** (0.002)
Maternal contraceptive use		-0.008 (0.012)	-0.011*** (0.001)	-0.005 (0.008)	-0.008*** (0.001)
Health care use		0.063*** (0.018)	0.003 (0.002)	0.062*** (0.017)	0.003 (0.002)
Maternal migration		0.001 (0.001)	0.000 (0.000)	0.003 (0.004)	0.001** (0.000)
Household toilet facility		0.018* (0.010)	-0.001 (0.001)	0.014 (0.009)	-0.000 (0.001)
Rural community		0.015** (0.007)	-0.002*** (0.001)	0.043** (0.017)	-0.004*** (0.002)
Ethnicity x region		0.214***	0.006	0.091* (0.006)	0.003

(0.072)	(0.007)	(0.054)	(0.005)
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Notes: * $p < .10$; ** $p < .05$; *** $p < .01$. Estimates are weighted using adjusted sampling weights. Standard errors are adjusted for clustering at the community level. Basic controls: country; months since birth; female; other religion; percentage other religion in a community; twin; a constant term. Full controls: basic controls; rural; maternal education; paternal education; household wealth; maternal age at birth; maternal age at first birth; birth order, birth interval; number of siblings born; place of birth; received any vaccination (height-for-age-only); toilet facilities; maternal marital status; maternal contraceptive use; region and ethnicity. See Table 2 for details on variables. The Gelbach decomposition quantifies the impact of the respective sets of covariates on the difference in child outcomes between the reference categories (RC) and the respective religion and community-level religious composition categories or in the basic models. Data source: DHS, 2018b; UN, 2017.

After adding a complete set of covariates, the difference was reduced and was not statistically significant. Fertility and the use of contraceptives by mothers reduced the difference by 0.012 (33%) and 0.011 (31%), respectively. Household wealth further accounted for 0.006 (17%), and rural residence accounted for 0.002 (6%).

The third column in Table 3 shows that in the basic model, the height-for-age of Muslims living in mixed communities exceeded that of Muslims in all-Muslim communities by 0.626 z-scores, which was statistically significant. This difference was reduced after a complete set of controls was added, but it remained statistically significant at 0.211 z-scores. Household wealth, which had the greatest explanatory power, accounted for 0.170 (27%), whereas health care use, maternal education, and rural residence respectively accounted for 0.062 (10%), 0.048 (8%), and 0.043 (7%).

The results in the fourth column of Table 3 show that Muslims in mixed communities had 0.049 lower linear probability of mortality than Muslims in all-Muslim communities. This difference was reduced and was not statistically significant after a complete set of controls was added. Of the covariates, fertility had the greatest explanatory power at 0.018 (37%), followed by mothers' use of contraceptives and household wealth, each of which accounted for 0.008 (16%). Rural residence accounted for 0.004 (8%), and the migrant status of mother reduced the difference by 0.001 (2%).

Figures 4 and 5 depict the robustness checks. Figure 4 shows the country-specific results for height-for-age using basic control variables. The previously observed pattern in which Muslims in all-Muslim communities had the lowest height-for-age was most apparent in Benin, Burkina Faso, Nigeria, and Togo. There was no substantial difference between Muslims and Christians in mixed communities in all of the countries, with the exception of Chad, where height-for-age were lower among Muslims, though the difference was not statistically significant. Figure 5 shows the country-specific results for under-five mortality. The previously observed pattern in which Muslims in all-Muslim communities had the highest mortality was most apparent in Burkina Faso, Cote d'Ivoire, Mali, Ghana, Nigeria, and Sierra Leone. There was no substantial difference between Muslims and Christians in mixed communities in all of the countries, with the exception of

Chad and Guinea, where Muslims had lower mortality than Christians in mixed communities, and in Ghana, where Muslims had higher mortality than Christians in mixed communities. However, the difference was statistically significant only in Chad.

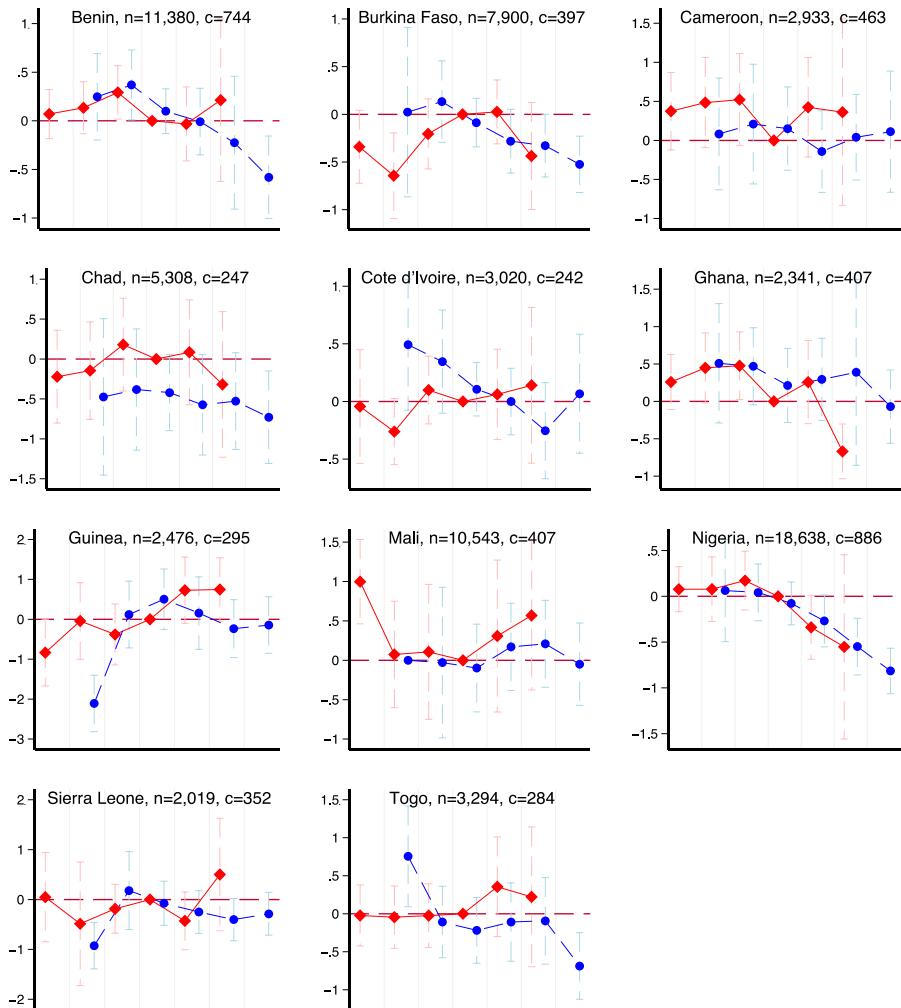


Figure 4. Differences in height-for-age by religion and community level religious composition with Christians in mixed communities as a reference category (RC) for each country

Notes: See Figures 2 and 3 for axes labels. Estimates are weighted using adjusted sampling weights. 95% confidence intervals are adjusted for clustering at the community level. Basic controls: months since birth; female; other religion; percentage other religion in a community; twin; a constant term. See Table 2 for details on variables. Note differences in y-axis scales. Data source: DHS, 2018b; UN, 2017.

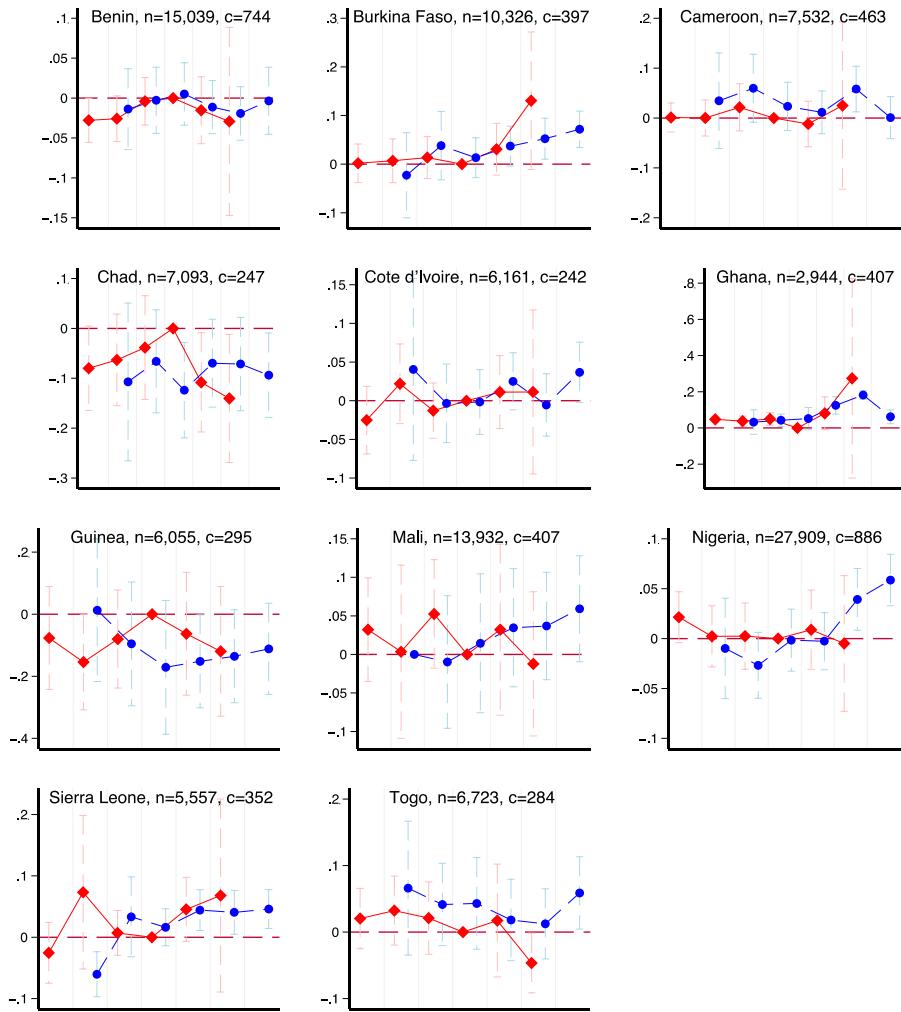


Figure 5. Differences in mortality by religion and community level religious composition with Christians in mixed communities as a reference category (RC) for each country

Notes: See Figures 2 and 3 for axes labels. Estimates are weighted using adjusted sampling weights. 95% confidence intervals are adjusted for clustering at the community level. Basic controls: months since birth; female; other religion; percentage other religion in a community; twin; a constant term. See Table 2 for details on variables. Note differences in y-axis scales. Data source: DHS, 2018b; UN, 2017.

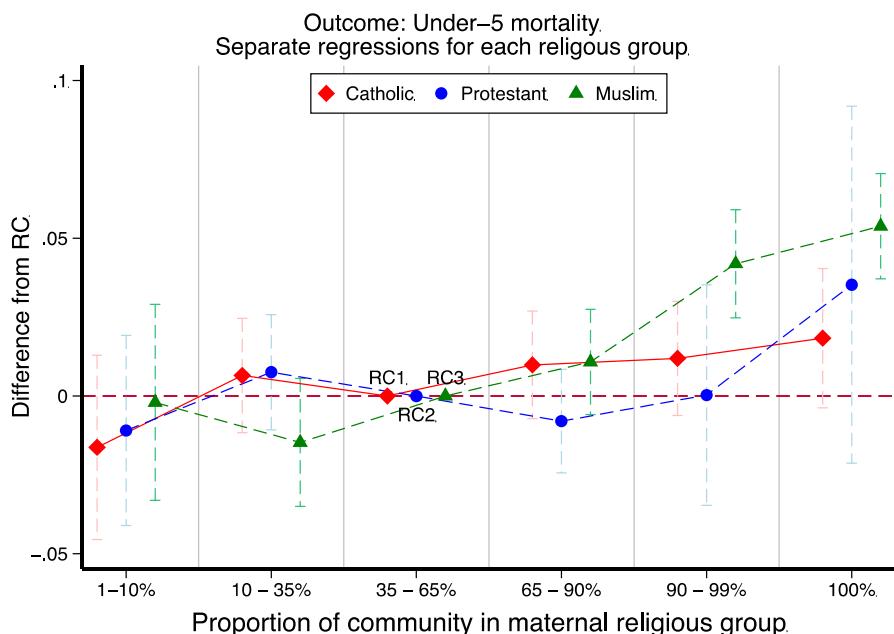
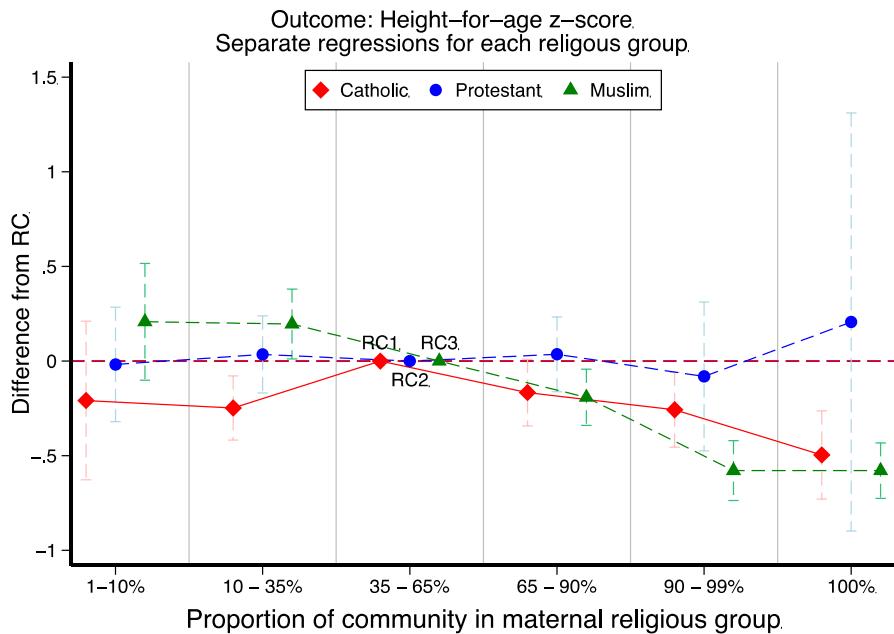


Figure 6. Differences in outcomes by community-level religious composition

Notes: Estimates are weighted using adjusted sampling weights. 95% confidence intervals are adjusted for clustering at the community level. Controls: country; months since birth; female; other religion; percentage other religion in a

community; twin; a constant term. Each line is obtained from a single regression, one for each religion and outcome. The reference categories are communities that have 35–65% of the respective religions (also including those of religions other than Muslims or Christians). Lines should be interpreted separately and do not indicate differences between the different religions. A separate regression shows that children born to Catholics and Protestants do not have statistically different outcomes, while children born to Muslims have worse outcomes (Table A3). Data source: DHS, 2018b; UN, 2017.

Figure 6 reveals the implications of the community-level religious composition when separating the category for Christians into Protestants and Catholics. Children born to Catholics and Muslims living in religiously homogenous communities appeared to have lower height-for-age compared to Catholics and Muslims in mixed communities, respectively. Muslims who constituted minority groups within their communities appeared to have higher height-for-age, whereas Catholics who constituted minority groups within their communities had lower height-for-age when compared to Catholics and Muslims in mixed communities, respectively. All religious groups residing in religiously homogenous communities appeared to have higher mortality compared with those who lived in more religiously heterogeneous communities, although the difference was statistically significant only for Muslims. It should be noted that separate models were performed for each religion, so point estimates do not indicate differences between religions. A separate analysis (see Table A3) showed that there was no statistically significant difference in child health outcomes between Protestants and Catholics, whereas Muslims had lower health outcomes that were statistically significant.

Discussion

Religion is influential in West and Central Africa, where most inhabitants are either Muslims or Christians of various denominations. Religion can impact attitudes, norms, and behaviors, as well as reflecting social status and living standards. Previous studies have suggested that religious affiliation is causally linked to child health through differences in behaviors and attitudes stipulated by religious doctrines. For example, the emphasis on hygiene within Islam and Judaism could be incidentally beneficial for child health, implying causality. Similarly, attitudes toward marriage, reproduction, modern health care, education, and other factors could vary between religious groups, thereby affecting child health via various pathways. Numerous examples can be found, illustrating how attitudes and behaviors influenced by religious affiliation can be beneficial or detrimental to child health. However, observed differences in child health between religious groups may reflect differences in access to resources and living standards.

Children born to Muslims have been found to have worse health outcomes compared with children born to Christians in several countries in West Africa

(Antai, 2009; Gyimah, 2007; Gyimah et al., 2006; Soura et al., 2013). However, even though the two religious groups coexist in the region, and within national borders, their living environment is only shared to a limited extent, which may determine their living standards and access to resources. This study is the first to test differences in child nutrition and under-five mortality associated with religious affiliation in 11 religiously heterogeneous countries in West and Central Africa and to explore the implications of the geographic clustering of religious groups and community-level religious composition. In addition, observed differences in child health were decomposed, and the statistical impacts of demographic characteristics and various dimensions of living standards were quantified.

The implications of geographic clustering were explored by assessing the differences between Muslims and Christians, first in communities that were exclusively Muslim or Christian and second in communities where the proportions of Christians and Muslims were similar. The Muslim disadvantage was apparent in all-Muslim communities, with no difference in child health found between Muslims and Christians living in religiously heterogeneous communities. The unadjusted difference in outcomes between all-Muslim and all-Christian communities was substantial at 53% of the mean deficit in the height-for-age within the sample, and 33% of the mean under-five mortality. The observed mortality disadvantage of children within all-Muslim communities, both in comparison to Muslims in mixed communities and to Christians in all-Christian communities, was mostly accounted for by covariates, of which fertility, mothers' use of contraceptives, and household wealth demonstrated the greatest explanatory power. Observable characteristics explain well over half of the observed deficit in the height-for-age for Muslims in all-Muslim communities, both in comparison to Christians within all-Christian communities and to Muslims within mixed communities in which household wealth, health care use, and maternal education had the most explanatory power. Additionally, ethnicity and region explained a substantial part of the difference between all-Muslim and all-Christian communities.

These results are in line with those of a study conducted in Ghana, where children born to Muslims were found to have worse survival outcomes than children born to Christians, attributed to socioeconomic status (Gyimah, 2007). Other studies from West Africa on health behaviors, immunization, and mothers' utilization of health care facilities have revealed residual associations, suggesting that religious affiliation not just reflect differences in living standards (Antai, 2009; Gyimah et al., 2006; Soura et al., 2013). In this study, the statistical impact of health care use on observed differences in child health between religious groups was quantified. Indeed, the measures of health care use were found to contribute to the observed disadvantage in height-for-age of Muslims within all-Muslim communities, which were similar in magnitude when compared to both Christians in all-Christian

communities and Muslims in mixed communities. These results suggest that the use of healthcare facilities is particularly limited in all-Muslim communities, rather than being limited among Muslims in general.

Additionally, the study explored the implications of the community-level religious composition. It was hypothesized that religiously homogenous communities could have higher levels of religiosity (Durkheim, 1961) or more social capital (Putnam, 2007) and that religious minorities could experience worse health outcomes as a result of discrimination and psychosocial stress (Williams et al., 1997) or, conversely, better health outcomes through social isolation which limits exposures to infections (van Poppel et al., 2002). Last, it was suggested that religious minorities might adapt to the majority group, as has been observed regarding fertility behaviors (Johnson, 1993). Religious minorities did not demonstrate any substantial differences in child health compared to the majority group within the same communities. Therefore, it is unlikely that they were discriminated against or socially isolated. Previous studies have suggested that levels of social capital, which can be beneficial for health (Kawachi et al., 2008) could be higher in homogenous communities (Putnam, 2007). The findings of this study indicated that health outcomes in religiously homogenous communities are worse compared with those in religiously diverse communities, indicating that social capital is either not greater in religiously homogenous communities or not an important mechanism in relation to child health in these contexts.

Religious groups do have similar child health outcomes within communities with the same religious composition, which could imply that minorities adapt to the behaviors of the majority group. This pattern can, however, also be attributed to living standards, signaled by community-level religious composition. Children, especially Muslim children, appear to have progressively worse health outcomes as the proportion of Muslims within a community increases. No clear residual differences remained for mortality after a complete set of covariates was added, while a residual difference remained for height-for-age. The higher mortality observed for Muslims in all-Muslim communities compared with Muslims in mixed communities were primarily explained by the covariates for fertility and mothers' contraceptive use. If reproductive behavior favoring higher fertility and lower contraceptive use are influenced by religious doctrine, then this negative influence appears to be greater for Muslims within religiously homogenous communities, possibly leading to higher mortality. However, it may also be the case that higher mortality in religiously homogenous Muslim communities results in higher fertility, which may explain the absence of any impact of fertility on the differences in height-for-age. The lower height-for-age of Muslims within all-Muslim communities was mostly explained by household wealth, health care use, maternal education, and rural residence, but residual differences may relate to religiosity. Although heightened religiosity is a possible mechanism that underlies

the worse health outcomes for Muslims in all-Muslim communities compared with Muslims in mixed communities, unobserved variations in living standards cannot be ruled out as an explanation.

Customs and practices vary between groups within the same religion, and this variation is not captured within the broad categories of Islam and Christianity. Local practices and traditional beliefs have been incorporated into these two major religions (Lugo and Cooperman, 2010). Although the core analysis conducted for this study showed results from a pooled sample, the implications of varying contexts were explored through an examination of the relationships between religion and child health within each country. Although patterns vary, in general, Muslims have worse health outcomes than Christians, especially within religiously homogenous communities. However, in most countries, there is no difference in child health outcomes between Muslims and Christians within religiously heterogeneous communities. Additionally, Christianity entails many denominations, which to some extent were addressed in this study through separate explorations of the implications of community-level religious composition for Catholics and Protestants. Although no differences were found in mean child health outcomes between Catholics and Protestants, the religious composition appears to be more important for Catholics, whose mortality, like those of Muslims, are higher within religiously homogenous communities.

A limitation of this study was the lack of information on religiosity. Differences relating to religious affiliation were measured based on statements of the mothers, but the extent to which specific religious doctrines were followed and whether fathers were of the same religion was not known. It is, however, reasonable to surmise that reported adherence to a specific religion would be correlated with that religion's prescribed beliefs and behaviors. Implications resulting from differences in religiosity according to community-level religious composition were part of the posited theoretical reasoning for explaining differences in outcomes between religiously diverse and homogenous communities.

To conclude, the findings of this paper were that in communities in which Muslims and Christians coexisted, there were no differences in child health outcomes between Muslims and Christians, indicating that religious affiliation had no direct effects, but that previously observed differences in child health in West Africa were related to the geographical clustering of religious groups. The observed differences in child health outcomes between Muslim and Christian communities in West and Central Africa were explained by covariates reflecting household living standards, health care and contraceptive use, and fertility, which may be underlying explanatory factors.

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Appendix

Table A1. Sample deduction

Remaining sample	Deduction	Remark
111,595		Full sample
110,816	2,149	Missing independent variables
109,446	175	Single birth within ethnicity and region
109,271		Final mortality sample
109,271	12,009	Died
97,262	18,986	Missing height measurement
78,276	8,205	Flagged height measurement
7,0071	127	Missing vaccination information
69,944	93	Single height measure within ethnicity and region
69,852		Final height-for-age sample

Table A2. Full regression outputs for EQ1 and EQ2

VARIABLES	EQ1		EQ2		EQ1		EQ2	
	Height-for-age z-score		Under-5 mortality		RC		RC	
Christians in mixed communities	RC		RC		RC		RC	
Christians in all-Christian communities	0.117 (0.0801)		0.0623 (0.0791)		0.0151* (0.00804)		-0.00213 (0.00904)	
Christians in predominantly-Christian communities	0.0905 (0.112)		0.0445 (0.0888)		0.00434 (0.00964)		-0.00335 (0.0101)	
Christians in majority-Christian communities	0.193* (0.104)		0.116 (0.0815)		0.00502 (0.0102)		0.00641 (0.00979)	
Christians in majority-Muslim communities	-0.155 (0.110)		-0.0879 (0.0980)		0.0122 (0.0120)		0.00621 (0.0123)	
Christians in predominantly-Muslim communities	-0.240 (0.289)		-0.118 (0.263)		0.0136 (0.0218)		-0.00399 (0.00939)	
Muslims in all-Muslim communities	-0.651*** (0.0813)		-0.224*** (0.0848)		0.0508*** (0.00826)		0.00804 (0.00939)	
Muslims in predominantly-Muslim communities	-0.407** (0.0978)		-0.148 (0.0933)		0.0327*** (0.00954)		0.00114 (0.00989)	
Muslims in majority-Muslim communities	-0.190** (0.0895)		-0.0583 (0.0845)		0.0104 (0.00886)		-0.00872 (0.00915)	
Muslims in mixed communities	-0.0254 (0.0732)		-0.0135 (0.0776)		0.00156 (0.00952)		-0.00732 (0.0100)	
Muslims in majority-Christian communities	0.133 (0.105)		0.110 (0.0975)		-0.00981 (0.0109)		-0.0148 (0.0116)	
Muslims in predominantly-Christian communities	0.163 (0.176)		0.168 (0.186)		0.00553 (0.0167)		-0.0138 (0.0169)	
Percentage other religion in community			-0.00299** -0.00797***		0.000334*** 0.000116			

Other religion	(0.00123)	(0.000112)
	-0.167**	-0.122
	(0.0849)	(0.0794)
First of multiple births	-0.481***	-0.841***
	(0.0865)	(0.0870)
Second or higher of multiple births	-0.513***	-0.492***
	(0.0837)	(0.0814)
Female	0.240***	0.246***
	(0.0206)	(0.0201)
Maternal age at interview	0.0163***	0.351***
	(0.00175)	(0.0183)
In(Months since birth)	-0.657***	-1.235***
	(0.0142)	(0.0300)
Maternal education		0.00405
Maternal education ²		(0.0101)
	0.000688	0.000129
Paternal education		(0.000740)
Missing paternal education		-0.0327***
Paternal education ²		(0.00936)
	0.00226***	0.0200
Wealth z-score		(0.000641)
Wealth z-score ²		0.176***
Maternal age at birth		(0.0290)
Maternal age at birth ²		-0.00293
	(0.00935)	(0.00935)
	-0.311***	0.00109
	(0.0230)	(0.00111)
	-0.000285	0.00758***
	(0.00282)	(0.000201***)

Maternal age at first birth	(2.93e-05)	
	-0.0135**	
Birth interval	(0.00628)	(0.000803)
	0.00749***	-0.00259**
Birth interval ²	(0.00152)	(0.000199)
	-3.57e-05***	1.28e-05**
Birth order	(9.80e-06)	(1.30e-06)
	-0.356 ***	-0.0932***
Number of siblings ever born	(0.0276)	(0.00420)
	0.327***	0.0986***
Firsborn	(0.0249)	(0.00375)
	0.228***	-0.0633***
Public health care birth	(0.0537)	(0.00694)
	0.0988***	0.0104***
Private health care birth	(0.0304)	(0.00373)
	0.0979**	0.00261
Other health care birth	(0.0479)	(0.00565)
	-0.0463	0.0190
Place of delivery unknown	(0.120)	(0.0132)
	-0.0318	0.114***
Received any vaccinations	(0.190)	(0.0132)
	0.0739**	
Never migrated	(0.0349)	(0.0349)
	-0.0190	-0.00700**
Rural	(0.0270)	(0.00308)
	-0.120***	0.0122***
Pit toilet	(0.0435)	(0.00408)
	-0.0940*	0.000208
No toilet	(0.0555)	(0.00674)
	-0.115*	-0.0115

Other toilet	(0.0047)	0.0389	(0.105)	-0.0105	(0.0133)
Using modern method		-0.00375	(0.0422)	-0.0335***	(0.00478)
Using traditional method		-0.00686	(0.0542)	-0.0370***	(0.00565)
Non-user - intends to use later		-0.0330	(0.0273)	-0.0177***	(0.00341)
Never in union		-0.232**	(0.104)	0.0192*	(0.0111)
In union: One cowife		-0.0740**	(0.0329)	0.0117***	(0.00375)
In union: More than one cowife		-0.0783*	(0.0419)	0.0280***	(0.00588)
Formerly in union		0.0938	(0.0657)	0.0523***	(0.00849)
Ethnicity and region means					
Christians in all-Christian communities	0.0312	0.0223	(0.256)	(0.0268)	
Christians in predominantly-Christian communities	-0.432	0.0381	(0.291)	(0.0318)	
Christians in majority-Christian communities	-0.0789	-0.000454	(0.317)	(0.0342)	
Christians in majority-Muslim communities	-0.243	-0.00634	(0.389)	(0.0426)	
Christians in predominantly-Muslim communities	0.171	0.0103	(0.776)	(0.0501)	
Muslims in all-Muslim communities	-0.137	0.0302	(0.261)	(0.0270)	

Muslims in predominantly-Muslim communities	-0.221	(0.283)
Muslims in majority-Muslim communities	-0.266	(0.289)
Muslims in mixed communities	0.342	(0.380)
Muslims in majority-Christian communities	-0.910**	(0.389)
Muslims in predominantly-Christian communities	-0.440	(0.582)
Percentage other religion in community	0.00342	(0.00311)
Other religion	-0.182	(0.354)
First of multiple births	-0.180	(1.246)
Second or higher of multiple births	-0.223	(1.159)
Female	-0.143	(1.259)
Maternal age at interview	-0.0720	(0.128)
In(Months since birth)	0.0140	(0.205)
Maternal education	0.0502	(0.0499)
Maternal education²	-0.00287	(0.00422)
Paternal education	-0.0183	(0.0448)
		(0.00507)

Missing paternal education	0.0608*	(0.0348)
Paternal education ²	-0.000253	(0.000361)
Wealth z-score	0.000572	(0.0102)
Wealth z-score ²	0.00101	(0.00328)
Maternal age at birth	-0.00789	-0.0402**
Maternal age at birth	(0.160)	(0.0195)
Maternal age at birth ²	0.00159	-1.18e-05
Maternal age at first birth	(0.00177)	(0.000190)
Maternal age at first birth	0.0564	0.00930*
Birth interval	(0.0455)	(0.00544)
Birth order	-0.0141	0.00138
Birth interval ²	(0.0136)	(0.00155)
Number of siblings ever born	0.000146*	-5.33e-06
Firstborn	(8.28e-05)	(7.63e-06)
Private health care birth	-0.527*	-0.00170
Public health care birth	(0.275)	(0.0396)
Other health care birth	0.178	0.0938

Place of delivery unknown	-0.185**	
Received any vaccinations	(3.188)	(0.0745)
Never migrated	-0.0378	
Rural	(0.160)	
Pit toilet	-0.0977	-0.00253
No toilet	(0.105)	(0.0109)
Other toilet	-0.265**	0.00337
Using traditional method	(0.134)	(0.0135)
Non-user - intends to use later	-0.0433	-0.0155
In union: One cowife	(0.271)	(0.0264)
In union: More than one cowife	-0.0232	-0.00748
Formerly in union	(0.299)	(0.0282)
Constant	0.782**	0.0385
	(0.369)	(0.0444)
	-0.134	0.0185
	(0.213)	(0.0243)
	-0.0291	0.0193
	(0.226)	(0.0240)
	-0.0639	0.0339**
	(0.124)	(0.0136)
	0.295	0.00425
	(0.462)	(0.0523)
	0.205	0.0249
	(0.164)	(0.0192)
	0.345*	0.00427
	(0.187)	(0.0230)
	0.382	-0.0435
	(0.359)	(0.0412)
	-1.463**	0.109***
	(0.0174)	(0.00163)

Observations	69,852	69,852	109,271
R-squared	0.114	0.169	0.023

Estimates are weighted using adjusted sampling weights. Standard errors are adjusted for clustering at the community level. Robust standard errors in parentheses. ** p<0.01, *** p<0.001, * p<0.1. All independent variables were demeaned from a survey specific mean of included observations. Ethnicity and region mean refers to means for all valid observations.

Table A3. Regressions separating Christians into Catholics and Protestants

VARIABLES	EQ1 ¹	EQ2 ¹	EQ1 ¹	EQ2 ¹
	Height-for-age z-score		Under-5 mortality	
Protestant	RC	RC	RC	RC
Unknown Christian	-0.478*** (0.0967)	-0.0439 (0.112)	0.0159 (0.0115)	-0.0228* (0.0126)
Catholic	-0.0180 (0.0473)	-0.0650 (0.0459)	0.00572 (0.00455)	0.000417 (0.00469)
Muslim	-0.569*** (0.0441)	-0.131** (0.0516)	0.0271*** (0.00434)	-0.00620 (0.00551)
Other	-0.335*** (0.0502)	-0.190*** (0.0525)	0.0226*** (0.00608)	0.00563 (0.00607)
Observations	69,852	69,852	109,271	109,271
R-squared	0.106	0.168	0.021	0.063

Notes: Estimates are weighted using adjusted sampling weights. Standard errors are adjusted for clustering at the community level. EQ1: country; months since birth; female; other religion; percentage other religion in a community; twin; a constant term. EQ2: basic controls; rural; maternal education; paternal education; household wealth; maternal age at birth; maternal age at first birth; birth order, birth interval; number of siblings born; place of birth; received any vaccination (height-for-age only); toilet facility; maternal marital status; maternal contraceptive use; region-specific ethnicity fixed effects. ¹Models are as EQ1 and EQ2 without distinguishing between different community-level religious compositions (cr). Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

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Growth and Survival

Sound physical health is a critical component of the human development process, wherein early life and childhood are pivotal periods. Although child health in sub-Saharan Africa has been improving for the past few decades, the region still has the highest mortality rate for children less than five years old, as well as high levels of child morbidity. The most immediate causes of these poor health outcomes are infectious diseases and undernutrition while more remote factors, such as resources available to parents, are also known to play a role in child health. Parents are further embedded in an economic, social and epidemiological environment, which commonly reflects parents' past and present resources, as well as aiding them in or inhibiting them from ensuring the healthy development of their children. The primary aim of this dissertation is to first study the consequences of an adverse environment in infancy on human development; and secondly, to explore disparities in child health as they relate to several parental factors – maternal health, parental education, and religious affiliation. Specifically, the dissertation examines the overlap and interaction between these parental factors and contextual factors in their relationship with child health in sub-Saharan Africa and low- and middle-income countries.



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Department of Economic History
School of Economics and Management

Lund Studies in Economic History 88
ISBN 978-91-87793-50-9
ISSN 1400-4860



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