

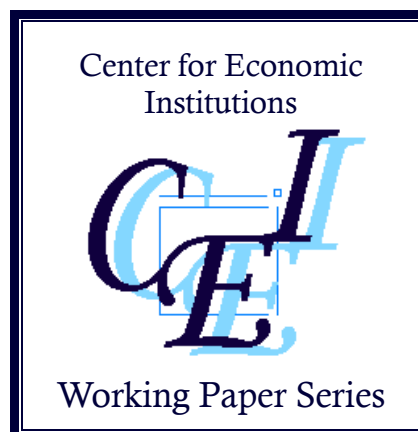
Center for Economic Institutions
Working Paper Series

No. 2018-22

**"Length of maternal schooling and child's risk of malaria
infection in Uganda: evidence from a natural experiment"**

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March, 2019



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Length of maternal schooling and child's risk of malaria infection in Uganda: evidence from a natural experiment

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Abstract

Background: An estimated 216 million cases of malaria occurred worldwide every year. Cross sectional studies have reported negative association between maternal education and child malaria risks, but no randomized trial has confirmed a causal relationship between these two factors. I utilized the free primary education reform in Uganda to assess the causal effects of maternal schooling on the child's risk of malaria infection.

Methods: Malaria biomarkers of children aged < five years were collected from the 2009 and 2014 Uganda Malaria Indicator Surveys (N = 5,316). In 1997, the government eliminated tuition requirement in primary schools and this increased the educational attainment of the affected cohorts. Using exposure to the reform as an instrumental variable, I used a two-stage least squares approach to estimate the causal effects of maternal year of education on the probability that a child would contract malaria at the time of the survey. I also evaluated the cost-effectiveness of primary schooling as a malaria control intervention.

Findings: One extra year of maternal education reduced child's risk of malaria infection by 7.5 percentage points ($p=0.057$), from a baseline 34.6% among the children of 1980 pre-reform cohort. The length of maternal education was also positively associated with insecticide-treated bednets usage. The results were robust to a variety of sensitivity tests. Primary schooling for women was a cost-effective intervention to reduce child's malaria infection.

Interpretation: Improving access to primary education could be a cost-effective measure to reduce malaria prevalence among children aged < five years in malaria-endemic countries.

Funding: None.

Introduction

Malaria remains a major global health issue worldwide, with an estimated 216 million cases in 2016.¹ While existing cross-sectional studies suggest that formal education for mothers could reduce children's risk of a malaria infection, little is known about the causal relationship between these two factors.²⁻⁴

Mothers' education may reduce the incidence of malaria infection among their children for several reasons: 1) improved ability to gather and process information on the transmission mechanism and vector control strategies,⁴⁻¹¹ 2) access to more financial resources through one's own labor force participation,^{9,10} 3) having a partner with a high socio-economic status,^{9,10} and 4) having more decision-making power within one's marriage and household.^{9,10} These changes may result in better use of vector control strategies, including insecticide-treated bednets (ITNs),^{6,12,13} indoor residual spraying (IRS), and anti-malaria drug treatments.^{4,6,7,14} In addition to the implications concerning controlling childhood malaria, this study contributes to the debate on the causal effects of maternal education on the health of children in general.¹⁵⁻¹⁷

Identifying the causal effects of maternal education on malaria incidence among their children is empirically not straightforward because mothers' educational attainment is likely to be correlated with various confounding factors, (i.e., household socio-economic status, risk preference, and innate ability), which can be difficult to control for in observational studies. For this reason, bivariate or multivariate regression analyses may produce a spurious association between maternal education and childhood malaria, and causal relationships cannot be inferred.² To my knowledge, no randomized trial has ever confirmed whether, or to what extent, length of maternal formal education reduces the incidence of malaria infection among their offspring.

To estimate the causal effects of maternal education on childhood malaria control, this study exploited across cohort variation in women's educational attainment generated by exposure to the 1997 free primary education reform in Uganda. In January 1997, the government abolished tuition and Parent-Teacher Association fees in public primary schools. Consequently, the educational attainment of the affected cohort increased¹⁸. Specific cohorts, born in 1982 or later, have been disproportionately affected, and their child's risk of malaria infection has been affected only through the change in the length of maternal formal education. Thus, this reform -- as a natural experiment -- provides an ideal setting to study the effects of maternal education on childhood malaria control. To uncover the potential channel through which maternal education affects children's risk of malaria infection, I estimated its effects on ownership and use of ITNs. I also evaluated the cost-effectiveness of primary schooling as a malaria control intervention.

Methods

Study population and data source

Uganda is the one of the most severe malaria endemic country, where malaria incidence rate was 272 per 1,000 people in 2016.¹⁹ *Plasmodium falciparum* is the predominant *Plasmodium* species found in Uganda.^{20, 21}

The data used in this study came from the 2009 and 2014 Uganda Malaria Indicator Surveys (UMIS), which were nationally representative, repeated, cross-sectional, household surveys that included malaria biomarker collection for children younger than five years of age.^{22,23} In the 2009 and 2014 surveys, 4,760 and 5,802 households were interviewed, and the response rates were 96% and 97%, respectively. All girls/women aged 15-59 years were eligible for the individual interview. In addition, I obtained data from the UMIS concerning testing for malaria in children aged 0-59 months using blood obtained by finger-prick. Blood slides were read in the laboratory for the presence of *Plasmodium* parasites and to determine the parasite species. Verbal, informed consent for each test was granted from the children's parents. All children aged 0-59 months in the households selected for UMIS were eligible for biomarker collection, and 97% of eligible children were tested for malaria with microscopy in both years.^{20, 21} Key information on birth year, age, and mothers' educational attainment were available for 99% of the respondents.

I excluded children of mothers born in 1973 or earlier and those born in 1992 or later because those women were likely to be exposed to other education reform.¹⁰ Children of mothers who were aged < 18 years were also excluded because they may still have been receiving formal education at the time of the survey. I also excluded women with no children aged < five years because my primary interests were the effects of maternal education on childhood malaria control.

Procedure and statistical analyses

Primary exposure was defined as mothers' educational attainment (in years) at the time of the survey. The primary outcome was their children's malaria status at the time of the survey. I defined malaria status based on the results of the microscopy testing to determine the presence of *Plasmodium* infection. I define binary variable which takes value of unity if the respondent woman born in a cohort exposed to 1997 reform, otherwise zero. Considering the share of individuals attending primary school

in 1996 by age, those born in 1982 or later (i.e., ≤ 14 -years-old as of January 1st 1996) were defined as exposed.¹⁰ In the analysis, I controlled for maternal year of birth, maternal ethnicity, children's age in months, and year of the survey.

For comparison, I first evaluated the association between maternal education and child's malaria status using a multivariate ordinary least squares (OLS) regression analysis; Next, I present results from the two-stage least squares (2SLS) instrumental variable (IV) models.²² This study reports the analysis in three steps. First, I tested whether women from the exposed cohorts disproportionately attained more education compared with those who were not exposed (first stage). I estimated the effects of the reform on mothers' educational attainment using an OLS regression model (linear probability model). I also graphically present whether the reform increased the probability that women completed grades 1–7 of primary education or greater. Second, I evaluated intention-to-treat (ITT; or reduced form) effects of the reform on child's malaria status. A quasi-experimental design (natural experiment), under the assumption that cohorts near the exposure cut-off are plausibly similar, allows me to study whether the probability that a child contracts malaria at the time of the survey declined among the exposed cohort compared to the unexposed cohort. Third, I present the results from the 2SLS estimates using dichotomous variables for the 1997 reform exposure as an (IV) to show how an additional year of maternal education affects children's malaria risks. Under several assumptions, the estimated effect size was interpreted as local average treatment effects (LATE) and described as the ratio of the ITT effects to the difference in the maternal educational attainment caused by the reform (IV estimate = ITT estimate/first stage estimate). LATE describes the average treatment effects on a subset of the population—so-called compliers—who attend school longer if they are exposed to the reform, but do not attend in the absence of the reform²³.

To isolate the treatment effects of maternal education on children's risk of malaria infection from the confounding factors, I first controlled for the full set of children's age in months indicators to explain non-linear relationships between the child's age and malaria risk. Second, I included the indicators for mothers' ethnicity to account for biological diversity in the multivariate regression analyses. Third, I included the indicators for survey year. Lastly, the regression model included mothers' year of birth in a linear form to adjust for gradual and continuous trends in maternal educational attainment (in the first-stage model), and in children's risk of malaria infection (in the ITT and IV models). Reform exposure effects are described as the change in the intercept for the cohort exposed to the reform (i.e., born ≥ 1982) compared with those who were not exposed (i.e., born ≤ 1981). When I explored the behavioral mechanism through which maternal education reduces the child's risk of malaria infection, the outcome variable was replaced by a binary indicator for using ITNs last night, and sleeping under the bed net last night. A separate model was estimated for each outcome.

To interpret the estimated effects as causal effects for the compliant population, four identification assumptions must be satisfied (Appendix p. 6). First, the IV (Z) must explain sufficient variation in individual educational attainment (S). This condition is empirically testable, and the results suggests that IV has large, positive, and significant effects on maternal formal education. Second, IV (Z) must be exogenous to unobserved confounding factors (U), such as household income, psychological traits, and innate ability, conditional on the observed covariates (X). This implies that a cohort born *just* before 1983 and those born *just* after 1983 are plausibly similar in the absence of the program after controlling for a cohort linear trend, survey year and ethnicity. A combination of the two survey rounds allowed me to control for survey year periodic effects on women's educational attainment (in the first-stage model) and child's risk of malaria infection (in the ITT and IV models). The model implicitly controls for maternal age effects by including the full set of the survey indicators and linear birth cohort trend.

To test the robustness of the results to the presence of non-linear cohort trends, I also included quadratic terms for maternal year of birth, allowed the slope of the trend to differ between exposed and unexposed cohorts, and limited the window of the observation to the narrower set of birth cohort. A key condition for my identification strategy was that no other interventions or reform would have affected the children's risk of malaria infection among mothers who were exposed to the 1997 reform. To test if the child's risk of malaria infection changed even in the absence of the reform, a placebo test was conducted—I assessed the effects of reform exposure on the children of mothers with no education who were unlikely to be affected by the reform. I also exploit the across-district variation in the primary school completion rates among the unexposed women to estimate the treatment effects using the difference-in-differences method. The idea behind this strategy is that the reform should have had larger impacts in the district where more women failed to complete seven years of primary education in the absence of the reform than in the district where most women completed it in the absence of the reform.⁹

Third, to obtain a consistent estimate of the causal effects of maternal education on the child's risk of malaria infection, I assumed that the IV (Z) affects the primary outcome (Y) only through its effects on maternal year of education (S). This so-called exclusion restriction is plausible because the timing and content of the reform were exogenously designed and implemented by the supply side (i.e., government), and it should not have resulted in cohort-specific effects other than expanding primary school access. Fourth, to interpret the estimated treatment effects as LATE for complier, monotonicity

assumption must be maintained. Namely, it must be maintained that the reform either increased (complier) or had no effects (never-taker or always taker) on women’s educational attainment, and no one in the exposed cohort reduced the length of schooling because of exposure to the reform (i.e., defier).²³ The presence of a defier is theoretically possible; however, it is practically very unlikely given that the reform vastly reduced the price of receiving primary education. To evaluate the cost-effectiveness of the primary schooling to prevent the child’s malaria infection, I calculated the cost of providing an extra year of primary education in Uganda to estimate the cost per malaria incidence averted and per disability-adjusted life-year (DALY) averted.

The present study was reviewed by the Hitotsubashi University Institutional Review Board and considered exempt from full ethics review because the analysis was based on a publicly available, anonymized dataset.

Role of funding source

This study received no funding.

| | MIS (2009) (n=2501) | MIS (2014) (n=2815) |
|-----------------------------------|------------------------|------------------------|
| Child malaria positive | 1002 (40.1%) | 528 (18.8%) |
| Child age (months) | 27.5 (16.9) | 30.1 (17.0) |
| Mother's age (years) | 26.2 (4.6) | 29.8 (4.8) |
| Mother's year of schooling | 4.8 (3.6) | 4.8 (4.1) |
| Has at least 7 years of schooling | 759 (30.3%) | 891 (31.7%) |
| Child contracts moderate anemia | 1064 (42.6%) | 799 (28.4%) |
| Owens mosquito net last night | 1689 (67.5%) | 2725 (96.8%) |
| All <5y children slept under net | 922 (37.0%) | 2076 (74.0%) |

Note: mother at least 18 years of at the time of the survey, born between 1974 and 1991, with valid test for her child’s malaria test. Data shows n (%) or mean (SD).

Table 1: Participants characteristics

Results

Malaria biomarker for children aged 0-59 months are available for 5316 children (Figure 1, Table 1). Unadjusted association between the length of maternal formal education and child malaria infection are almost monotonically negative (Figure 2).

Figure 1: Study participant flow

Shows the flow of participants through the Uganda Malaria Indicator Surveys in 2009 and 2014

Figure 2: Probability that children contracted malaria at the time of the survey by maternal education

The sample includes children aged 0–59 months at the time of the survey and their mothers, born between 1974 and 1991, and who had valid results for microscopy malaria testing.

This negative association between maternal education and the children’s risk of malaria infection remained strong even in the estimates using a naïve multivariate linear regression model (Appendix p. 9). This suggests that an extra year of maternal formal education is associated with a 2.1 percentage point (SE = 0.2) decrease in the probability that her child will contract malaria at the time of the survey. This significant association, however, may be not causal because of the possible association between observed maternal education (S) and unobserved confounding factors (U). Therefore, I exploited the exogenous variation in the length of maternal education as if it was randomly caused by the 1997 reform to obtain a 2SLS estimate of its causal effects on the children’s risk of malaria infection.

The first-stage model shows that the reform increased maternal educational attainment by 0.7 years on average (SE = 0.29; $p < .0001$; Table 2). Figure 3 depicts that share of mothers completing each grade of primary education grew slowly until the birth cohort born in 1981. This share disproportionately increased among the ≥ 1982 cohort. By contrast, I did not find the increase in the probability that women completed the 8th grade (1st grade of secondary education) or more among the ≥ 1982 cohort.

| | Exposure | Outcome | Effect estimate (SE) |
|--|--------------------|---|----------------------|
| Schooling (n=5316; 4.4 years of education pre-reform) | | | |
| Model 1: first stage | Reform indicator | Years of schooling | 0.65‡ (0.20) |
| Malaria (n=5316; 34.9% Malaria positive pre-reform) | | | |
| Model 2: intention-to-treat | Reform indicator | Malaria positive | -4.8† (2.4) |
| Model 3: 2SLS (IV) | Years of schooling | Malaria positive | -7.5* (3.9) |
| Anemia (n=5316; 41.2% Moderate or serious anemia pre-reform) | | | |
| Model 2: intention-to-treat | Reform indicator | Anemia | -5.4† (2.5) |
| Model 3: 2SLS (IV) | Years of schooling | Anemia | -8.2* (4.3) |
| Vector control (n=5316; 77.5% Owns net pre-reform; n=4558; 47.9% all <5y children slept under net) | | | |
| Model 3: 2SLS (IV) | Years of schooling | Own mosquito net | -0.5 (2.9) |
| Model 4: 2SLS (IV) | Years of schooling | All <5y children slept under net last night | 10.1† (4.8) |

For malaria, anemia, and vector control outcome, regression coefficient and standard errors are multiplied by 100 and reported on a percentage point scale. Percent malaria positive refer to the prevalence among children of mothers in the 1980 cohort. Model 1 is estimated by Ordinary Least Square (OLS) method, model 2 by OLS linear probability method, model 3 by instrumental variable two stage least square (IV-2SLS) method, in which binary indicator for being born 1982 or later was used as an excluded instrumental variable for maternal years of schooling, and model 4 by IV-2SLS with sample owns mosquito net. All model controls for full set of child age in month indicators, a linear term of maternal year of birth, indicator for survey year, indicator for survey month, indicator for child sex, indicator for ethnic groups, and age of the mother. * $p < 0.1$. † $p < 0.05$. ‡ $p < 0.01$. No weights were used. Kleibergen-Paap Wald rk F statistic in the first stage models were 10.4.

Table 2: Regression results

Figure 3: Maternal educational attainment by birth cohort

Shows the probability that a mother completed certain grade of education by the time of the survey

The second row of Table 2 shows the ITT effects of the reform on children's risk of malaria infection. The reform reduced the probability that children of exposed mothers will contract malaria at the time of the survey by 7.5 percentage points, on average ($p = .057$). In the 2SLS model (row 3 of Table 2), the probability that a child contracts malaria decreased by 4.8 percentage points as mothers attained one extra year of education on average ($p = .042$). Compared to the probability that children of mothers born in 1980 contract malaria (35.3%), this effect size is as large as a 21 percent reduction per additional year of maternal schooling.

I conducted multiple sensitivity analyses for the 2SLS estimates (Table 4). Overall, the results are robust to different observation windows, and functional form of the birth cohort trend in exposure and outcome. The difference-in-differences estimates were qualitatively similar although different IV theoretically provides different LATE because compliers are different. The placebo test indicates that the reform had negligible effects on the women with no schooling who were unlikely to be exposed to the reform. To test the external validity, I used the 1994 free primary education reform in Malawi for replication, and the results are qualitatively similar (Appendix Table S3).

UNESCO estimated the cost per primary student per year as US \$34.2 in Uganda between 2010 and 2013 on average.²⁴ Since an additional year of maternal education reduced the probability that her child contracts malaria at the time of the survey by 7.5 percentage point, the cost per malaria infection among children aged < five years averted amounts to US \$456. Referring to the World Health Organization standard, the intervention costs less than the GDP per capita per DALY averted are considered a very cost-effective intervention. Given the calculation shown in the Appendix, a malaria infection at five-years-old in Uganda led to 0.5 DALY lost. Given the total fertility rate of 5.6 in Uganda,²⁵ the resulting cost-effective ratio per DALY averted of maternal primary schooling is US \$162.9. According to the World Bank, GDP per capita in Uganda in 2017 was US \$604²⁵, suggesting that promoting primary education is a very cost-effective intervention to prevent child malaria morbidity. Compared

with existing interventions, primary schooling for mothers is more expensive than chemoprophylaxis; Long Lasting ITNs; intermittent preventive-treatment in infants, and antimalarial drugs, including parenteral artemunate, dihydroartemisinin piperazine, and artemether lumefantrine; but being comparable to IRS and RTS, S/AS01 vaccine.^{26–29}

| | Exposure | Outcome | Effect estimate (SE) |
|--|--------------------|------------------|----------------------|
| Model 1: 2SLS, slope change in YOB | Years of schooling | Malaria positive | -6.3† (2.6) |
| Model 2: 2SLS, quadratic in YOB | Years of schooling | Malaria positive | -6.3† (2.9) |
| Model 3: 2SLS, quadratic in maternal age | Years of schooling | Malaria positive | -6.4* (3.9) |
| Model 4: 2SLS, control birth order | Years of schooling | Malaria positive | -9.3* (5.4) |
| Model 5: 2SLS, narrow window | Years of schooling | Malaria positive | -8.2* (4.8) |
| Model 6: 2SLS, exclude 1982 cohort | Years of schooling | Malaria positive | -7.4† (3.4) |
| Model 7: 2SLS, Malawi | Years of schooling | Malaria positive | -7.1‡ (2.6) |
| Model 8: ITT, education=0 | Reform exposure | Malaria positive | -0.7 (5.0) |

Coefficient and standard errors are multiplied by 100 and reported on a percentage point scale. Model 1-6 is estimated by instrumental variable two stage least square (IV-2SLS) method, in which binary indicator for being born 1982 or later was used as an excluded instrumental variable for maternal years of schooling. Model 7 is estimated by IV 2SLS method, in which binary indicator for being born 1979 or later was used as an excluded instrumental variable for maternal years of schooling. Model 8 is estimated by Ordinary Least Square (OLS) method. All model controls for full set of child age in month indicators, a linear term of maternal year of birth, indicator for survey year, indicator for survey month, indicator for child sex, indicator for ethnic groups, and maternal age. *p<0.1. †p<0.05. ‡p<0.01. No weights were used.

Table 3: Robustness check

Discussion

The present study revealed that length of maternal education reduces the probability that her children will contract malaria in Uganda. Length of maternal education was positively associated with better use of ITNs. The results suggest an empirical association between maternal schooling and the children’s risk of malaria infection previously reported in the existing observational studies potentially underestimated the true causal effects. The current results are interpreted as causal because exposure to the reform is considered random, and the reform is likely to change child malaria risk only through its effects on maternal schooling.

Theoretically, average treatment effects estimated by the IV 2SLS method are interpreted as “local” treatment effects for a subset of the population. First, I exploited exogenous variation in women’s educational attainment caused by the reform at the level of primary education. Therefore, the estimate is local to the population who are induced to stay longer at primary education. It may be surprising that even a low level of education (i.e., primary) had modest protective effects on childhood malaria control, and the effects of higher education may be even stronger. Second, the present estimates are local to the population—so-called compliers—who attained more schooling if the reform was implemented when they were 14-years-old, but not in the absence of the reform. Third, the estimated effects are local to the context of malaria-endemic countries. Thus, the association between maternal education and childhood malaria control may differ in countries with a lower prevalence of malaria in children. Fourth, the findings are local to the context, in which ITNs; IRS; and malaria treatment, such as artemisinin-based combination therapy, are plausibly available. When the survey was conducted in 2014, 90% of households owned at least one ITN and 45% of pregnant women received at least two doses of IPT.^{20,21} Therefore, it may not be plausible to generalize the findings to the earlier cohorts who were raised before malaria control interventions had not rolled out sufficiently in Uganda. However, given that these malaria control methods are becoming practically available in malaria-endemic countries, the results should inform present and future malaria control strategies. Having a consistent evidence from Malawi supports the high generalizability of the findings.

The 2SLS estimates outweigh the OLS estimates for distinct reasons other than the LATE interpretation. One possible explanation is that unobserved confounding factors that positively affect length of maternal schooling are also positively correlated with the child’s malaria risk, or vice versa. For example, a mother from an urban area may attain more education because the distance to the nearest primary school is short; simultaneously, her child may be more likely to contract malaria, potentially because the household has less access to ITNs.^{20,21} In this scenario, estimate from naïve OLS method is biased toward zero.

The current study had several limitations. First, participation rates for a child malaria blood test was not perfect, albeit very high (97%). No discontinuity in participation rates were observed around the cut off. Thus, the composition of mother whose

child provided the malaria biomarker samples was comparable between the exposed and the unexposed cohort. Second, some children aged <5 years may contract malaria before his/her mother completes education. This is theoretically possible, but empirically very unlikely, because we exclude women who were younger than 18 years old at the time of the survey, and few women attend primary schools after the age of 18.

Third, the reform may reduce maternal mortality, and thus the composition of the observed sample may systematically differ between the exposed and the unexposed individuals. To test if this is the case, I compared the cohort size by birth cohort, and found no significant increase in the cohort size among the 1982 or later cohorts compared to 1981 or earlier cohorts (Figure S3). Thus, selective mortality of the mother is unlikely to bias my estimate. Fourth, the reform may reduce the child mortality. Thus, the composition of the observed sample of children may differ between the exposed cohort and the unexposed cohort. However, as reported in the existing study, the reform was shown to have no effects on the probability of infant mortality. Hence, selective child mortality is also unlikely to bias the estimates.

Fifth, available data allowed me to observe children's probability of contracting malaria only up to 59-months-old. Although limited, this is a crucial period, when malaria often causes loss of life and neurocognitive impairment³⁰. Since the benefits of maternal education on malaria control are likely to be applicable to older children, and even to adults, the true cost-effectiveness of the intervention may be even greater than the current estimates. Lastly, because of the survey dates and inclusion criteria, the children of the first exposed 1982 cohort were born when the mothers aged 21–31 years at the time of the survey. This is the period during which many women are pregnant in Uganda. Although teenage pregnancies are less common than pregnancies among women in their 20s.^{20,21}, future research should examine the effects of maternal education on malaria incidence in these younger mothers.

Promoting access to primary education for women had a substantial effect on childhood malaria control in Uganda. The cost-effectiveness ratio per DALY averted of the primary schooling intervention is high. Hence, reform to expand access to primary education for girls should be an effective means to improve malaria control, and it should be combined with other malaria vector control interventions since maternal education increased the appropriate use of bednets for young children.

Contributor: KM contributed to the conception, design of the work, data analysis and interpretation, drafting the article, critical revision of the article, and final approval of the text.

Declaration of interest: none exist

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Appendix

Study Population

Maternal age in the study population ranges from 18 to 41, with mean of 28.1 (SD 5.0) in the full sample. Mean child age was 28.9 months. Mean length of maternal schooling was 4.8 years (SD 3.6) in 2009 UMIS, and 4.9 years (SD 4.1) in 2014 UMIS, respectively.

Description of the 1997 Education reform in Uganda

Uganda's formal education consists of primary (7 years), lower secondary (4 years), and upper secondary education (2 years). The national law stipulates that children start seven years of primary education at age six, but before the reform started in 1997, delayed school entry and grade repetition were not uncommon. The primary level gross enrolment rate was 70 percent in 1996.¹ Public primary schools had financially relied on private resources. Parents contributed to the majority of school inputs such as tuitions, Parents Teacher Association (PTA) fees, and uniform costs. These costs per child at public primary school amounted to 62 percent of the average annual household expenditure. This financial burden made it difficult for many, particularly poor, parents to afford school costs.²

The issue of free primary education received a high-level political support when the then-candidate for the first president, Museveni, made it one of his platform issues during the campaign for the election in May 1996. After being elected, fee abolition was written into a government manifesto in December 1996, and it was announced that implementation would begin in January 1997.^{3,4} In 1997 January, the government abolished tuition and PTA fees in public primary schools. This nationwide education subsidy effectively boosted the primary-level gross enrollment ratio from 70 percent in 1996 to 120 percent in 2009.¹ and this increase was particularly large among girls.^{2,5-7}

Detail of Cost-Effective Calculations

I followed a simple model to calculate the number of DALY resulting from malaria infection at age five. I assumed that all the malaria incidence occurs at age of five, and incidence lasts for seven days. Disability weights were obtained from the 2010 Global Burden of Disease.⁸ Life expectancies at age five for males and females were obtained from WHO life table.⁹ The case fatalities rate of malaria incidence under age five in Uganda was obtained from the Annual Malaria Report.¹⁰

Mechanism

Vector control

-net ownership and access

The use of mosquito nets to prevent malaria is a key strategy supported by the Ugandan government. In this study, I focused on whether mothers were more likely to own and use mosquito nets if they

were more educated.

-IRS

Indoor residual spraying is another effective vector control strategy supported by the WHO. Due to the high burden of its cost, only 14 high-burden districts out of 110 districts were covered in 2014. Although the regional coverage is exogenously determined by the government regardless of maternal level of education, here I tested if such spraying was more likely to be done in a household with educated mothers.

Management of fever in children

The current policy suggests the diagnosis with microscopy or Rapid Diagnosis Test for all fever cases in children. The parasitological confirmation of the malaria species presence is required before the prescription of the anti-malarial drug.

The present study tests if children under the age of five, and with fever symptoms, were more promptly diagnosed and treated if they were raised by more educated mothers.

Malaria Knowledge

One possible pathway through which maternal schooling may change the behavior of the mother is because longer grade completion may lead to improved maternal knowledge on vector control and case management of fever in children. Here, I show whether educated females hold more information on causes of malaria, ways to avoid malaria, and the case management of children with fever.

Estimated results of mechanism analysis

Figure S3 shows the analysis of the mechanism through which maternal schooling reduced the incidence of malaria among children. An extra year of maternal schooling was not associated with ownership of the mosquito net, but it was positively associated with net usage for children aged <5 years. An additional year of maternal schooling increases maternal knowledge on the method to avoid malaria. Maternal schooling was positively associated with the prompt treatment of children with fever, but this did not reach the conventional benchmark of statistical significance.

Economic explanation for behavioral change

Through what mechanism did mothers improve vector control and management of fever in children as they are educated? Although a detailed analysis is beyond the scope of the paper, I provide several economic explanations behind these behavioral change among educated mothers. First, longer years of maternal education may change the behavior of the mother, and reduce the child's malaria risk through the following mechanisms: 1) expanded access to financial resource through labor force

participation, partnering with a husband from a higher SES; 2) higher ability to gather and process information, and better knowledge on health in general, and; 3) stronger decision making power within a marriage.^{6, 7}

Robustness checks

Non-random attrition and consent rates

One possible source of bias to the current estimate is that the consent rates for child malaria blood testing may be non-random, and correlated with the exposure to the 1997 reform. It is also possible that the reform reduced the mortality of females, and selective mortality may bias the estimate. Figure S4 shows the probability of missing a child malaria biomarker, and it shows the size of the cohort by maternal birth year. It provides no evidence that either of these non-random selection elements biased my results. Furthermore, I tested whether the probability of missing a child malaria biomarker significantly increased in the 1983 cohort compared to the earlier cohorts in a regression. The results in Table S2 suggest no evidence supporting such concern.

Alternative explanation

The reform may improve fathers' educational attainment in the same cohort, and the risk of child malaria may decline owing to paternal, rather than maternal, education. However, an existing study has shown that educational attainment of the male cohort born ≥ 1982 did not disproportionately increase compared to the cohort born ≤ 1981 .⁷ Therefore, the observed decline in the probability of children's risk of malaria infection was not mediated by the change in fathers' education.

Spillover effects

The reform may have spillover effects on the children of mothers who were not exposed to the reform. For example, the risk of malaria increases with the density of infected mosquitos in the neighborhood, and educated mothers may use better vector control methods. Under such a scenario, the risk of malaria infection may decline even in households in which mothers were not exposed to the reform. These mothers may be affected if they live in a neighborhood with exposed mothers. It is also possible that information spillover may affect unexposed mothers' behavior, therefore further reducing the malaria risk. However, both scenarios produce an underestimate of the true effect size of maternal education on child's malaria control. Therefore, the true effect of maternal education and cost-effectiveness of primary schooling intervention may be even greater than the current estimate.

Exclusion of pivotal cohort

Given the proportion of females attending primary education by age in 1996, I define the first cohort exposed to the reform as 1982 cohort. I, however, observed, in Figure 2 (main text), the rise in the probability of completing primary schooling among 1982 cohort were evident, but partial and were even more evident among 1983 cohort. Therefore, I tested the robustness of the estimates by excluding 1982 cohort as a sensitivity test.

Controlling for birth order

The reform may change women's fertility pattern and thus composition of the children may differ between exposed and non-exposed cohort. To obtain a conservative estimate, I tested the robustness of my findings by controlling for the full set of birth order indicators to control for the birth-order-specific effects on children's risk of malaria infection. In sum, the results are similar, further confirming that maternal education improves child malaria control.

Estimated results of robustness checks

Table S3 shows the robustness of the findings to using additional controls, using a narrower bandwidth, excluding the pivotal cohort, and the placebo test. Using a linear term in year of birth with reform indicator interaction, quadratic term of year of birth, extra year of maternal schooling was associated with 6 percentage point reduction in child's malaria risk. Controlling for birth order of the children, or by implementing a narrower window size, an additional year of maternal schooling reduces the probability that her child contracts malaria by 8-9 percentage points on average. Excluding the 1982 cohort, an additional year of maternal schooling reduces the probability that a child contracts malaria by 7 percentage points. I found no effects of the reform on females with no schooling, whereas I found the exposure to the reform reduced the incidence of the child malaria by 5 percentage points in the full sample.

Lastly, using the exposure to Malawi 1994 free primary education reform as the instrumental variable, IV 2SLS estimates shows that an extra year of maternal schooling in Malawi was associated with a decline in the probability that a child would contract malaria at the time of the survey by 8.8 percentage points. (Details are discussed in the next subsection)

Generalizability: Replication in Malawi

Malawi is another country where the malaria endemic is most severe. The malaria incidence rate was 188.8 per 1,000 people in 2015.¹¹ *Plasmodium falciparum* is the predominant

Plasmodium species found in Malawi. ¹²⁻¹⁴

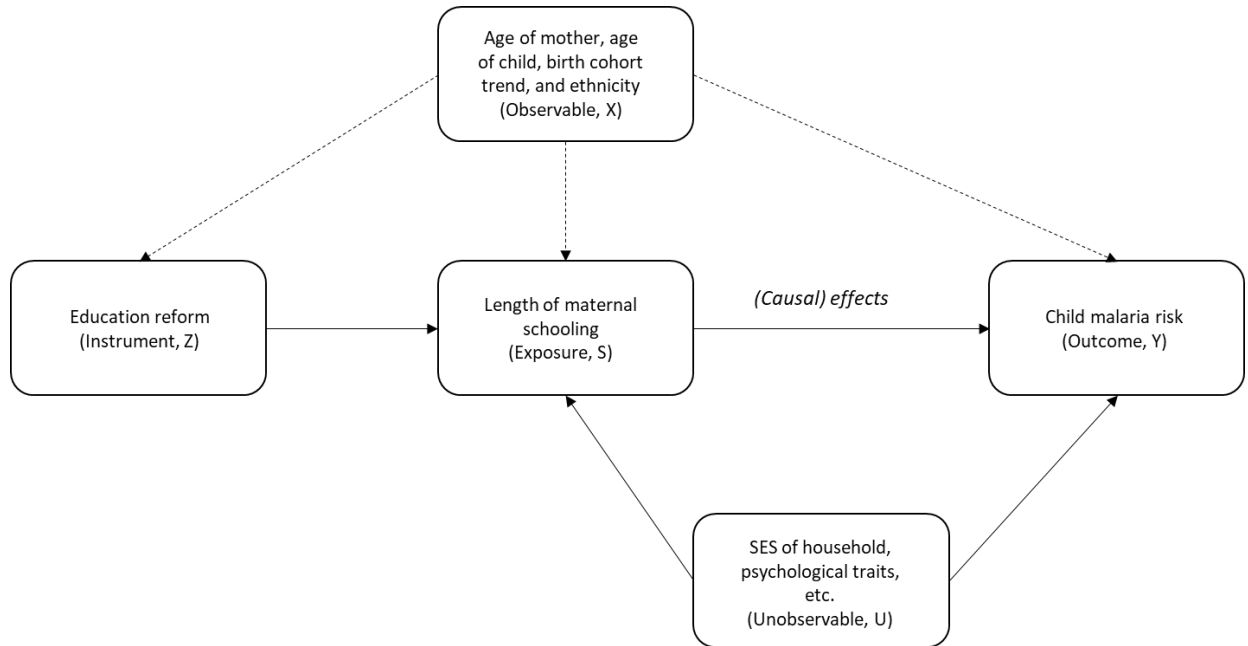
The data used in this replication came from the 2012, 2014 and 2017 Malawi Malaria Indicator Surveys (UMIS), which were nationally representative, repeated, cross-sectional, household surveys that included malaria biomarker collection for children younger than five years of age.¹²⁻¹⁴ In the 2012, 2014 and 2017 surveys, 3,404, 3,405, and 3,729 households were interviewed, respectively. The response rates were 98%, 99% and 99.8%, respectively.¹²⁻¹⁴ All girls/women aged 15–59 years were eligible for individual interviews. In addition, I obtained data from the MMIS concerning testing for malaria in children aged 0–59 months using a finger-prick blood collection method. All children aged 0–59 months in the households selected for MMIS were eligible for biomarker collection, and 97% for 2012 and 2014 MIS, and 99% for 2017 MIS of eligible children were tested for malaria with microscopy in both years.¹²⁻¹⁴ Key information on birth year, age, and mothers' educational attainment were available for 99% of the respondents.

I excluded children of mothers born in 1970 or earlier and those born in 1989 or later because those women were likely to be exposed to other education reform programs. Children of mothers who were aged < 18 years were also excluded because they may still have been receiving formal education at the time of the survey. I also excluded women with no children aged < five years because my primary interests were the effects of maternal education on childhood malaria control.

Primary exposure was defined as mothers' educational attainment (in years) at the time of the survey. The primary outcome was their children's malaria status at the time of the survey. I defined a binary variable which takes value of unity if the respondent woman born in a cohort exposed to 1994 reform, otherwise zero. Considering the share of individuals attending primary school in 1994 by age, those born in 1980 or later (i.e., \leq 13-years-old as of January 1st 1994) were defined as exposed. I excluded the 1979 pivotal cohort in the main analysis (Makate and Makate, 2016). I also tested the sensitivity of the results by including the 1979 cohort as a robustness check. In the analysis, I controlled for maternal year of birth, maternal ethnicity, children's age in months, sex, and month and year of the survey.

Table S4 presents the results of replication in Malawi.

Figure S1 Causal diagram



The diagram explains the identification assumptions under 2SLS method. I assume that conditional on X , Z is valid instrument if 1) Z is strongly correlated with S (relevance), and 2) Z is not associated with U , and Z affects Y only through its effects on S (exclusion restriction). Under the additional assumption 3) Z changes S only in one direction (monotonicity), the estimated treatment effects are interpreted as Local Average Treatment Effects (LATE).

Figure S2: Changes in maternal education and child malaria by maternal birth cohort in Uganda

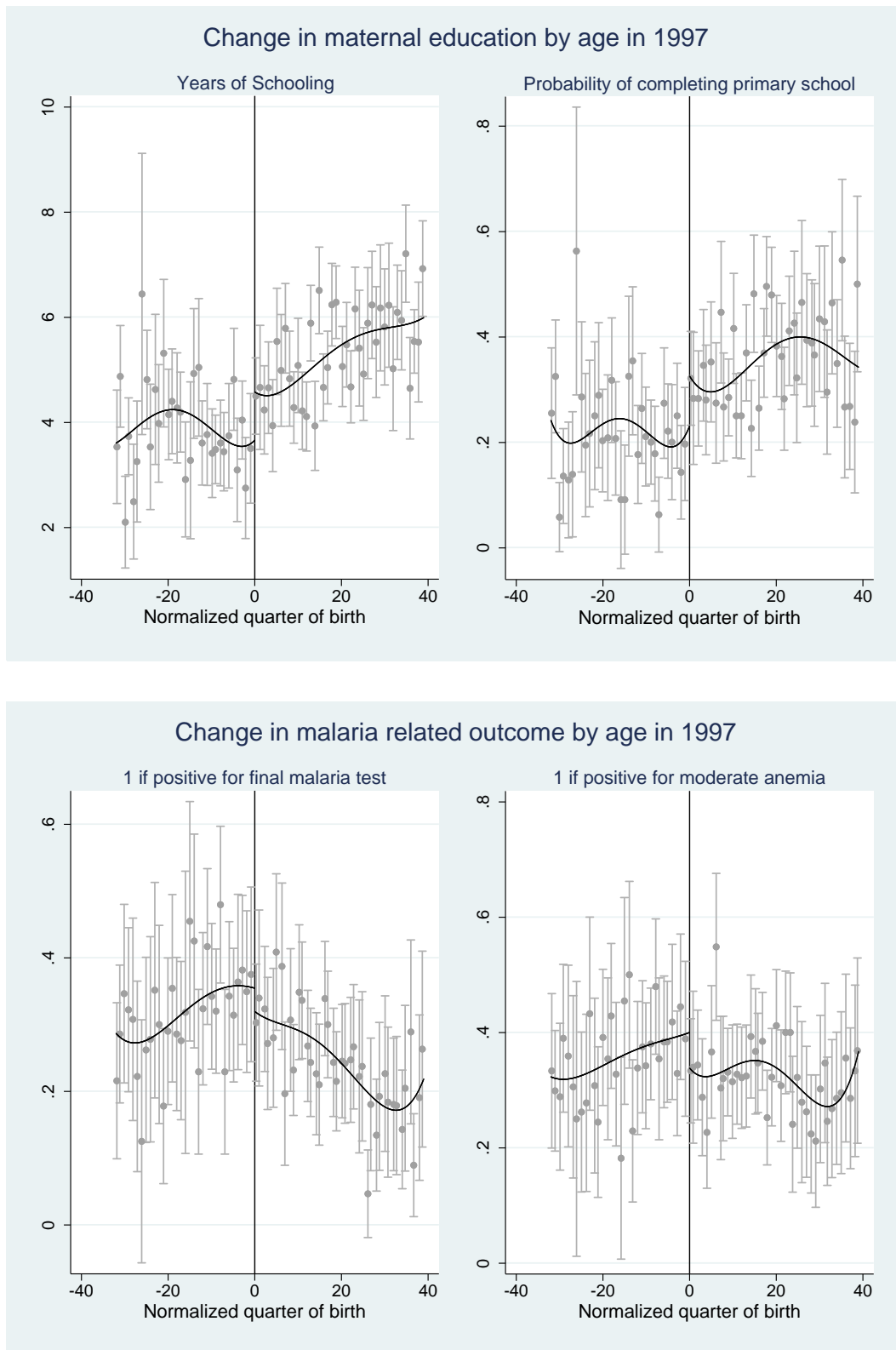


Figure S3 The effects of maternal schooling on the mechanism outcome

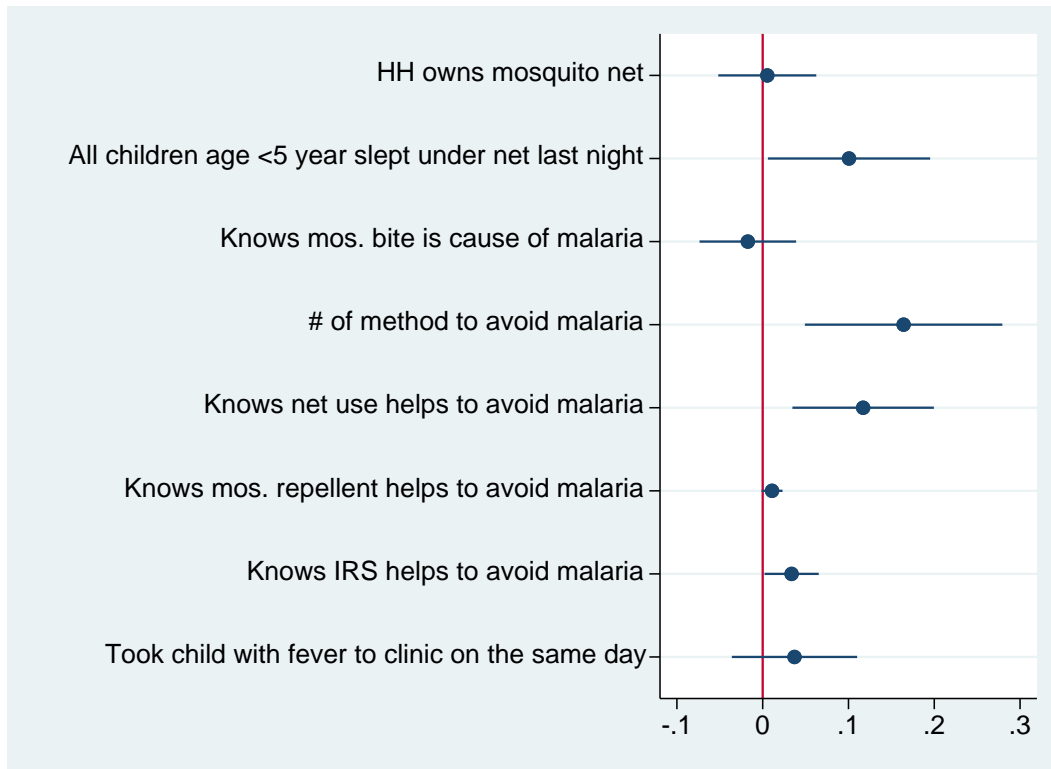


Figure shows estimated coefficient of the “maternal schooling” with 95% confidence interval. Models were estimated by instrumental variable two stage least square (IV-2SLS) method, in which binary indicator for being born 1982 or later was used as an excluded instrumental variable for maternal years of schooling. All model controls for full set of child age in month indicators, a linear term of maternal year of birth, indicator for survey year, indicator for survey month, indicator for child sex, indicator for ethnic groups, and maternal age. No weights were used.

Figure S3 Change in the cohort size and probability of missing child malaria biomarker by maternal birth year

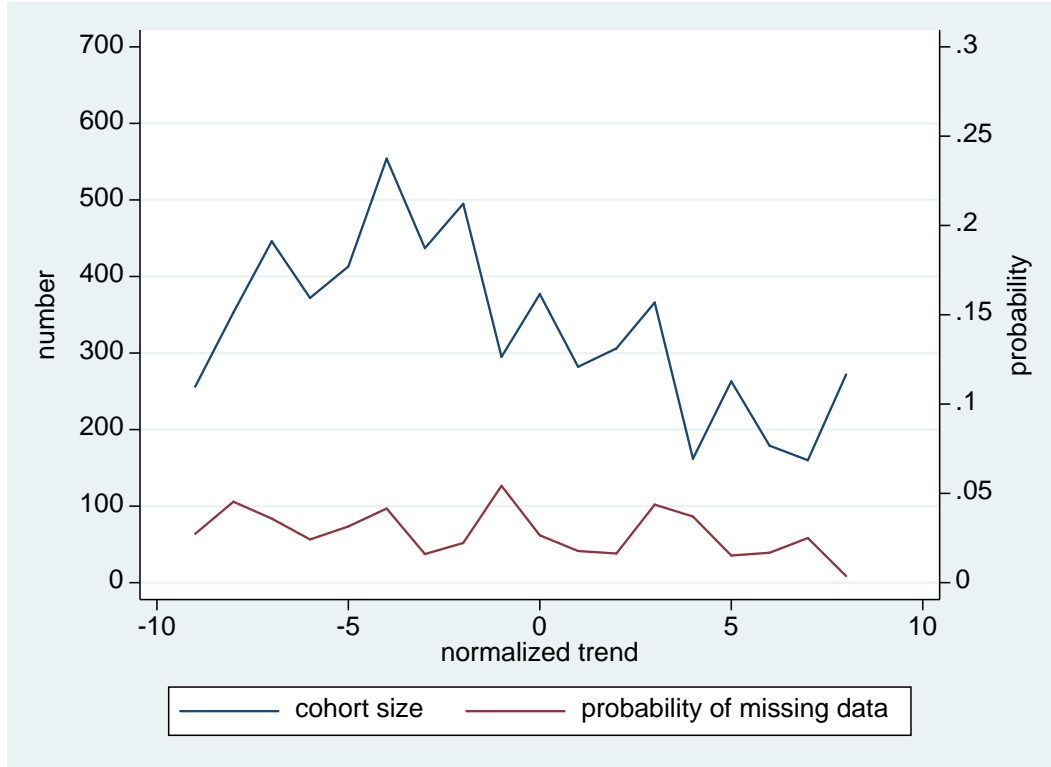


Table S1 Association between length of maternal schooling and child malaria status

| Outcome: child malaria positive | (1) | (2) | (3) |
|--|----------------------|----------------------|----------------------|
| Years of education | -0.022*** (0.001) | | |
| 1 if completed at least 7 years of schooling | | -0.152*** (0.012) | -0.094*** (0.019) |
| 1 if completed at least 6 years of schooling | | | -0.055** (0.025) |
| 1 if completed at least 5 years of schooling | | | 0.048 (0.029) |
| 1 if completed at least 4 years of schooling | | | -0.039 (0.032) |
| 1 if completed at least 3 years of schooling | | | -0.087** (0.035) |
| Observation | 5,316 | 5,316 | 5,316 |

Table S2 ITT effects of the reform on the probability of missing child malaria biomarker

| Outcome: missing malaria data | | (1) |
|-------------------------------|------------------------------|------------------|
| Reform indicator | (1 if born in 1983 or later) | 0.000 (0.000) |
| Observation | | 5,316 |

Note: MIS 2009, 2014 provides information. Robust standard errors in parenthesis.

Table S3 Uganda

| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) | (9) |
|----------------------------|---------|----------|----------|---------|---------|---------|----------|---------|----------|
| Years of education | -0.075* | -0.063** | -0.063** | -0.064* | -0.093* | -0.082* | -0.074** | -0.007 | -0.048** |
| | (0.039) | (0.026) | (0.029) | (0.036) | (0.054) | (0.048) | (0.034) | (0.050) | (0.024) |
| 1 if born in 1982 or later | | | | | | | | | |
| <i>Basic covariates</i> | | | | | | | | | |
| i.child age | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| c.YOB | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| i.survey year | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| i.survey month | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| i.ethnicity | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| c.mother's age | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| <i>Additional controls</i> | | | | | | | | | |
| c.YOB#i.reform | | ✓ | | | | | | | |
| c.YOB^2 | | | ✓ | | | | | | |
| c.mother's age^2 | | | | ✓ | | | | | |
| i.birthorder | | | | | ✓ | | | | |
| <i>Narrower bandwidth</i> | | | | | | | | | |
| 1973<YOB<1992 | ✓ | ✓ | ✓ | ✓ | ✓ | | | | |
| 1974<YOB<1991 | | | | | | ✓ | | | |
| exclude 1982 cohort | | | | | | | ✓ | | |
| sub sample, no schooling | | | | | | | | ✓ | |
| all sample | | | | | | | | | ✓ |
| Observation | 5,316 | 5,316 | 5,316 | 5,316 | 5,316 | 4,841 | 4,974 | 1,249 | 5,316 |

Table S4 Malawi

| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) | (9) |
|----------------------------|---------------------|--------------------|--------------------|--------------------|---------------------|---------------------|--------------------|-------------------|---------------------|
| Years of education | -0.088** (0.043) | -0.111* (0.067) | -0.112* (0.064) | -0.098* (0.058) | -0.068** (0.031) | -0.135** (0.062) | -0.064* (0.038) | | |
| 1 if born in 1980 or later | | | | | | | | -0.033 (0.088) | -0.074** (0.031) |
| <i>Basic covariates</i> | | | | | | | | | |
| i.child age | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| c.YOB | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| i.survey year | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| i.survey month | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| i.ethnicity | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| c.mother's age | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| <i>Additional controls</i> | | | | | | | | | |
| c.YOB#i.reform | | ✓ | | | | | | | |
| c.YOB^2 | | | ✓ | | | | | | |
| c.mother's age^2 | | | | ✓ | | | | | |
| i.birthorder | | | | | ✓ | | | | |
| <i>Narrower bandwidth</i> | | | | | | | | | |
| 1973<YOB<1992 | ✓ | ✓ | ✓ | ✓ | ✓ | | | | |
| 1974<YOB<1991 | | | | | | ✓ | | | |
| exclude 1982 cohort | | | | | | | ✓ | | |
| sub sample, no schooling | | | | | | | | ✓ | |
| all sample | | | | | | | | | ✓ |
| Observation | 3,185 | 3,185 | 3,185 | 3,185 | 3,185 | 2,822 | 3,343 | 543 | 3,185 |

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