The socio-demographic promise of expanded female education across sub-Saharan Africa

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Nuffield College

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Thesis Abstract

Sub-Saharan Africa (SSA) is highly vulnerable to both early childhood mortality and weather shocks as a cause of climate change. The region also exhibits very low levels of women's status. The demographic literature suggests that maternal education could help reducing child mortality and increasing women's status, as well as reducing vulnerability to the risks associated with climate change. This thesis contributes to this literature and expands it in several ways. The first essay demonstrates the causal role of maternal education in shaping under-five mortality in Malawi and Uganda. It finds a negative effect that operates through reduced financial barriers to medical care, increased attitudes towards modern health services, and rejection of domestic violence. Moreover, being more educated enhances proximity to a health facility and knowledge about the transmission of AIDS in Malawi, and wealth and improved personal illness control in Uganda. By providing a spatial portrait of male decision-making dominance across SSA, the second essay highlights considerable within and between country heterogeneity in women's status. Using spatial panel data methods, it also finds evidence that diffusion of norms about family decision-making helps explain declining reports of male decision-making dominance over time and that women's education and urbanisation are the main channels through which this diffusion occurs. The latter suggests that women's education and urbanisation are important for the social interaction process leading to the diffusion of norms about family decision-making. The third paper focuses on the in-utero exposure to growing-season droughts and finds that it increases under-five mortality and that the lack of nutritious food among mothers during pregnancy could be driving this effect. It also provides evidence that maternal education may protect against drought-related fatality in early childhood.

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Chapter 1. Introduction[1]

For decades, national governments and the international community have put great emphasis on policies that increase access to education at all levels for all. Education features high in the United Nations' 2030 Agenda for Sustainable Development as one of its goals and through its links to most of the remaining ones. The push to achieve education at all levels and, most importantly, the expansion of access to education particularly for women and girls were promoted not only as desirable ends, but also with the hope that they would accelerate progress to achieve other goals. The latter are aimed at addressing poverty, nutrition, health and wellbeing, and gender equality. Women's education is thus considered important for women as individuals, mothers, and wives, and also a necessary precondition for human and social development.

The promise of expanded female education is also supported by the demographic literature, which demonstrates the relevance of female education as a contributing factor of social and demographic change. In particular, female education is found to be correlated with lower fertility, child and maternal mortality, and higher ages at marriage and first birth (Caldwell 1979, 1980; Cleland and Van Ginneken 1988; Hobcraft 1993; National Research Council 1999). Some of the proposed mechanisms linking education to demographic outcomes include improved women's socioeconomic status, empowerment, and health knowledge, attitudes, and behaviours (Aslam and Kingdon 2012; Basu and Stephenson 2005; Caldwell 1979; Glewwe 1999; Jejeebhoy 1992; Rosenzweig and Schultz 1989).

The empirical association between changes in education and changes in social and demographic processes can be found in many countries and at different educational levels. Such association, however, does not necessarily reflect a causal effect of education and is more complex than usually considered.

[1] Number of words (excluding references and appendices): 32,554.

Because education appears to be most amenable to public policy interventions, an understanding of causality is particularly important from a policy perspective. When a causal relationship between education and socio-demographic outcomes actually exists, an empirical association can help build the case for a role of education in shaping these outcomes. However, in the absence of a causal link, policy changes, such as the elimination of education fees intended to increase access to schooling, might not have the desired positive impacts.

Establishing a causal link is difficult given the many confounders and longitudinal data, that are more suited to this end, are few. Cross-sectional data, that are instead more widely available, could also be employed to test causal relationships with the use of experimental or quasi-experimental designs. Yet, most research to date uses non-experimental cross-sectional designs, thereby not contributing to an understanding of causality.

The relationship between education and socio-demographic outcomes is also complex in the sense that it is not exhaustive of the wide range of effects that education may have. In addition to having direct effects on socio-demographic outcomes, education can also have positive externalities (or spillover indirect effects) that spread into other areas. The direct effects of education mean that a woman's education has an effect on the demographic behaviour of that same woman. Spillover indirect effects instead imply that the demographic behaviour of a woman can also depend on the education of other women living in the same community or in neighbouring communities. This may occur because through social interactions, women exchange information and acquire knowledge from other more educated community members, and this can in turn have positive impacts on certain social and demographic processes (Behrman et al. 2002; Bongaarts and Watkins 1996; Kohler et al. 2001; Montgomery and Casterline 1996).

Another type of education externality can result from the role education might have in protecting from external (contextual) risks that negatively impact socio-demographic outcomes. The ongoing debate about climate change adaptation has highlighted the central role that education, and in particular female education, can have in mitigating the negative impacts of extreme climate events in developing countries (Lutz et al. 2014; Muttarak and Lutz 2014). Educated individuals and societies are "more empowered and hence more adaptive in their response to, preparation for, and recovery from disasters" (Muttarak and Lutz 2014). At the individual level, schooling may serve to reduce vulnerability to natural disasters and act as a substitute of disaster experience in promoting the take-up of preventive measures by improving abstract reasoning and anticipation skills (Hoffmann and Muttarak 2017; Lutz et al. 2014). Similarly, educated societies have better social, economic, and institutional capabilities to successfully adapt to climate change (Kc and Lutz 2014). Despite their importance, the externalities of education are much less investigated compared with the direct effects; as a result, the total effects of education on socio-demographic outcomes may be larger than what is often observed.

Against this background, women's education is likely to remain one of the central elements of the policy debate about how to trigger social and demographic change, especially in low- and middle-income countries where most progress needs to occur. It appears evident that additional research on two fronts is needed. First, more longitudinal or experimental research is necessary to ascertain if a causal relationship between education and socio-demographic outcomes exists. Second, further investigation of the externalities of education can help assessing the total impact of education.

This thesis contributes to filling the aforementioned research gaps. With respect to the causal effects of female education on socio-demographic outcomes, it offers two main contributions. The first essay applies a quasi-experimental design to cross-sectional data from Malawi and Uganda in order to examine the role of maternal education in reducing

child mortality. It also aims at exploring the mechanism linking the two in these two countries. The second essay employs longitudinal data from a set of sub-Saharan African countries to explore whether women's education is causally linked to increases in women's status in the family over time. These essays provide the basis for assessing whether the socio-demographic promise of expanded female education is being achieved.

With respect to the positive externalities of education, this thesis extends the literature on the indirect effects of education in two ways. Again, the second essay distinguishes between direct and spillover indirect effects of women's education on women's status and thus measures the total education effects. This is done by using methodologies from spatial analysis. The third essay provides evidence of the positive externalities of women's education in drought-related fatality reduction in early childhood in SSA. By doing so, this essay supports that education may mitigate the negative consequences of climate change.

The research in this thesis is particularly viable and relevant in SSA for two main reasons. The first relates to the introduction of mass schooling policies aimed at expanding access to education. Many national governments in SSA have been implementing policies for primary education over the past decades and will continue implementing them for secondary and higher education. On average, the female primary school gross enrolment rate in SSA has increased from 66% in 1990 to 96% in 2017 (UNESCO Institute for Statistics). Their implementation allows to identify a causal link of education with socio-demographic outcomes. The second relates to climate change, which has the potential to offset some of the progress in social development achieved so far. As a consequence of climate change, extreme weather events are expected to increase in duration, frequency, and intensity (Collier et al. 2008; Masih et al. 2014). The implications of climate change will be felt mainly among the poorest and most vulnerable segments of the world population, that is people who live in low- and middle-income countries. Therefore,

investigating both the demographic implications of climate change and the possible adaptation is particularly relevant in sub-Saharan African countries, where climate change is expected to exacerbate the vulnerabilities of people.

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Chapter 2. The Causal Effect of Maternal Education on Child Mortality: Evidence From a Quasi-Experiment in Malawi and Uganda[2] [3]

[2] This chapter presents joint work co-authored with Christiaan Monden. Materials from this chapter have been published as: Andriano, L. and Monden, C.W.S. (2019). The Causal Effect of Maternal Education on Child Mortality: Evidence From a Quasi-Experiment in Malawi and Uganda. Forthcoming in *Demography*. http://dx.doi.org/10.1007/s13524-019-00812-3

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Abstract

Since the 1980s, the demographic literature has suggested that maternal schooling plays a key role in determining children's chances of survival in low- and middle-income countries; however, few studies have successfully identified a causal relationship between maternal education and under-5 mortality. To identify such a causal effect, we exploited exogenous variation in maternal education induced by schooling reforms introducing universal primary education in the second half of the 1990s in Malawi and Uganda. Using a two-stage residual inclusion approach and combining individual-level data from Demographic and Health Surveys with district-level data on the intensity of the reform, we tested whether increased maternal schooling reduced children's probability of dying before age 5. In Malawi, for each additional year of maternal education, children have a 10% lower probability of dying; in Uganda, the odds of dying for children of women with one additional year of education are 16.6% lower. We also explored which pathways might explain this effect of maternal education. The estimates suggest that financial barriers to medical care, attitudes towards modern health services, and rejection of domestic violence may play a role. Moreover, being more educated seems to confer enhanced proximity to a health facility and knowledge about the transmission of AIDS in Malawi, and wealth and improved personal illness control in Uganda.

Keywords: Child mortality, Maternal education, Universal primary education, Instrumental variable method, Africa

Introduction

Child survival is a key indicator of social development and remains a serious challenge for developing countries. Although child mortality has decreased dramatically since 1990, still more than 40 children per 1,000 live births died before their fifth birthday in 2016 (United Nations Inter-agency Group for Child Mortality Estimation (U.N. IGME) 2017). Most

under-5 mortality occurs in sub-Saharan Africa, which has experienced a slower reduction in child mortality and currently has the highest under-5 mortality rate in the world (79 per 1,000 in 2016) (U.N. IGME 2017).

Maternal education is considered to be a key factor in reducing child mortality. Gakidou et al. (2010), for instance, estimated that increases in maternal education could account for more than 50% of the worldwide reduction in under-5 mortality between 1970 and 2009. Many others have documented the strong negative association between maternal education and child mortality (e.g., Bicego and Boerma 1990; Caldwell 1979; Cleland et al. 1992), and this association has been a driver for big investments in the education of girls (Schultz 1993). Despite the extensive literature on maternal education and child mortality, few studies have assessed the causality of this relationship. Only four studies have used instrumental variable (IV) methods to study the nature of this relationship in just three sub-Saharan African countries: Uganda (Keats 2016; Makate 2016), Malawi (Makate and Makate 2016), and Zimbabwe (Grépin and Bharadwaj 2015). These studies show mixed results, and only three of them deal with primary education. Our analysis builds on the Universal Primary Education (UPE) reforms that were implemented in Malawi and Uganda in the 1990s. This study contributes to the existing body of work in four ways.

First, we combined individual-level survey data with official district-level statistics on the number of primary schools in order to exploit differences in district reform intensity and birth cohort as instruments for maternal education. Exposure to the UPE reforms depended on both the year and district of birth. Previous studies did not differentiate between districts in Malawi but instead controlled for four regions (Makate and Makate 2016); for Uganda, 10 large regions (Makate 2016) or an indicator for Kampala (Keats 2016) were used to control for region-specific trends. We differentiated between 27 and 38 districts that were responsible for the provision of primary schooling in Malawi and

Uganda, respectively. In this way, we accounted for district-specific trends that affected both women's educational attainment and children's health in both countries.

Second, previous studies of Uganda and Malawi did not fully take into account censoring, thereby potentially introducing selection and recall biases. Our study provides the first IV estimates of the maternal education and under-5 mortality relationship while taking into account right-censoring. We used a Cox proportional hazard model in an IV approach, and doing so affected the results. Although linear specifications—both in this and previous studies (Keats 2016; Makate 2016)—did not provide evidence for a significant effect, we did find, using a Cox model, a significant impact of maternal education in Uganda.

Third, we added consistency to this field, with mixed designs and mixed results, by applying the same IV approach to two countries. And, finally, we added to earlier analyses by exploring a consistent set of mechanisms for the effect of maternal education in both Malawi and Uganda.

Prior Research on the Causal Effect of Maternal Education

The negative relationship between maternal education and child mortality may be at least partly due to omitted variables, such as maternal ability, family background and resources, and community infrastructure, each of which predicts both maternal education and child mortality. Because few studies have addressed this issue empirically, we included other regions than sub-Saharan Africa in our overview.

Studies using family or household fixed effects have suggested that the effect of maternal education on child health outcomes may be overestimated. After unobserved family factors were controlled for, the association of maternal education with child outcomes was attenuated or no longer significant (see, e.g., Horton 1988; Strauss 1990; Wolfe and Behrman 1987).

Another way of addressing omitted variable bias is to use an exogenous source of variation in education that is not related to child mortality—an IV approach. IVs exploit "situations where the forces of nature or government policy have conspired to produce an environment somewhat akin to a randomized experiment" (Angrist and Krueger 2001:73). We focus on this type of quasi-experimental study. To the best of our knowledge, only six studies have examined the causal effect of maternal education on child mortality: Breierova and Duflo (2004), Chou et al. (2010), Grépin and Bharadwaj (2015), Keats (2016), Makate (2016), and Makate and Makate (2016).

Breierova and Duflo (2004) used a large-scale school construction program in Indonesia in the mid-1970s to identify the effect of parental schooling on child mortality. They measured program intensity as the number of new primary schools relative to the number of school-aged children. Using regional differences in program intensity and birth cohorts as instruments for average years of education, they found that an increase in parental education led to a significant reduction in the number of children who died and that there was no significant difference between the effect of mother's and father's schooling. In most cases, their two-stage least squares (2SLS) effects of parental schooling were larger than their ordinary least squares (OLS) effects.

Chou et al. (2010) exploited the 1968 expansion of compulsory schooling in Taiwan to estimate causal effects of parental educational attainment on birth outcomes. The authors instrumented parental education with the interaction between treatment status and program intensity, measured by the number of new schools relative to school-aged children. They found that maternal and paternal schooling significantly reduced the probability of neonatal, post neonatal, and infant mortality. All but one weighted 2SLS (W2SLS) effects were statistically significant, and some were larger than the weighted least squares effects.

Grépin and Bharadwaj (2015) used age-specific exposure to the 1980 expansion of access to secondary education in Zimbabwe to instrument for maternal secondary education. The expansion was part of a much wider package of reforms, introduced by the new independent government. Their IV estimates, based on the Zimbabwe Demographic and Health Surveys (DHS), indicated that maternal secondary education statistically lowered a child's probability of dying by the time of the survey, and before ages 1 and 5. All their IV coefficients exceeded the corresponding OLS coefficients. They found that an additional year of maternal secondary schooling reduced child deaths by about 21%. Although relevant, this study misses the majority of the population because throughout sub-Saharan Africa, especially in the 1980s, the most important and most numerous differences in education were between having no education and having a primary education.

In a recent working paper, Keats (2016) used the 1997 Ugandan UPE reform (described later) to examine the impact of female education on child health outcomes. He employed a dichotomous indicator for treatment status as instrument for maternal schooling, with women aged 7–14 in 1997 serving as the treatment group and women aged 15–22 in 1997 being the control group. His analysis, based on the Uganda DHS, was restricted to (1) women who had completed their education and (2) first births that had happened in the five years prior to the interview. The IV findings showed that an increase in women's education affected their firstborns with regard to immunisations, preventive care, and chronic malnutrition. The IV results for infant mortality (Keats 2016: 23, table 7) instead showed no evidence of significant differences between the firstborns of exposed and non-exposed women. Keats suggested that the null effect of women's education on mortality could be because the firstborn child subsample is likely biased downward as a result of negative selection.

Makate (2016) also used the Uganda DHS and the 1997 Ugandan UPE to estimate the effect maternal education had on child mortality. He compared outcomes of children born to women aged 5–13 in 1997 (treatment group) with those of children born to women aged 17–25 in 1997 (control group). He limited the samples for infant and under-5 mortality to children who had been fully exposed to under-1 and under-5 mortality, ignoring right-censored cases still at risk of dying. This restriction introduced potential selection and recall bias. His IV estimates revealed that an additional year of maternal primary schooling negatively affected infant mortality and child death by the survey date but had no impact on under-5 mortality. The significant IV coefficients exceeded the corresponding OLS coefficients. Makate (2016) found that a one-year increase in maternal education caused a decrease of between 17.5% and 19.3% in child mortality; however, he warned that these results should be interpreted with caution because they were imprecise and may have lacked robustness.

Finally, Makate and Makate (2016) used the Malawian UPE reform of 1994 to examine the impact of maternal education on child mortality. Following Grépin and Bharadwaj (2015), they exploited age-specific exposure to the reform to instrument for maternal primary education, considering women aged 6–13 in 1994 as the treatment group, and women aged 17–24 in 1994 as the control group. Like Makate (2016), they too limited their samples for infant and under-5 mortality to children who had been fully exposed, thus introducing potential selection and recall bias. Using data from the Malawi DHS, they showed that the IV estimates of the effect of maternal schooling on the mortality of infants, under-5 children, and children of mothers aged 19 or younger were negative and larger in absolute value than the OLS estimates. For neonatal mortality, the IV coefficient was not statistically significant. They concluded that an additional year of maternal primary schooling reduced infant mortality by 34% and under-5 mortality by 36%. They also concluded that the mechanisms through which maternal education affects child survival

were increased frequency of prenatal care, improved literacy levels, increased father's educational attainment, and altered fertility behaviours.

Pathways of Influence

Many different channels through which maternal schooling contributes to improved child survival and health have been suggested. Socioeconomic factors, such as income and wealth, are the most consistently and frequently studied mechanisms (Alemayehu Azeze and Huang 2014; Cleland and Van Ginneken 1988; Desai and Alva 1998; Frost et al. 2005; Grépin and Bharadwaj 2015; Keats 2016). However, maternal education may also increase children's health outcomes through a range of other factors, such as attitudes, knowledge, and women's power. Drawing on empirical studies, we identify six major pathways through which schooling may influence child survival. The proximate variables are not mutually exclusive, and this is not an exhaustive list of all potential mechanisms but rather a preliminary explanatory model to be built upon in future research.

The first pathway concerns socioeconomic position. Evidence from Uganda suggests that more-educated women are likely to have better jobs and more wealth (Keats 2016). Uneducated people tend to work in the informal market, whereas more-educated workers in the formal sector receive higher wages (El Badaoui and Rebière 2013).

Second, schooling may alter attitudes towards modern health services through a shift from traditional practices to acceptance of modern medicine and rational explanations of disease. Education in rural Bolivia resulted in a decrease in the use of traditional treatment practices, such as withholding fluids during diarrhoea (Bicego and Boerma 1990). Basu and Stephenson (2005) argued that even slightly educated women were more likely to seek and obtain effective health care to treat their children's illnesses, especially the common ones (which account for the bulk of children's illnesses and mortality).

Third, education may foster personal illness control via preventive care. Results from Basu and Stephenson's (2005) study of India suggest that completed primary education results in higher odds of various measures of personal illness control (e.g., seeking treatment for their child's cough/fever, receiving prenatal care in pregnancy, and receiving prenatal care in the first trimester). Children of more-educated mothers in Uganda are more likely to be immunised and to receive vitamin A supplements to prevent blindness, diarrhoea, and measles (Desai and Alva 1998; Keats 2016).

Fourth, schooling may affect environmental factors via good use of sanitation and hygiene practices in the home and greater access to health facilities. Previous studies (Hatt and Waters 2006; Hobcraft 1993) found that more-educated mothers are more successful at reducing the prevalence of diarrheal diseases. Moreover, women with higher levels of education tend to migrate to communities with (better) amenities and to have (greater) access to medical facilities (Desai and Alva 1998).

Fifth, schooling may facilitate the acquisition of health knowledge (Glewwe 1999). Education is related to knowledge of contraceptive methods and their more efficient use (Rosenzweig and Schultz 1989) as well as healthy behaviours during pregnancy (Grossman 1972). Agüero and Bharadwaj (2014) found that education increases the knowledge of HIV-preventive behaviours and HIV transmission in Zimbabwe.

Sixth, schooling may open new opportunities for empowerment and autonomy in the household. Increased female education has been shown to have a positive effect on women's relative position in the household (Jejeebhoy 1992; Thomas 1990). For instance, evidence from Ethiopia suggests that education positively influences women's participation in household decision-making (Behrman 2015).

Universal Primary Education Reforms in Malawi and Uganda

During the 1990s, several African governments started to eliminate primary school fees (World Bank 2009). Malawi and Uganda were among the earliest countries to adopt UPE policies in sub-Saharan Africa and are the only countries for which the available data allows a comparison between women who did and did not experience free primary education and who are old enough to have completed their schooling and have had children.

In Malawi, the reform's launch coincided with the introduction of multiparty democracy. School fees were abolished, grade by grade, starting in 1991. In September 1994, a so-called big bang approach was adopted, and free primary education was strongly enforced (Avenstrup et al. 2004). Besides the abolition of tuition fees (between US$2.35 and US$5.18 per pupil per year), uniforms were made optional, and the government paid for textbooks and exercise books (Kadzamira and Rose 2003; Rose 2002). The government also funded learning materials, classrooms, furniture, teachers' houses, sanitation facilities, and boreholes (World Bank 2009). Primary school is made up of eight years, and the entry age is 6. In 1994, there were three school terms per year for primary schools, running generally from September to December, January to April, and April to July. The reform was implemented in September 1994, and tuition fees were abolished for all grades of primary school. Therefore, girls who were born in 1980 or earlier were not exposed to the reform, whereas girls who were born in 1981 or later were exposed to the reform.

In Uganda, a UPE policy reform was prepared in 1987, but lack of resources and political constraints impeded its implementation. The Free Primary Education reform was finally implemented in January 1997. Initially, free primary education was provided for up to four children per family, and at least two of the four children had to be girls, with priority given to disabled children. In practice, the abolition of school fees (of US$4.62 to US$7.48 per pupil per year) was applied to all children in primary school (Grogan 2006;

Riddell 2003). The government assigned each school a capitation grant for instructional materials, extracurricular activities, maintenance and utilities, and administration. In addition, a facilities grant was made available to assist schools in the construction of classrooms, latrines, and teachers' houses, and for buying furniture (Essama-Nssah et al. 2008; Grogan 2006). Primary school consists of seven years of education. The academic year starts in February and ends in December, and the entry age for primary school is 6. All children who turn 6 years old in the calendar year start school, yet the age of entry varies between ages 5 and 7 (UNESCO 2015). Therefore, girls who were born in 1983 or earlier were not exposed to the reform, whereas girls who were born in 1984 or later were exposed to the reform.

Note that using these calendar-year cut-offs would assume no grade repetition or late entry into primary school. This might seem a very strong assumption, and we found that it is, in fact, not plausible in either country: empirical evidence shows that the actual age of exit from primary school was 17 years in Malawi and 16 years in Uganda (see Fig. 2.A1 in the online appendix). Using the same approach as Grépin and Bharadwaj (2015), we excluded women outside the official primary school age and thus only partially exposed to UPE in order to get a better picture of the schooling discontinuity. Therefore, our analysis compares the outcomes for women who were born between 1981 and 1987 with the outcomes for women who were born between 1969 and 1976 for Malawi, and the outcomes for women who were born between 1984 and 1991 with the outcomes for women who were born between 1974 and 1980 for Uganda.

Evidence from both countries indicates that the UPE reforms substantially improved enrolment. In Malawi, enrolment in primary education increased by 51% in the first year, from 1.9 million in 1993 to 2.9 million in 1994 (Al-Samarrai and Zaman 2002; Inoue and Oketch 2008). In Uganda, it rose by 68%, from 3.4 million in 1996 to 5.7 million in 1997, and jumped by 140% over six years (Avenstrup et al. 2004).

Evidence also suggests that the quality of education declined because of the rapid expansion of primary school enrolment (World Bank 2009). In particular, in Malawi, the government responded to the extraordinary increase in the enrolment rate in 1994–1995 with a more than proportional increase in the number of teachers; however, about one-half of the new teachers were untrained (compared with 16% in 1993–1994), and the other half had inadequate training (Avenstrup et al. 2004; Education Management Information Systems (EMIS) 1994; Inoue and Oketch 2008). In addition, a shortage of books, school materials, and permanent classrooms led to a more intensive use of existing facilities (Inoue and Oketch 2008; Kadzamira and Rose 2003). In Uganda, the access shock to education caused an initial decrease in various indicators, referring to the resources available to pupils. In particular, the increase in the sectoral budget in the years following the reform was not sufficient to reduce the textbook/pupil and teacher/pupil ratios. According to the national education statistics, the number of primary school teachers and classrooms, respectively, increased by only 71% and 55% between 1996 and 2002 (Avenstrup et al. 2004).

To accommodate the expected increase in primary school enrolment, governments in both countries opened new primary schools. The number of primary schools increased by 15% in Malawi, from 3,216 in the 1993–1994 school year to 3,706 in 1995–1996 (EMIS 1994, 1996a); the corresponding increase in Uganda was 55%, from 8,526 in the 1995 school year to 13,218 in 2001 (EMIS 1996b, 2001).

A notable aspect of the school construction program in both countries is that its intensity varied across districts (see the online appendix for more details). To capture the intensity of the program, we focused on the change in the number of primary schools between 1994 and 1996 in Malawi, and between 1995 and 2001 for Uganda. This measure, calculated as $\frac{(primaries_1 - primaries_0)}{primaries_0}$, is based on official statistics provided by the respective ministries for education (see the online appendix) and reflects the scale of the

changes when the baseline level is taken into account. Previous studies have used similar approaches (Breierova and Duflo 2004; Chou et al. 2010; Duflo 2001; Osili and Long 2008). In Malawi, the mean and standard deviation of program intensity are 0.18 and 0.24, respectively; in Uganda, these are 0.65 and 0.96, respectively. Changes in the number of enrolled children in primary school were also not distributed evenly. In Malawi, the mean and standard deviation of the relative difference between 1994 and 1996 are 0.55 and 0.35, respectively; in Uganda, the mean and standard deviation of the relative difference between 1995 and 2001 are 1.35 and 0.51, respectively.

The introduction of UPE had a major impact on pupil participation and the number of primary schools. In Malawi, districts that saw the largest relative increase in the number of primary schools (i.e., high-intensity districts) experienced the largest increase in the number of enrolled children in primary school. On the other hand, in Uganda, high-intensity districts experienced an increase in the number of children enrolled in primary schools but this was not as high as that experienced by low-intensity districts. In both countries, the allocation of additional schools is weakly correlated (<.2) with the number of school-aged children—based on official statistics (EMIS 1994, 1996a,b, 2001) and the 1991 Uganda census data (Minnesota Population Center 2017)—in the district before the reform. This correlation, however, is based on small samples (27 and 38 districts for Malawi and Uganda, respectively), and population density and school size vary considerably between and within districts. We could not identify a systematic allocation pattern of additional schools. Reform intensity, however, clearly varied substantially and affected enrolment, as documented in official statistics. We want to emphasise that although the quality of education deteriorated after the reform, it did not decrease more in high-intensity districts: we could not find any systematic relation between reform intensity and changes in quality of education as measured by relative differences in the pupil/teacher

ratio between 1994 and 1996 in Malawi and between 1996 and 2000 in Uganda (EMIS 1994, 1996a,b; Uganda Bureau of Statistics 2002a).

Empirical Strategy

Data

The individual-level data are from the DHS (see the online appendix for additional information). For Malawi, we used pooled data from the 2000, 2004, and 2010 DHSs. For Uganda, the DHS surveys from 2000–2001, 2006, and 2011, and the DHS–Malaria Indicator Survey (MIS) from 2009 were pooled. Although all the individual surveys we selected were designed to be nationally representative, the actual sampling procedures vary between surveys, which may introduce uncertainty about the extent to which the final sample is nationally representative. However, we cannot be sure about the size or direction of the potential bias. Pooling is necessary to allow for adequate sample sizes of women of the all ages for our design. In order to consider only those women who had completed their education, we included women who were at least 18 or 19 years old when the survey was conducted in Malawi and Uganda, respectively. The percentage of women who continue their studies after age 18 or 19 is lower than 15% in both countries (see the online appendix for calculations). In addition, we excluded all children who were not living with their mothers at the time of the survey. We also excluded visitors to the household. Table 2.1 shows the descriptive statistics of the pooled samples.[4]

[Table 2.1 here]

Figure 2.1 charts educational attainment by mother's year of birth. On average, treated women in Malawi and Uganda had more education than untreated women. (See Fig. 2.A2 in the online appendix for further evidence of the differences in educational attainment.)

[4] Descriptive statistics were weighted by DHS sample weights.

[Figure 2.1 here]

Figure 2.2 shows the mortality of children under age 5 born to all women in our sample, by mother's year of birth. In both countries, mortality was lower for children of treated mothers.

[Figure 2.2 here]

Instrumenting Maternal Education

We took advantage of the timing of the UPE reform to instrument maternal education using exposure to the reform. The mother's year of birth and the administrative unit in which she started and completed primary school jointly determined exposure to the reform.

Using information on districts assumes that women living in a particular district at the time of the interview also had acquired their education in that district. DHS does not collect information on the district where women started and completed primary school or on where they were born. Thus, if women had changed their district of residence since primary school age, we were unable to assign an exact value of program intensity. We used current district of residence as a proxy for district of education, under the assumption that women had not moved since they started primary school. This seems to be a plausible assumption: the last censuses in Malawi (2008) and Uganda (2002) showed that internal migrants accounted for, respectively, 16% and 13% of the total population of the country (National Statistical Office 2009; Uganda Bureau of Statistics 2002b). In the Results section, we provide further evidence that using district of residence did not affect our results and conclusions. Nevertheless, available data did not allow us to analyse and control for fostering during childhood. Child fostering is a common childcare practice across sub-Saharan Africa and partly serves as a mechanism for households to enhance schooling opportunities for children (Lloyd and Desai 1992). It could affect our results if

women were sent to live in a different household in a different district when they were school-aged. We should consider this limitation when interpreting our results.

The combined effect of the UPE program, through the increase in number of primary schools and elimination of primary school tuition fees, provided a quasi-natural experiment that allowed us to instrument maternal schooling and evaluate its impact on under-5 mortality. The first-stage regression model that we estimated for years of schooling attained by women in cohort *a* (with the oldest cohort as the omitted cohort) and district *k* reads as follows:

$$S_{iak} = \sum_a C_a\gamma_a + \sum_k X_k\alpha_k + \sum_a(C_aP_k)\beta_a + \sum_a(C_aE_k)\delta_a + \sum_a(C_aN_k)\theta_a + \boldsymbol{R}_{iak}\boldsymbol{\eta} + \nu_{iak}, \quad (1)$$

where S_{iak} is the endogenous variable, comprising years of education of woman *i*, born in year a and district *k*; C_a is a dummy variable for cohort a; X_k is a dummy variable for district k; P_k is program intensity in district *k*; E_k is the number of girls in primary school before UPE in district *k*; N_k is the number of primary school–aged children before UPE in district *k*; $\mathbf{R}_{iak}$ is the categorical variable religion (Catholic, Presbyterian, Muslim, other Christian, no religion, and other; not available for Uganda); and ν_{iak} is the error term. By including the number of primary school–aged girls enrolled before UPE and the number of primary school–aged children before UPE, each interacted with the cohort dummy variables, we controlled for time- and district-varying factors correlated with pre-program enrolment and captured yearly and district differences in the demand for education.

Panel a of Fig. 2.3 shows the coefficients and the confidence intervals of the interactions between year of birth and program intensity in the woman's district of residence in Malawi. Panel b illustrates the corresponding coefficients in the Uganda equation. In both countries, the coefficients are 0 for the oldest cohorts and are statistically different from 0 for the youngest cohorts. These results suggest that the program intensity

effects were restricted to the treatment group and that cohorts in the control group were not affected by the program.

[Figure 2.3 here]

The *F* ratio of the test—in which the coefficients of the interactions between year of birth and program intensity for the youngest cohorts are statistically significant as a set—is 32.2 for Malawi and 93.9 for Uganda. Because the coefficients of the interactions between year of birth and program intensity are 0 for the oldest cohorts, and different from 0 for the youngest cohorts, we could reduce the number of program intensity coefficients that must be estimated by imposing a simple restriction. This can reduce the degree of multicollinearity among the independent variables and improve the precision and efficiency of the estimates of the effect of the program. The model reads as follows:

$$S_{iak} = D_a\gamma + \sum_k \mathrm{X}_k\alpha_k + (D_aP_k)\beta + (D_aE_k)\delta + (D_aN_k)\theta + \mathbf{R}_i\boldsymbol{\eta} + \nu_{iak}, \qquad (2)$$

where D_a is a dummy variable taking the value 1 if the mother is in the treatment group, and 0 otherwise.

Cox Specification

In linear models, the 2SLS approach is used to address endogeneity by replacing the endogenous value with the predicted value for education from the first-stage estimation. Because our outcome was risk of death up to age 5, we used a survival model to account for the right-censoring of the data (Allison 1982). When survival models are used, the two-stage residual inclusion (2SRI) approach, which is identical to the 2SLS in a linear setting, has been shown to yield consistent estimates (Atiyat 2011; Terza et al. 2008). Unlike the 2SLS, in the second stage of the 2SRI regression, both the first-stage residuals, S_{v_i}, and the

endogenous variable, S_i, are included in the model to be fitted. We estimated a Cox proportional hazards model for right-censored data[5] (Cox 1972):

$$h(t) = h_0(t)e^{(\sum_k X_k\alpha_k + S_i\rho_1 + S_{v_i}\rho_2 + (D_aE_k)\delta + (D_aN_k)\theta + \mathbf{R}_i\boldsymbol{\eta} + G_j\pi + B_j\tau + \sum_y C_y\lambda_y + \varepsilon_{jyak})}, \quad (3)$$

where $h_0(t)$ is an unspecified baseline hazard function, G_j is child sex, B_j is child birth order, and C_y is a dummy variable for child cohort y. We controlled for child birth order with indicator variables for (1) first birth and (2) second birth and more. Child cohort was represented by indicator variables for born in Malawi in 1995–1999, 2000–2004, and 2005–2010, and for born in Uganda in 1995–2000, 2001–2006, and 2007–2011. Given the timing of the surveys and our sample selection (i.e., births occurred five years prior to the survey), child cohort further captured potential effects from pooling data across surveys. All coefficients are log hazard ratios, and ρ_1 is a consistent estimate for the true effect of maternal education on under-5 mortality. Therefore, $\exp(\rho_1)$ is the hazard ratio associated with a one-year increase in maternal education, and $(\exp(\rho_1) - 1)$ is the effect of an additional year of maternal schooling on the probability of dying before age 5 for children of compliers (i.e., mothers going to primary school if eligible, and not going if not eligible). If $\exp(\rho_1)$ is smaller than 1 and statistically different from 0, there is a causal negative relationship between maternal education and under-5 mortality. The ρ_2 is the effect of the first-stage residuals on under-5 mortality; its interpretation is equivalent to that of the Wu-Hausman test in a 2SLS framework, wherein a statistically significant coefficient indicates endogeneity in the relationship between maternal education and under-5 mortality. Moreover, the Cox model assumes that the hazards are proportional over time. Proportionality of the effect of years of education was confirmed by testing the slope of Schoenfeld residuals. Furthermore, simulations have shown that unadjusted standard errors are accurate when the 2SRI approach is used (Atiyat 2011:27).

[5] The Cox proportional hazards model is preferred over a discrete-time event history model, which is widely used to estimate child mortality. The exogeneity of excluded instruments is hard to assess in the second model given its complex structure.

To quantify the magnitude of the effect of maternal schooling, we estimated the population attributable fraction (PAF) of under-5 mortality associated with maternal schooling (Chen et al. 2010)—that is, the fraction of under-5 mortality cases that would not have occurred if mothers had had some education. In the presence of confounders, $\boldsymbol{W}$, we used the following formula:

$$PAF = \frac{pr(D=1) - \sum_{k=1}^{m} pr(\boldsymbol{W}=\boldsymbol{w}_k) pr(D=1|Z=0, \boldsymbol{W}=\boldsymbol{w}_k)}{pr(D=1)}, \quad (4)$$

where D is the binary status variable (alive or dead), Z is a binary exposure indicator, and $\boldsymbol{w}_1, \ldots, \boldsymbol{w}_m$ are the m levels of $\boldsymbol{W}$. In our case, where the exposure variable is continuous, an analogous formula involves integration of the exposure level distribution. To calculate excess deaths associated with low maternal education, the PAF is then multiplied by the total number of under-5 deaths (data U.N. IGME, 1990–2016) in Malawi occurring in the 1995–2010 birth cohort in 2003; and in Uganda, occurring in the 1995–2011 birth cohort in 2004, which was the mean year of death in the samples.

Pathways

We studied six pathways through which maternal education might reduce child mortality. We used the 2SLS strategy and regressed the pathway indicators on the predicted value of maternal education from the first stage. Table 2.2 gives descriptive statistics for the pathway indicators.

[Table 2.2 here]

We used two indicators for socioeconomic status, the first pathway: (1) the DHS comparable wealth index, based on ownership of durable goods and quality of housing; and (2) a binary variable indicating whether the woman did not consider money a barrier to obtaining medical care (1 = money not a barrier).

For the second pathway—attitudes towards modern health services—we used a binary variable of whether the woman used modern contraception (1 = yes).

For the third pathway—personal illness control—we used a latent variable, based on a factor analysis of three variables: (1) the number of tetanus injections received during pregnancy; (2) whether the woman had seen a health professional during pregnancy; and (3) the number of antenatal visits during pregnancy. Higher scores indicate higher personal illness control.

The fourth pathway explored the impact of maternal education through environmental factors, as measured by a binary variable of whether the distance to a health facility was not a big problem (1 = not a big problem).

The fifth pathway—health knowledge—was measured by three indicators: knowledge about contracting AIDS; knowledge about transmitting AIDS; and knowledge about the ovulatory cycle (see the online appendix for exact questions). Three questions captured knowledge about contracting AIDS, two questions were related to knowledge about its transmission, and a single question tested the women's knowledge about ovulation. Factor analysis showed that the five questions on AIDS loaded on two factors, with an eigenvalue larger than 1. Higher scores mean greater health knowledge.

Finally, we used two indicators for the sixth pathway, women's empowerment. Eight questions capturing women's empowerment were consistently available across DHS surveys. Based on a factor analysis showing two factors with an eigenvalue greater than 1, we distinguished two indicators. The first, decision-making, used three questions about the husband's and wife's roles in decision-making in the household with regard to woman's health care, large household purchases, and visits to family and friends. The second indicator, empowered domestic violence, was based on five questions on whether a man is justified in beating his wife/partner under various circumstances: if she (1) went out without telling him; (2) neglected the children; (3) argued with him; (4) refused to have sex with him; and (5) burned the food. The five items were coded as 1 if the man is not viewed

as justified, and 0 otherwise. Higher scores on both indicators represent higher empowerment for women.

Results

Exposure to UPE and Maternal Education

Did the UPE reform affect maternal education? Table 2.3 shows the effects of UPE on maternal education in Malawi and Uganda. In Malawi, gains in educational attainment were larger in high-intensity districts compared with low-intensity districts. In Uganda, however, educational attainment increased more in high-intensity districts compared with low-intensity districts. The estimates suggest that a 1 percentage point increase in reform intensity is associated with an increase in years of education for the youngest cohorts of 1.7 (i.e., 1.644 + 0.01) and 1.5 (i.e., 1.490 – 0.002) years in Malawi and Uganda, respectively.

For UPE exposure to be used to instrument for maternal schooling, it must be strongly correlated with maternal years of schooling. An instrument is considered relevant if the *F* statistic on the excluded instrument in the first stage is greater than 10 (Staiger and Stock 1997); however, this rule of thumb is invalid when multiple instruments are used in the first-stage regression. In this case, two more powerful tests are the Cragg–Donald Wald *F* statistic and its robust counterpart, the Kleibergen–Paap Wald *rk F* statistic. Stock and Yogo (2005) compiled critical values for these statistics for 2SLS estimators: if the test statistics are below these critical values, the instruments are considered weak. We tested the strength of the set of instruments by comparing the Kleibergen–Paap Wald *rk F* statistic with the Stock and Yogo (2005) critical value of 19.93 for 10% maximal test size distortion.[6] The *F* statistics of 57.1 and 23.3 indicate that our instruments are not weakly correlated with schooling.

[Table 2.3 here]

[6] The Stock–Yogo test of maximal bias is not available unless the number of overidentifying restrictions is at least 2.

The use of district of residence as a proxy for district of birth introduced concerns about migration. Was district of residence exogenous to the reforms? Cross-district migration patterns in census data suggest that among districts with high internal migration rates, those experiencing a net gain in population (i.e., having more immigrants than emigrants) were both high- or low-intensity districts and that districts experiencing a net loss in population (i.e., having more emigrants than immigrants) were both high- or low-intensity districts (National Statistical Office 2009; Uganda Bureau of Statistics 2002b). In other words, internal migrants had not moved to high-intensity districts only, and the decision to migrate to one district rather than another was not associated with the reform. Nevertheless, two main sources of bias may arise from measurement error. One source of bias could be introduced by child fostering. In particular, if women were sent to live in a different household in higher-intensity districts during childhood, we might have overestimated the value of β from (Eq. (2)). We could not control for this mechanism in our analysis, and this might limit our results. Furthermore, women might have migrated after completing primary education, and this might have resulted in some bias in our β estimate from (Eq. (2)). In particular, if women had migrated to higher-intensity districts, we might have underestimated the value of β, the opposite being true if women had migrated to lower-intensity districts. The districts with a sizable share of immigrants were Lilongwe City (Malawi) and Kalangala (Uganda): 52% and 38% of their populations, respectively, migrated from other districts. Therefore, we performed an analysis without Lilongwe City and Kalangala to account for possible measurement error. Table 2.4 shows that the bias in β was negligible and that the results were robust to excluding Lilongwe City and Kalangala.

[Table 2.4 here]

Maternal Education and Child Mortality

We now move to the effect of maternal education on child mortality. We estimated two Cox models for each country: one model treating maternal schooling as exogenous, and another estimated using the 2SRI approach that treats maternal education as endogenous. Table 2.5 shows the results of the impact of maternal education on child mortality.

In line with the literature, the simple Cox model suggests that increased female education had a negative impact on child mortality in both Malawi and Uganda (columns 1 and 3, Table 2.5). In particular, in Malawi, the odds of dying for children of women with one additional year of education were 2.9% lower (column 1). The model in column 2 accounts for the endogeneity of education and shows that the effect of maternal education on child mortality was even larger, with a one-year increase in education significantly reducing the probability of dying before age 5 by about 10.0%. In Uganda, the odds of dying for children of women with one additional year of education were 4.9% (column 3). Here, too, the causal relationship between maternal education and child mortality was even stronger when we controlled for the endogeneity of the education variable: for each additional year of education, children had a 16.6% lower probability of dying (column 4). The first-stage residuals had a positive impact on under-5 mortality, revealing that more-educated women had, on average, unobservable characteristics that were associated with a higher level of child mortality (columns 2 and 4) (i.e., negative selection). The effect, however, is not statistically significant (p value of ρ_2 is .15 for Malawi and .18 for Uganda), which indicates both that maternal education was not endogenous and that the conventional Cox estimator was consistent. These results further indicate that the conventional Cox estimate is smaller than the 2SRI Cox estimate, although not significantly, which suggests that omitted variable bias was not a significant factor.

[Table 2.5 here]

Furthermore, as a robustness check, we performed the same analysis excluding Lilongwe City (Malawi) and Kalangala (Uganda). Table 2.6, columns 1 and 3, indicate that the results are robust to excluding Lilongwe City and Kalangala. In addition, for the sake of comparing our results with those in the three previous studies analysing the relationship between maternal education and child mortality (Keats 2016; Makate 2016; Makate and Makate 2016), we specified a linear model estimation for under-5 mortality (i.e., we used a 2SLS approach). The findings show that the negative impact of maternal education on under-5 mortality lost statistical significance in both countries (Table 2.6, columns 2 and 4). These findings indicate that ignoring the right-censoring issue of the data can lead to misleading conclusions.

[Table 2.6 here]

We now turn to the possibility that our instruments are correlated with under-5 mortality. The exclusion restriction assumption says that the reform did not affect under-5 mortality directly, only through its effect on maternal schooling. We tested this restriction following the methodology described by Bollen et al. (1995)—that is, by testing the null hypothesis that the instruments are uncorrelated with the error term in the second stage. The test involves including all but one excluded variable in the equation that controls for endogeneity of maternal education and testing that the variable is a significant predictor (Bollen et al. 1995). A rejection of the null hypothesis would cast doubt on the validity of the instruments as well as on the quality of the second-stage estimates. The test statistic failed to reject the null hypothesis of instrument exogeneity in both countries (*p* value was .1 for Malawi and .7 for Uganda; see Table 2.5), pointing to the validity of the overidentifying restrictions.

We also calculated estimates of attributable mortality and excess deaths to low maternal education. Given both the loss in efficiency associated with 2SRI and the consistency of the conventional Cox estimator in both countries, we used the hazard ratios

from models in columns 1 and 3 of Table 2.5 to calculate the PAF. The median PAF was –12.2% (95% confidence interval (CI): –18.1% to –6.6%) for Malawi and –23.9% for Uganda (95% CI: –33.7% to –14.9%). An increase in maternal education would have led to a 12.2% and 23.9% reduction in under-5 mortality risk in Malawi and Uganda, respectively. This translates into 8,477 (of 69,569) and 39,296 (of 164,406) excess deaths in Malawi and Uganda, respectively.

The UPE reform may have affected both the quantity and quality of education. This matters for our interpretation of the effect of education on child mortality. In particular, if the quality of schooling had increased after the reforms, this would affect our interpretation of ρ_1 from (Eq. (3)). If UPE significantly affected both the quantity and quality of education, ρ_1 would still capture the effect of the reform but not the effect of an additional year of education. However, we found no evidence that the quality of education provided improved after the reforms. In fact, it seems more likely that education quality indicators decreased for Malawi and Uganda in the late 1990s and early 2000s (Avenstrup et al. 2004). To examine whether UPE affected quality of education, we compared the returns to education for the oldest cohorts in our treatment group (i.e., Treatment 1 (T1): 11–13 years old at UPE) with the returns for the youngest cohorts in our treatment group (i.e., Treatment 2 (T2): 8–10 years old at UPE) with the returns for younger cohorts (i.e., Treatment 3 (T3): 5–7 years old at UPE). Individuals were differently affected by UPE: compared with women in T1, those in T2 were exposed to the reform longer, and those in T3 were exposed to the program since the beginning of their education. If quality of education had increased, we would expect the effects of education on child mortality to be statistically different. The coefficients for T1, T2, and T3 are not statistically different from one another, however. Values of the *t* test of the difference in the education coefficients between T1 and T2, between T1 and T3, and between T2 and T3 are –0.05, 1.2, and 1.3 in the Malawian sample; these values for the Ugandan sample are –0.4, –1.2, and –0.9. This

finding suggests that the UPE policy affected child mortality mainly through the quantity of education.

Maternal Education and Pathways of Influence

Next we examined various mechanisms through which maternal education might reduce child mortality. These mechanisms are grouped into broader categories in Table 2.7 (see the online appendix for reduced-form estimates). With regard to socioeconomic position, schooling had a significant positive effect on wealth in Uganda only, and having more education increased the probability of not considering money to be a barrier to medical care in both countries.

In both countries, schooling had a significant effect on the odds of use of modern contraceptive methods. The reduced-form results confirm that exposed women were more likely than non-exposed women to use modern contraceptive methods. With regard to personal illness control, increased education had a significant effect in Uganda only.

For the environmental pathway, exposure to the reform was associated with increased proximity to a health facility. In Uganda, the effect was not statistically significant.

Did education affect health knowledge? For Malawi, schooling increased knowledge about the transmission of AIDS, but there was no significant effect for Uganda. The reduced-form results confirm that exposed women had more knowledge about the transmission of AIDS. Counterintuitively, maternal schooling decreased correct knowledge about ovulation in Malawi; however, it is not clear that the given answer categories allowed for a clear distinction and weighting between correct, semi-correct, and incorrect answers (see the online appendix). Therefore, we should be very cautious in generalising such a specific result.

Finally, we examined whether female empowerment was shaped by schooling. In both countries, schooling had no significant effect on women's participation in household

decision-making; however, more-educated women were more likely to reject domestic violence. The reduced-form results suggest that exposed women were more likely than non-exposed women to reject domestic violence.

[Table 2.7 here]

Conclusions

We investigated the causal effect of maternal education on child mortality by taking advantage of educational reforms that introduced free primary education in Malawi and Uganda in 1994 and 1997, respectively. In both countries, the reform caused an increase in maternal schooling. We modelled child mortality using a Cox model to take censoring into account. Findings from these models suggest that each additional year of education caused a 10.0% and 16.6% lower probability of dying before age 5 in Malawi and Uganda, respectively. These estimates are higher than, but not significantly different from, conventional estimates. Therefore, our findings do not support the idea that the relationship between maternal education and under-5 mortality is biased upward as a result of omitted variables. These findings are in line with those of similar studies exploiting exogenous variation in education in developing countries, which obtained IV estimates that were greater than OLS estimates (Breierova and Duflo 2004; Chou et al. 2010; Grépin and Bharadwaj 2015; Makate and Makate 2016). Our PAF estimates (of 12% and 24% for Malawi and Uganda, respectively) are consistent with the range of estimates reported in these studies (i.e., PAFs of 8% to 36%).

Our findings differ from those reported by Keats (2016) and Makate (2016), however, who found a nonsignificant effect of maternal education on child mortality. There are two main possible explanations for this inconsistency. First, these prior studies used different samples, which could have biased their estimates. In particular, Keats (2016) considered only firstborn children, and Makate (2016) instead analysed all fully exposed

births. As discussed earlier, using different samples leads to selection bias. Previous studies have shown that birth-history data in sub-Saharan African DHSs can suffer from omission and backward displacement of births, and they are thus vulnerable to the underreporting of births and deaths of children for five years before the survey (Pullum et al. 2013; Schoumaker 2011); this is particularly true for less-educated mothers (Schoumaker 2011). Second, Makate (2016) and Keats (2016) used a linear specification in the second-stage model; we used a Cox model, and this leads to different results.

We also assessed whether quality of education might have influenced our results, but we found that the UPE policy affected child mortality mainly through quantity of education. Despite the established causal relationship, we did not question whether the quality of education was unnecessary in triggering demographic change. The UPE policy was meant to increase the quantity of education, and this analysis considered only the effects of the expansion in school participation and, in doing so, tried to isolate those effects. The lack of detailed information about the efficiency of the two reforms, local administrative wastage, and corruption—and their district variation—limits our understanding of the role of quality of education and its relationship with reform intensity, which could introduce some bias in the modelling. More in-depth studies on the *quality* of education are needed to examine how the causal effect was brought about.

Our study provides evidence of the potential mechanisms underlying the impact of maternal education on child mortality. We showed that financial barriers to medical care, attitudes towards modern health services, and rejection of domestic violence may play a role. Moreover, being more educated seems to have enhanced proximity to a health facility and knowledge about the transmission of AIDS in Malawi, and wealth and improved personal illness control in Uganda. We could explore only a limited number of variables in DHS, and we had to rely on very specific indicators for each pathway. Hence, we have to be careful in generalising the results. Where we do not find evidence for a pathway, the

lack of evidence might be due to the particular way of measuring the pathway. Further studies are needed to examine the causal relationship between education and possible mediating variables in more detail. In particular, more work is required to understand how health knowledge is shaped by education.

We focused exclusively on maternal education, thus ignoring paternal education, even though it may be important, too (Chou et al. 2010). However, we had only one reform to instrument education with, and instrumenting two variables in one model was not feasible. Moreover, it is well known that the education of husbands and wives is strongly correlated (Smits et al. 1998), which would complicate identifying any independent effects. Also, including fathers would have reduced the sample size, and it is not always certain that the woman's partner is actually the child's father. Nevertheless, we acknowledge that the estimates for maternal education might be overestimated due to omission of the variable paternal education (Breierova and Duflo 2004; Chou et al. 2010).

To conclude, our findings show that the reforms in Malawi and Uganda were effective in increasing maternal education and, as a consequence, in decreasing child mortality. They suggest that in other sub-Saharan countries, which have more recently started to eliminate primary education fees as part of the UNESCO Education for All initiative, free primary education for girls could have similar implications for reducing child mortality. Finally, the UPE policy induced variation in access to schooling only at the primary school level; however, the effect of more schooling at the secondary level may be different. In 2007, the government of Uganda was the first in sub-Saharan Africa to implement a free universal secondary education policy. This reform provides an important opportunity to assess the causal effect of continued education and address some of the aforementioned limitations and remaining questions.

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Fig. 2.1 Average years of education by mother's year of birth (averages and polynomial trend lines).

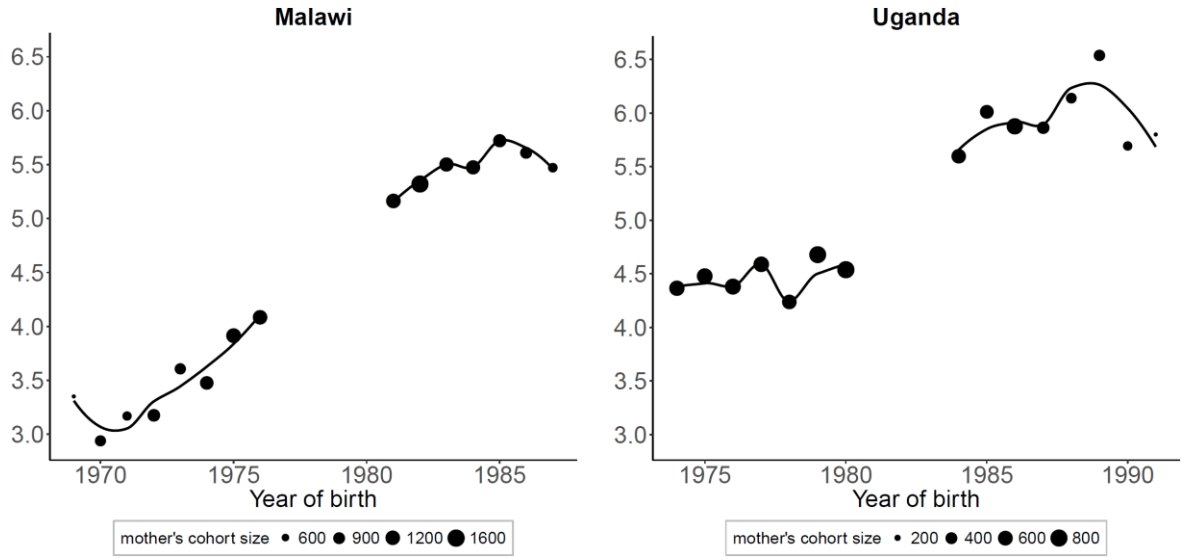

Fig. 2.2 Child mortality by mother's year of birth (averages and polynomial trend lines).

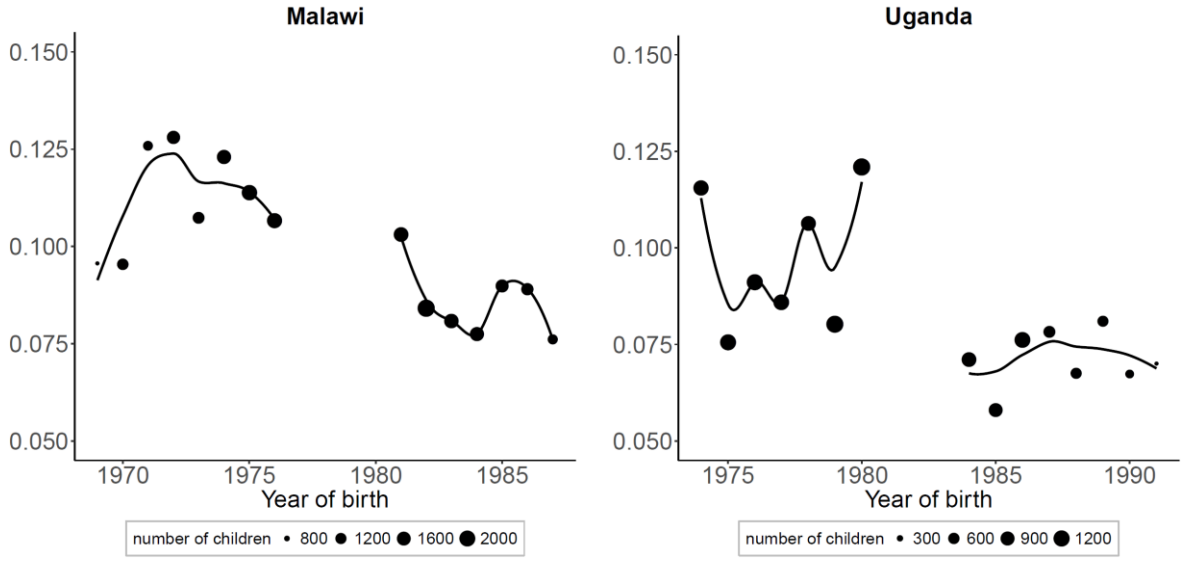

Fig. 2.3 Coefficients of the interactions year of birth × program intensity in the district of residence in (Eq. 1).

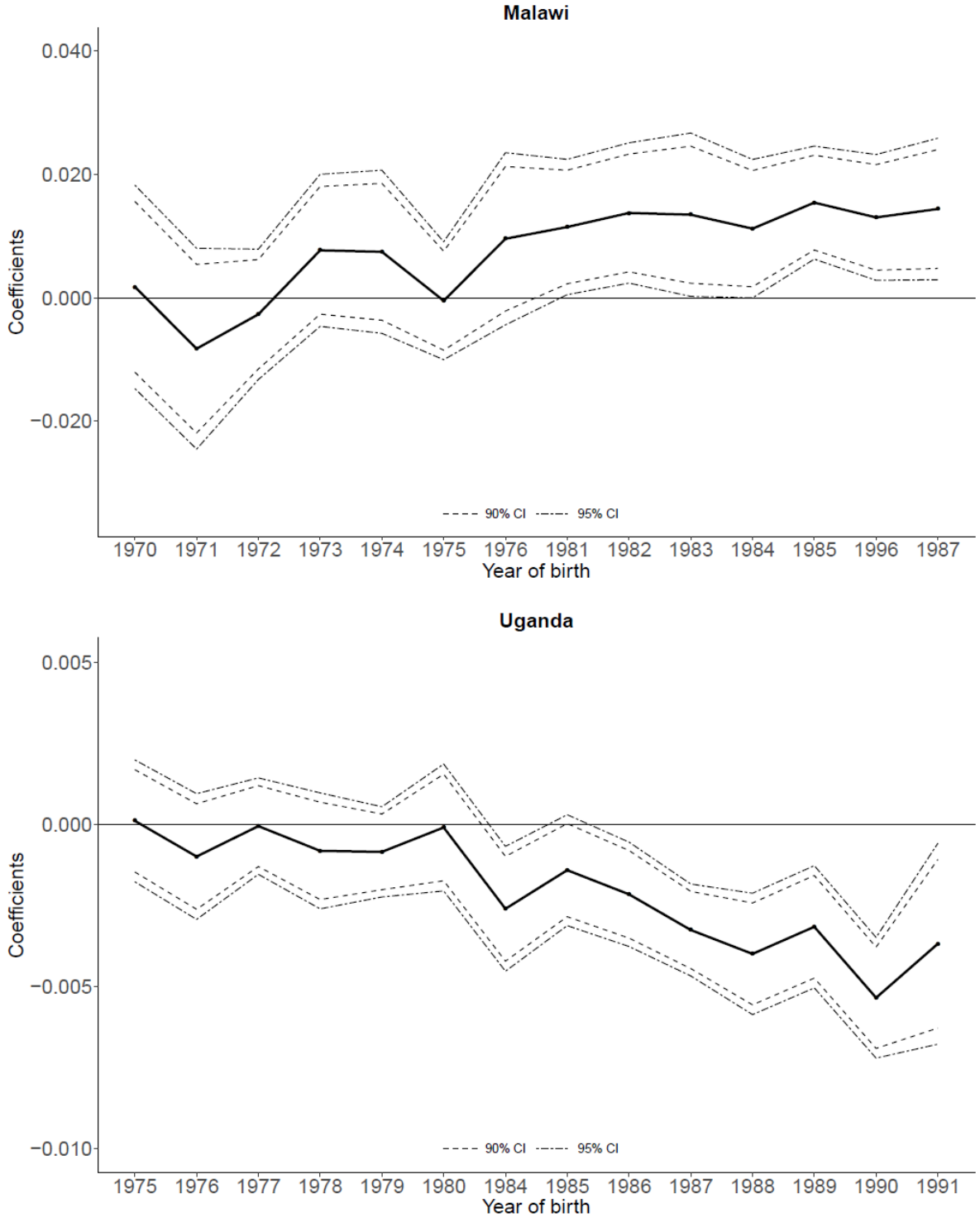

Table 2.1 Sample characteristics.

	Overall		Treatment		Control	
	Mean	SD	Mean	SD	Mean	SD
A. Malawi (treatment: born 1981–1987; control: born 1969–1976)						
Characteristics of the mother						
Years of education	4.51	3.62	5.45	3.44	3.53	3.53
Age in 1994	15.54	5.68	10.38	1.91	20.97	2.22
Sample size	15,484		7,880		7,604	
Characteristics of the child						
Child is dead	0.10	0.30	0.09	0.28	0.11	0.32
Birth year	2003.8	4.25	2005.7	3.1	2001.9	4.37
Child is female	1.50	0.50	1.51	0.50	1.50	0.50
Birth order	1.82	0.38	1.70	0.46	1.94	0.23
Sample size	23,087		11,517		11,570	
B. Uganda (treatment: born 1984–1991; control: born 1974–1980)						
Characteristics of the mother						
Years of education	5.08	3.76	5.93	3.45	4.48	3.85
Age in 1997	15.86	5.18	10.24	2.10	19.88	2.03
Sample size	8,319		3,455		4,864	
Characteristics of the child						
Child is dead	0.09	0.28	0.07	0.26	0.10	0.30
Birth year	2005.0	4.21	2007.6	2.36	2003.2	4.27
Child is female	1.50	0.50	1.50	0.50	1.50	0.50
Birth order	1.79	0.41	1.65	0.48	1.89	0.32
Sample size	13,779		5,514		8,265	

Means are based on weighted data. Sample sizes are unweighted.

Table 2.2 Summary statistics of the pathway indicators.

	Overall		Treatment		Control	
	Mean	SD	Mean	SD	Mean	SD
A. Malawi (treatment: born 1981–1987; control: born 1969–1976)						
Socioeconomic status						
Wealth index (0 to 5.1)	0.73	0.60	0.75	0.64	0.71	0.56
Medical care: money (n = 12,010)	0.42	0.49	0.46	0.50	0.37	0.48
Attitudes towards modern health services						
Use of modern contraception (n = 13,427)[a]	0.42	0.49	0.43	0.49	0.41	0.49
Personal illness control						
Personal illness control (0 to 8) (n = 15,274)[b]	3.73	0.78	3.72	0.72	3.74	0.83
Environmental characteristics						
Close to health facility (n = 12,010)	0.41	0.49	0.43	0.49	0.37	0.48
Health knowledge						
Knowledge about contracting AIDS (0 to 2.5) (n = 15,042)	1.96	0.67	1.98	0.66	1.93	0.66
Knowledge about transmission of AIDS (0 to 2.5) (n = 15,042)	1.98	0.64	2.02	0.60	1.95	0.67
Knowledge about ovulation (0 to 1) (n = 15,465)	0.44	0.33	0.45	0.32	0.44	0.34
Empowerment						
Decision-making (0 to 2.5) (n = 14,491)	0.92	0.81	0.91	0.80	0.92	0.83
Empowered domestic violence (0 to 3.6) (n = 14,491)	3.19	0.92	3.24	0.87	3.13	0.96
B. Uganda (treatment: born 1984–1991; control: born 1974–1980)						
Socioeconomic status						
Wealth index (0 to 3.9)	1.21	0.72	1.40	0.65	1.07	0.74
Medical care: money (n = 6,827)	0.41	0.49	0.45	0.50	0.38	0.49
Attitudes towards modern health services						
Use of modern contraception (n = 5,661)[a]	0.27	0.44	0.25	0.43	0.28	0.45
Personal illness control						
Personal illness control (0 to 7.3) (n = 6,679)[b]	2.69	0.77	2.71	0.70	2.68	0.82
Environmental characteristics						
Close to health facility (n = 6,827)	0.51	0.50	0.52	0.50	0.50	0.50
Health knowledge						
Knowledge about contracting AIDS (0 to 2.6) (n = 6,637)	2.16	0.71	2.18	0.71	2.15	0.71
Knowledge about transmission of AIDS (0 to 2.1) (n = 6,637)	1.42	0.67	1.41	0.68	1.43	0.66
Knowledge about ovulation (0 to 1) (n = 6,831)	0.43	0.33	0.42	0.32	0.43	0.33
Empowerment						
Decision-making (0 to 2.4) (n = 6,076)	1.22	0.84	1.17	0.84	1.25	0.83
Empowered domestic violence (0 to 2.7) (n = 6,076)	1.57	0.88	1.64	0.89	1.53	0.87

Means are based on weighted data. Sample sizes are unweighted.
[a] Excluding pregnant women.
[b] Excluding less recent births.

Table 2.3 First-stage results: Impact of UPE on maternal education.

	Years of Education	
	Malawi	Uganda
Young	1.644***	1.490***
	(0.214)	(0.318)
Young × Program Intensity	0.0109***	–0.00228***
	(0.003)	(0.0004)
Kleibergen–Paap *rk* Wald *F* Statistic	57.105	23.261
N	15,484	8,319
R^2	.710	.717

The standard errors, shown in parentheses, are clustered at the district level. All regressions include religion (Malawi only), district fixed effects, interaction between the dummy variable for young and the district-specific number of girls in primary school before UPE, and interaction between the dummy variable for young and the number of primary school–aged children before UPE. The variable program intensity is expressed as percentage.
$^{\dagger}p < .10$; $^{*}p < .05$; $^{**}p < .01$; $^{***}p < .001$

Table 2.4 First-stage results: Robustness checks excluding districts with high internal migration.

	Years of Education	
	Malawi Excluding Lilongwe City	Uganda Excluding Kalangala
Young	1.630***	1.517***
	(0.214)	(0.322)
Young × Program Intensity	0.0108***	–0.00227***
	(0.003)	(0.0004)
Kleibergen–Paap *rk* Wald *F* Statistic	54.530	23.121
N	15,167	8,298
R^2	.708	.716

The standard errors, shown in parentheses, are clustered at the district level. All regressions include religion (Malawi only), district fixed effects, interaction between the dummy variable for young and the district-specific number of girls in primary school before UPE, and interaction between the dummy variable for young and the number of primary school–aged children before UPE. The variable program intensity is expressed as percentage.
$^{\dagger}p < .10$; $^{*}p < .05$; $^{**}p < .01$; $^{***}p < .001$

Table 2.5 Second-stage results: Maternal education and child mortality.

	Child Is Dead			
	Malawi		Uganda	
	Non–2SRI	2SRI	Non–2SRI	2SRI
	Odd Ratio (95% CI)	Odd Ratio (95% CI)	Odd Ratio (95% CI)	Odd Ratio (95% CI)
Years of Education	0.971***	0.900*	0.951***	0.834†
	(0.958–0.984)	(0.811–0.998)	(0.934–0.969)	(0.687–1.012)
First-Stage Residuals		1.080		1.142
		(0.973–1.199)		(0.941–1.385)
Overidentification Test (*p* value)		.1		.7
N	23,087	23,087	13,779	13,779

The overidentification value is the *p* value of the overidentification test. All regressions include religion (Malawi only), child sex, birth order, child year of birth fixed effects, district fixed effects, interaction between the dummy variable for young and the district-specific number of girls in primary school before UPE, and interaction between the dummy variable for young and the number of primary school–aged children before UPE.

†$p < .10$; *$p < .05$; ***$p < .001$

Table 2.6 Second-stage results: Robustness check and linear specification.

	Child Is Dead			
	Malawi		Uganda	
	2SRI Excluding Lilongwe City	2SLS[a] Full Sample	2SRI Excluding Kalangala	2SLS[a] Full Sample
	Odd Ratio (95% CI)	Linear Coefficient (SE)	Odd Ratio (95% CI)	Linear Coefficient (SE)
Years of Education	0.897* (0.807–0.996)	–0.008 (0.006)	0.846† (0.698–1.026)	–0.011 (0.007)
First-Stage Residuals	1.083 (0.974–1.204)		1.126 (0.928–1.365)	
Overidentification Test (*p* value)	.1	.2	.7	.6
N	22,635	23,087	13,741	13,779

The overidentification value is the *p* value of the overidentification test. All regressions include religion (Malawi only), child sex, birth order, child year of birth fixed effects, district fixed effects, interaction between the dummy variable for young and the district-specific number of girls in primary school before UPE, and interaction between the dummy variable for young and the number of primary school–aged children before UPE.

[a] The coefficient is the linear effect of an additional year of maternal schooling on child mortality. The adjusted standard errors, shown in parentheses, are clustered at the district level.

†$p < .10$; *$p < .05$

Table 2.7 Second-stage results: Maternal education and 10 pathways indicators (one model per indicator per country).

Years of Education on	Malawi 2SLS	Uganda 2SLS
Socioeconomic Status		
Wealth index	0.008	0.151***
	(0.010)	(0.029)
Medical care: money	0.029**	0.075**
	(0.010)	(0.023)
Attitudes Towards Modern Health Services		
Use of modern contraception	0.028**	0.027†
	(0.009)	(0.015)
Personal Illness Control		
Personal illness control	–0.004	0.079†
	(0.014)	(0.043)
Environmental Characteristics		
Close to health facility	0.031**	0.009
	(0.010)	(0.020)
Health Knowledge		
Knowledge about contracting AIDS	0.006	0.052
	(0.013)	(0.037)
Knowledge about transmission of AIDS	0.023*	0.001
	(0.010)	(0.029)
Knowledge about ovulation	–0.015†	–0.006
	(0.008)	(0.015)
Empowerment		
Decision-making	0.001	–0.066
	(0.022)	(0.045)
Empowered domestic violence	0.095***	0.204***
	(0.015)	(0.061)

The standard errors, shown in parentheses, are clustered at the district level. All regressions for mothers include religion (Malawi only), district fixed effects, interaction between the dummy variable for young and the district-specific number of girls in primary school before UPE, and interaction between the dummy variable for young and the number of primary school–aged children before UPE. All regressions for children further include child sex, birth order, and child year of birth fixed effects. Sample excludes missing information and Uganda DHS–MIS 2009 because these questions are not included in the DHS–MIS questionnaire (DHS restriction). First-stage results are unaffected by the reduction in the sample size.
†$p < .10$; *$p < .05$; **$p < .01$; ***$p < .001$

Appendices

Treatment and control groups: Malawi and Uganda

When examining the role of women's education on fertility and child health outcomes in Uganda, Keats (2016) considered women aged 7–14 in 1997 as the treatment group and women aged 15–22 in 1997 as the control group. Keats justified this by arguing that previous studies (Grogan 2006, 2009) have proved that UPE in Uganda had an impact on school starting age: UPE increased the probability of a child entering school before age 8 by 9%. Furthermore, Breglia et al. (2008) and Tamusuza (2011) found that children attended primary school when they were older than the officially recommended age as a result of grade repetition, long-term absenteeism, and late entry into school, and that the age of exit from primary school was 16 years (Tamusuza 2011:125). A similar argument applies to Malawi: empirical evidence suggests that 15% of girls who were 14 years old (i.e., the official age of exit from primary school) were in standards (years) 1 to 5 instead of standard 8 (EMIS 2004:17).

Using the 1992 Malawi DHS and the 1995 Uganda DHS Household Surveys, we found that the primary school attendance of girls starts to decline considerably at the age of 16 years in Malawi and 15 years in Uganda (Fig. 2.A1), which suggests that the actual age of exit from primary school was effectively 17 years in Malawi and 16 years in Uganda.

[Figure 2.A1 here]

The administrative structure of the educational sector in Malawi and Uganda

Malawi is divided into three administrative regions: Northern, Central and Southern. In 1994, there were twenty-four districts, five in the Northern Region, nine in the Central Region, and ten in the Southern Region; and in each district there are Traditional Authorities and Villages. The provision of primary schooling and primary school teacher training was a responsibility of the Ministry of Education, Science and Technology.

However, the administrative structure of education was assigned to six education divisions, namely Northern, Central Eastern, Central Western, South Eastern, Shire Highlands, and South Western, and to the respective 28 district education offices (Chimombo et al. 2000; EMIS 2006). The latter included 24 districts and 4 urban cities (Mzuzu city, Lilongwe urban, Zomba urban, and Blantyre urban). Because in the 2000 and 2010 DHSs Mzuzu city and Mzimba district were combined, we could identify 27 districts instead of 28.

In 1996, Uganda was divided into four statistical (not administrative) regions: Central, Eastern, Northern and Western. The country was further divided into thirty-nine administrative districts, which were subdivided into counties, sub-counties and parishes (and sub-parishes in most cases). Under the local Government Act of 1997, public services such as nurseries, primary schools, special schools, and technical schools fell under the administration and management of District Councils. These councils had the authority to formulate, approve, and execute their own development plan, register UPE children, and distribute textbooks. Moreover, monthly remittances for schools from central government were transmitted to local government via the district administration officer, who in turn passed them on to schools (Uganda Ministry of Education and Sports 2004 cited in Nakabugo et al. (2008); Nambalirwa (2010)). Because in the 2000–2001 DHS Ntungamo district was combined with Mbarara district, we could identify 38 districts instead of 39. We used GPS data to identify the location of households within districts.[7]

Education management information systems database of the Ministry of Education

The district-level data are from the education management information systems database of the ministries of education of Malawi and Uganda. The ministries of education in both countries published data from school records submitted by primary school administrations. These data might have some limitations. It has been shown that the estimates on gross

enrolment rate were different from the survey estimates either because: (1) some school did not respond to the Ministry's request for information; (2) government school fund allocations were based on enrolment, so school administrators may have been motivated to inflate pupil numbers (Policy and Operations Evaluation Department (IOB) 2008 cited in Tamusuza 2011). However, in our analysis we used the number of actual primary schools (as opposed to the number of responding schools). Therefore, it could be that the number of primary schools was underreported, but this would likely be true in both academic years considered and thus would not substantially affect the difference in the number of schools that we consider in this paper.

Because the Uganda Ministry of Education and Sports did not collect data on actual primary schools for 1996, we used data on 1995, which illustrates the situation as of December 1995.

Demographic and Health Surveys

The DHS are national household sample surveys measuring population, health, socioeconomic and, for most of them, anthropometric indicators, which emphasise maternal and child health in developing countries. The DHS provides the most reliable data source on child mortality across developing countries in terms of coverage, comparability, and data quality using extensive interviewer training, standardised measurement instruments and techniques, and instrument pretesting to ensure standardisation and comparability across space and time (ICF Macro 2009). In order to realise the datasets, the DHS uses a two-stage stratified design: in the first stage, a number of clusters are selected with a probability proportional to the size from a frame list of enumeration areas (EAs) created from the most recent population census and in the second stage, a fixed number of households are selected from the complete list of households in each of the selected EAs.

[7] Clusters in the DHS we selected were geo-referenced, that is their coordinates were collected during the

The households in a survey area are stratified according to type of residence (urban/rural) crossed by administrative/geographical regions (Aliaga and Ren 2006). All household members in a certain age group (usually women of reproductive age 15–49 and men age 15–59) in the selected households are interviewed. In this paper, the household members of interest were the mother, for which a complete birth and death history of her children was collected (including children's birth date and death age, when applicable) and the children under age 5 born in the 5-year period before the survey.

The DHS–MIS was developed by the Monitoring and Evaluation Working Group of Roll Back Malaria, an international partnership coordinating global efforts to fight malaria, and collects national and regional or provincial data from a representative sample of respondents.

Women continuing education after age 18 or 19

Using the DHS household data, we calculated the percentage of women who continue their studies after age 18 or 19 as the ratio between the total number of *de jure* female members aged 18 or 19 or older who were attending school during the school year at the time of the survey and the total number of *de jure* female members between 18 or 19 years old and 24 years old. In Malawi, 12.1%, 10.4%, and 14.0% of women continued their studies after 18 years old in 2000, 2004, and 2010, respectively; in Uganda, 10.7%, 9.5%, and 10.5% of women continued their studies after 19 years old in 2000–2001, 2006, and 2011, respectively (we could not calculate the percentage for the 2009 survey due to limitations of the DHS–MIS data).

Evidence of the differences in educational attainment

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survey sample listing process using GPS receivers.

Figure 2.A2 shows the reverse cumulative distribution functions of years of education by mother's year of birth for the treated and untreated women and the difference in these functions. In both countries, it is evident that the change in the distribution was greater among exposed women than among non-exposed women and that the former had more education compared with the latter.

Indicators for the health knowledge pathway

The first indicator, knowledge about contracting AIDS, was based on three questions on whether using condoms and having one sex partner can reduce the chances of contracting AIDS and whether a healthy-looking person can have AIDS. The second indicator, knowledge about transmission of AIDS, used two questions on whether a person can get AIDS by being bitten by a mosquito and by sharing food with a person who has it. The two items were coded 1 if the person cannot get AIDS under these circumstances and 0 otherwise. The third indicator, knowledge about ovulation, was based on one question on the timing of the ovulatory cycle and was coded 1 = correct answer "middle of cycle", 0.5 = "after period ends" or "before period begins", and 0 = incorrect.

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Fig. 2.A1 Fraction of girls in primary school by age in 1992 (Malawi) and 1995 (Uganda)

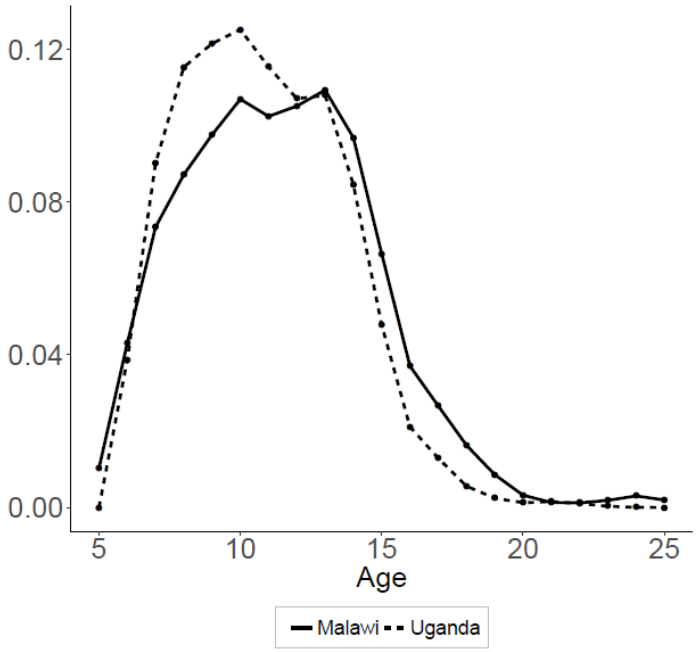

Fig. 2.A2 Reverse empirical cumulative distribution functions of education by mother's year of birth

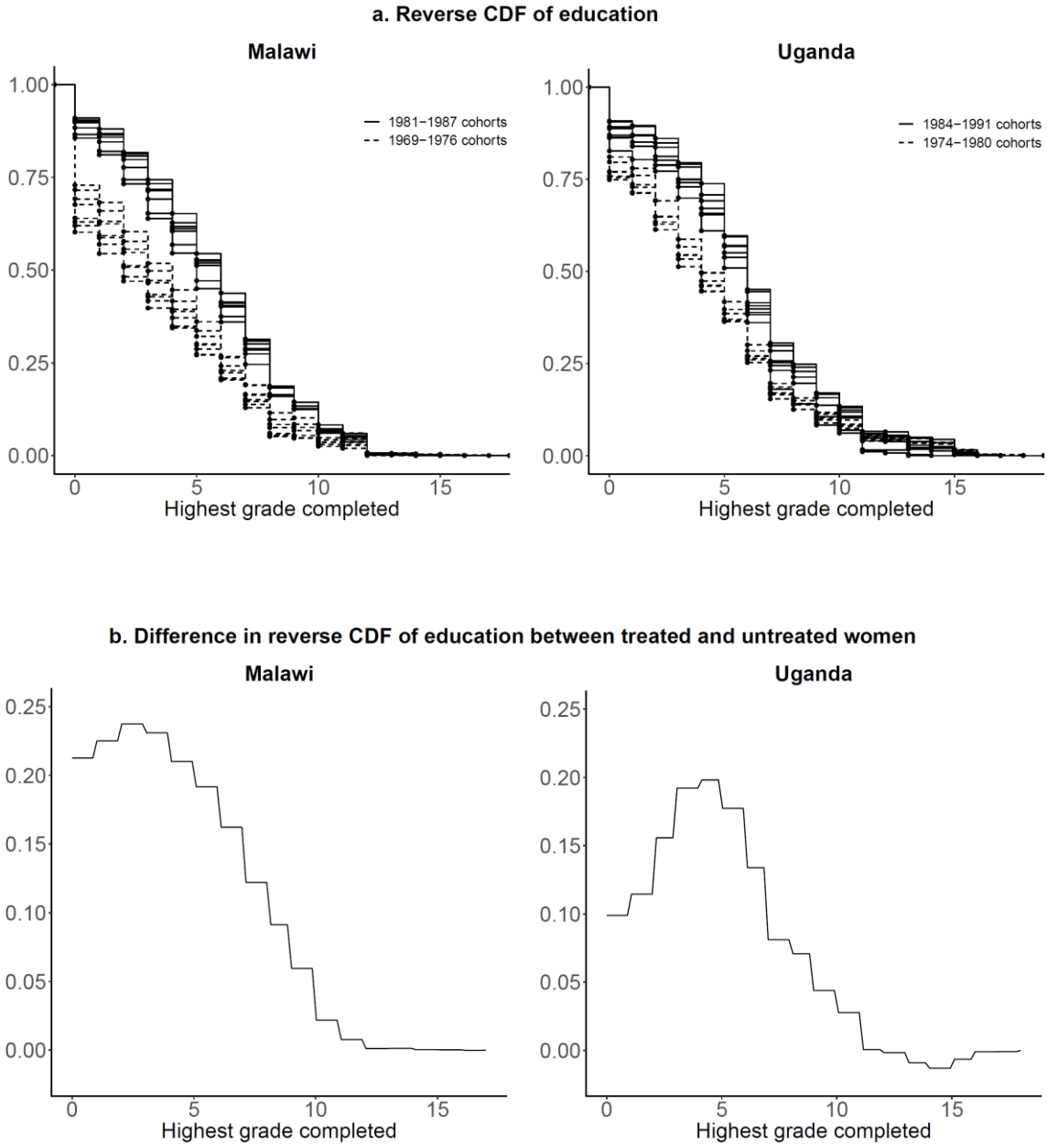

Table 2.A1 Reduced form OLS estimates: Impact of UPE on 10 pathway indicators (one model per indicator per country).

	Malawi		Uganda	
Pathway indicator	Young	Young x Program Intensity	Young	Young x Program Intensity
Socioeconomic Status				
Wealth index	0.033	-0.0004†	0.210***	-0.0004***
	(0.023)	(0.0003)	(0.048)	(0.00001)
Medical care: money	0.059*	0.0001	0.117**	-0.0002*
	(0.024)	(0.0004)	(0.040)	(0.0001)
Attitudes Towards Modern Health Services				
Use of modern contraception	0.081***	-0.001†	0.022	-0.0003***
	(0.021)	(0.0003)	(0.025)	(0.0001)
Personal Illness Control				
Personal illness control	-0.052	0.108†	0.067	-0.058***
	(0.034)	(0.056)	(0.068)	(0.010)
Environmental Characteristics				
Close to health facility	0.092***	-0.001*	0.024	0.0001
	(0.022)	(0.0003)	(0.038)	(0.0001)
Health Knowledge				
Knowledge about getting AIDS	0.009	0.0001	0.075	-0.0001
	(0.023)	(0.001)	(0.059)	(0.0001)
Knowledge about transmission of AIDS	0.070**	-0.001	-0.013	-0.0001
	(0.026)	(0.0005)	(0.046)	(0.0001)
Knowledge about ovulation	-0.044***	0.0003	-0.026	-0.0001†
	(0.013)	(0.0003)	(0.027)	(0.0001)
Empowerment				
Decision-making	-0.005	0.0002	-0.146*	-0.0002†
	(0.038)	(0.001)	(0.068)	(0.0001)
Empowered domestic violence	0.142***	0.001**	0.301***	-0.0003*
	(0.030)	(0.001)	(0.055)	(0.0002)

The standard errors, shown in parentheses, are clustered at the district level. All regressions for mothers include religion (Malawi only), district fixed effects, interaction between the dummy variable for young and the district-specific number of girls in primary school before UPE, and interaction between the dummy variable for young and the number of primary school–aged children before UPE. All regressions for children further include child sex, birth order, and child year of birth fixed effects. Sample excludes missing information and Uganda DHS–MIS 2009 since these questions are not included in the DHS–MIS questionnaire (DHS restriction). The variable program intensity is expressed as percentage.
$^{\dagger}p < .10$; $^{*}p < .05$; $^{**}p < .01$; $^{***}p < .001$

Chapter 3. Spatial and Temporal Variation in Family Decision-Making: A Diffusion Perspective in Sub-Saharan Africa[8]

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Abstract

This paper maps spatial and temporal variation in family decision-making norms and analyses the diffusion of male decision-making dominance using pooled Demographic and Health Surveys from 28 countries in sub-Saharan Africa. Applying adaptive bandwidth kernel density estimation, we find considerable spatial heterogeneity in male decision-making dominance both between and within countries in an earlier (i.e. 2001–2005) and later (i.e. 2010–2014) period. Using a spatial panel fixed effects model approach, we find evidence that diffusion of norms about family decision-making helps explain declining reports of men's decision-making dominance over time. We also find that schooling and urbanisation are the main channels through which this diffusion occurs and speculate that this is because schools and urban areas are spaces where people may be exposed to new ideas about gender norms.

Keywords: Africa, decision-making, families, spatial analysis, diffusion.

Introduction

Norms about women's status are central to demographic understandings of health and wellbeing of women and their families (Balk 1994; Kuate Defo 1997; Hindin, 2000; Oppenheim-Mason, 1987; Upadhyay & Hindin 2005). In low-income countries, improving women's status has been a central policy concern that features heavily in the United Nations (UN) Millennium Development Goals and UN Sustainable Development Goals. This has given rise to measures—such as the UN Development Programme (UNDP) women's empowerment indicators—that provide cross-national comparisons of changes in women's status over time. For example, the UN's gender inequality index suggests that there have been overall improvements in women's status cross-nationally (UN 2019). While such indicators provide valuable measures of aggregated trends, they are only available at the national level, and thus may potentially hide considerable within country

heterogeneity, such as between urban and rural areas, and across ethno-linguistic and administrative boundaries. Aggregated indicators also reveal very little about the social and demographic processes through which norms about women's status spread across time and space.

This paper offers a new perspective on the diffusion of norms related to women's status by utilising cutting edge spatial analysis techniques to map spatial variation in women's reports about their husbands' dominance in decision-making about their own health, which, for shorthand, we refer to as men's decision-making dominance[9], throughout sub-Saharan Africa (SSA). Although demographers have long hypothesised that diffusion of norms are important catalysts of demographic change (Coale & Watkins 1986), it has been challenging to empirically document how and why diffusion occurs. To address this, we use spatial regression methods to test whether diffusion helps explain declining reports of men's decision-making dominance and explore the pathways through which diffusion occurs.

SSA makes an insightful case study for this topic because African countries have some of the lowest reported levels of women's participation in family decision-making throughout low-income countries (Pesando & GFC Team 2018). Urbanisation and mass education— two major pathways through which demographers posit diffusion of norms occurs—have increased in many places in SSA, raising questions about whether women's status has changed alongside these factors. Because industrialisation has been largely absent in SSA, and thus cannot explain declines in reported male decision-making dominance, diffusion of norms about women's status could represent an important explanation for changes observed at the aggregate level.

We start by exploring spatial trends in male decision-making dominance in SSA by producing high resolution maps that illuminate spatial patterns of male decision-making

dominance using pooled Demographic and Health Surveys and AIDS Indicator Surveys from across sub-Saharan Africa in the early 2000s and approximately ten years later. Next, we explore the contextual factors that predict men's decision-making dominance using panel fixed effects spatial regression methods. Following approaches used by Vitali et al. (2015) and Vitali and Billari (2017), we test for evidence of diffusion of norms about women's status using panel fixed effects spatial regression methods that allow for spatial dependence in both the dependent and explanatory variables. Importantly, this allows us to assess whether diffusion processes play a role in explaining declines in male decision-making dominance and explore whether key spatial explanatory variables such as education and urbanisation matter for the diffusion of norms about family decision-making.

Women's Status and Family Decision-Making

In this paper, we focus on women's reports about their husbands' dominance in making decisions about their own health as an indicator of gender norms about women's status within the family. Decision-making measures have been widely-used to assess women's abilities to make strategic choices that impact personal and family well-being (Hindin 2000; Peterman et al. 2015; Smith et al. 2003). Studies have validated the use of women's decision-making to measure status by showing that women's reported participation in family decision-making is associated with higher levels of contraceptive use, improved child health and nutrition, and higher probability of women's rejection of wife beating across diverse contexts in sub-Saharan Africa (Amugsi et al. 2016; Hindin 2000; Smith et al. 2003; Uthman et al. 2010). Furthermore, alternative measures of women's status—including age, education, assets, and income—are highly predictive of women's participation in family decision-making in sub-Saharan Africa (Behrman 2017; Kishor &

[9] The term 'men's decision-making dominance' is used interchangeably with 'male decision-making

Subaiya 2008; Kritz & Makinwa-Adebusoye 2018), thus suggesting that reports about decision-making adequately capture a latent construct of women's status.

Nonetheless, gender inequality is complex and multi-dimensional and there is no one way to measure women's status (Oppenheim-Mason 1986). It is notoriously difficult to fully understand the "black box" of what actually happens in the family (Haddad et al. 1997) and there are many important critiques of what decision-making measures actually capture. For example, husbands and wives sometimes provide different responses to questions about decision-making and property ownership (Behrman & Frye 2018; Doss et al. 2014; Kilic & Moylan 2016) and variations in wording about women's decision-making leads to differences in estimates of the prevalence of women's decision-making across contexts (Peterman et al. 2015). Some of these discrepancies in reports about decision-making may be due to social desirability bias because women answer questions based on how they think they should answer as opposed to what actually transpires in the family.

Although we acknowledge that our analysis is limited to reported decision-making given that we do not observe intra-familial dynamics, we nonetheless maintain that this measure provides important information on gender norms about women's status. In sub-Saharan Africa, discourse about women's "empowerment" and "modern" gender relations in the family are often the subject of development policies and programming that are widely dispensed through media, publicity, and NGO workers among others (Thornton et al. 2015; Thornton et al. 2014). Thus, it is highly plausible that norms about whether it is "acceptable" to report that men dominate decision-making might change more quickly than actual behaviours. Although social desirability bias might also play a role in women's responses, these reports would nonetheless capture important normative change related to the socially desirable role for women and men to play in family decision-making, that could eventually lead to the adoption of new behaviours.

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dominance' and 'husbands' decision-making dominance'.

A Diffusion Perspective on Spatial and Temporal Variation in Women's Status and Family Decision-Making

Diffusion of new ideas and norms are central to theories of demographic change. Most notably, a large demographic literature explores how the diffusion of ideas about smaller family sizes and norms about the acceptability of birth control were key to the timing of the fertility transition in Europe and the United States (Bocquet-Appel & Jakobi 1998; Coale & Watkins 1986; Goldstein & Klüsener 2014; Tolnay 1995). For example, in the Princeton Fertility Project—which laid the foundation for many of these ideas—Coale, Watkins and others showed that fertility decline in Europe occurred first in places with cultural, ethnic, and linguistic similarities, rather than in places that were forerunners of industrialisation. This suggests that diffusion of norms about the values and costs of childbearing and acceptability of contraceptive use among culturally similar groups were essential to eventual fertility-related behavioural change (Knodel & van de Walle 1979).

Diffusion of norms and ideas about fertility and family have also played a role in scholarship that explores how and why demographic change occurs in contemporary contexts. The literature on the Second Demographic Transition suggests that ideational change valuing individual autonomy, self-fulfilment, and the diffusion of new demographic behaviours such as cohabitation as an alternative to marriage, divorce, and non-martial childbearing were important precursors of the low and lowest low fertility observed in many parts of contemporary Europe (Lesthaeghe 2014; Van de Kaa 2001). The theory of developmental idealism—which suggests an idealised version of Western nuclear family is equated with "high" levels of economic development—has diffused from European elite to non-elite actors throughout the world, with important implications for family and fertility change (Thornton 2001; Thornton et al. 2015). Because diffusion is difficult to measure quantitatively without considering the role of geographic space,

researchers have increasingly adopted spatial approaches to document evidence of diffusion of norms related to fertility, marriage and cohabitation, nonmarital childbearing, and reproductive health in high and low income contexts (Mita & Simmons 1995; Nazio & Blossfeld 2003; Vitali & Billari 2015; Vitali et al. 2015).

There are myriad explanations for the processes through which diffusion of norms occurs. At the micro-level, social interaction plays an important role in spreading new ideas and conceptions about what demographic behaviour is admissible or preferable (Bongaarts & Watkins 1996; Kohler et al. 2002; Montgomery & Casterline 1996). Social networks can also be consequential for fertility and demographic decision-making (Balbo & Barban 2014). In contemporary contexts, technological changes—such as the spread of cell phones and internet—have become important for the diffusion of new ideas and norms (Billari et al. 2017). In low income countries, the spread of new ideas can also occur through development programming and outreach which often centres around an idealised set of norms and values set by external international actors (Pierotti 2013; Thornton et al. 2014; 2015). The rise of global media and entertainment may also contribute to the diffusion of norms. For example exposure to radio and television programming that included family planning messaging has been shown to have an impact on fertility, family planning, and divorce in Africa, Asia, and the Americas (Chong & La Ferrara 2009; Dewi et al. 2013; Jensen & Oster 2009; La Ferrara et al. 2012; Westoff & Koffman 2011).

While most studies of demographic diffusion focus on diffusion of norms related to fertility and marriage (Reed et al. 1999), gender norms about women's status may also be subject to diffusion processes. For example, the introduction of cable television was associated with improvements in women's status (as measured by declines in the acceptability of wife beating and son preference) in India (Jensen & Oster 2009) and a cross-national study documents a large decline in the acceptability of wife-beating throughout low and middle-income countries at the start of the twenty first century

(Pierotti 2013). In the latter study, urbanisation, education, and media exposure are all associated with increased rejection of wife beating, but because compositional changes in these variables cannot fully explain the trend of increased rejection of wife beating the author attributes these findings to the promulgation of global cultural scripts.

In our analysis, we focus on schooling and urbanisation as the main channels through which diffusion of norms occurs in this context. While there may be other relevant channels as well—such as media, social networks, etc.—we hypothesise that many of these mediating processes occur through the primary channels of schooling and urbanisation, particularly since schooling and urbanisation have increased considerably in Africa in recent decades (Fox 2012; Lopus & Frye 2018; Psaki et al. 2017). Both schools and urban areas are spaces where people come into contact with new ideas about gender norms via exposure to new media, exposure to new people from other backgrounds, regions or countries and exposure to women in new roles such as teachers, medical professionals, etc. (Caldwell, 1980). Both schools and urban areas are also sites where individuals have the opportunities to interact with others in new ways and to perform new gender roles for the first time, and have been shown to have important implications for aspirations, identities, and norms (Frye 2012; Johnson-Hanks 2006).

Data and Measures

Our analysis uses Demographic and Health Surveys (DHS) data, which is collected by ICF international in collaboration with host-country governments. Since the 1980s the DHS Program has collected standardised nationally representative cross-sectional surveys on reproductive health, women's status, and demographic wellbeing across low- and middle-income countries. The DHS uses a two-stage sampling procedure that first identifies primary sampling units (PSUs) (also known as clusters), and then randomly selects households within those clusters for interviews. All women in the household aged 15–49

are interviewed, and sampling weights can be applied so that the sample is nationally representative of women of reproductive age. Starting in the 1990s, but most systematically since the early 2000s, GPS coordinates of the clusters were also collected, which allow linking interviewed women to their geographic location at time of survey. The GPS coordinates assigned are the central points of the clusters in which the women live. We use these coordinates to create what is referred to in the spatial literature as a "grid-cell" (e.g. 0.50 x 0.50-degree grid cell—about 50 km by 50 km at the equator), which is our principal unit of analysis.[10] Spatial analysis methods rely upon using grid-cells—as opposed to clusters—because grid cells are squares that can be used for spatial interpolation and regression.

For this analysis, we pool micro-level DHS data from 28 sub-Sahara African countries using 15 DHS surveys collected in the period 2001–2005 and 32 DHS surveys collected in the period 2010–2014. We use all DHS survey waves for SSA that include GPS data[11], providing us with a total of 360,931 women living in 21,795 clusters (see Figure 3.1 for spatial distribution of clusters). Table 3.1 presents additional information about the characteristics of the samples (all data are weighted using the DHS sampling weights).

[Figure 3.1 and Table 3.1 here]

The main outcome of interest is constructed based on a question where the female respondent is asked who in the family is the main decision-maker about her own health. This is a commonly used measure of women's status that has been used and validated throughout the demographic literature (Pesando & GFC Team 2018), although as we

[10] To maintain respondent confidentiality, DHS randomly displaces the latitude/longitude position of clusters up to 2 km for urban clusters and up to 5 km for rural clusters, with 1% of the rural clusters being displaced up to 10 km. This displacement may cause some clusters to lie outside the country boundaries. We change the coordinates of the clusters outside of national boundaries to be the nearest point on the country's border. In order to do this, we use administrative boundary shapefiles obtained from the freely available Database of Global Administrative Areas (GADM) and projected using the World Geodetic System 1984 projection.

[11] These also include the DHS–AIDS Indicator Survey (AIS), a standardised survey collecting data on HIV/AIDS indicators from a representative sample of respondents.

discussed in the literature review there are limitations to this measure, such as reporting bias. We construct a variable for the share of households in a given grid cell in which the women's partner/husband is the sole decision maker on the women's health. In our analytical strategy section (below), we provide further detail on the construction of cell-level indicators. We focus on women—rather than men's—reports because male respondents are not consistently asked this decision-making question over survey rounds and countries.

In our main analysis, we investigate two social trends that we hypothesise play a key role in diffusion: (1) the spread of women's education; and (2) urbanisation. We measure women's education by creating a variable for the percentage of women in the grid cell who have at least some education at the time of survey using DHS data (e.g. have ever been to school). We focus on ever attending school (as opposed to primary completion etc.) because we hypothesise that school attendance—as opposed to attainment—is particularly important for diffusion of norms. Furthermore, in many parts of Africa schooling levels are so low that many women have not attended school. We measure urbanisation by creating a variable for night-time light intensity in the grid cell, i.e. lights from cities, towns, and other sites with persistent lighting, including gas flares. Night-time light intensity is a commonly used measure of urban growth (Schneider et al. 2010) and economic activity (Ghosh et al. 2010), and is preferred to the DHS measure of urbanisation, which cannot be used in this analysis because it is dichotomous and not continuous (because our maps are prevalence maps, we need a continuous measure of urbanisation). Data on night-time light intensity are taken from the freely available dataset of Global DMSP-OLS Nighttime Lights of the National Geophysical Data Center within the U.S. National Oceanic and Atmospheric Administration and are available for each year 1992 to 2013 at a spatial resolution of 30 arc-second grids (about 1 km by 1 km at the

equator). For our analysis, we use data for the years in which surveys were conducted[12], and aggregate them at a 0.50 x 0.50-degree resolution by taking the mean across all 30 arc-second grid cells.

Methods

Spatial Interpolation

The first step of our analysis is to explore spatial and temporal heterogeneity in men's decision-making dominance in SSA. To this end, we apply spatial interpolation methods to estimate the family decision-making indicator for each grid cell across time. Spatial interpolation is the process of using spatial points (e.g., DHS clusters, Figure 3.1) with known values to estimate values for all cells on the map and thus obtain gridded data. We adopt kernel density estimation (KDE) technique with adaptive bandwidths encompassing an optimal number of persons surveyed through the DHS.[13] The KDE is a non-parametric method for estimating grid cell densities based on observed data. It produces a density surface around each spatial point that is highest at the point and diminishes with distance (Larmarange et al. 2017). The estimated value at each spatial point is then used to create prevalence surfaces showing the spatial variations in the variable of interest. Prevalence surfaces are choropleth maps in which grid cells are shaded proportional to the measurement of the variable displayed. In order to have reliable maps, we remove unpopulated cells, e.g. cells where there are lakes, rivers, deserts (using data from the freely available Database of Gridded Population of the World, Version 4 of the Center for International Earth Science Information Network). In appendix, we show that our descriptive findings are both valid and robust to different N_{opt} and spatial resolution.

[12] This does not apply to surveys conducted in 2014 for which we use data from 2013.

[13] Because the optimal N parameter (Larmarange et al., 2011) is a function of survey-specific parameters, it varies by survey (Table 3.1). Formally, it is described as follows:

$$N_{opt} = 14{,}172 * n^{0.419} * p^{-0.361} * g^{0.037} - 91.011, \qquad (1)$$

Using these methods, we create high-resolution maps of family decision-making that allow us to visually assess how men's decision-making dominance varies geographically. We also explore how male decision-making dominance varies temporarily by creating maps for both the earlier (e.g. early 2000s) and later (e.g. 2010s) rounds of the DHS, and also creating a map of change in male decision-making dominance between the earlier and later rounds, whereby change is the percentage variation from 2001–2005 to 2010–2014. The latter is done only for cells in the 15 countries that have *both* an earlier and a later round of the DHS. In order to understand corresponding changes in education—which we think might be central to the diffusion of norms about family decision-making—we create comparable a map for the education variable. We cannot create a map of change for the light intensity variable (our proxy for urbanisation) because in some cells light intensity is zero and has remained stable over time; however, changes can still be detected by visually comparing the maps between the earlier and later rounds.

We also measure the within-country versus between-country variation in husbands' decision-making dominance in each time period. This is important, because many national boundaries in SSA were artificially imposed during the colonial period, thus national boundaries frequently cut across ethno-linguistic groups and encompass highly heterogenous populations. Given enormous within country ethnic and social heterogeneity, it is plausible that there might be as much variation in norms about family decision-making within countries as between countries. To empirically asses the importance of between versus within country variance we regress our main variable of interest—male decision-making dominance—on a set of country indicators using ordinary least-squares regression (Burke et al. 2016). The R-squared of this regression represents the proportion of total variation in family decision-making explained by differences across countries.

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where p is the sample prevalence, n is the number of persons surveyed in the sample, and g specifies the

Spatial Panel Data Modelling

After establishing spatial and temporal trends in male decision-making dominance, the next step of our analysis is to assess whether there is evidence of diffusion in family decision-making norms and the drivers behind this diffusion, which we do using spatial panel data modelling methods following the approach of Vitali et al. (2015) and Vitali and Billari (2017). The key novelty of our spatial panel modelling scheme is that it allows for spatial autocorrelation in both the dependent (e.g. male decision-making dominance) and the explanatory variables (e.g. women's education and urbanisation). Spatial autocorrelation in the dependent variable establishes the extent to which male decision-making dominance in any given cell depends on male decision-making dominance in other neighbouring cells. There is evidence of diffusion when low levels of male decision-making dominance in forerunner cells spread to other neighbouring cells over time (e.g. when levels of male decision-making dominance in neighbouring cells become more similar to those in forerunner cells with lower-levels of male decision-making dominance). Given significant spatial autocorrelation in the dependent variable, the autocorrelation on the explanatory variables enables us to disentangle the extent to which decreases in male decision-making dominance are driven directly by the cell's own characteristics or indirectly by the characteristics of neighbouring cells. In what follows, we go into further detail in the models used to explore these issues.

We start by reviewing a panel data fixed effects model, which can be formally described as follows:

$$y_{it} = \boldsymbol{x}_{it}\boldsymbol{\beta} + \mu_i + \varepsilon_{it}, \qquad (2)$$

where our dependent variable y_{it} is the proportion of women in cell i and year t reporting that their husband/partner is the sole decision maker on the women's health, $\boldsymbol{x}_{it}$ is the vector of independent variables (proportion of women in the cell with at least some

number of sample clusters.

education and urbanisation level in the cell), $\boldsymbol{\beta}$ is the matching vector of coefficients, μ_i denotes cell-specific fixed effects, and ε_{it} is the error term.

The panel data fixed effects model produces unbiased parameter estimates provided that our observations (in our case, the cells) are independent. The assumption of independence does not hold if, instead, observations are spatially dependent. In this case, models including spatial effects are more suitable (see appendix for more details). Spatial effects are generally introduced into the model using a spatial weighting matrix, W_i, a positive matrix where the rows and columns correspond to the cross-sectional observations, representing the neighbouring structure across cells. Neighbours are here defined on the basis of a contiguity criterion, according to which two cells are neighbours if they share a common edge or a common vertex. An element of the matrix, w_{ij}, equals $1/\pi_i$ if $j \in N(i)$ and 0 otherwise, where $N(i)$ defines the set of all neighbours of i, and π_i is the number of neighbours of i, and expresses the existence of a neighbour relation between i and j.

After spatial dependence is established, the simple panel data fixed effects model can be extended to include spatial effects. We show results using the spatial Durbin (SDM) panel model specification because supplementary analyses suggest it best describes our data (see appendix for further detail).

The SDM panel model is expressed as follows:

$$y_{it} = \lambda \sum_{j=1}^{N} w_{ij} y_{jt} + \boldsymbol{x}_{it}\boldsymbol{\beta} + \sum_{j=1}^{N} w_{ij}\boldsymbol{x}_{ijt}\boldsymbol{\gamma} + \mu_i + \varepsilon_{it}, \quad (3)$$

where λ is the coefficient of the spatially lagged dependent variable and referred to as the spatial autocorrelation coefficient in the dependent variable, $\boldsymbol{x}_{ijt}$ is the vector of independent variables measured in cell j and year t, and $\boldsymbol{\gamma}$ is the matching vector of coefficients. This set-up allows male decision-making dominance in cell i and year t, y_{jt}, to depend on male decision-making dominance observed in neighbouring cell j and year t, y_{jt}. A positive estimate of λ indicates that decreases in male decision-making dominance

in neighbouring cells are associated with decreases in male decision-making dominance in the reference cell i. It thus informs us about the existence of a diffusion mechanism, according to which reductions in male decision-making dominance spread across cells, after controlling for observed characteristics and unobserved time-invariant characteristics. Unlike the cross-sectional spatial models, where λ represents a spatial pattern reflecting a diffusion process, the introduction of the temporal dimension in spatial panel models reinforces the interpretation of λ as reflecting diffusion.

The other advantage of this model is that it allows men's decision-making dominance in each cell i to depend on a set of characteristics measured in the same cell and on an average of the same characteristics measured in neighbouring cells. The latter expresses the extent to which male decision-making dominance in cell i is affected by women's education and urbanisation averaged over its neighbouring cells, and thus it allows identifying the drivers behind the diffusion of new norms about family decision-making.

Unlike the fixed-effects model, the parameter estimate in spatial panel models cannot be interpreted as the marginal effect of a variation in the explanatory variable on the dependent variable. Because a change in a single cell associated with any given explanatory variable will influence the cell itself (direct impact) and potentially influence all the other neighbouring cells indirectly (indirect impact), the total marginal effect is the combination of the direct effect and the indirect effect (LeSage and Pace 2009). LeSage and Pace (2009) show that estimated direct and indirect impacts are based on the partial derivatives of y on the explanatory variable and thus calculated using the parameter estimates λ, $\boldsymbol{\beta}$, and $\boldsymbol{\gamma}$. Estimated impacts differ from the parameter estimates because they include feedback effects. In particular, for the direct effect, a change in one explanatory variable in cell i has an influence on the dependent variable in cell i also through an effect from cell i to neighbouring cell j, and then back to cell i, via the spatial autocorrelation on

the dependent variable. This occurs because each cell is its neighbour's neighbour. A change in one explanatory variable in cell i can also have an influence on the dependent variable in cell j. This impact is referred to as indirect or spatial spillover effect. To offer a richer interpretation of spatial spillovers, we report both the point estimates and the impact estimates.

Results

Descriptive Findings

In this section, we start by presenting prevalence maps of men's decision-making dominance in the 2000s and 2010s both at the country (left panel, Figure 3.2) and local levels (right panel, Figure 3.2). The country level maps—which would be consistent with aggregated indicators of women's status that average across the country—show there is marked heterogeneity across countries in male decision-making dominance. For example, in first period (2001–2005) the prevalence of male decision-making dominance ranges from a low of 17% of households in Zimbabwe to a high of 74.9% households in Burkina Faso and in the second period the prevalence of male decision-making dominance ranges from 9% of households in Lesotho to 83.6% of households in Mali. Country level estimates suggest Western African countries have higher prevalence of male decision-making dominance in both time periods than Eastern and Southern countries (with a few important exceptions).

Unlike the country-level maps (left), the local-level maps (right) demonstrate considerable within country heterogeneity in the prevalence of male decision-making dominance. For example, in the country-level estimates Ghana appears to be a regional outlier with a lower prevalence of male decision-making dominance compared to neighbouring countries. However, the local level maps show that in the first time period southern Ghana (which borders the ocean) has a low prevalence of male decision-making

dominance, but Northern Ghana has much higher prevalence of male decision-making dominance that more closely resembles its neighbours. Likewise, the country level estimates suggest that in Mali the prevalence of male decision-making dominance increased between the first and second time periods. However, the local level maps show that this was largely concentrated in the northern regions of the country, whereas the southern regions of the country remain less changed. In addition to these examples, the local level maps show that the southern regions of Guinea and Nigeria had particularly low proportions of men's decision-making dominance compared to other parts of the country, whereas the Eastern areas of Ethiopia and Kenya had higher proportions of men's decision-making dominance relative to other parts of the country in the 2000s. Similarly, in the 2010s, there were very high proportions of men's decision-making dominance relative to other areas of the country in the Northern areas of Burkina Faso, Chad, Nigeria, and Mali and in Central Congo.

[Figure 3.2 here]

The maps in Figure 3.2 visually reveal that cells with high values of men's decision-making dominance are clustered geographically and the same is true for cells with low values of men's decision-making dominance, which may be indicative of spatial autocorrelation in the decision-making indicator. Formally, the presence of global spatial autocorrelation is tested using the Moran's *I* index, a cross-product statistic between a variable and its spatial lag that tests whether the value of a variable observed in a given location is independent of the value observed in a neighbouring location.[14] In our data, the Moran's *I* index equals .89 (p-value < 0.001) in the 2000s and .92 (p-value < 0.001) in the 2010s. This indicates a strong and positive spatial interdependence in our indicator, in

[14] The Moran's *I* index is the slope of a linear regression of the spatially lagged variable (a weighted average of the value of the variable in the neighbouring cells) on the original variable (in standardised form). It thus examines whether the spatial pattern is clustered, dispersed, or random. It is expressed as follows:

$$I = \frac{N\sum_{i=1}^{n}\sum_{j=1}^{n} w_{ij}(y_i-\bar{y})(y_j-\bar{y})}{\left(\sum_{i=1}^{n}\sum_{j=1}^{n} w_{ij}\right)\sum_{i=1}^{n}(y_i-\bar{y})^2}, \qquad (4)$$

other words, that cells with similar values of male decision-making dominance tend to be concentrated geographically[15] (see appendix for results from Local Moran's I, i.e. Local Indicator of Spatial Autocorrelation).

We further explore the role of heterogeneity within versus between countries in men's decision-making dominance by calculating the R-squared of a regression of our indicator on a set of country dummies. We find that between-country variation accounts for 58.6% of the total variation in male's decision-making dominance in 2001–2005 and 68.5% of the total variation in male's decision-making dominance in 2010–2014. Similarly, between-country variation accounts for 14.2% of the total variation in changes in male decision-making dominance between 2001–2005 and 2010–2014. Therefore, around 85.8% of the variation in changes in male decision-making dominance is attributed to factors that vary over space and time within countries, thus highlighting the importance of a spatial approach that takes into account this within country spatial heterogeneity.

Next, we present maps of the independent variables that we use in our empirical model, i.e. women's education and urbanisation. Figure 3.3 shows substantial cross-country variation in women's education in both periods. At the national level, the proportion of women with at least some education ranges from a low of 19.7% in Burkina Faso to a high of 98.0% in Lesotho in the first period and from a low of 24.2% in Mali to a high of 99.0% in Lesotho in the second period (maps on the left). The high-resolution maps allow to clearly visualise the marked within-country variations (maps on the right). The maps of women's education and men's decision-making dominance seem to follow

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where $\bar{y}$ is the sample mean. The significance of this spatial correlation can be assessed by means of a permutation approach.

[15] We also calculate the Moran's I index for the raw dataset, that is a dataset in which each observation, representing the DHS cluster, has one value for the variable men's decision-making dominance. This variable is measured as the proportion of women in a given cluster who report that their husband is the sole decision-maker about their health. We do this to show that the spatial interpolation has not introduced spatial dependence in men's decision-making dominance and thus that spatial dependence was already existent. In other words, we want to prove that our findings are not an artefact of interpolation. Because clusters are spatial points (e.g., a single coordinate pair) and therefore cannot share a common edge or vertex, a neighbouring location is defined as the point that lies within 25 km from the reference point. The Moran's I index is equal to .55 (p-value < 0.001) in the 2000s and to .61 (p-value < 0.001) in the 2010s.

the same pattern. That is, cells with high proportions of women with some education appear to have low proportions of men's decision-making dominance, and so on. Figure 3.4 shows the spatial distribution of the urbanisation variable in 2002 and 2013. The variable ranges from 0 to 28 and can be interpreted as the level of urbanisation per cell. As expected, higher levels of urbanisation are more common around large cities (e.g. Lagos, Nairobi, and Harare).

[Figures 3.3 and 3.4 here]

In Figure 3.5, we present maps of change in men's decision-making dominance (left) and women's education (right) at the country and local levels between the first and second time period. Maps showing the temporal change at the country-level suggest that male decision-making dominance has decreased over time in most countries. However, the local-level maps show that decreases over time have not been homogenous within countries. In particular, the local-level maps show that some areas have experienced a decline over time in male decision-making dominance (red cells), other areas have experienced an increase over time (blue cells). In particular, Mali, Senegal, Central and Western Guinea, Eastern Burkina Faso and Zimbabwe, Northern Nigeria and Uganda, as well as Southern Cameroon have had the most increases in prevalence of male decision-making dominance between the first and second time periods. Similarly, the maps of change in women's education show that within-country variation over time is high. Nonetheless, although there are a few areas where the proportion of women with at least some education has decreased over time (red cells), it has increased over time in most areas (blue cells).

[Figure 3.5 here]

Estimation Results

In this section we present the results from the SDM panel fixed effects model estimated for the 3,186 cells with a value in both earlier and later time periods. The appendix shows the presence of spatial dependence within the data, which suggests that panel data models with a spatial effect are preferred.

To further test the appropriateness of a spatial model specification, we estimate a panel SDM model, shown in Table 3.2. The spatial autocorrelation coefficient in the dependent variable λ and the Wald test statistics are reported at the bottom of the table. The former is equal to .91 (p-value < 0.001), indicating spatial dependence of men's decision-making dominance across cells. This means that a decrease in men's decision-making dominance in neighbouring cells leads to a decrease in men's decision-making dominance in the reference cell. This provides support to a diffusion perspective that norms about family decision-making spread over space and time.

[Table 3.2 here]

We turn to the interpretation of the direct effects based on the panel SDM model (Column 3), which can be interpreted as the effects of urbanisation or education within the cell on male decision-making dominance. We find a one-unit increase in the percentage of women's education in the reference cell decreases the percentage of men's decision-making dominance in that same cell by 0.204 (p-value < 0.001). Thus, cells in which women are, on average, more educated tend to have lower values of male decision-making dominance. Urbanisation also has a negative effect on male decision-making dominance, although the effect is not statistically significant.

We now compare the direct effect estimate (direct effect from Column 3) with the point estimate (β from Column 1) of the panel SDM model to measure the feedback effect, which arises as a result of impacts passing through neighbouring cells and back to the cell that the change originated from; the feedback effect is measured as the difference between

the estimate of the direct effect and the point estimate. This is the reason why in Table 3.2 the direct effect estimates and the point estimates for women's education and urbanisation are different from one another. The estimated coefficient of women's education is, in absolute value, slightly higher for the direct effect estimate of –0.204 than the point estimate of –0.190. The higher coefficient estimate for the direct effect implies that men's decision-making dominance is slightly more responsive to an increase in women's education than is found with the point estimate of the panel SDM model. The same applies to the estimated coefficient of urbanisation that is slightly higher for the direct effect estimate of –0.548 than the point estimate of –0.159. The feedback effects are positive and equal to 0.014 (in absolute value) for women's education and to 0.389 (in absolute value) for urbanisation, which indicates that feedback effects increase the importance of changes in these variables on male decision-making dominance.

We next look at the indirect effects of women's education and urbanisation on men's decision-making dominance (Column 4), which can be interpreted as the effect of an increase in women's education and urbanisation in all neighbouring cells. In other words, the indirect effects measure the extent to which the characteristics of neighbouring cells influence the dependant variable in a given cell. For example, we find that the proportion of men's decision-making dominance in a given cell decreases by 0.356 points as the proportion of women with at least some education in all neighbouring cells increases by 1 percentage point. As for urbanisation, we find that a 0.31-point (the mean of the variable urbanisation in our sample) increase in urbanisation levels in all neighbouring cells decreases the proportion of male decision-making dominance in a given cell by 3.2 (i.e., $10.324 * 0.31$) percentage points. These results suggest that men's decision-making dominance in a given cell decreases over time because women's education and urbanisation are becoming more widespread in neighbouring cells.

The role of these two variables differs in that female education has a strong direct and indirect effect, whereas urbanisation affects male decision-making dominance only through the indirect effect. The former means that changes in the proportion of women with at least some education in a given cell have an effect on not only its own level of men's decision-making dominance, but also that of its neighbouring cells. The latter means that the level of men's decision-making dominance of a given cell is not affected by the level of urbanisation of that cell, but only by the level of urbanisation of all neighbouring cells. This finding could be because more urbanised areas are more likely to house infrastructure such as hospitals or healthcare centres, that is places where people may interact with each other and be exposed to new ideas about gender norms. The last column of Table 3.2 shows the estimated results for the total (sum of the direct and indirect effects) effects, which can be interpreted as the total marginal effects of a variation in the women's education and urbanisation on men's decision-making dominance. Our results show that decreases in male decision-making dominance are driven by both women's education and urbanisation. Both factors have a negative total effect on men's decision-making dominance.[16]

Conclusions

Drawing on demographic literature on the importance of women's status in the family for health and wellbeing, we used reports of men's dominance in decision-making about as a measure of women's status within the family. We adopted a spatial approach to map spatial and temporal variation in male decision-making dominance both within and between countries in sub-Saharan Africa. We also explored whether diffusion of new ideas about family decision-making helped account for observed declines in male decision-

[16] We estimate additional alternative models (not shown) as a means of robustness checks. These are models where the spatial weighting matrix is defined differently and, in particular, it is based on the rook's contiguity criterion, that is on shared boundaries only, as opposed to shared edges and vertexes. Results are robust when

making dominance between the early 2000s and 2010s and identified some of the drivers behind this diffusion. In doing so, our analyses made a number of contributions to the demographic literature on women's status, norms, and social diffusion.

A first contribution of the paper was to document the advantages of using a spatial approach to explore variation in women's status both within and between countries. While aggregated estimates of women's status, such as those produced by UNDP, provided important baseline information our analyses showed how aggregate estimates masked enormous spatial heterogeneity within countries. Indeed, we found that about 86% of the variation in changes in men's decision-making dominance across space and time could be attributed to factors that changed within (as opposed to between) countries. This suggested that within country heterogeneity played a meaningful role in shaping observed patterns of male decision-making dominance, which made sense in light of enormous socio-cultural and linguistic heterogeneity within African countries. The spatial perspective has important implications for researchers and policy makers, and the latter group may be particularly interested in using spatial approaches to identify and target geographic areas in need of policy interventions.

A second contribution of the paper was to use spatial regression methods to provide a formal test of whether diffusion of norms helped account for aggregate-level declines in men's decision-making dominance. Although demographers have long hypothesised that diffusion of norms are important catalysts of demographic change (Coale & Watkins 1986) particularly in the African context where industrialisation has remained low, it has been challenging to empirically document how and why diffusion occurs. Our analysis showed that grid cells with similar values of male decision-making dominance tended to be concentrated geographically, thus suggesting a role for processes of social diffusion in spreading of norms about family decision-making. This was further confirmed in our

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spatial panel analysis where we showed that a decrease in men's decision-making dominance in neighbouring cells leads to a decrease in men's decision-making dominance in the reference cell, providing empirical support to a diffusion perspective.

A third contribution of our paper was to provide insight into the pathways through which diffusion may have occurred. Our spatial panel analysis showed that prevalence of women's education and urbanisation in grid cell were negatively associated with men's decision-making dominance (although only the former was statistically significant), which suggested that women's education in the community was associated with declines in male decision-making dominance. Furthermore, we found that men's decision-making dominance in a given cell decreased over time when women's education and urbanisation became more widespread in neighbouring cells. This provided further support to the diffusion perspective and supported work by Caldwell, Thornton, and others about how the spread of women's education and urbanisation play an important role in the diffusion of family decision-making norms.

Although our study made important contributions to the demographic literature on diffusion of norms related to women's status, it had a number of limitations. First, it was plausible that there were geographic boundaries that were not observed in our data (e.g. lakes, mountains) that would make it difficult to interpret spatial trends in decision-making. While this is a general concern will all spatial analyses of this type, we deal with this to the best of our ability by removing unpopulated cells with known lakes, deserts etc. Another constraint of our analysis was that we were only able to focus on women's responses to questions about decision-making because men were not asked about decision-making consistently over DHS survey rounds. Given the prominence of the DHS many demographic studies in SSA focus on women's responses and not men's, and a fruitful area of further enquiry would be to use or collect additional data sources that allows for the

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our neighbours) and by the rook contiguity of first and second order.

comparison of both male and female responses. A further limitation of our analysis was the possibility of omitted variable bias in our panel fixed effects models. Nonetheless, we speculate that it is highly plausible that potential mediators would occur through the primary channels of schooling and urbanisation. A final concern with our panel models was reverse causation, and the possibility that increasing levels of women's decision-making led to increases in women's education and so on. While it is highly plausible that increased women's decision making contributes to increased female education in the next generation, since we focus on the DHS population of women of reproductive age most women in our sample will have completed schooling prior to the survey.

Our analysis demonstrated the importance of considering spatial heterogeneity in measures of women's status—in addition to aggregated indicators or summary statistics—to provide a more complete assessment of changes in women's status over time and space. In doing so, we extended literature which has used spatial methods to map changes in mortality, and education in Africa (Burke et al. 2016; Golding et al. 2017; Graetz et al. 2018; Neal et al. 2016) to explore spatial trends in other dimensions of family dynamics. To the best of our knowledge, this is the first-time spatial methods such as these have been applied to assess the spatial distribution and geographic diffusion of a measure that captures intra-familial gender norms such as decision-making as opposed to morbidity or reproductive health. Given that the availability of geocoded data is growing, this presents a promising avenue for future explorations of women's status by policy makers and researchers alike.

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Fig. 3.1 Maps of DHS respondents' geographic location in 2001–2005 (left) and 2010–2014 (right).

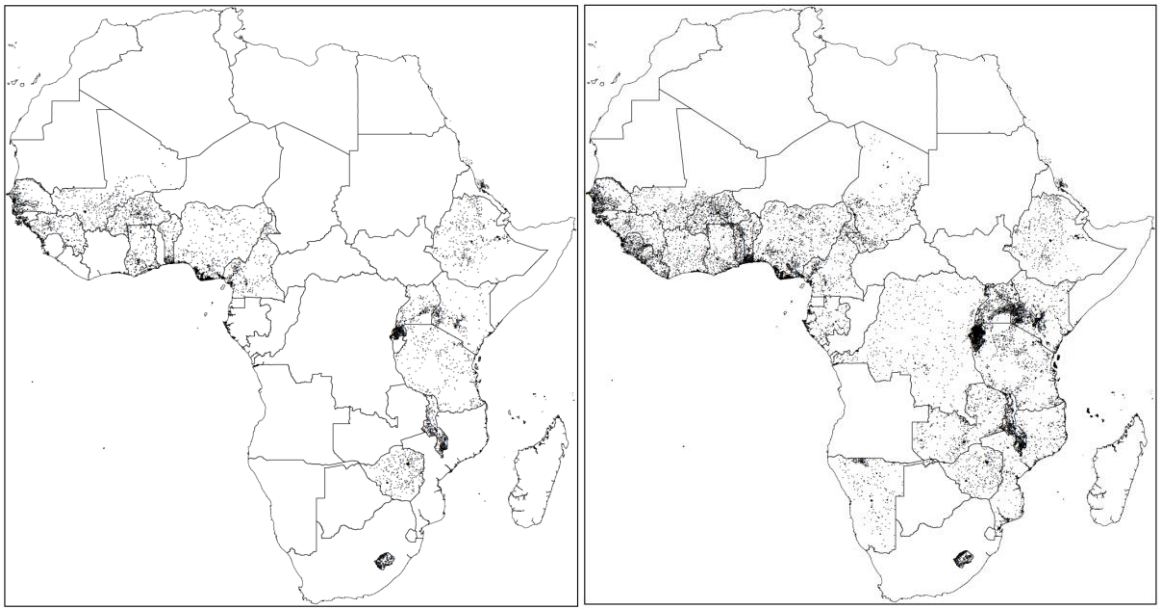

Fig. 3.2 Maps of prevalence of male decision-making dominance in 2001–2005 at the national (top left) and local (top right) levels and in 2010–2014 at the national (bottom left) and local (bottom right) levels.

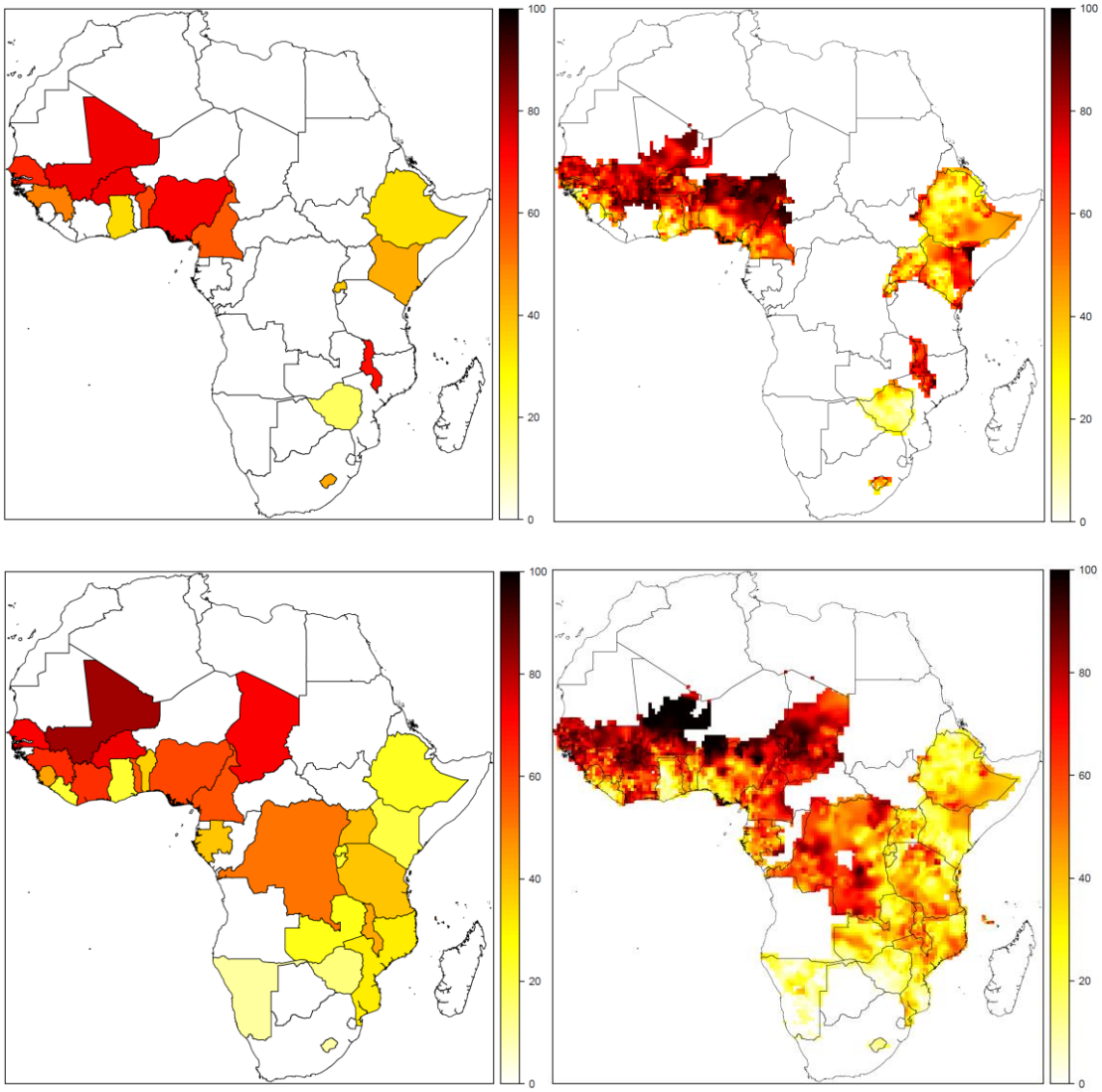

Fig. 3.3 Maps of prevalence of women's education in 2001–2005 at the national (top left) and local (top right) levels and in 2010–2014 at the national (bottom left) and local (bottom right) levels.

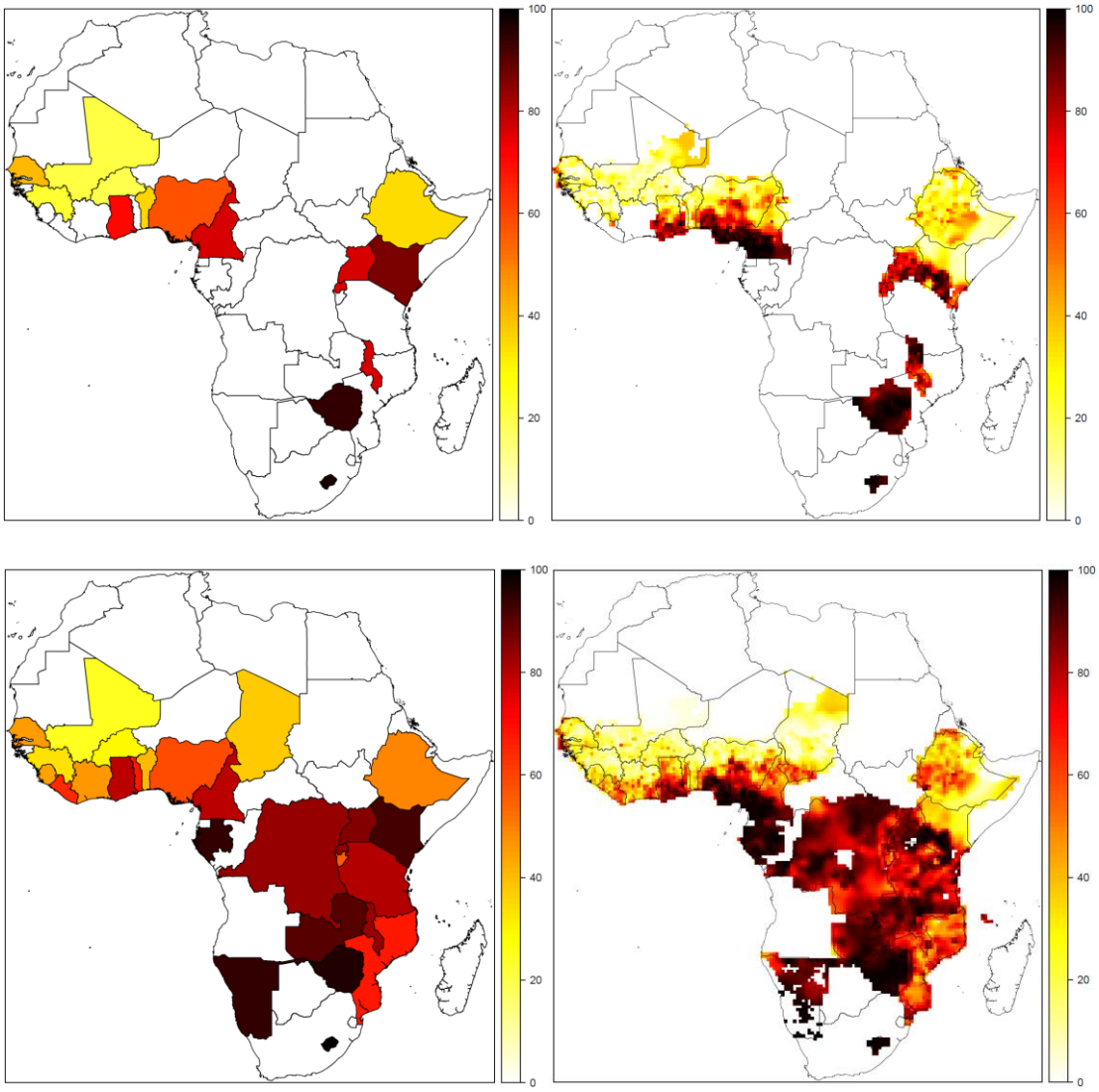

Fig. 3.4 Spatial distribution of urbanisation in 2001–2005 (left) and 2010–2013 (right).

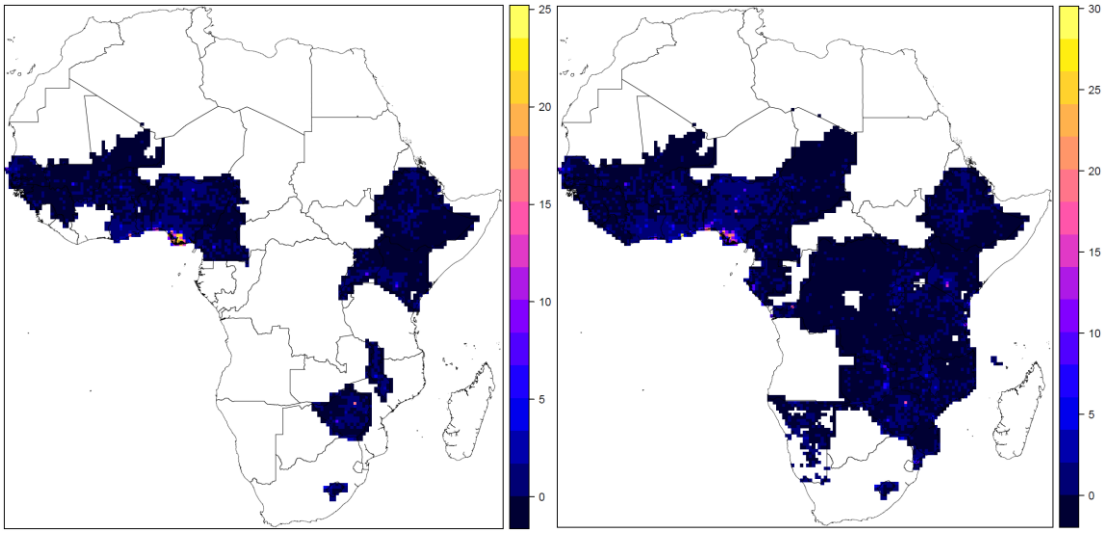

Fig. 3.5 Maps of change between 2001–2005 and 2010–2014 in male decision-making dominance at the national (top left) and local (bottom left) levels and in women's education at the national (top right) and local (bottom right) levels.

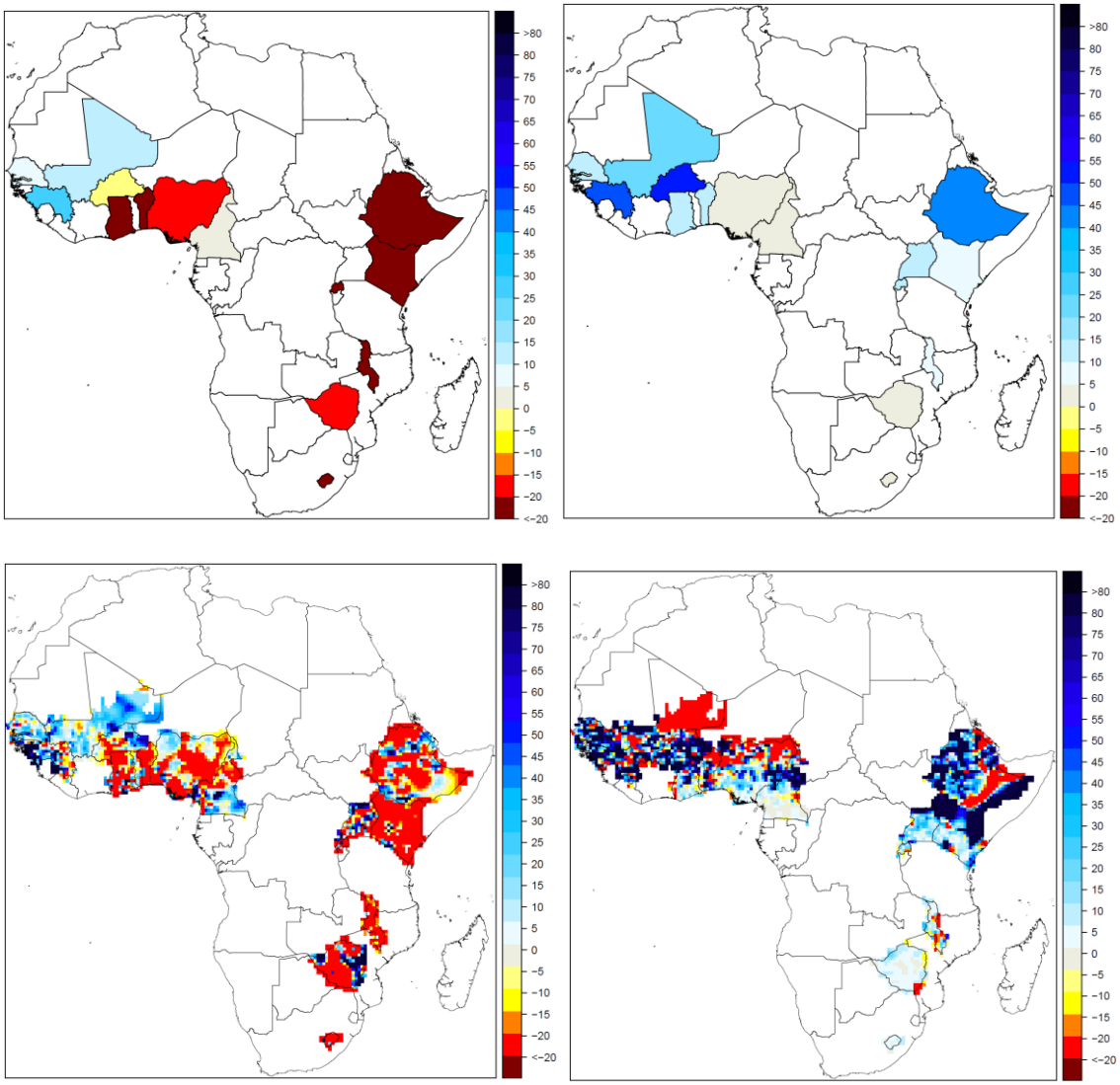

Table 3.1 Characteristics of the DHS samples.

Country	Year	Survey Type	Number of Clusters	N_{opt}	
				Men's decision-making dominance	Women's education
Benin	2001	DHS	247	78	94
Benin	2011	DHS	746	138	177
Burkina Faso	2003	DHS	397	166	146
Burkina Faso	2010	DHS	541	231	234
Burundi	2010	DHS	376	83	173
Cameroon	2004	DHS	464	121	195
Cameroon	2011	DHS	577	145	268
Cote d'Ivoire	2011	DHS	341	112	116
Chad	2014	DHS	624	225	184
Comoros	2012	DHS	242	46	80
Democratic Republic of the Congo	2013	DHS	492	148	227
Ethiopia	2005	DHS	528	110	149
Ethiopia	2011	DHS	571	136	183
Gabon	2012	DHS	331	60	189
Ghana	2003	DHS	410	89	110
Ghana	2014	DHS	423	79	173
Guinea	2005	DHS	291	93	120
Guinea	2012	DHS	300	118	115
Kenya	2003	DHS	398	118	129
Kenya	2014	DHS	1579	130	364
Lesotho	2004	DHS	380	108	121
Lesotho	2014	DHS	399	96	168
Liberia	2013	DHS	322	98	147
Malawi	2004	DHS	520	140	139
Malawi	2010	DHS	827	165	255
Mali	2001	DHS	399	175	164
Mali	2012	DHS	413	215	137
Mozambique	2011	DHS	609	114	152
Namibia	2013	DHS	547	57	221
Nigeria	2003	DHS	360	120	139
Nigeria	2013	DHS	889	329	486
Rwanda	2005	DHS	456	177	161
Rwanda	2010	DHS	984	186	235
Rwanda	2014	DHS			
Senegal	2005	DHS	366	149	164
Senegal	2010	DHS			
Senegal	2012	DHS	782	294	276
Senegal	2014	DHS			
Sierra Leone	2013	DHS	435	136	165
Tanzania, United Republic of	2010	DHS	458	76	342
Togo	2013	DHS	330	95	117
Uganda	2000	DHS	266	100	79
Uganda	2011	DHS	667	105	332
Uganda	2011	AIS			
Zambia	2013	DHS	719	129	225
Zimbabwe	2005	DHS	396	98	159
Zimbabwe	2010	DHS	393	101	241

Table 3.2 Results from panel SDM.

	Parameter estimates		Marginal effects		
	β	γ	Direct effect	Indirect effect	Total effect
Women's education	-0.190***	0.140***	-0.204***	-0.356*	-0.560***
	(0.022)	(0.027)	(0.021)	(0.139)	(0.141)
Urbanisation	-0.159	-0.787	-0.548	-10.324**	-10.873**
	(0.323)	(0.485)	(0.340)	(3.825)	(3.950)
λ			0.911***		
			(0.008)		
H_0: γ=0			27.99***		
H_0: γ+λβ=0			13.91**		
AIC			21,883.08		
BIC			21,919.48		
Log-likelihood			-10,935.54		
N			6,372		

+p<.1; *p<.05; **p<.01; ***p<.001

Appendices

Spatial panel data models

In this paper, we consider three types of spatial panel specifications: the spatial lag (SAR) panel model, the spatial error (SEM) panel model, and the spatial Durbin (SDM) panel model. The SAR panel model is expressed as follows:

$$y_{it} = \lambda \sum_{j=1}^{N} w_{ij} y_{jt} + \boldsymbol{x}_{it}\boldsymbol{\beta} + \mu_i + \varepsilon_{it}, \qquad \text{(A1)}$$

where λ is the coefficient of the spatially lagged dependent variable and referred to as the spatial autocorrelation coefficient in the dependent variable. This set-up allows male decision-making dominance in cell i and year t, y_{jt}, to depend on male decision-making dominance observed in neighbouring cell j and year t, y_{jt}. A positive estimate of λ indicates that decreases in male decision-making dominance in neighbouring cells are associated with decreases in male decision-making dominance in the reference cell i. It thus informs us about the existence of a diffusion mechanism, according to which reductions in male decision-making dominance spread across cells, after controlling for observed characteristics and unobserved time-invariant characteristics. Unlike the cross-sectional spatial models, where λ represents a spatial pattern reflecting a diffusion process, the introduction of the temporal dimension in spatial panel models reinforces the interpretation of λ as reflecting diffusion.

The SEM panel model is described as follows:

$$y_{it} = \boldsymbol{x}_{it}\boldsymbol{\beta} + \mu_i + u_{it}, \qquad u_{it} = \rho \sum_{j=1}^{N} w_{ij} u_{jt} + \varepsilon_{it}, \quad \text{(A2)}$$

where u_{it} is the spatially autocorrelated error term. This model allows the error term to be spatially autocorrelated, that is it allows for spatial clustering in unobservable characteristics.

To determine whether the panel SAR model or the panel SEM model better describes the data than a model without any spatial interaction effects, the Lagrange Multiplier (LM) tests for a spatially lagged dependent variable and for spatial error

autocorrelation may be used. The robust LM tests (Anselin, Bera, Florax, & Yoon, 1996) may then be used to identify both which spatial effects (λ and/or ρ) is relevant and the existence of one type of spatial dependence conditional on the other (Elhorst, 2014). These tests test the hypothesis that the panel spatial model specifications improve the fit and are based on the residuals of the non-spatial panel data (e.g., pooled OLS) model, each following a chi-square distribution with one degree of freedom. If all hypothesis are rejected, then the non-spatial panel data model is dismissed in favour of the panel SAR model or the panel SEM model. The four tests shown in Table 3.A1 point to a spatial model specification. Nevertheless, one should be careful to select one of these two models. We explain why below.

[Table 3.A1 here]

The SDM panel model extends the SAR panel model by adding spatially lagged independent variables and is expressed as follows:

$$y_{it} = \lambda \sum_{j=1}^{N} w_{ij} y_{jt} + \boldsymbol{x}_{it}\boldsymbol{\beta} + \sum_{j=1}^{N} w_{ij} \boldsymbol{x}_{ijt}\boldsymbol{\gamma} + \mu_i + \varepsilon_{it}, \quad \text{(A3)}$$

where $\boldsymbol{x}_{ijt}$ is the vector of independent variables measured in cell j and year t, and $\boldsymbol{\gamma}$ is the matching vector of coefficients. The advantage of this model is that it allows men's decision-making dominance in each cell i to depend on a set of characteristics measured in the same cell and on an average of the same characteristics measured in neighbouring cells. The latter expresses the extent to which male decision-making dominance in cell i is affected by women's education and urbanisation averaged over its neighbouring cells, and thus it allows identifying the drivers behind the diffusion of new norms about family decision-making.

The panel SDM model is used to test whether the model can be simplified to a panel SAR model or to a panel SEM model since these are nested within the panel SDM model (Elhorst, 2013; LeSage & Pace, 2009). In particular, the panel SDM model is used to test the hypotheses $H_0: \gamma = 0$ and $H_0: \gamma + \lambda\beta = 0$. Both tests follow a chi-square

distribution with K degrees of freedom, where K are the number of explanatory variables. If both hypotheses are rejected, then the panel SDM model best describes the data. On the contrary, if the first hypothesis cannot be rejected, the panel SDM model can be simplified to the panel SAR model (Burridge, 1981), provided that the (robust) LM tests also pointed to the panel SAR model. Likewise, if the second hypothesis cannot be rejected, the panel SDM model can be simplified to the panel SEM model (Burridge, 1981), provided that the (robust) LM tests also pointed to the panel SEM model. Elhorst argues that if the (robust) LM tests point instead to another model than the Wald tests, then the panel SDM model should be adopted. This is because the panel SDM model is a more general spatial model. According to the tests presented in the paper (Table 3.2), both the hypothesis $H_0: \gamma = 0$ and $H_0: \gamma + \lambda\beta = 0$ are significantly rejected. We can therefore infer that the panel SDM model best describes the data.

Local Indicator of Spatial Autocorrelation

The Local Indicator of Spatial Autocorrelation (LISA) allows us to identify local clusters and local spatial outliers (Anselin 1995) by indicating if neighbouring cells have high or low values. In particular, cells can be deemed as to be not significant or a cluster of 'high-high', 'low-low', 'high-low', and 'low-high' values relative to neighbouring cells at a probability level of 0.05 or less (significance values based on permutation). In Figure 3.A1, we show one scatterplot map of the LISA for 2001–2005 (top) and one for 2010–2014 (bottom). Cells in the West are defined as 'high-high' areas, that is areas where male decision-making dominance is persistently high across neighbouring cells. On the contrary, cells in the East are 'low-low' areas, that is areas in which male decision-making dominance is persistently low across neighbouring cells. The map also identifies very few local outliers, defined as 'low-high' and 'high-low' areas, that is cells that have low values

of men's decision-making dominance and neighbours with high values of men's decision-making dominance, and vice versa.

[Figure 3.A1 here]

Robustness of descriptive findings

In order to show the robustness and validity of our descriptive findings, we conduct three robustness checks. First, we perform the same analyses using different N parameters – i.e., $N_{opt} * 2$ and $N_{opt}/2$ – and verify that our results do not depend on the choice of N_{opt}. Second, we apply the spatial interpolation methods to the same indicators to create maps at a lower resolution (i.e., 1 x 1-degree grid cell) and find that our results and inferences are not affected by the number of grid cells in the country. Third, we validate the results of our gridded data by following three steps. We first create measures of male decision-making and women's education at the country level aggregating the cell-level estimates at the country level, that is by taking the mean across cells within country (country boundary shapefiles are used). Next, we create additional measures of male decision-making and women's education at the country level by calculating, respectively, the proportion of women in a given country in which the partner/husband is the sole decision maker on the women's health and the proportion of women in a given country who have at least some education. Then, we compare the distributions of the former measures with the distributions of the latter measures. If they are similar, then the difference between measures is small and the aggregated measures are good and reliable indicators of men's decision-making and education at the country level. In Figure 3.A2, we present density plots of men's decision-making dominance (left) and women's education (right) for the 2001–2005 and 2010–2014. Expect for few values located at the lowest (left) end of the distribution of women's education, the density functions are very similar. The measures are

also highly correlated, i.e., 0.98 for men's decision-making dominance and 0.96 for women's education.

[Figure 3.A2 here]

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Fig. 3.A1 Maps of local indicator of spatial autocorrelation in male decision-making dominance in 2001–2005 (top) and 2010–2014 (bottom).

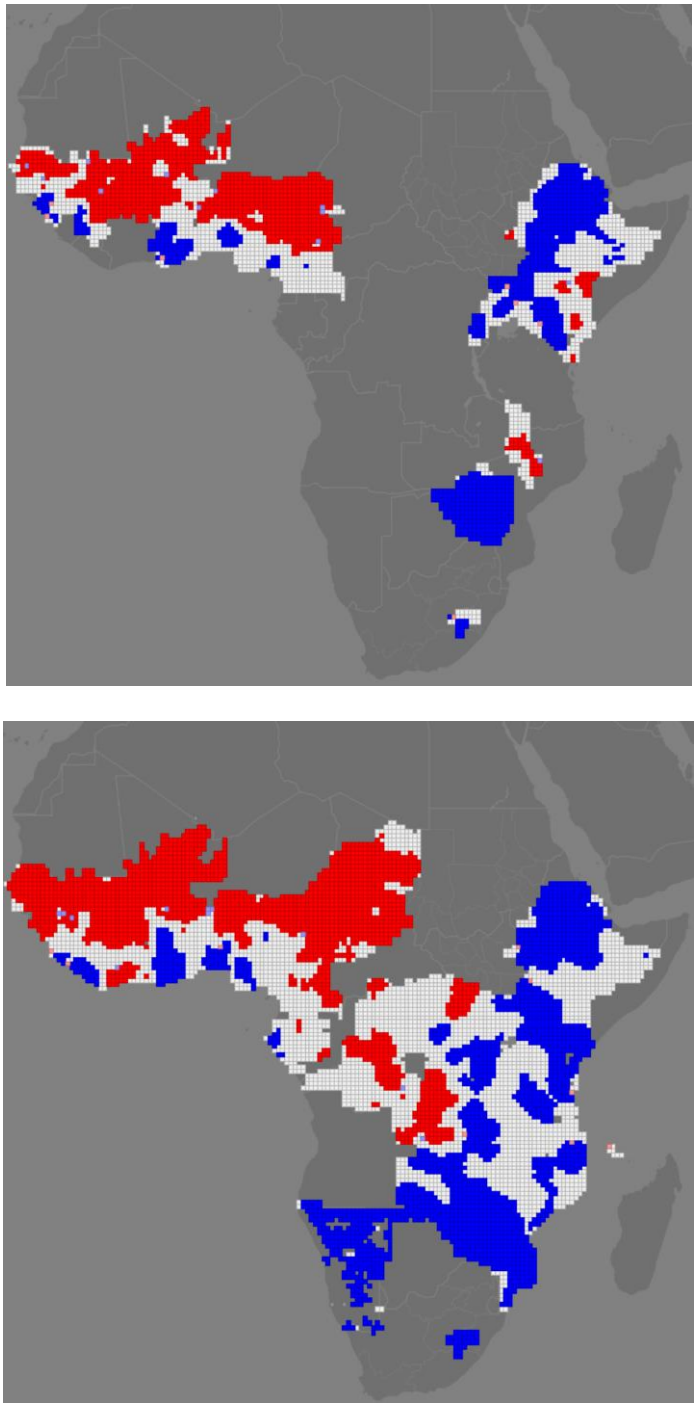

Fig. 3.A2 Density plots of country-level estimates and aggregated cell-level estimates of male decision-making dominance in 2001–2005 (top left) and 2010–2014 (bottom left) and of women's education in 2001–2005 (top right) and 2010–2014 (bottom right).

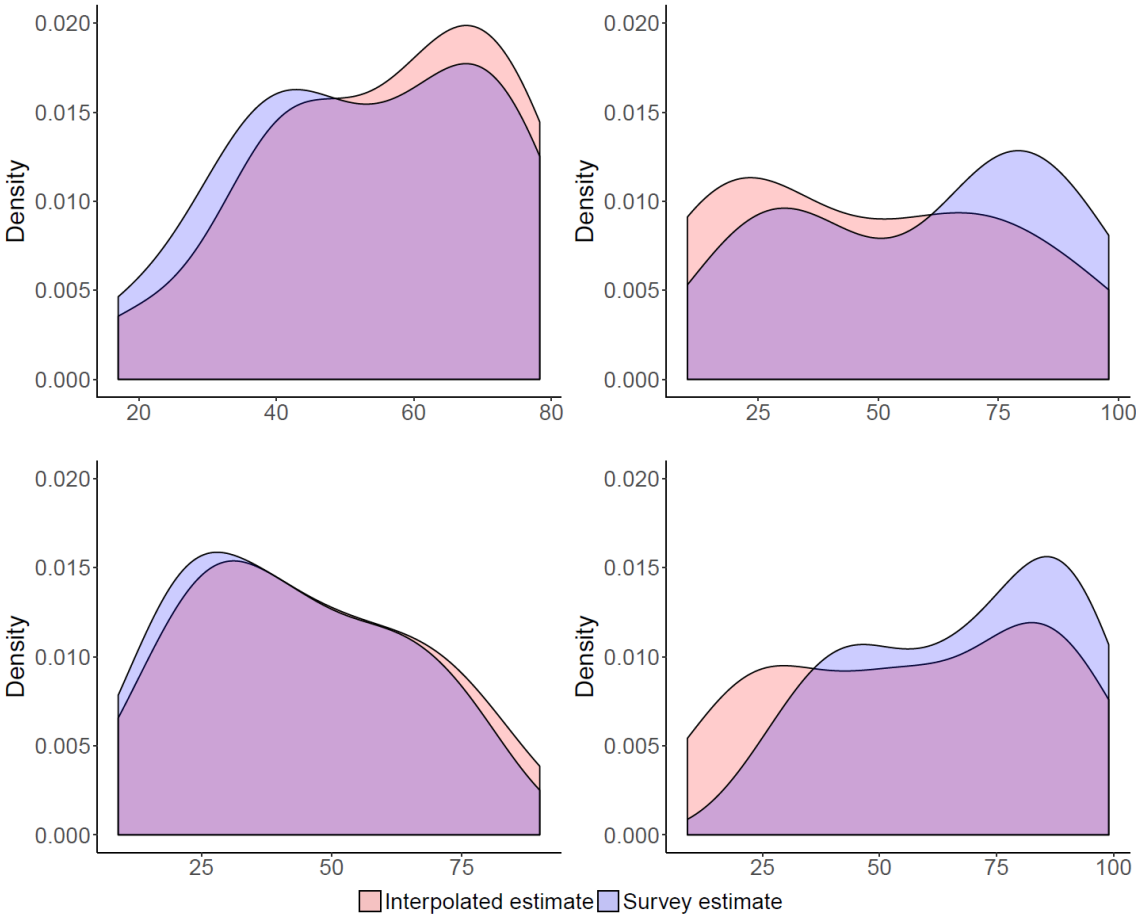

Table 3.A1 Pooled OLS estimates and Lagrange Multiplier tests.

	Men's decision-making dominance
Women's education	-0.442***
	(0.009)
Urbanisation	0.553**
	(0.181)
Constant	72.371***
	(0.460)
LM spatial lag	*17,008.939***
LM spatial error	*17,776.909***
Robust LM spatial lag	*29.541****
Robust LM spatial error	*797.511****
N	*6,372*

+p<.1; *p<.05; **p<.01; ***p<.001

Chapter 4. The Effect of In-Utero Exposure to Growing-Season Droughts on Under-Five Mortality and the Role of Maternal Education in Sub-Saharan Africa[17]

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Abstract

Limited research explores how extreme weather affects child mortality in Sub-Saharan Africa, although the region is highly vulnerable to both weather shocks and early childhood mortality. I use innovative methods to explore whether in-utero exposure to growing-season droughts has an effect on early childhood mortality and whether maternal education is protective against drought-related fatality in early childhood. Combining individual-level data from 98 Demographic and Health Surveys from 32 countries in sub-Saharan Africa with high-resolution climate and calendar crop data, I develop a measure of drought shocks based on the Standardised Precipitation Evapotranspiration Index. Using a high-dimensional fixed-effects approach that exploits the exogenous timing of droughts, I find that: i) in-utero exposure to growing-season droughts significantly increase child mortality, especially in rural rainfed areas; ii) in-utero exposure to growing-season droughts is associated with significantly higher mortality for children of low-educated mothers than for children of high-educated mothers. I speculate that this is because more-educated mothers have greater access to nutritious food which is protective in times of hardship.

Keywords: droughts, weather shocks, child mortality, women's education, sub-Saharan Africa.

Introduction

Climate change is expected to exacerbate the already existing vulnerabilities of the poorest people, who depend on agriculture for livelihood and sustenance. In particular, as a consequence of climate change, the intensity, frequency, and duration of weather shocks will increase (Collier et al. 2008; Masih et al. 2014), and this will adversely affect agricultural productivity and human health. This will particularly have huge implications for the wellbeing and health of the most vulnerable segments of the rural population, such

as pregnant women and children from poor households. The literature shows that adverse weather shocks prior to birth can severely affect child health outcomes and infant mortality through their effect on the disease environment, and on maternal malnutrition and stress (Cornwell and Inder 2017; Flatø and Kotsadam 2014; Kudamatsu et al. 2012; Rocha and Soares 2015). Despite the huge importance of climate change for population processes, there has been much less progress in analysing the effects that exposure to weather shocks prior to birth have on early childhood mortality.

In light of climate change, scholars and policy makers are increasingly looking for ways to mitigate the negative consequences of weather shocks. Public investments in universal education in developing countries are often proposed as promising way to improve child survival chances (Gakidou et al. 2010), and are also considered as a top priority for reducing natural disaster vulnerability and enhancing adaptive capacity vis-à-vis future climate change (Lutz et al. 2014; Muttarak and Lutz 2014). Despite its policy relevance, there is only limited research that empirically explores whether maternal schooling can enhance adaptation to weather shocks during pregnancy in developing countries.

Drawing on these gaps in the literature, this paper explores the effects of in-utero exposure to droughts during growing seasons on under-five mortality, and the role maternal education plays in mitigating children's susceptibility to droughts in utero in sub-Saharan Africa. Because of high agriculture dependence and limited capacity to adapt, Sub-Saharan Africa is one of the most vulnerable regions to climate change and variability, and in particular, to frequent and severe droughts (Boko et al. 2007; Collier et al. 2008; Esikuri 2005). Due to climate change, the frequency and intensity of droughts in the region will increase (Collier et al. 2008), as will the susceptibility due to fast growing populations, expected increasing water demands, and degradation of land and environmental resources (Masih et al. 2014). The worrisome implications for water resource sustainability and food

security are likely to have a large impact on population health and wellbeing. The impact is expected to be especially pronounced on children, as children experience 88% of the disease burden induced by climate change (Zhang et al. 2007). In recent years there have been substantial improvements in early childhood mortality in sub-Saharan Africa, but weather shocks have the potential to offset this progress.

My paper makes three main contributions to the demographic literature on the effect of in-utero exposure to weather shocks on early childhood mortality. Past empirical literature limits the analysis of the mortality consequences of in-utero exposure to growing-season droughts to infants (Flatø and Kotsadam 2014; Kudamatsu et al. 2012; Rocha and Soares 2015), while overlooking its effects on under-five mortality. Because in developing countries childhood deaths occur at high rates between ages one and five, focusing on infant mortality might underestimate the negative consequences of droughts. In this paper, I provide new estimates of the effect of in-utero exposure to growing-season droughts on under-five mortality. I exploit the exogeneous timing of weather events to develop a measure of drought shocks based on the mother's geographic location and employ a fixed effects design that allows to disentangle the effect of droughts from seasonal effects on under-five mortality and from common and unobserved factors that affect both weather and under-five mortality.

The second contribution is methodological and relates to both the measure of drought shocks and the period in which shocks occur. Previous research exploring the effect of in-utero exposure to droughts on child health outcomes generally defines it as lack of rainfall in the months prior to birth. The literature, however, argues that decreases in precipitation are not the only cause of droughts and that increases in evaporation from soil and vegetation which are driven by global warming also play a role (Masih et al. 2014; WMO 1975). In this paper, I use a more comprehensive indicator of droughts that takes into account more climate parameters than just precipitation: the Standardised Precipitation

Evapotranspiration Index (SPEI). The SPEI is viewed as the most complete and robust agricultural drought index in Africa (WMO 2012). My analysis is the first combining it with data on early childhood mortality. As a further step, I measure drought shocks during the growing season of the main crop cultivated in the area where mothers reside, where instead most studies use monthly and yearly averages in the twelve months before birth. The growing season is the period when crops are most vulnerable to unfavourable weather conditions and is of crucial important for crop productivity, especially in sub-Saharan Africa, where a negligible part of the land is irrigated (i.e., 6.4% in 2004 (Frenken 2005)) and yields and prices are determined by changes in weather.

The third contribution of the paper is to explore for the first time the role that maternal education plays in mitigating children's vulnerability to weather shocks during pregnancy. Previous research from the US only focuses on siblings' health endowments at birth to explore whether more educated mothers are more protective of their less healthy child. Specifically, Hsin (2012) finds that more educated parents are more likely to compensate birthweight differences, while Datar et al. (2010) do not find any evidence supporting this argument. Given the large expansion of women's education in sub-Saharan Africa in recent decades, maternal education may be a potentially very important mitigator of the impact of weather shocks on early childhood mortality.

The Effects of Weather Shocks on Early Childhood Health and Mortality: Existing Evidence Base and Theoretical Perspectives

Prior Research on the Effects of Adverse Shocks

Health-damaging exposure to environmental risks can begin before birth, negatively affecting the growth of the foetus (WHO 2002). It is known that adverse environmental shocks in utero can have detrimental consequences on health at birth (Almond and Currie 2011; Almond and Mazumder 2011; Andalón et al. 2016; Camacho 2018; Currie and

Rossin-Slater 2013; Dorélien 2019; Torche 2011) and that parents tend to invest less in less healthy children (Almond et al. 2009; Almond and Mazumder 2013). Beyond the worrisome implications on children's health and mortality later in life, the latter finding might confound the impact of in-utero exposure to negative shocks on child mortality. Therefore, if in-utero conditions trigger a number of events that affect individual outcomes in childhood, we should place attention to this critical period. This work expands on the foetal origins hypothesis according to which the intrauterine environment, and, in particular, maternal nutrition, programs the foetus to have certain characteristics which can lead to future complications (Barker 1990).

A growing literature is focusing on the effect of intrauterine exposure to weather shocks on infant mortality and early childhood health. To my knowledge, only three studies analyse the effect of in-utero exposure to weather shocks on infant mortality. Kudamatsu et al. (2012) find that exposure to droughts in utero increases mortality among infants born in the hungry season in arid areas in Africa via its effect on maternal malnutrition and undernutrition. Their findings show that droughts in the two rainy seasons before birth raise infant mortality by 23.1 per 1,000 live births. Flatø and Kotsadam (2014) show that in sub-Saharan Africa, extreme droughts in the rainy season prior to birth affect female infant mortality only, causing the deaths of 11.9 girls per 1,000 live births. They also argue that the gender difference in infant mortality following droughts is due to discrimination against daughters. In a study from the semiarid Northeast Brazilian region, Rocha and Soares (2015) find that exposure to a drought twelve months before birth increases mortality among infants by 3.3 points and the effect is mainly concentrated on intestinal infections and malnutrition.

With regard to the health consequences of weather shocks among children, the literature shows mixed evidence. One study from Indonesia finds that rainfall during gestation significantly affects height-for-age among urban children, and in particular, that

an average monthly rainfall difference of 200 mm translates into a z-score difference of 0.27 (Cornwell and Inder 2017). Another studies from India reports that drought exposure in the year before birth reduces weight-for-age z-score by about 0.1 standard deviations and increases the probability of being underweight or severely underweight by about 2 percentage points, but finds marginal effects on anaemia (Kumar et al. 2016). On the contrary, no significant effects of in-utero exposure to excess rainfall on children's height and weight are found in rural Mexico (Aguilar and Vicarelli 2011).

Another stream of literature analyses the effects of weather shocks on health at birth. One study in the US finds that exposure to extreme hot temperatures during the second and third trimester of pregnancy worsens health at birth by reducing birthweight between 7.5 and 11.5 g (Deschênes et al. 2009). Another study from the semiarid Northeast Brazilian region finds that being exposed to positive rainfall shocks twelve months prior to the birth improves the health of new-borns (Rocha and Soares 2015). In particular, a one standard deviation increase in rainfall is associated with an increase of 1.6 g in birthweight and 0.3 percentage point in the fraction of full-term pregnancies. Finally, in Colombia exposure to moderate heatwaves during the third trimester of pregnancy is found to reduce birthweight of infants by 4.1 g, while exposure to moderate cold shocks during the first and second trimesters of pregnancy reduces the length at birth by 0.014–0.018 cm (Andalón et al. 2016). The same study finds that in-utero exposure to heatwaves reduces the normal Apgar score by –0.2 to –0.6 percentage points.

Drought and Early Childhood Mortality: Pathways of Influence

There are a number of reasons why in-utero exposure to droughts may effect early childhood mortality. During droughts, households experience reductions in quantity, quality, and variety of crops produced as well as large increases in food prices (e.g., the 1991–1992 drought in Zimbabwe is found to increase food prices by 72% (Stern 2006))

(Rabassa et al. 2012). In order to cope with reductions in food access (i.e., food availability and affordability), vulnerable households tend to prioritise calorie-rich but nutrient-poor food (Bloem et al. 2010). The subsequent declines in dietary quality and quantity increase micronutrient malnutrition of pregnant women and exacerbate their pre-existing vulnerabilities. Pregnancy is in fact the most nutritionally demanding period in a woman's life (Gebremedhin and Enquselassie 2011), and maternal malnutrition and undernutrition during pregnancy are a known cause of low birthweight which is a major cause of death among children (Strauss and Dietz 1999).

Moreover, droughts change the disease environment and increase susceptibility to disease. In particular, droughts decrease quantity and quality of water and increase vulnerability to infections associated with polluted water supplies (De Waal 1989). Because pregnancy causes a naturally induced immune suppression, pregnant women are more vulnerable to water-borne illnesses. Amongst these, chronic diarrhoea, caused by unsafe water use, leads to anaemia and other micronutrient deficiencies which in turn hamper both maternal and new-born health (Chen et al. 2012; Watt and Chamberlain 2011). The positive association between water scarcity and higher prevalence of diarrhoea seems to be limited to the dry season (Bandyopadhyay et al. 2012), and there is no clear indication of how this relationship persists in the growing season which I consider in this work. Recent research also shows that droughts increase HIV prevalence among women and identifies transactional sex as one of the pathways through which this effect operates (Burke et al. 2014). Engaging in transactional sex before and during pregnancy can increase the risk of in-utero transmission from an HIV-infected mother to her children and their subsequent risk of death in the absence of antiretroviral therapy (WHO 2010).

Finally, droughts affect both stress levels and energy expenditure of pregnant women. Maternal stress leads to the production, in both the mother and the foetus, of hormones and cortisol related to premature delivery (Glynn et al. 2001; Hobel 2004;

Lockwood 1999). Previous studies find that maternal stress can negatively affect birthweight both by shortening gestational age (Copper et al. 1996; Nordentoft et al. 1996; Torche 2011) and by its effects on intrauterine growth restriction (Wadhwa et al. 2004). Droughts can also increase the distance required to access clean water. Previous evidence shows that carrying large heavy water loads can have detrimental effects on women's health through musculoskeletal disorders and related functional disability (Geere et al. 2010); however, little is known about these effects during pregnancy (Watt and Chamberlain 2011). Additionally, as a consequence of lower yields of crops following droughts, women may work less in agriculture and diversify into non-agricultural activities and thus have more than one job (Branco and Feres 2017). The subsequent harder labour performed may even increase the risk of spontaneous abortion (Boserup 1989).

The Moderator Effect of Maternal Education

In response to climate change, scholars and policy makers are increasingly interested in factors that might lessen the negative consequences of weather shocks. Given the vast expansion in women's education in sub-Saharan Africa over the past few decades, maternal education is particularly relevant. Women's education is an important determinant of child survival chances; previous studies find a causal link between maternal education and child mortality (Andriano and Monden 2019; Breierova and Duflo 2004; Chou et al. 2010; Grépin and Bharadwaj 2015; Makate and Makate 2016). The literature suggests that this relationship persists during famines when food prices rise dramatically (Kiros and Hogan 2000, 2001), and that women's education both reduces vulnerability to weather shocks and increases the propensity to prepare against natural disaster (Hoffmann and Muttarak 2017; Lutz et al. 2014).

Schooling could enhance adaptation to droughts during pregnancy through a number of pathways both at the individual and societal level. First, women's education can

act as the catalyst for changes in the health system and as a complement to health provision (Caldwell 1986). Schooling may affect the environment in which women are embedded by both living in communities with better amenities (e.g., water and sanitation systems) and enhancing hygiene practices at home (Desai and Alva 1998; Hatt and Waters 2006). Second, more educated women may also acquire command over food and medical care in harsh conditions. As argued by Sen (1981), whether food access decline leads to starvation depends on capability failure which in turn depends on market access and households' social, economic, and political entitlements. Education is considered as one of the most important publicly-conferred entitlements (Frenk et al. 1991). Empirically, schooling is found to improve the socioeconomic position of women, reduce their financial barriers to medical care, and enhance their relative position in the household (Andriano and Monden 2019; Behrman 2015; Caldwell 1986; Jejeebhoy 1992; Keats 2016; Thomas 1990). Finally, women's education may also favour healthy behaviours during pregnancy (Grossman 1972). As an example, previous research shows that it increases ownership and use of insecticide-treated nets among pregnant women (Singh et al. 2013).

Data

I combine three sources of data (1) Demographic and Health Survey micro data on child mortality and maternal education; (2) climate data produced by the European Centre for Medium-Term Weather Forecasting; and (3) calendar crop data from the Global Monthly Irrigated and Rainfed Crop Areas dataset. Each of these data sources is discussed in detail below.

Individual Data

The individual-level data come from 98 Demographic and Health Surveys (DHS), Malaria Indicator Surveys (MIS), and AIDS Indicator Survey (AIS) from 32 countries in sub-

Saharan Africa. All DHS, MIS, and AIS available as of late 2016 that include latitude and longitude information on women's geographic location at survey are used (see Supplementary Material for more information on data collection and the list of surveys included). Women's geographic location at survey serves as proxy for women's geographic location during pregnancy. Only children born in the 5-year period are considered to avoid recall bias due to omission and backward displacement of births in sub-Saharan Africa (Pullum et al. 2013; Schoumaker 2011). Children not living with their mothers at the time of the survey are excluded (i.e., 4% of the total number of children) since their geographic location does not correspond with their mothers. Table 4.1 provides information on the pooled sample. The compiled data contain over 36,928 clusters, with an average of 27 children per cluster (see Fig. 4.1 for a map of the DHS clusters). In total, my analytic sample covers 714,580 children born in the 5-year period before the DHS survey.

[Table 4.1 and Figure 4.1 here]

Climate Data

The climate data used to compute the Standardised Precipitation Evapotranspiration Index (SPEI) are taken from the high-quality re-analysis dataset ERA-Interim archive produced by the European Centre for Medium-Term Weather Forecasting (ECMWF) (Dee et al. 2011) (see Supplementary Material). The data are previously validated by other studies that explore the effect of weather shocks on infant mortality and conflict incidence (Flatø and Kotsadam 2014; Harari and La Ferrara 2018).

The variables used for the computation of SPEI are specified in Supplementary Material. The data contain information on each weather parameter for every six hours from 1 January 1979 to 31 August 2016, on a global grid of parallels and meridians at a 0.75 × 0.75-degree resolution (about 80 km by 80 km at the equator). In other words, for each grid cell a time-series of each variable is available.

Calendar Crop Data

The calendar crop data are used to derive the growing season of the main crop in the area in which each woman resides. I use the Global Monthly Irrigated and Rainfed Crop Areas (MIRCA2000), which is a dataset of monthly growing seasons of 26 irrigated and rainfed crops at different latitudes and longitudes, with a spatial resolution of 5 arc-minute grids (about 9.2 km by 9.2 km at the equator) (Portmann et al. 2010).

For each grid cell, I identify the main crop cultivated as the crop with the greatest harvested area within the grid cell, and its growing season, defined as the time interval between the last month of the planting period and the first month of the harvesting period. On average, the cultivated area of the main crop accounts for 43% of the total cultivated area within the grid cell. Fig. 4.2 maps the main crop cultivated across Africa and provides a full picture of the cultivation pattern, which exhibits high spatial variation. In grid cells where no crops are cultivated and thus no information on the growing season are available (i.e., blank grid cells), the main crop's growing season of the closest grid cell was used. In the model estimation, I will include a binary variable if the growing-season period of a close grid cell is used in order to account for such approximation.

Using the latitude and longitude data in the DHS, each child is matched to the calendar crop grid cell (MIRCA2000 cell) and weather grid cell (ERA-I cell) in which it falls.

[Figure 4.2 here]

Methods

Growing-Season Droughts

In this section, I first discuss the measure of agricultural droughts and then explain how the in-utero exposure to growing-season droughts indicator is constructed. Agricultural

droughts are associated with a shortage of water for plant growth and assessed as insufficient soil moisture to replace evapotranspirative losses (WMO 1975), which in turn negatively affects crop production (Boken 2005; Sivakumar et al. 2010; Wu et al. 2011). Several indexes have been developed to measure them, but the SPEI has been identified as the most complete and robust agricultural drought index in Africa (WMO 2012) ((Sivakumar et al. 2010) for a review of all agricultural drought indices). This index measures drought severity, intensity, and duration, and unlike precipitation-based drought indices, it also allows for comparisons of drought severity through space and time since it can be calculated over a wide range of climates (Vicente-Serrano et al. 2010). The SPEI also has the advantage to be multiscalar, that is it can be computed at different time scales over which water deficits accumulate to reflect different drought types, with short time scales referring to soil water content and river discharge and long time scales relating to changes in groundwater storage (Vicente-Serrano et al. 2010). For this analysis, I choose to calculate the SPEI at a 3-month time scale (SPEI3) because it reflects short and medium-term moisture conditions which provide a seasonal estimation of climate relevant for agriculture (WMO 2012). As additional analysis, I provide evidence on the impact of SPEI3 on factors capturing the economic and agricultural productivity (see Supplementary Material). In Table 4.A1, I demonstrate that droughts decrease GDP growth by 0.26 percentage point (Column 1) and that main crop's yields are about 23% lower following droughts (Column 2). These results are statistically significant ($p < .001$) and suggest that drought shocks, as measured by the 3-month SPEI, exert substantial influence on both economic performance and agricultural outcomes. Below, I explain how the in-utero exposure to growing-season droughts is derived.

First, I calculate the potential evapotranspiration (PET), that is the amount of water that could be evaporated and transpired if a sufficient water source were available, and the 3-month SPEI for each ERA-I cell and month from January 1979 to August 2016 (see

Supplementary Material for details on the derivation of PET and SPEI). SPEI3 is expressed in units of standard deviation from the cell average and has mean 0 by construction. In accordance with WMO definition of drought intensity, I define an extreme drought event when SPEI3 is smaller than –2, that is when the index is below normal levels by 2 standard deviations (WMO 2012). In this analysis, I hypothesise that substantial increases in child mortality levels stemming from droughts will occur only after extreme shocks. Therefore, I choose to focus on droughts below –2 standard deviations from the mean, but also present results for droughts between the –1 and –1.5 level (i.e., moderate drought) and between the –1.5 and –2 level (i.e., severe drought).

Second, for babies born in location g and date t, I calculate the proportions of consecutive growing-season months in which there is an extreme drought event during the last and second-to-last completed growing-season months prior t. A similar approach is used by von Uexkull et al. (2016) to explore the effects of growing-season drought on conflict.

Finally, I construct the in-utero exposure to growing-season droughts indicator as:

$$gsdrought\_ei_{g,t} = \omega_{g,t} * gsdrought\_ei_1^{g,t} + \left(1 - \omega_{g,t}\right) * gsdrought\_ei_2^{g,t}, \quad (1)$$

where $gsdrought\_ei_1^{g,t}$ and $gsdrought\_ei_2^{g,t}$ are the proportions of consecutive growing-season months in which there is an extreme drought event during the last and second-to-last completed growing-season months prior t. Weight $\omega_{g,t}$ is given by $\omega_{g,t} = \frac{t - h^{g,t}}{12}$, where $h^{g,t}$ is the running month of the last harvest preceding date t in location g.

The in-utero exposure to growing-season droughts indicator is a 0–1 continuous variable where 0 indicates that the mother did not experience any growing-season droughts event in the two years prior to the child's birth and 1 indicates that the mother experienced droughts in every growing-season month in the two years prior to the child's birth. It measures how growing-season droughts affect mothers' nutritional intake during pregnancy.

Control Variables and Model Estimation

In this analysis, I explore the effect of in-utero exposure to growing-season droughts on child mortality, as well as examine whether maternal education is protective against drought-related fatality in early childhood. First, I identify the effect of in-utero exposure to growing-season droughts on child mortality by estimating a high-dimensional fixed-effects model of how droughts affect the probability of dying before age five years. Second, I investigate how maternal schooling modifies this effect by interacting the in-utero exposure to growing-season droughts indicator with the variable years of maternal education.

Before going in detail about the modelling, I discuss the potential confounders of this relationship and how I take into account their effect in the model. Variations in climate depend on weather systems that are spatially correlated in which for example places on one side of a lake receive the same shock and places on the other side receive another shock. The presence of these natural boundaries is correlated with certain characteristics (e.g., culture or ethnicity), which are in turn correlated with child mortality (Flatø and Kotsadam 2014). Therefore, I control for cell-specific levels of mortality by including ERA-I cell fixed effects, that is I compare children born in the same location. Because long-run trends in rainfall and temperature might be correlated with long-run trends in child mortality, there may be indirect macroeconomic or institutional effects of weather shocks on child mortality at the national level (Kudamatsu et al. 2012). Thus, I control for time-variant indirect effects by including country specific year of birth fixed effects. In other words, I compare children born in the same year within the same country.

I also add child-level controls of in-utero exposure to extremely wet growing-season conditions[18], sex, birth order (defined as 1 = first birth; 2 = 2–4 births; 3 = 5+ births), type of residence (defined as 1 = rural; 0 = urban), and season of birth (defined as 1 = hungry; 0 = harvest). Additionally, I control for maternal characteristics, specifically, years of education, age at birth, and age at birth squared. In order to capture potential effects from pooling data across surveys, I also include survey-specific fixed effects. The specification of the model is as follows:

$$p_{igjkcy} = \beta_1 gsdrought\_ei_i + \boldsymbol{x}_i\boldsymbol{\delta} + \boldsymbol{w}_j\boldsymbol{\gamma} + \boldsymbol{\alpha}_g + \boldsymbol{\alpha}_{cy} + \boldsymbol{\alpha}_k + \varepsilon_{igjkcy}, \quad (2)$$

where p_{igjkcy} is a binary variable for under-five mortality of child i born in ERA-I cell g to mother j interviewed in survey k from country c and year y. $gsdrought\_ei_i$ is the in-utero exposure to growing-season droughts, $\boldsymbol{x}_i$ is the vector of child characteristics, and $\boldsymbol{w}_m$ is the vector of maternal characteristics.

The vectors of ERA-I cell fixed effects, country-specific year-of-birth fixed effects, and survey fixed effects are respectively $\boldsymbol{\alpha}_g$, $\boldsymbol{\alpha}_{cy}$, and $\boldsymbol{\alpha}_k$; and ε_{igjkcy} is a mean-zero error term. I allow for correlation of error terms across children born in a wet month in the same ERA-I cell and use survey-specific sampling weights, calculated as the ratio between the woman's sample size for survey in which the child appears and the population of her country in the year of that survey (population data from World Bank World Development Indicators, 1960–2016). The latter ensures results are representative of the individuals living in the 32 countries in sub-Saharan Africa.

β_1 quantifies the impact of in-utero exposure to growing-season droughts on under-five mortality. As long as the drought is exogenous, the estimate β_1 is unbiased and provides an indication of the causal effect of in-utero exposure to growing-season droughts

[18] Even though the focus of my analysis is on in-utero exposure to growing-season droughts, I control for the effect of in-utero exposure to extremely wet growing-season conditions. In accordance with WMO definition of drought intensity, I use a symmetric variable to control for events where SPEI3 is greater than 2 standard deviations. In my sample, the in-utero exposure to growing-season droughts and the in-utero exposure to extremely wet growing-season conditions are not correlated (i.e., –0.02).

on under-five mortality. Below I first provide a discussion about the several potential channels that may confound this effect and then show that the results are not confounded by these biases.

In order to study whether maternal schooling modifies the effect of in-utero growing-season droughts on under-five mortality, the interaction between the variable $gsdrought\_ei_i$ and the variable for maternal schooling is added to the model in (Eq. (2)) in a second analysis. Due to the cross-sectional design, no causality can be established. Therefore, the coefficient associated with this variable measures whether the in-utero exposure to growing-season droughts is associated with significantly lower or higher mortality for children of less-educated mothers than for children of more-educated mothers.

Concerns about Coefficient Bias

One potential source of bias in my estimate is selection in utero. If a weaker foetus is more likely to die in utero due to a drought, then the stronger foetuses survive until birth and are more likely to survive in childhood. This is likely to underestimate the true effect of in-utero exposure to growing-season droughts on child mortality. I test this selection on foetal strength by using a pregnancy outcome in the survey data. I regress a variable which takes value one if a pregnancy is terminated before birth for any reason on the growing-season droughts indicator, where t in (Eq. (1)) is either the date the pregnancy ended or the date of the child's birth. A significant and positive impact of in-utero exposure to growing-season droughts on having a termination will suggest that foetal loss increases following growing-season droughts and that the estimate of in-utero exposure to growing-season droughts on child mortality is underestimating the true effect.

Another source of bias is migration after conception. Because the DHS / AIS / MIS data contain women's geographic location at survey, I cannot be exactly sure that women

and children are exposed to the weather conditions in the same area (i.e., ERA-I cell) since two years before birth. The direction of the bias is unclear, but if, for example, a woman moves from an area with unfavourable climatic conditions for crop growth to another with more favourable climatic conditions between two years before child's birth and survey time, the estimate might be underestimating the true effect of in-utero exposure to growing-season droughts on child mortality. This is an important check for the validity of results since empirical evidence shows that families tend to migrate as a response to droughts (Pedersen 1995). I check the robustness of my results to migration, using a question on how long the mother has lived in the same cluster in the survey data. Specifically, I exclude all children who have not lived in the same cluster since two years before their birth. This allows to be exactly sure of capturing the correct weather conditions children have been exposed to while in utero.

Results

Table 4.1 presents summary statistics of the variables used in this analysis. On average in my sample, child mortality rate is 0.09 suggesting that 90 children per 1,000 live births die before their fifth birthday and mothers have 4 years of education. Children are evenly distributed by sex, and born in 2005 to mothers aged 27 years. The proportion of children varies by birth order, ranging from 23% at birth order 1 to 47% at birth order 2, and 30% at birth order 3. Seventy-two percent live in rural areas and 81% are born in the hungry season.

In addition, a small proportion of children have in utero exposure to growing-season droughts. In particular, only 2.8% of children were exposed to any growing-season droughts which also suggests that growing-season droughts are rare events. Among exposed children, the average proportion of growing-season months in which there were

any droughts was 0.18 (SD = 0.13), which indicates that children have in utero exposure to droughts for 18% of the months of the last two growing seasons.

Main Model Results and Heterogeneity of the Effect

Table 4.2 shows the results of the model described in section 5.2, employing various samples.[19] The overall effect of in-utero exposure to droughts during the last two growing seasons using the full sample is positive but statistically significant at unconventional levels ($p < .10$) (Column 1). Next, I explore whether there are different effects of in-utero exposure to growing-season droughts based on the place of residence (urban/rural) and find that the effect is concentrated in rural areas. I cannot reject the hypothesis that urban effects are zero (Column 2; linear combination), and find that the difference between estimates for rural and urban areas is statistically significant ($p = .002$). Restricting the sample to children living in rural areas only (Column 3), I find that in-utero exposure to growing-season droughts has a meaningful effect. Specifically, going from no exposure to exposure for 20% of the months of the last two growing seasons causes the deaths of 7 (i.e., $36 * .20$) under-five children per 1,000 live births, an effect that is significant at the 5% level and that corresponds to a 8% increase in child mortality rates given a mean of 90 deaths per 1,000 live births. This effect is particularly outstanding when compared with the effect size of maternal education. A one-year increase in maternal education is associated with a decrease in under-five mortality of 1.5 per 1,000 live births which translates into an effect of –1.6%, that is only 20% of the droughts effect.

[Table 4.2 here]

I now turn to a more detailed analysis of the effect, focusing on one source of heterogeneity. To capture differential effects by type of crop, I exploit the richness of the calendar crop data that allow to distinguish between irrigated and rainfed crops. If the

[19] For simplicity, only coefficients to growing-season droughts and years of education are reported.

drought variable is a measure of how past weather has affected crops, I should find that the effect in areas where crops are mainly dependent on the quantity of rainfall is positive and greater than it is in areas where crops are irrigated. I estimate two models on irrigated and rainfed areas separately. Column 4 shows the estimation for the sample in which crops are irrigated. Because of low irrigation development in sub-Saharan Africa, the sample size reduces considerably. In irrigated areas, in-utero exposure has a negative and statistically insignificant effect on under-five mortality. Focusing the analysis on rainfed areas, instead, I find that increased in-utero exposure significantly increases under-five mortality (Column 5). In these areas, moving from no exposure to exposure for 20% of the months of the last two growing seasons increases under-five mortality by 7 (i.e., $33 * .20$) per 1,000 live births. Given a mean of 90 deaths per 1,000 live births, the effect corresponds to a 7.3% increase in child mortality.

Lack of Access to Nutritious Food during Pregnancy as a Mechanism

I now provide evidence supporting the lack of access to nutritious food among mothers during pregnancy as the main mechanism through which exposure to growing-season droughts increases child mortality (Columns 1–3, Table 4.3). Ideally, the in-utero-malnutrition mechanism should be studied more directly by using data on malnutrition. However, individual data on malnutrition is either scarce or completely lacking. The usual indicator measuring malnutrition in the DHS data is birth weight, but, unfortunately, it has high rates of missingness in 71 surveys (i.e., 65%), while it is completely lacking in the remaining ones. The literature, however, argues that birthweight, and, in general, measures of birth size are a poor proxy for the underlying effects of nutritional shocks on embryonic or foetal development (e.g., Franko et al. 2009; Gluckman and Hnason 2005; Schulz 2010). Therefore, this remains an empirical issue to be investigated with datasets other than DHS or when more accurate DHS data become available. Meanwhile, other studies have used

infant mortality rates to proxy intrauterine nutritional shocks. I use infant mortality as proxy for capturing nutritional shocks in utero and regress it on the same set of variables specified in section 5.2. I find that in-utero exposure to growing-season droughts causes 25 infant deaths per 1,000 live births and the effect is statistically significant (Column 1, Table 4.3, $p < .05$), which is consistent with similar research (Kudamatsu et al. 2012). This finding gives preliminary support of the in-utero-lack-of-access-to-nutritious-food mechanism to explain the positive relationship between in-utero exposure to growing-season droughts and under-five mortality.

Next, I check whether in-utero exposure to growing-season droughts increases child mortality regardless of the period in which the main crop is growing. I look at the weather conditions over the entire two years prior to birth rather than over the last two growing seasons of the main crop. This analysis allows to compare the performance of the growing-season droughts indicator with a simpler measure – the two-year droughts indicator – to provide evidence that mothers' nutritional intake during pregnancy mainly depend on the weather conditions during the two growing seasons prior to childbirth. It further allows to speculate about the mechanism through which the relationship operates. In particular, if droughts are causing crop failure and food shortage, we should find droughts concentrated during the growing season to matter the most, while droughts outside the growing season to at least not be as relevant.[20] The evidence supports this implication. I find that in-utero exposure to droughts has a positive impact on child mortality, but the effect is smaller in magnitude than that of in-utero exposure to growing-

[20] In order to test that droughts within the growing season matter more than droughts outside the growing season, the ideal model would include two separate variables of droughts, one for growing-season droughts and the other for non-growing-season droughts, and then test if coefficients are statistically different from one another. Because in the sample some crops are annual crops (i.e., growing season starts in January and ends in December), it is not possible to create a variable of non-growing season droughts and estimate such model. I could instead create a variable for the two-year droughts as the exposure to droughts over the entire two years prior to birth. Therefore, the only viable strategy is to estimate one model including the two-year droughts indicator and compare its coefficient with the coefficient of the growing-season droughts indicator in Column 5, Table 4.2.

season droughts (30 vs 33 per 1,000 live births)[21] and not statistically significant (Column 2, Table 4.3, p = .26)[22]. This finding indicates that the growing-season droughts indicator captures mothers' nutritional intake during pregnancy better than the two-year droughts indicator. It also indicates that the positive impact of in-utero exposure to droughts on child mortality prevails when they occur during the growing seasons, which in turn suggests that the effect may also operate through the lack of access to nutritious food in utero.

Different In-Utero Exposures

I also explore whether different strengths of growing-season droughts have similar negative consequences on children's survival chances. In particular, I compare the effect of in-utero exposure to moderate and severe growing-season droughts. As under-five mortality is an extreme outcome, we should observe substantial increases in its levels after extreme weather shocks. On the one hand, extreme droughts could potentially have larger effects through households' lack of access to nutritious food if they destroy harvests and increase food prices. On the other hand, extreme droughts could also trigger coping mechanisms – e.g., support from formal and informal safety networks – in the households that aim at protecting pregnant women from the negative impacts related to their health and stress. I create additional continuous variables that indicate the degree of intensity of the shock by constructing variables for –1 to –1.5 SDs from zero, –1.5 to –2 SDs, and –2 and less to define, respectively, moderate, severe, and extreme growing-season droughts. I find that the effect of in-utero exposure to moderate growing-season droughts is negative and statistically insignificant, while that of severe droughts is positive and smaller than that of

[21] Because the model specification in Column 5, Table 4.2, is not the same as that in Column 2, Table 4.3, I cannot statistically test that coefficients are different from one another. Although the difference in coefficients is small, the results still show that the coefficient of droughts is not statistically different from zero (as opposed to the coefficient of growing-season droughts).

[22] Because this model does not include control of season of birth, standard errors are clustered only by ERA-I cell.

extreme droughts, albeit statistically insignificant (Column 3, Table 4.3). Moderate and severe growing-season droughts, indeed, are not important in shaping child mortality.

Finally, it is not clear whether more recent in-utero exposure to growing-season droughts should have larger or smaller effects on under-five mortality than less recent one. This analysis could help disentangle the stage in pregnancy in which droughts have an effect on under-five mortality. In particular, less recent droughts affect maternal nutritional intake around conception and in the first period of pregnancy (i.e., embryogenesis and initial foetal development), as opposed to more recent droughts that influence maternal nutrition during the perinatal period. I use the terms from the right-hand side of (Eq. (1)) to denote, respectively, more and less recent in-utero exposure to growing-season droughts and include them in the analysis as separate variables (symmetrical variables for more and less recent in-utero exposure to extremely wet growing-season conditions are also included in the model). The coefficients presented in Column 4, Table 4.3, indicate that the impact of in-utero exposure to growing-season droughts is positive; however, this positive impact is statistically significant for less recent exposure, whereas the coefficient is also positive, but not statistically significant, in the case of more recent exposure.[23] Thus, less recent exposure to growing-season droughts during pregnancy leads to an increase by 49 under-five deaths per 1,000 live births, suggesting that exposure to growing-season droughts during the initial intrauterine period is more detrimental to child survival.

[Table 4.3 here]

[23] The coefficients for less recent exposure and more recent exposure are not statistically different from one another (i.e., p-value of test of equality of coefficients is 0.29).

Robustness of Primary Result

In this section, I examine whether the primary result – the large effect of growing-season droughts in rural and rainfed areas shown in Table 4.2, Column 5 – is robust to issues of selection or specification. The findings are shown in Table 4.4.[24]

I test whether the result is biased by selection in utero by using data on termination available in the surveys. I find that foetal loss does not significantly increase because of droughts (Column 1). This suggests that weaker foetuses are not being selected out during droughts and that foetal loss is not affecting the result. The estimate might also be biased by migration after conception. Excluding all children who have not lived in the same cluster since two years prior to their birth leads to a reduction in the sample size to 213,163 mainly because this information is not included in all available surveys (only 54 out of 98 surveys, initial sample size is 263,220[25]). Removal of these children from the sample does not alter the sign and significance level of the coefficient (Column 2).

I also show that the result is robust to removing the in-utero exposure to extremely wet growing-season conditions control (Column 3) and the child and maternal controls (Column 4). A further analysis excludes women aged 18 years and younger at child's birth. This is done because the schooling variable refers to the time of survey and might have changed over time. The coefficient remains unchanged (Column 5) and this is plausible since it is very likely that women drop out of school once they get pregnant. Finally, the DHS imputes the full date of the child's birth when the mother is not able to provide it or when dates are inconsistent. Therefore, in an additional analysis I exclude children for which the date of birth is not complete or consistent in order to account for possible measurement error. The result is robust to removing these observations (Column 6).

[Table 4.4 here]

[24] Because the models in Columns 1 and 4 do not include control of season of birth, standard errors are clustered only by ERA-I cell.

[25] In Supplementary Material, I show robustness of the primary result when estimating the model using all surveys with data on migration (see Table 4.A2).

Moderating Effect of Maternal Education

The previous analysis showed that in-utero exposure to growing-season droughts increase under-five mortality. In the next analysis, I explore whether maternal education moderates the effect of in-utero exposure to growing-season droughts, by including an interaction between this variable and the variable for maternal schooling, defined as years of education, in the model estimation. Table 4.5 shows the results of estimation for the rural and rainfed sample.

I find that among children with same in-utero exposure to growing-season droughts, maternal schooling is associated with lower under-five mortality; however, the relationship is not statistically significant at conventional levels ($p = .105$). On the other hand, I reject the hypothesis that the education effect is zero (Column 1; linear combination which tests the hypothesis that in-utero exposure to growing-season droughts has a null marginal effect on under-five mortality at the sample mean for maternal education).

Next, does the effect vary by mother/household characteristics? I focus on two sources of heterogeneity. One is education of the mother: I define less educated a woman with less than 7 years of education; I choose a cut-off of 7 years since there is a substantial increase in the distribution of child mortality below this level of education. The other is occupation and I define a child's household agricultural when both the mother and her partner/husband work in the agricultural sector at the time of the survey. There are two sources of measurement errors that could bias the results in the latter estimation. One is that parents may have changed their occupation since their child's conception, and another lies in the definition of agriculture in the DHS data which also includes forestry and fishery. The moderator role of maternal schooling in the relationship between in-utero exposure to growing-season droughts and under-five mortality seems to be particularly

pronounced among children of less educated women and children living in an agricultural household (Columns 3–4). In particular, I find that among children with the same in-utero exposure to growing-season droughts, a one-year increase in schooling is associated with lower mortality of 14 and 19 children per 1,000 live births, respectively. The estimates respectively correspond to a 15% and 19% decrease in mortality rates of equally-exposed children given a mean of 94 and 99 deaths per 1,000 live births. Again, the hypothesis that the education effect is zero is rejected (Column 3–4; linear combination) as is the hypothesis that the effect of growing-season droughts is the same across years of education ($p = .06$, Column 3; $p = .04$, Column 4).

A further analysis concerns the possibility that the effect of maternal education in this relationship in non-linear and that having at least some education is necessary to offset the positive impact of growing-season droughts. I define a new binary indicator of schooling (1 if the woman has more than five years of education) and interact it with the in-utero exposure to growing-season droughts indicator. The results suggest that among children of high-educated women, in-utero exposure to growing-season droughts has a non-significant effect on under-five mortality (Column 2; linear combination), but reject the hypothesis that the effect of growing-season droughts is the same across different levels of education ($p = .005$). The former finding highlights that the negative consequences of in-utero exposure to growing-season droughts are mainly concentrated among children of low-educated mothers. The latter finding indicates that that among children with the same in-utero exposure to growing-season droughts, high levels of maternal education are associated with lower mortality of 79 children per 1,000 live births.

[Table 4.5 here]

Conclusions

In sub-Saharan Africa, rainfed agriculture provides about 90% of the food and feed (Rosegrant et al. 2002) and diversification in rural income generating activities is lower than in other regions (Davis et al. 2010). Agriculture is the main source of livelihood for more than 70% of the population, while also contributing to about 25% of GDP (Dixon et al. 2001; Hellmuth et al. 2007). During the last decades, the region has experienced an intensification of the frequency, intensity, and geospatial coverage of droughts (Masih et al. 2014). Droughts can cause, among others, crop failure and food shortage and are thus likely to have large impacts on the most vulnerable segments of the rural population, such as pregnant women and children.

This paper aims at identifying the impact of in-utero exposure to growing-season droughts on under-five mortality and exploring if maternal education is protective against drought-related fatality in early childhood. I combine individual-level data from 98 Demographic and Health Surveys from 32 countries in sub-Saharan Africa with local weather and calendar crop data and create a measure of drought shocks during the two growing seasons prior to the child's birth using the child's date of birth and geographic location at survey. I assume that the geographic location at survey coincides with the geographic location during the two years prior to child's birth. By imposing this restriction, I assume that there is not selective migration that confounds the primary result, an assumption that is supported by the data.

Because agricultural droughts should mainly have an effect on rural and agriculture-dependent population, I restrict the sample to rural households. I find that in-utero exposure to growing-season droughts significantly increases under-five mortality and provide evidence suggesting that this effect mainly operates through the lack of access to nutritious food among mothers during pregnancy. Sensitivity analyses show that these results do not seem to be driven by foetal loss or migration. I also find that droughts do not

significantly affect child mortality in irrigated areas which could point to the expansion of irrigation as an important strategy to reduce vulnerability during droughts. It could be not so straightforward, however. The literature suggests that water resources are highly dependent on, and influenced by, climate (Shiferaw et al. 2014) and that expansion of irrigated schemes are accounted responsible for child mortality increases via spread of diseases such as malaria and bilharzia (Farah and Preston 1982).

Next, I explore whether maternal education moderates the positive impact of in-utero exposure to growing-season droughts on child mortality. Given the large expansion of women's education in sub-Saharan Africa, which alone is considered an important determinant of child survival, and the emerging literature suggesting that female education is key to enhanced adaptation to climate change and reduced natural disasters vulnerability, maternal education may lessen the effect of in-utero exposure to weather shocks on child mortality. I find that in-utero exposure to growing-season droughts is associated with significantly higher mortality for children of low-educated mothers than for children of high-educated mothers; this finding is particularly pronounced for less educated and agricultural households. It suggests that high-educated mothers have certain characteristics that can potentially moderate the impact of in-utero exposure to growing-season droughts on child mortality. Because droughts affect the access to nutritious food among mothers during pregnancy, it may be that high-educated mothers have greater access to nutritious food during pregnancy which is protective in times of hardship.

This study is subject to a number of limitations. I exploit a weather shock as an exogenous source of crop failure and food shortage, but there could be alternative paths of influence that are not tested in this paper, such as the changed disease environment and increased maternal stress and energy expenditure. Further research is needed to examine these alternative paths of influence in more detail. In addition, the causal nature of the moderator role of maternal education in the relationship between droughts and child

mortality is far from assessed in this study. I do not exploit exogenous variations in maternal education, but instead it is likely that variations in education are not orthogonal to weather. Therefore some caution is warranted when interpreting these results.

Another limitation of this study is that it ignores the role of paternal education in lessening the positive impact of in-utero exposure to growing-season droughts. Previous literature shows that paternal education is important for the survival chances of children (Breierova and Duflo 2004; Chou et al. 2010), thus it might play a role in the moderating the positive droughts-mortality relationship too. Due to limitations of the DHS / MIS / AIS, which collect data on the woman's partner, who is not necessarily the child's father, I cannot include paternal education in the analysis. Nevertheless, it is important to acknowledge that ignoring the role of paternal education may lead to overestimate the role of maternal education in the droughts-mortality relationship. Future research should examine the potential moderator role of paternal education in more detail using in-depth data.

In spite of these limitations, the findings are relevant in relation to programs enhancing societies' adaptation to climate change. First, they contribute to a body of evidence showing that exposure to adverse shocks in utero affects early childhood mortality. Second, they contribute to an emerging body of research suggesting that education reduces natural disaster vulnerability and enhances adaptive capacity vis-à-vis future climate change. The results of this analysis suggest that maternal education could reduce the susceptibility of children to extreme weather triggered by climate change and that continued expansion of women's education in sub-Saharan Africa can be highly beneficial for child survival.

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Fig. 4.1 Spatial distribution of DHS clusters by type of residence.

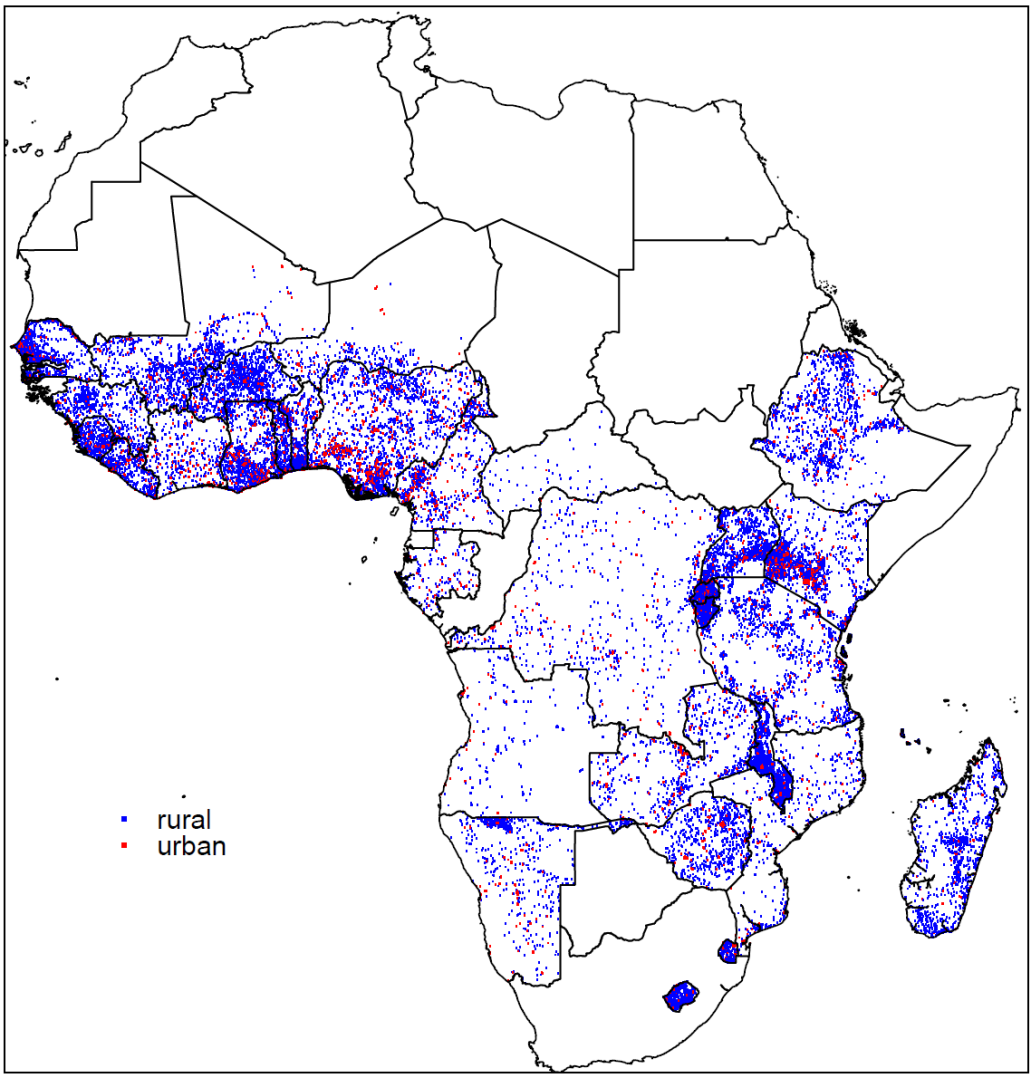

Fig. 4.2 Spatial distribution of main crop across Africa.

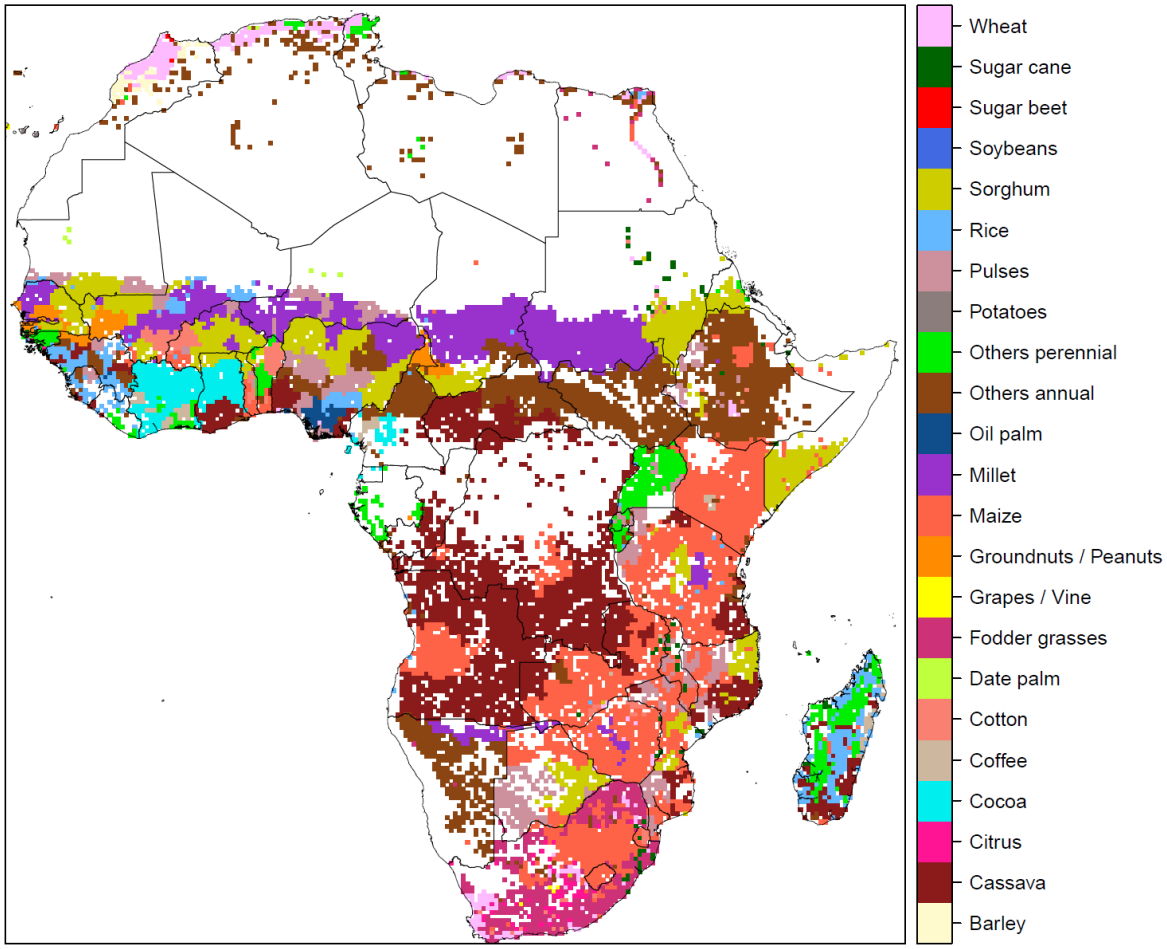

Table 4.1 Sample characteristics.

Variable	Value
Under-five mortality	0.09
	(0.28)
Years of education	3.80
	(4.2)
Mother's age at birth	27.00
	(6.9)
Child's year of birth	2005
	(5.6)
Survey year	2008
	(5.5)
Sex (%)	
Female	0.49
Male	0.51
Type of residence (%)	
Rural	0.72
Urban	0.28
Child's birth order (%)	
1	0.23
2–4	0.47
5+	0.30
Season of birth (%)	
Hungry	0.81
Harvest	0.19
N	714,580

Proportions and means are based on weighted data, standard errors in parenthesis; *N* is unweighted.

Table 4.2 Growing-season droughts on under-five mortality (multiplied by 1,000).

	All	All	Rural	Irrigated	Rural and rainfed
	Model 1	Model 2	Model 3	Model 4	Model 5
Growing-season droughts	22.00^{+}	42.00**	36.00*	-9.00	33.00*
	(12.00)	(15.00)	(16.00)	(37.00)	(16.00)
Growing-season droughts x Urban		-72.00**			
		(24.00)			
Years of education	-1.90***	-1.90***	-1.50***	-1.70**	-1.50***
	(0.19)	(0.19)	(0.22)	(0.53)	(0.23)
Interaction p-value		0.002			
Linear combination		-29.86			
		(18.85)			
Mean of dependent variable	85	85	90	78	90
N	714,580	714,580	526,065	57,008	492,754
R^2	0.03	0.03	0.04	0.04	0.04

$^{+}$p<.1; *p<.05; **p<.01; ***p<.001

Column headers indicate sample employed. Control variables: in-utero exposure to extremely wet growing-season conditions, child's sex, child's birth order, type of residence (not included in Models 3 and 5), child's season of birth, mother's age at birth, mother's age at birth squared, ERA-I cell fixed effects, survey fixed effects, country specific year of birth fixed effects, and a binary variable if using the growing-season period of a close grid cell. Standard errors are clustered by ERA-I cell and season of birth. 'Interaction p-value' is the p-value for growing-season droughts x urban. 'Linear combination' is the sum of coefficients (growing-season droughts) + (growing-season droughts x urban).

Table 4.3 Growing-season droughts on infant mortality and under-five mortality (multiplied by 1,000).

	Infant mortality	Under-five mortality		
	Model 1	Model 2	Model 3	Model 4
Extreme growing-season droughts	25.00*		33.00*	
	(12.00)		(16.00)	
Severe growing-season drought			5.10	
			(8.80)	
Moderate growing-season drought			-4.90	
			(5.70)	
Droughts		30.00		
		(27.00)		
Growing-season droughts (t–1)				20.00
				(19.00)
Growing-season droughts (t–2)				49.00*
				(23.00)
Years of education	-0.85***	-1.50***	-1.50***	-1.50***
	(0.19)	(0.25)	(0.23)	(0.23)
Mean of dependent variable	67	90	90	90
N	492,754	492,754	492,754	492,754
R^2	0.02	0.04	0.04	0.04

+p<.1; *p<.05; **p<.01; ***p<.001

Rural and rainfed sample. Column headers indicate dependent variable. Control variables: in-utero exposure to extremely wet growing-season conditions (not included in Model 3 and included as more and less recent exposure in Model 4), child's sex, child's birth order, child's season of birth (not included in Model 2), mother's age at birth, mother's age at birth squared, ERA-I cell fixed effects, survey fixed effects, country specific year of birth fixed effects, and a binary variable if using the growing-season period of a close grid cell (not included in Model 2). Standard errors are clustered by ERA-I cell and season of birth (Models 1 and 3–4) and by ERA-I cell (Model 2).

Table 4.4 Growing-season droughts on under-five mortality and termination (multiplied by 1,000) – Robustness checks.

	Termination	Under-five mortality				
	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Growing-season droughts	4.20	49.00*	33.00*	35.00*	38.00*	32.00*
	(18.00)	(22.00)	(16.00)	(16.00)	(17.00)	(15.00)
Years of education	-0.21	-1.30***	-1.50***		-1.40***	-1.30***
	(0.16)	(0.39)	(0.23)		(0.24)	(0.23)
Mean of dependent variable	49	100	90	90	87	86
N	518,610	213,163	492,754	492,754	457,543	479,232
R^2	0.43	0.04	0.04	0.03	0.03	0.03

+p<.1; *p<.05; **p<.01; ***p<.001

Rural and rainfed sample. Column headers indicate dependent variable. In models 2, 5, and 6, the sample is restricted to children living in the same cluster since two years prior to birth, children of old mothers, and children with complete date of birth, respectively. Control variables: in-utero exposure to extremely wet growing-season conditions (not included in Model 3), child's sex (not included in Models 1 and 4), child's birth order (not included in Models 1 and 4), child's season of birth (not included in Models 1 and 4), mother's age at birth (not included in Model 4), mother's age at birth squared (not included in Model 4), ERA-I cell fixed effects, survey fixed effects, country specific year of birth fixed effects, and a binary variable if using the growing-season period of a close grid cell. Standard errors are clustered by ERA-I cell (Models 1 and 4) and by ERA-I cell and season of birth (Models 2–3 and 5–6).

Table 4.5 Moderating effect of maternal education in the growing-season droughts – under-five mortality relationship (multiplied by 1,000).

	All Model 1	All Model 2	Less educated Model 3	Agricultural Model 4
Growing-season droughts	48.00**	52.00**	49.00**	66.00*
	(19.00)	(18.00)	(19.00)	(29.00)
Growing-season droughts x Years of education	-7.40		-14.00+	-19.00*
	(4.50)		(7.30)	(9.30)
Growing-season droughts x High education		-79.00**		
		(28.00)		
Years of education	-1.40***		-1.30***	-0.92+
	(0.23)		(0.36)	(0.49)
High education		-7.60***		
		(1.70)		
Interaction p-value	0.105	0.005	0.056	0.045
Linear combination	40.93*	-27.02	35.28*	47.35+
	(16.73)	(24.44)	(17.09)	(27.48)
Mean of dependent variable	90	90	94	99
N	492,754	492,754	437,639	145,544
R^2	0.04	0.04	0.04	0.05

+p<.1; *p<.05; **p<.01; ***p<.001

Column headers indicate sample employed. Control variables: in-utero exposure to extremely wet growing-season conditions, child's sex, child's birth order, child's season of birth, mother's age at birth, mother's age at birth squared, ERA-I cell fixed effects, survey fixed effects, country specific year of birth fixed effects, and a binary variable if using the growing-season period of a close grid cell. Standard errors are clustered by ERA-I cell and season of birth. 'Interaction p-value' is the p-value for growing-season droughts x variable for education. 'Linear combination' is the sum of coefficients (growing-season droughts) + (growing-season droughts x education).

Appendices

Information on DHS data collection and list of surveys included

The Demographic and Health Survey project uses a two-stage stratified design, that is in a first stage, a number of clusters are selected with a probability proportional to the size from a frame list of enumeration areas created from the most recent population census, and in a second stage, a fixed number of households are selected. All women of reproductive age (in most cases women between 15 and 49 years old) in the selected households are interviewed and a complete birth and death history of their children is collected, including birth date and, when applicable, death age. Clusters are geo-referenced, that is their coordinates are collected during the survey sample listing process using GPS receivers.

The surveys included in this analysis are the following: Angola 2006–2007, Angola 2011, Benin 1996, Benin 2001, Benin 2011–2012, Burkina Faso 1993, Burkina Faso 1998–1999, Burkina Faso 2003, Burkina Faso 2010, Burkina Faso 2014, Burundi 2010, Burundi 2012, Cameroon 1991, Cameroon 2004, Cameroon 2011, Central African Republic 1994–1995, Comoros 2012, Congo Democratic Republic 2007, Congo Democratic Republic 2013–2014, Cote d'Ivoire 1994, Cote d'Ivoire 1998–1999, Cote d'Ivoire 2011–2012, Eswatini 2006–2007, Ethiopia 2000, Ethiopia 2005, Ethiopia 2011, Gabon 2012, Ghana 1993, Ghana 1998, Ghana 2003, Ghana 2008, Ghana 2014, Guinea 1999, Guinea 2005, Guinea 2012, Kenya 2003, Kenya 2008–2009, Kenya 2014, Kenya 2015, Lesotho 2004, Lesotho 2009, Lesotho 2014, Liberia 2007, Liberia 2009, Liberia 2011, Liberia 2013, Madagascar 1997, Madagascar 2008–2009, Madagascar 2011, Madagascar 2013, Malawi 2000, Malawi 2004, Malawi 2010, Malawi 2012, Malawi 2014, Mali 1995–1996, Mali 2001, Mali 2006, Mali 2012–2013, Mozambique 2011, Namibia 2000, Namibia 2006–2007, Namibia 2013, Niger 1992, Niger 1998, Nigeria 1990, Nigeria 2003, Nigeria 2008, Nigeria 2010, Nigeria 2013, Rwanda 2005, Rwanda 2007–2008, Rwanda 2010, Rwanda 2014–2015, Senegal 1992–1993, Senegal 1997, Senegal 2005,

Senegal 2008–2009, Senegal 2010–2011, Senegal 2012–2013, Sierra Leone 2008, Sierra Leone 2013, Tanzania 1999, Tanzania 2007–2008, Tanzania 2010, Tanzania 2011–2012, Togo 1998, Togo 2013–2014, Uganda 2000–2001, Uganda 2006, Uganda 2009, Uganda 2011, Uganda 2014–2015, Zambia 2007, Zambia 2013–2014, Zimbabwe 1999, Zimbabwe 2005–2006, Zimbabwe 2010–2011.

Information on climate data

The advantages of the ERA-Interim data are that: i) they are re-analysed using various sources such as satellites, radiosondes, pilot balloons, aircraft, wind profilers, ships, drifting buoys, and land stations; ii) they have less bias in rainfall outcomes in the arid and semi-arid areas of Africa compared to gauge data (Zhang et al. 2013).

The Standardized Precipitation Evapotranspiration Index

The SPEI is calculated following Vicente-Serrano et al. (2010).[26] The computation involves the following steps.

The PET is modelled in a first instance using the FAO-56 Penman-Monteith equation described in Allen et al. (1998, Eq. 6), considered to be a superior method to determine reference evapotranspiration:

$$ET_0 = \frac{0.480\Delta(R_n - G) + \gamma \frac{900}{T+273} u_2 (e_s - e_a)}{\Delta + \gamma(1 + 0.34u_2)}, \quad \text{(A1)}$$

where

ET_0 reference evapotranspiration [mm day^{-1}],

R_n net radiation at the crop surface [MJ m^{-2} day^{-1}],

G soil heat flux density [MJ m^{-2} day^{-1}],

T air temperature at 2 m height [°C],

u_2 wind speed at 2 m height [m s^{-1}],

e_s saturation vapour pressure [kPa],

e_a actual vapour pressure [kPa],

$e_s - e_a$ saturation vapour pressure deficit [kPa],

Δ slope vapour pressure curve [kPa °C^{-1}],

γ psychrometric constant [kPa °C^{-1}].

Given the paucity of some weather data, Allen et al. (1998) provide procedures for estimating missing climatic data. The (average) daily net radiation (MJ m^{-2} day^{-1}) can be derived from the (average) daily incoming solar radiation (MJ m^{-2} day^{-1}). The (average) daily saturation vapour pressure (kPa) can be calculated using the (average) daily maximum and minimum air temperature (°C), while the (average) daily dewpoint temperature (°C) can be used to derive the (average) daily actual vapour pressure (kPa). The atmospheric pressure required to calculate the psychrometric constant (kPa °C^{-1}) can be derived from the elevation above sea level (m). In order to calculate the monthly ET_0 with the FAO-56 Penman-Monteith equation, I use the following parameters: monthly mean daily net solar radiation (MJ m^{-2} day^{-1}), daily maximum and minimum air temperature (°C), monthly mean daily wind speeds at 2 m height (m s^{-1})[27], monthly mean daily dewpoint temperature (°C), elevation above sea level (m), and latitude (degrees).

After calculating PET, a measure of the water surplus or deficit as the difference between the precipitation P and PET for the month i is calculated as:

$$D_i = P_i - PET_i, \qquad \text{(A2)}$$

The calculated D_i values are aggregated at a time scale of 3.

[26] The SPEI-R package developed by Beguería and Vicente-Serrano (2013) is used to derive the PET and SPEI.

The D series are then standardised using a log-logistic distribution to obtain the SPEI series. The probability density function of a three-parameter log-logistic distributed variable is expressed as:

$$f(x) = \frac{\beta}{\alpha}\left(\frac{x-\gamma}{\alpha}\right)^{\beta-1}\left[1+\left(\frac{x-\gamma}{\alpha}\right)^{\beta}\right]^{-2}, \qquad \text{(A3)}$$

where α, β, and γ are scale, shape, and origin parameters, respectively, for D values in the range ($\gamma > D < \infty$).

Validation of the drought shocks measure

I validate the measure of drought shocks, as measured by SPEI3, by showing that it is negatively associated with GDP growth and yield of main crop. For this analysis, multiple datasets are combined and used to define a cell-year-level measure of drought shocks. Data on crops are from the MIRCA2000 dataset. Using data on the same crop across countries or at the country level would not allow to correctly determine the impact of droughts on the true cultivation in the area: as seen in Fig. 4.2, the pattern of cultivation varies substantially across the African continent. Data on GDP are from the Penn World Table (version 9.0), while data on the yield of crops are from the FAOSTAT database. I use data for 1979–2014 across 32 sub-Saharan African countries.

For each year and cell, I construct the measure of drought shocks by averaging the proportions of months in which SPEI3 is below –2 (i.e., extreme droughts) during the reference calendar year and the previous calendar year (a symmetric variable for extremely wet conditions is constructed and included in the model).[28] Then, I estimate two linear

[27] The original wind speed variable is measured at 10 m above the ground level. In order to adjust wind speed to the standard height of 2 m needed for this estimation, I use of the following calculation procedure: $u_2 = u_{10}\frac{4.87}{\ln(67.8*10-5.42)}$ (Allen et al. 1998, Eq. 47).

[28] Because in some grid cells the growing-season period starts in one year and ends in the next year, I cannot compute a measure of growing-season droughts for each year. Despite this limitation, this analysis is still useful to give an indication of how SPEI3 affects economic performance and agricultural outcomes.

regression models to analyse the impact of droughts on (log) GDP and (log) yield. ERA-I cell fixed effects, country fixed effects, and year fixed effects are included as control variables. Data are then weighted by yearly country population based on World Bank data (GDP regression) and by crop-specific harvested area (yield regression), and the standard errors are clustered by ERA-I cell.

[Table 4.A1 here]

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Table 4.A1 Effect of extreme droughts on economic and agricultural productivity.

	Log GDP per capita Model 1	Log yield Model 2
Extreme droughts	-0.26***	-0.23***
	(0.05)	(0.07)
Extremely wet conditions	0.13***	-0.01
	(0.03)	(0.04)
N	4,410,770	4,366,316
R^2	0.92	0.77

+p<.1; *p<.05; **p<.01; ***p<.001

Regressions include ERA-I cell specific fixed effects, country specific fixed effects, and year specific fixed effects, and are weighted by country population (GDP regression) and crop-specific harvested area (yield regression). Standard errors are clustered by ERA-I cell.

Table 4.A2 Growing-season droughts on under-five mortality (multiplied by 1,000) – Surveys including information on migration.

	All
Growing-season droughts	56.00**
	(20.00)
Years of education	-1.80***
	(0.33)
Mean of dependent variable	105
N	263,220
R^2	0.04

+p<.1; *p<.05; **p<.01; ***p<.001

Rural and rainfed sample. Control variables: in-utero exposure to extremely wet growing-season conditions, child's sex, child's birth order, child's season of birth, mother's age at birth, mother's age at birth squared, ERA-I cell fixed effects, survey fixed effects, country specific year of birth fixed effects, and a binary variable if using the growing-season period of a close grid cell. Standard errors are clustered by ERA-I cell and season of birth.

Chapter 5. Conclusions

This thesis contributed to the ongoing debate on the role of female education in triggering social and demographic change and reducing vulnerability resulting from climate change. Three essays were developed as stand-alone contributions to the literature; however, each contributes on its own to address the two research gaps (i.e., causality and externalities of education) identified in the introduction of this thesis.

With respect to the first research gap, the first and second essays used two different methodologies to overcome the limitations of cross-sectional data in establishing the causal impact of education on socio-demographic outcomes. Using a quasi-experimental design, the first essay found evidence of a causal link of maternal education to child mortality in Malawi and Uganda and showed that the mechanisms behind this effect differ across countries. It further demonstrated that education was not endogenous in this relationship, and that the conventional estimator was consistent. In other words, the instrument was not necessary and a correlational analyses would have produced a consistent estimate of the relationship between maternal education and child mortality. This does not mean that quasi-experimental designs are not necessary in general. Instead, quasi-experimental designs are useful to confirm (or not) that causal links exist. The second essay, instead, applied spatial interpolation methods to georeferenced cross-sectional surveys from 28 sub-Saharan African countries to *create* panel data on women's education and status—the latter measured by male dominance in family decision-making. These data were in turn used to create high-resolution maps that illuminate the spatial patterns of women's education and status. Most importantly, these data were used in a panel design to show that women's education is an important driver of increases in women's status over time.

With respect to the second research gap, the second and third essays tested for two different types of education externalities. The second essay used spatial models to look at the spillover indirect effects of women's education on women's status and found evidence

of their existence. In particular, it showed that women's status in a given area decreases as women's education in all neighbouring areas increases. In addition to this, evidence of spillover indirect effects of urbanisation on women's status was found too. The third essay, instead, found evidence of the role of maternal education in mitigating children's vulnerability to in-utero exposure to droughts across SSA. This was done in two steps. First, it was shown that in-utero exposure to droughts causes child deaths. Second, it was shown that in-utero exposure to droughts is associated with significantly higher mortality for children of less-educated mothers than for children of more-educated mothers.

Despite the contributions of this thesis to the literature on the role of the effects of education on social and demographic outcomes, further investigation is needed. There might be at least four main potential avenues for future research on this topic.

First, while the focus of the first essay is on the impacts of expanded female primary education, the impacts of expanded secondary education could be also investigated. To the best of my knowledge, only one study from one low- and middle-income country explores the causal effect of expanded secondary education on child mortality (Grépin and Bharadwaj 2015) and the estimate is very similar to the one generated by an expansion of primary education. However, this does not represent enough evidence on which level of education is more important in reducing child mortality and a better understanding in settings with high rates of child mortality is also relevant from a policy perspective. The same reasoning may be applied to the other two essays, where educated women were defined as those who have at least some years of education. While in the second essay this was done because the focus was on school attendance—as opposed to attainment—, the third essay would have missed most of the population if higher levels of education had been chosen. Given that overall the levels of education have increased in many low- and middle-income countries, and that many national governments have started to implement free universal secondary education policies, additional research aimed at

assessing the causal effects of secondary education now becomes more viable. The new policies may provide an important opportunity to further extend the literature on this topic.

Second, the literature on the causal impacts of education is limited to just few socio-demographic outcomes. On the one hand, a growing number of quasi-experimental studies on the relationship between education and fertility-related behaviours and intentions among adolescent girls provide causal evidence of this link (Behrman 2015; Breierova and Duflo 2004; Ferré 2009; Grant 2015; Keats 2016; Osili and Long 2008; Zanin et al. 2015). On the other hand, virtually no studies identify a causal effect of education on other socio-demographic outcomes such as for example women's experience of physical or emotional violence from an intimate partner or sex differences in child mortality. Given the importance of improving gender equality globally, a better understanding of the role of education in improving young women's and daughters' status and health in low- and middle-income countries is crucial from a policy perspective. Virtually all available studies exploring the relationship between female education and these outcomes are correlational. Although the first essay suggested that education is not endogenous in its relationship with child mortality and that a simple association is consistent, there is no certainty that this holds when analysing other outcomes. The only way to confirm this is to analyse the effects of education on these outcomes using a causal inference approach. As mentioned earlier, this thesis showed that when longitudinal data are not available in nature, there are at least two ways to go: 1) using quasi-experimental cross-sectional designs; 2) applying spatial interpolation methods to georeferenced cross-sectional surveys in order to create aggregate data at different points in time and applying a panel design.

Third, because the effects of education on demographic outcomes can operate through many channels, and because quasi-experimental estimates will capture the combined effect of these without explaining how this works, it is important—where

possible—to investigate the effects of education on each mechanism. One advantage of the analysis used in the first essay is that it assessed whether there are impacts of education on mortality by examining the effects of education on a set of mechanisms, one at a time. Another advantage is that by comparing two countries, it allowed for exploration of whether there was something universal about mass schooling that affected child mortality, or whether effects were context specific. This analysis can be, and needs to be, applied to the other social and demographic outcomes, such as for example women's experience of physical or emotional violence from an intimate partner or sex differences in child mortality.

Fourth, because of its attention to the role of space in explaining differences in population processes, demography is considered a spatial social science (Voss 2007). The increasing availability of geocoded data provides a great opportunity for demographers to emphasise the role of space to examine demographic processes. For example, in addition to reporting the geographic location of the sampled community where individuals live, micro data from low- and middle-income countries, such as the DHS, now also provide detailed geospatial information specific to each community. The latter include, but are not limited to, annual precipitation and temperature, population count, and proximity to national borders and water, and can be used to analyse their effects on a variety of socio-demographic outcomes. When these are not sufficiently detailed or additional covariates are needed, other geospatial covariates such as for example crop areas and yields, or urbanisation levels gathered from other sources can be combined with micro data based on individuals' geographic location. As an example, the second essay showed how information on nighttime light from satellites can be merged with data on women's status and used to demonstrate that urbanisation can help explain declining reports of male dominance in decision-making. The third essay, instead, showed how a cutting-edge measure of weather shocks can be combined with DHS data on child mortality to better

understand how weather shocks affect child mortality. In the coming decades, geocoded data are expected to become more accurate, more frequent, and more inexpensive to collect. This is the case of climate data, since they are already publicly available, have a long temporal range (usually from 1900 onwards), are released almost instantaneously, and are often re-analysed using various sources such as satellites, radiosondes, pilot balloons, etc. All this presents a promising avenue for future explorations of how environmental factors may affect social and demographic processes by policy makers and researchers alike. This looks particularly interesting to investigate in sub-Saharan African countries, as well as in other low- and middle-income countries, for two main reasons. First, given the enormous within country ethnic and social heterogeneity of SSA, it is likely that local factors, such as environmental conditions, can help explore the spatial and temporal variation in social and demographic outcomes more than country factors can. In this sense, the second essay highlighted the importance of a spatial approach that takes into account the within country spatial heterogeneity. Second, because weather shocks will increase in coming decades as a consequence of climate change, there is a crucial need for a better understanding of the relationship between climatic processes and the social and demographic outcomes, lives, and livelihoods of people in low- and middle-income countries. For example, research on the effects that weather shocks, such as hot temperatures, might have on gender-based violence as well as on family decision-making is both important and lacking in the literature. These are two main indicators of gender equality and have huge implications for women's and children's health and wellbeing, as well as that of other members in the family. Additional research on the effects of in-utero exposure to weather shocks on other health outcomes, such as birthweight, and the period in which these effects are concentrated is also needed to identify the most vulnerable gestational period. The new rounds of the DHS data now include detailed information on

child's date of birth—day, month, and year as opposed to month and year only—and would therefore allow a better identification of these effects.

To sum up, this thesis demonstrated the importance of the expansion of access to education for women and girls in shaping social and demographic processes across SSA. First, by showing that female education reduces child mortality and increases women's status, it provided solid empirical evidence that the socio-demographic promise of expanded female education is being fulfilled. The analyses also showed that the positive impacts of women's education may also extend to the aspect of vulnerability reduction during pregnancy and therefore have protective effects against drought-related fatality in early childhood.

These findings are relevant in relation to the policy agenda on sustainable development in low- and middle-income countries. The role of education in women's lives is a pivotal point of much discussion in the policy and research literature interested in low- and middle-income countries. Realising its importance in the achievements of the Sustainable Development Goals, education has been made a stand-alone goal, and education-related targets feature under other goals. In addition, recent literature has identified education as the most effective long-term defence against the risks of climate change. Therefore, it is possible that the expansion of education for all, and particularly for women and girls, will continue. From these results, it seems evident that public investments in education for women and girls have been producing some of the desired outcomes.

The question remains as to whether expanded female education will continue to hold the socio-demographic promise. On the one hand, other factors will contribute to triggering social and demographic change and mitigating the negative consequences of climate change. On the other hand, this thesis supported the role of women's education in promoting sustainable development. The continued expansion of female education over the

last decades could have important implications for triggering social and demographic change as well as reducing vulnerability vis-à-vis future climate change. This could be true in sub-Saharan African countries as well as in other low- and middle-income countries that have largely expanded female education.

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