# Disease and Human Capital Accumulation: Evidence from the Roll Back Malaria Partnership in Africa*

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We study the effect of the Roll Back Malaria Partnership's campaigns on human capital outcomes using microeconomic data from 27 countries in Sub-Saharan Africa. We first construct a theoretical framework to describe a household's human capital production. Then, using a difference-in-differences approach based on pre-campaign malaria risk and campaign timing and magnitude, we estimate the effect of anti-malaria campaigns on a set of human capital outcomes. Campaigns reduce infant mortality and fertility, while increasing adult labor supply and educational attainment. Our results underscore the importance of considering how effects extend beyond health when evaluating large-scale efforts to reduce disease.

*Keywords*: Health, education, fertility, labor supply, Africa, malaria

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# 1 Introduction

Despite decades-long efforts, malaria remains a life-threatening disease. In 2015 alone, there were roughly 214 million cases of malaria, resulting in an estimated 584,000 deaths.[1] Malaria has long been a topic of importance in the economics literature due to its deleterious relationship with economic growth. At a microeconomic level, reducing malaria leads to improvements in infant mortality and early childhood health (Lucas, 2013; Pathania, 2014). In turn, these changes have the power to substantially influence household decision-making. Empirical evidence from historic eradication campaigns shows that reductions in malaria can increase live births (Lucas, 2013), improve educational attainment, literacy, and cognition (Cutler et al., 2010; Lucas, 2010; Venkataramani, 2012; Barofsky, Anekwe and Chase, 2015; Burlando, 2015) and lead to greater incomes, consumption and labor productivity (Bleakley, 2010; Cutler et al., 2010; Hong, 2013; Barofsky, Anekwe and Chase, 2015). In this paper, we study the response of human capital outcomes to malaria control efforts in 27 countries in Sub-Saharan Africa.

In 1998, the World Health Organization (WHO) launched a new campaign to halve malaria deaths worldwide by 2010 (Nabarro and Tayler, 1998). With this goal came the need to establish a global framework for coordinated action against malaria — and the Roll Back Malaria (RBM) Partnership was born.[2] RBM serves as a conduit to harmonize resources and actions among the many national, bilateral and multilateral actors engaged in malaria control. By 2010, targeted funding from external actors had reached nearly $2 billion annually (Pigott et al., 2012). Sponsored control efforts focus on prevention and treatment among the most at-risk populations through artemisinin-combination therapies.[3] They also limit malaria transmission from mosquitoes to humans with insecticide treated nets and indoor residual spraying.[4] By 2014, just over a decade after the scale-up of these control efforts, worldwide malaria deaths had been cut in half.

[1] See the World Health Organization's website: http://www.who.int/mediacentre/news/releases/2015/report-malaria-elimination/en/. Accessed on 05/31/2016.

[2] More information can be found at the website of the Roll Back Malaria Partnership: http://www.rbm.who.int/.

[3] Artemisinin and its derivatives produce the most rapid action of all current drugs against *P. falciparum* malaria.

[4] These approaches are sometimes combined with larval control which eliminates mosquitoes at their larval stage. However, due to its detrimental environmental effects and poor cost-effectiveness, larval control is recommended only for specific settings.

This massive reduction in malaria-related mortality may have effects that reach beyond health. Improving early childhood health paves the way for greater educational attainment. But it also raises the opportunity cost of education by increasing a child's potential wages on the labor market (Bleakley, 2010). This, in turn, can influence adult fertility and labor decisions by reducing the cost of each additional child (Vogl, 2014). To untangle the relationship between malaria control campaigns and these outcomes, we construct a simple theoretical framework of a household's human capital production. We then estimate the impact of the RBM campaigns on infant mortality, fertility, adult labor market participation, and children's education from 2003 to 2014.

Our empirical strategy is a modified difference-in-differences analysis. We compare the outcomes of individuals treated by anti-malaria campaigns to the outcomes of individuals less treated by anti-malaria campaigns based on a continuous assignment to treatment. To do so, we combine geocoded household microdata from the Demographic and Health Surveys (DHS) with detailed maps of malaria risk generated by the Malaria Atlas Project (MAP) and country-year disbursements from the RBM campaign's largest donors. This innovative temporal and spatial structure allows us to cover a much larger range of countries than previously studied, which is important not only for statistical power but also for internal and external validity. Figure 1 displays the 27 countries in our sample.

Though similar in principle to other empirical studies, we make several departures from the standard difference-in-differences framework. First, we primarily observe individuals post-treatment. To assign individuals to treated or untreated groups, we make use of the fact that RBM targeted areas with the highest burdens of malaria, a feature determined largely by geographic and climactic characteristics. This measure differs from previous studies relying on similar household data which tend to use household control strategies to proxy for treatment. An area's pre-treatment (i.e. pre-RBM) malaria risk can therefore proxy for the likelihood that a given area was treated or untreated. Based on a respondent's geocoded cluster, we assign to each individual a pre-campaign malaria risk ranging between 0 and 1. This assignment, which is independent of survey year, determines a respondent's treated or untreated status.

Yet assigning treatment purely by an area's pre-treatment malariousness would be too reductionist in this context. Treatment depends predominantly on the timing and intensity

of RBM campagins in a given country. This implies that two treated individuals surveyed in two different years in the same country receive vastly different degrees of exposure to anti-malaria campaigns over their respective lifetimes. While we do not observe the same respondent in multiple surveys, we observe similar individuals — those in the same age cohort — across time. All members of a single age cohort in a given country-year experience the same intensity of RBM treatment, which we compute as the yearly amount per capita disbursed by RBM campaigns during an individual's lifetime. The chief innovation of this strategy is that it exploits several layers of variation in exposure, relying not only on cohort dates of birth but also the distribution of DHS surveys across time. Introducing differential treatment intensity within clusters has another, more practical, advantage: it allows us to control for cluster fixed effects as well as country-by-cohort-by-survey year fixed effects. These demanding restrictions help us to isolate an estimated effect of RBM that is driven by variation in assignment to treatment at the cluster level and variation in intensity of treatment at the country-cohort-survey year level.

Our results, which are not driven by pre-campaign catch-up effects between treated and untreated populations, show that RBM campaigns reduce infant mortality and fertility, while increasing adult labor supply and educational attainment. More specifically, a one dollar increase in RBM funding per capita per year reduces infant mortality by 6.3 percentage points and leads to an increase in roughly 0.40 years of schooling. The magnitude of these effects is in line with existing evidence. Furthermore, our results hold in falsification tests and alternative sub-samples as well as other robustness checks.

Other notable studies implement similar difference-in-differences analyses to estimate the effects of malaria control on various socioeconomic factors and find similar results. Bleakley (2010) analyzes malaria eradication in the United States (1920) and in Brazil, Colombia and Mexico (1950) to assess the impact of childhood exposure to malaria on labor productivity. Cutler et al. (2010), Lucas (2010), and Venkataramani (2012) estimate this impact on educational and/or cognitive outcomes in India, Paraguay and Sri Lanka, and Mexico, respectively. Lucas (2013) uses a difference-in-differences approach to study the effect of malaria elimination on fertility and child survival rates using the case of Sri Lanka. In Uganda, Barofsky, Anekwe and Chase (2015) find that malaria eradication raised educational attainment by about half a year for both males and females, increased girls' primary school completion and

generated an almost 40% increase in the likelihood of male wage employment. Finally, in Ethiopia, Burlando (2015) shows that education levels are lower in areas with more adverse disease environments.

Our approach complements these contributions in at least four ways. First, the scope of our analysis (millions of individuals from 27 countries) is unprecedented. While one of the advantages of a quasi-experimental approach over a randomized experiment is that it can be replicated over a larger population, the maximum number of countries covered by previous quasi-experimental studies is four (Bleakley, 2010). Second, contrary to most previous studies, we do not focus on the malaria periphery, i.e. the set of countries characterized by species of *Plasmodium* (*P. vivax*, *P. ovale* and *P. malariae*) relatively less harmful to health. We concentrate instead on Sub-Saharan Africa where *P. falciparum*, the most aggressive of all species, is dominant. Third, we study contemporaneous, international control efforts which are relevant to ongoing policy decisions. This allows us to make an important distinction from previous analyses that focus on historic malaria eradication efforts in the early to mid-1900s (Bleakley, 2010; Cutler et al., 2010; Lucas, 2010; Venkataramani, 2012; Barofsky, Anekwe and Chase, 2015). Finally, we focus on a rich set of outcomes: health, fertility, labor market participation and educational attainment. Our findings highlight the importance of evaluating large-scale health interventions with respect not only to their primary health outcomes but also to their secondary effects. As such, they shed further light on the benefits of subsidizing health interventions (Miguel and Kremer, 2004; Cohen and Dupas, 2010; Dupas, 2014; Tanaka, 2014; Cohen, Dupas and Schaner, 2015).

The paper proceeds as follows: Section 2 describes a simple theoretical model which clarifies the relationship between child health, fertility, adult labor supply and education. In Section 3, we provide background on malaria risk and control strategies in Sub-Saharan Africa. We also present our outcomes of interest. We outline our empirical strategy in Section 4. Section 5 displays our results, robustness checks and discussion. Finally, Section 6 summarizes our conclusions and highlights avenues for future research.

# 2 Theoretical framework

Major malaria control efforts like RBM target children under five and pregnant women (WHO, 2015). This is because acquired immunity, even in highly endemic areas, does not play an efficient protective role until the age of five. RBM, if effective, should therefore decrease younger children's mortality and morbidity. Existing micro-level evidence suggests that this is indeed the case: Bhattarai et al. (2007) show that RBM-sponsored interventions allowed for such a decrease in Zanzibar. These interventions also led to a significant drop (33%) in postneonatal mortality (death in the first 1-11 months of life) in malarious regions of Kenya (Pathania, 2014).

Such improvements to child survival and health alter the costs of raising children. They may thus affect household decisions to have children, to participate in the labor force, and to invest in offspring's human capital. We develop a simple, unified framework to illustrate the interplay between fertility, adult labor supply and educational choices. We then examine comparative statics when infant survival and early childhood health improve. Section 1 of the Supplemental Appendix describes this model in full. In what follows, we summarize its key predictions.

We model a unitary household of one adult and her potential surviving offspring. The household cares about its own consumption and leisure, as well as about the number and human capital of its children, since human capital in childhood is an important determinant of future earnings (Becker, 1975; Currie and Madrian, 1999; Currie, 2009; Hong, 2013). The human capital of a surviving child depends on his/her education and health. A lower (resp. higher) elasticity of substitution between these two inputs means that they have greater complementarity (resp. substitutability).

Solving the model supports a well-documented quality-quantity trade-off (Becker and Lewis, 1973; Rosenzweig and Zhang, 2009; Bleakley and Lange, 2009). An increase in child health raises a child's wage rate. Consistent with Bleakley (2010), this increases the opportunity cost of additional education. A parent must allocate her time between working and raising children. If each additional child costs less, a parent may reduce her own labor supply and increase her preferred number of children because a lower labor supply allows the parent to raise more children. Concomitantly, parents should invest less in schooling if

education and health are substitutes.

But the relationship between health and education is complex, not only within an individual's lifecycle but also through intergenerational dynamics (Vogl, 2014). Providing a child with one more unit of education should in fact generate a bigger increase in human capital when this child is healthy (Hazan and Zoabi, 2006). In our context, we expect the complementarity between health and education to be high, since reducing malaria can also improve learning through biological means. First, contracting malaria during pregnancy may cause foetal growth retardation which produces physical and cognitive impairments in children (Barreca, 2010). Second, complicated forms of malaria often develop rapidly during early childhood. Numerous studies quantify the detrimental effects of severe malaria, better known as cerebral malaria, on children's physical and cognitive abilities (see Mung'ala-Odera, Snow and Newton (2004) for a literature review). Even during late childhood, the protection conferred by acquired immunity is only partial. Clinical as well as asymptomatic malaria hampers educational achievement notably via school absenteeism and cognitive deficiencies (Clarke et al., 2008; Thuilliez et al., 2010; Nankabirwa et al., 2013).

If better health improves the returns to education, parents may invest more in schooling. This outcome can occur if the complementarity between education and health is sufficiently high. The cost of each additional child increases, and a parent's labor supply increases as her preferred number of births decreases.

These relationships illustrate important lesson — a decline in malaria can generate a wide range of outcomes, many of them potentially positive. Provided that the complementarity between health and education is strong enough, a drop in malaria risk does not only improve child survival rates and health. It also affects fertility, adult labor supply and educational investments in a way that is conducive to human capital accumulation. The effect of RBM on each of these outcomes is thus an empirical question, one that we address in the remainder of the paper.

# 3 Background and sample

In this section, we provide some background to our empirical strategy. As described in the introduction, we estimate the effect of RBM on human capital outcomes based on variation

in assignment to treatment at the cluster level and variation in intensity of treatment at the country-cohort-survey year level. We first present our measure of malaria risk and the evolution of malaria risk over time in our sample, paying careful attention to the change in malaria risk in areas with the highest burdens of malaria prior to RBM. We then briefly describe past and present malaria control efforts in Sub-Saharan Africa. Finally, we outline our main outcomes of interest. Further details on the construction of all variables are available in Section 2 of the Supplemental Appendix.

We select our sample countries from those which were surveyed at least once post-campaign, which received RBM disbursements, and which include geocoded clusters and all of our outcome variables.[5] Respondents are randomly selected within clusters, and clusters are randomly distributed across surveys, cohorts, years, and countries.

## 3.1 Malaria risk

Campaigns targeted areas with the greatest initial burden of malaria. Though information is not available about the specific treatment received by each cluster, we assume that campaigns targeted clusters with the highest pre-campaign malaria risk.

Our proxy for malaria risk is the *P. falciparum* parasite rate (PfPR) from the Malaria Atlas Project (MAP)[6] (Bhatt et al., 2015). For a given year, PfPR describes the estimated proportion of individuals in the general population aged 2 to 10 years old who are infected with *P. falciparum* at any given time. These estimates are generated by a geostatistical model that relies on parasite rate surveys as well as bioclimatic and environmental characteristics.[7] We also complement our results with treatment probabilities based on the coverage of specific malaria control strategies in our robustness checks.

We use MAP estimations of malaria risk in 2000 for grids of 1 km $\times$1 km over the African continent, assigning a pre-campaign malaria risk measure to each geocoded DHS cluster. This procedure allows us to cover 25,827 DHS clusters scattered over 27 Sub-Saharan African

[5]The absence of one or more of these characteristics prevents us from including additional countries, particularly those in less malarious regions which might serve as additional controls. In previous versions of this paper, we relied on countries with at least one pre-campaign round and one post-campaign round. Our current estimates are still robust using this sub-sample. We provide these results in the Supplementary Appendix.

[6]See http://www.map.ox.ac.uk/. We sincerely thank Peter Gething for providing the yearly data (from 2000 to 2012) through personal communication for an earlier version of this paper.

[7]Gething et al. (2011) and Bhatt et al. (2015) describe the estimation process.

countries. Table A1 reports descriptive statistics for PfPR in 2000. Figure 2 provides the spatial distribution of these DHS clusters and the level of malaria risk in 2000. All four Sub-Saharan African sub-regions, as defined by the United Nations geoscheme, are represented: Central Africa (Cameroon, DRC and Gabon), Eastern Africa (Burundi, Ethiopia, Kenya, Madagascar, Malawi, Mozambique, Rwanda, Tanzania, Uganda, Zambia and Zimbabwe), Southern Africa (Namibia and Swaziland) and Western Africa (Benin, Burkina Faso, Côte d'Ivoire, Ghana, Guinea, Liberia, Mali, Nigeria, Senegal, Sierre Leone and Togo).

We run several checks of the evolution of malaria risk over the 2000-2014 period for the 27 countries in our sample. First, we show that malaria risk declined and the application of control strategies increased, particularly from 2003 when the majority of RBM campaigns launched. Figure 3a shows a precipitous decrease in mean malaria risk, particularly from 2003 when the majority of RBM campaigns launched. Similarly, in Figures 3b to 3d, we examine the evolution of standard malaria control strategies, all of which increased over this time period.

Second, we show that these trends were strongest in areas with comparatively higher malaria risk prior to the scale up of anti-malaria campaigns. We create a panel of the 244 regions in our sample. To show the clear contrast between the pre-RBM period (2000-2002) and the post-RBM period (2003-2005), we plot the change in PfPR against the mean initial value of PfPR in 2000. Consistent with Bhatt et al. (2015), Figures 4a and 4b show that initial PfPR and the change in malaria risk are not correlated prior to 2002, while they are negatively and significantly correlated after 2003. Conditioning the use of malaria control techniques on initial malaria risk produces a similar result. The higher the level of malaria risk in 2000, the greater the increase in insecticide treated net usage in Figures 5a and 5b (which use two different bednet measures from the DHS and MAP, respectively). In Figures 5c and 5d, we examine the trade-off between drugs administered for fever to children under five. Due to its lower effectiveness, chloroquine waned in popularity as a first-line treatment, and the prescription of ACTs increased instead (Flegg et al., 2013).[8] Though our panel of countries with drug information is more limited, we see that, consistent with this substitution, the popularity of chloroquine decreased in the most malarious regions

[8]Malawi was the first African country to replace chloroquine in 1993, followed by Kenya in 1998 and Tanzania in 2000 (see Mohammed et al. (2013)).

while the use of artemisinin combination therapies grew weakly.

Taken together, these plots provide suggestive evidence that treatment probability, measured by PfPR or by control strategies, depends on an area's initial burden of malaria. We will refer to PfPR as malaria risk for the remainder of the paper.

## 3.2 Malaria control efforts in Sub-Saharan Africa

The WHO launched the first worldwide malaria eradication program in 1955. Malaria reduction strategies revolved primarily around vector control (surveillance and spraying) and antimalarial drug treatments. However, many of the most malarious areas, such as the newly-independent states of Sub-Saharan Africa, did not see any benefits (Alilio, Bygbjerg and Breman, 2004). As described in 2002 by the final report of the External Evaluation of Roll Back Malaria:

> *"Prior to RBM's launch, a series of unsuccessful initiatives to curb the growing burden of malaria contributed to a sense of skepticism and disillusionment among international health experts. The WHO Malaria Eradication Programme (1955-69) resulted in widespread disappointment and failure, after 15 years of a coordinated, multinational effort. On a more modest national scale, the WHO-sponsored vector control projects in Cameroon, Nigeria and elsewhere in Africa in the 1960s were also largely ineffective. During the 1980s and 90s, especially in Africa, malaria control programmes fell into disrepair or were abandoned entirely. Problems were compounded by growing resistance to insecticides and drugs, general weaknesses in the health care infrastructure, and economic shocks that reduced government spending per capita on health care. The malaria situation worsened, and fatalism and resignation towards the disease became widespread."*

The RBM Partnership formed in reaction to the deteriorating state of malaria control efforts. RBM's first major disbursements occurred in 2003, driven by the Global Fund to Fight AIDS, Tuberculosis and Malaria following its establishment in 2002. There is a general consensus that RBM-sponsored efforts have been achieving a measure of success. As the WHO expert group Malaria Policy Advisory Committee notes:

*"The scale-up of malaria control efforts in recent years, coupled with major investments in malaria research, has produced impressive public health impact in a number of countries, and has led to the development of new tools and strategies aimed at further consolidating malaria control goals."*

Sub-Saharan Africa, home to the heaviest burden of malaria, saw malaria cases decrease by 42%, with death rates dropping by 66%, between 2000 and 2015. Bhatt et al. (2015) estimate that malaria control interventions have averted 663 million clinical cases since 2000, of which 68%, 22% and 10% are attributable to insecticide treated nets, artemisinin combination therapies, and indoor residual spraying, respectively.[9]

We present the increasing trend of RBM disbursements in our sample from 2000 to 2014 in Figure 6. To do so, we use disbursements from the three primary external funders of the RBM campaigns: the Global Fund (since 2003), the President's Malaria Initiative (since 2006), and the World Bank Booster Program for Malaria Control in Africa (since 2006). We observe disbursements at the level of the country-year. We use this information to compute a respondent's exposure as the yearly amount per capita[10] disbursed at the country level during an individual's lifetime by these three primary funders. An individual's lifetime is defined as the difference between the DHS survey year and his or her year of birth, from which we subtract one year. We consider exposure to begin in utero (though defining the start of exposure with the year after birth does not alter our results). (See the Supplemental Appendix for further details.)

An individual's exposure depends on his or her date of birth which is difficult to predict. Furthermore, we use in many cases multiple surveys per country, the timing of which is also difficult to systematically anticipate with respect to high-level DHS, organizational, and national priorities. This produces an exposure to treatment which varies from $-0.162$[11] to 8.918 with a standard deviation of 0.787. Table A1 presents these descriptive statistics, and Table A2 presents descriptive statistics for exposure separately for age, date of birth, country and survey year.

[9]The authors note that *"these proportional contributions do not necessarily reflect the comparative effectiveness of different intervention strategies but, rather, are driven primarily by how early and at what scale the different interventions were deployed."*

[10]Yearly population data come from the World Development Indicators.

[11]Negative values are possible in a small number of cases of young children when a country was required to return disbursed funds.

It is important that the timing and intensity of the RBM disbursements were not anticipated by the target population. If households anticipated better child health outcomes, for example, they could have modified decisions on fertility, labor supply or educational investments prior to the campaign's start. But the likelihood that the average citizen would have predicted the scale-up of RBM campaigns is low. The establishment of the Global Fund in 2002 marked RBM scale-up. The Global Fund itself evolved out of a series of high-level discussions between donors and multilateral agencies that began toward the end of 1999. These discussions notably culminated with the sixth of the eight Millennium Development Goals established following the Millennium Summit of the United Nations in 2000: "To combat HIV/AIDS, malaria, and other diseases." Moreover, it was only in 2011 that the Global Fund began to advertise its activities in countries of operation.[12] It is thus doubtful that the establishment of this Global Fund and its subsequent disbursements were anticipated by the general population of beneficiary countries.

## 3.3 Outcomes of interest

The DHS provide our outcomes of interest, and we report descriptive statistics in Table A1 of the Supplemental Appendix. Following Pathania (2014), we use infant mortality (death within the first year of life among live births) as a proxy for child survival rates and health. We construct this variable based on the questionnaire conducted among women of reproductive age (15-49) which includes complete reproductive history and childhood mortality. More precisely, we define infant mortality only for cohorts born at least one year before the survey date since it is undefined for cohorts younger than one year. Moreover, in order to avoid recall bias, we restrict the sample to live births that took place at most 5 years before the date of interview. We complement infant mortality with two additional indicators: neonatal mortality and postnatal mortality which represent the probability of death within the first month and within months 1-11 respectively.

We note that evidence is broadly supportive of decreases in infant mortality over time. Figure 6 tracks the risk of mortality from malaria. From 1980 to the early 2000s, Figure 6 shows a steady increase in the cumulative probability of dying from malaria among children

[12] A green leaf logo is printed on Global Fund-provided malaria treatments from the Affordable Medicines Facility-malaria program to highlight negotiated price reductions from artemisinin combination therapy manufacturers.

under five.[13] But Figure 6 also depicts a turning point in mortality risk, one that occurs in the early 2000s. This drop is consistent with the scale-up of global malaria control efforts. While suggestive, this estimate of mortality risk faces its own limitations, and it is therefore important to investigate this trend with a method using more precise household data.

To measure fertility, we rely on one question from the women's questionnaire: the number of children ever born. We also use two questions from the women's and men's questionnaires to proxy for adult labor supply: (i) whether the respondent has been employed in the last 12 months (self-employment included) and, if so, (ii) whether he or she was paid in cash. We use the latter information as a proxy for the probability of being involved in a market-oriented rather than subsistence labor.

Finally, for all individuals in the person-level recode, we compute education in single years. We also use this variable to identify whether the respondent has completed at least the full number of years of primary education (5, 6 or 7) in her country's educational system.

We note that DHS surveys contain child labor modules in 15 of our 27 countries (Benin, Burkina Faso, Burundi, Côte d'Ivoire, DRC, Cameroon, Gabon, Guinea, Mali, Malawi, Rwanda, Sierra Leone, Senegal, Togo, and Uganda). From these surveys, we rely on the number of hours worked by a child (ages 5 to 14) over the previous week in order to estimate how RBM affects child labor. While results (available upon request) are consistent with our framework (i.e. RBM campaigns reduce child labor), we view them as exploratory given that we cannot rely on our full sample.

# 4 Empirical strategy

## 4.1 Baseline specification

We aim to isolate the treatment effect by comparing the outcomes of individuals with characteristics that command (or would command) high and low treatment intensities in the treated and untreated groups. Without a standard panel structure, we adapt a difference-in-differences approach to our context.

[13] This measure refers to the total number of children under five out of 1,000 who are likely to die from malaria in the absence of all other causes from the Institute for Health Metrics and Evaluation. Murray et al. (2012) describe the estimation process which relies on all available data on sub-national malaria mortality.

Our baseline specification regresses a respondent's outcome on an interaction term between the probability of belonging to the treated group and the treatment intensity. The former assigns a measure of malaria risk to individuals at a localized geographic level (the DHS cluster). The latter exploits, conditional on treatment, variation in the timing and amount of RBM disbursements relative to respondents' birth cohorts and DHS survey years. Interacting these variables allows us to identify a causal pathway from RBM campaigns to human capital outcomes. More precisely, we fit the following econometric model:

$$\begin{aligned} &\mathrm{y}_{ijct} = \alpha + \beta.(\mathrm{malaria}_{2000j} \times \mathrm{exposure}_{Nct}) + \mathbf{X_{ijct}}'.\mathbf{\Gamma} \\ &+\delta_{Nct} + \delta_{Nc} + \delta_{ct} + \delta_{Nt} \\ &+\delta_j + \delta_c + \delta_t + \epsilon_{ijct} \end{aligned} \tag{1}$$

where $y_{ijct}$ is an outcome of individual $i$ in DHS cluster $j$, who belongs to cohort $c$ (the group of individuals born in year $c$) and is surveyed in year $t$.[14]

As the coefficient of the interaction term between $\mathrm{malaria}_{2000j}$ and $\mathrm{exposure}_{Nct}$, $\beta$ identifies the treatment effect. The variable $\mathrm{malaria}_{2000j}$ measures the level of the PfPR in 2000 in DHS cluster $j$, hence its pre-campaign malaria risk. We use this pre-campaign malaria risk to provide a continuous probability (from 0 to 1) of a DHS cluster's treatment by RBM campaigns. While effective at distinguishing the treated from untreated, cluster-level assignment to treatment masks substantial variation in exposure to treatment among those treated.

For this reason, we interact $\mathrm{malaria}_{2000j}$ with $\mathrm{exposure}_{Nct}$. The latter captures individual exposure to RBM and, conditional on treatment, treatment intensity. It measures in a given country $N$, the yearly amount per capita disbursed during an individual's lifetime by the three primary external funders of the RBM campaigns. As a function of country $N$, year of birth cohort $c$, and DHS survey year $t$, $\mathrm{exposure}_{Nct}$ is defined by substantial variation but also randomness to the extent that births, DHS surveys, and campaign start dates are difficult to predict.

To control for each element of the interaction term and its correlates, we introduce DHS cluster fixed effects $\delta_j$ as well as country-by-cohort-by-DHS year fixed effects, $\delta_{Nct}$, and their

[14]To focus on a post-colonization time frame, and therefore avoid concurrent shocks to health and educational policies, we restrict respondents to those born after 1960. However, we show in Table 6 that our results hold when this restriction is lifted.

subcomponents: $\delta_{Nc}$, $\delta_{ct}$, $\delta_{Nt}$, $\delta_N$ (these country fixed effects drop due to the concomitant control for DHS cluster fixed effects), $\delta_c$ and $\delta_t$. Finally, the vector $\mathbf{X_{ijct}}$ includes individual covariates gender and wealth (age is already captured by $\delta_{ct}$).

Adopting this restrictive parameterization isolates a treatment effect based on assignment to treatment at the cluster level and considerable variation in treatment intensity by a respondent's country, cohort and survey year. We further amend this empirical strategy with several terms to combat the potential for bias due to omitted variables.

## 4.2 Potential threats to validity

### 4.2.1 Straightforward omitted variables bias

By definition, an individual's exposure to RBM campaigns depends negatively on age (i.e. the difference between DHS survey year and the respondent's date of birth). As a consequence, a negative correlation exists between ($\text{malaria}_{2000j} \times \text{exposure}_{Nct}$) and ($\text{malaria}_{2000j} \times \text{age}_{ct}$). Yet, because the effect of pre-campaign malaria risk is likely to vary by age, ($\text{malaria}_{2000j} \times \text{age}_{ct}$) could be correlated to our outcome variables.[15]

Pre-campaign malaria risk may also be correlated to pre-campaign outcomes. For instance, there is surely a correlation between ($\text{malaria}_{2000j} \times \text{exposure}_{Nct}$) and the interaction term between pre-campaign educational outcomes at the cluster level and $\text{exposure}_{Nct}$. But initially more educated individuals are more likely to adopt malaria prevention strategies (see Nganda et al. (2004); Rhee et al. (2005); Hwang et al. (2010); Graves et al. (2011)).[16] Therefore, the impact of exposure to malaria control campaigns may vary depending on pre-campaign educational outcomes.

To mitigate these potential omitted variables biases, our tables display three columns of results per dependent variable. They report coefficient $\beta$ (i) when Equation (1) is estimated; (ii) when the interactions between $\text{age}_{ct}$ and region fixed effects are included;[17] (iii) and when, additionally, the interactions between $\text{exposure}_{Nct}$ and region fixed effects are added.[18]

[15] Younger cohorts are more heavily treated than older cohorts in each treated cluster. If these cohorts have positive spillovers on older cohorts (by reducing malaria risk), we will underestimate the effects of RBM campaigns on the outcomes of older cohorts.

[16] See also Kenkel (1991) and Dupas (2011) for the relationship between education and health behavior.

[17] We rely on region rather than cluster fixed effects to avoid multicollinearity. However, our results remain substantively unchanged if we rely on the interactions between $\text{age}_{ct}$ and cluster fixed effects.

[18] We obviously cannot control for the interaction term between $\text{exposure}_{Nct}$ and cluster fixed effects since

### 4.2.2 Pre-campaign catch-up effects

Before proceeding to results, we first rule out the possibility that changes in our outcome variables between more and less exposed individuals began prior to RBM scale-up. Otherwise, we will be unable to ascertain if $\beta$ in Equation (1) captures the impact of the RBM campaigns or if it simply reflects pre-campaign trends.

To test for pre-campaign catch-up effects, we perform a falsification test. We estimate Equation (1) over individuals who were exposed to the campaign but whose outcomes could not be affected by the campaign. We examine three outcomes: (i) Height-For-Age z-scores based on WHO reference standard, a proxy for health conditions during childhood; (ii) the number of years of education completed; (iii) whether the respondent completed primary school. Relying on Equation (1), we study the Height-For-Age z-scores among individuals who had completed their growth at the campaign's start date (above age 20) and the educational outcomes for those who had completed their education by the campaign's start date (above age 24).

The results are reported in Table 1. The coefficient $\beta$ is never positive. In other words, prior to the RBM campaigns, the difference in health and educational outcomes between more and less exposed individuals is not greater in treated relative to untreated areas. It is, in fact, lower (and often statistically significant). If anything, the pattern observed during the pre-campaign period runs against us finding a positive impact of the RBM campaign on human capital accumulation.

# 5 Results

Regressing our outcomes on the variation[19] in malaria prevalence and incidence as well as coverage by standard control strategies yields Table A3 in the Supplemental Appendix. Except for the probability of being involved in a market-oriented rather than subsistence work, a clear pattern emerges: variations in malaria prevalence and incidence are positively (resp. negatively) correlated with infant mortality and fertility (resp. adult labor supply and education), while the reverse is true for the variations in coverage by malaria control

this would drop the main variable of interest in our analysis, i.e. $(\text{malaria}_{2000j} \times \text{exposure}_{Nct})$.

[19] With the exception of the variation in artemisinin combination therapies which is provided by MAP at the country level, these changes are measured at the regional level.

methods. Put differently, the results are consistent with an impact of the RBM campaign that is conducive to human capital accumulation.

Table 2 and Figure 7 support this interpretation. Table 2 reports the results of a difference in means analysis. It shows that the decrease (resp. increase) in infant mortality and fertility (resp. adult labor supply and education) between the post- and pre-campaign periods is greater in DHS clusters that show high rather than low pre-campaign malaria risk. Figure 7 displays, for each cohort, the standardized coefficient of pre-campaign malaria risk on infant mortality (Figure 7a), the total number of births (Figure 7b), the probability of being employed (Figure 7c) and the number of years of education completed (Figure 7d).[20] For relevant age ranges, the detrimental effects of pre-campaign malaria risk appear to decrease with exposure to RBM campaigns: younger cohorts have better outcomes compared to older cohorts. We further investigate these preliminary findings by estimating Equation (1) in the following section.

## 5.1 Infant mortality

Columns 1 through 6 of Table 3 report the OLS estimates of Equation (1) for infant mortality, without (odd columns) and with (even columns) exposure-by-region fixed effects.[21] Our results are also robust to using a simpler specification with a dichotomous version of the continuous $\text{exposure}_{Nct}$, which is equal to 1 if $\text{exposure}_{Nct}$ is strictly positive and to 0 otherwise.[22] Results are available upon request.

Irrespective of whether we rely on a binary or continuous measure for exposure, a marginal increase in the interaction term reduces the probability of infant mortality by between 14-18 percentage points. The coefficients of neonatal and postnatal mortality are roughly the same in magnitude, suggesting that the definition of mortality matters little in our specification. We further interpret these results in Section 5.4.

[20] The coefficient of pre-campaign malaria risk is standardized to allow for comparison across cohorts and outcomes. Controls for gender, age and wealth as well as DHS year fixed effects are included.

[21] We do not control for age-by-region fixed effects since the age range is small (from 0 to 4).

[22] This dichotomization is possible only with infant mortality. For the other variables, it would require that we distinguish between individuals surveyed before and after the campaign's start. Yet, contrary to the respondent's date of birth, the DHS survey year almost never varies within DHS clusters since very few DHS clusters were surveyed twice. In other words, relying on a binary variable to capture exposure to the RBM campaign would prevent us from controlling for DHS cluster fixed effects.

## 5.2 Fertility, adult labor supply and education

The remainder of Table 3 reports the OLS estimates of Equation (1) for fertility, adult labor supply and education. We introduce age-by-region fixed effects and exposure-by-region fixed effects sequentially. Table 3 confirms the preliminary results from Table 2 and Figure 7: the RBM campaign reduces total fertility and increases adult labor force participation as well as the probability of being involved in market-oriented activities. More precisely, holding malaria risk in 2000 constant, an incremental increase in RBM spending reduces the total number of live births by nearly 4, and increases the probability of being employed and of being paid in cash by roughly 50 and 18 percentage points respectively. Exposure to RBM also improves educational outcomes, increasing both the probability of completing primary school and the actual years of education completed. These results are consistent with a model of household production in which health and education operate as complementary inputs. They are furthermore robust to non-linear transformations of RBM exposure and to analysis by gender. We interpret the magnitude of the result on educational attainment further results in Section 5.4.

## 5.3 Robustness

### 5.3.1 Concurrent public policies

During the early 2000s, the Millennium Development Goals led many governments to draft sweeping anti-poverty plans. Government expenditures on social services increased. If these increases correlate closely to RBM disbursements, we risk that our results pick up the effects of increases in public expenditure, leading us to overestimate the purported effects of RBM campaigns. We obtain expenditure on public education as a percentage of GDP from the World Bank EdStats, Education Statistics: Core Indicators. Health and military expenditure come from the World Development Indicators. To compute total public expenditure per capita during a respondent's lifetime in each of these categories, we rely on GDP in current USD and total population (both from the World Development Indicators). In this way, exposure to public expenditure mirrors our primary measure of exposure to the RBM campaign.

In Table 4, we control for exposure to concurrent government expenditure on education,

health, and military interacted with $\text{malaria}_{2000j}$ for all outcome variables. We then add all expenditures simultaneously. For all outcomes other than infant mortality, we also control for the percentage of a respondent's life elapsed since the start of Free Primary Education. Our results hold, though the magnitudes decrease slightly, as we net out the spillovers of government policies.

### 5.3.2 Alternative treatment probabilities

While our estimated measure of pre-RBM malaria risk is both spatially and temporally precise, it is still an estimate. And, as an estimate, it is only as strong as the information on which it is based. In Tables 5a and 5b, we subject our results to modifications of our treatment probability (the level of malaria risk in 2000) in case of measurement error. Specifically, we exploit variation in malaria risk, artemisinin combination therapies, and insecticide treated nets between surveys. We focus our attention on these two curative and preventative measures as indoor residual spraying is typically limited to specific geographic areas.[23] We substitute each of these measures for the fixed value of pre-campaign malaria risk in our baseline estimation. However, as these changes can be endogenous, we instrument each of them by malaria risk in 2000.

Consistent with our baseline findings, our results show that the interaction term positively affects infant mortality and fertility, while it negatively affects labor and educational variables. On the contrary, when using variation in artemisinin combination therapies and bednets, the interaction term negatively affects infant mortality, fertility, and positively labor and educational variables. The effect of artemisinin combination therapies appears to be globally higher on our outcomes but consistent with our results overall.

### 5.3.3 Additional checks

In Table 6, we place various restrictions on our sample population. First, to account for the possibility of migration, we restrict our sample to individuals whose head of household had lived in the same DHS cluster for at least ten years. This information is available only for older DHS surveys which drastically reduces our sample size. Nevertheless, we observe

[23] Artemisinin combination therapies vary at the national level, in which case regional covariates are removed from corresponding estimations. Further description of these variables can be found in the Supplemental Appendix.

that results hold for the majority of dependent variables, though infant mortality outcomes remain negative but not significant. The RBM effect on the probability of being engaged in wage employment also tends to zero. We then lift the restriction requiring all respondents to have been born post-1960. Finally, we impose a restriction that all individuals must be above the age of five. Censoring the sample makes little difference in the statistical significance or the magnitude of the coefficients. In a slightly different sub-sample test (results for a sub-set of dependent variables reported in Table A6), we test the robustness of our baseline results by dropping potential outlying countries from the sample. These countries include those with large populations (DRC, Ethiopia, Kenya, Nigeria, Tanzania) as well as those which experienced major conflict during the 2000 to 2014 time frame (Cote d'Ivoire, Liberia, Sierra Leone). We also restrict our attention to countries with at least two DHS survey rounds available. Our results are not driven by any country or subset of countries.[24]

Finally, we run two different types of falsification tests in Tables A4 and A5 of the Supplemental Appendix. For infants, we exchange mortality for two health outcomes unrelated to malaria: acute respiratory infections and diarrhea. Estimating Equation (1) with these new dependent variables shows no effect of RBM exposure. Replicating this approach for adults is not feasible. Instead, we create an artificial RBM intervention by shifting the start date of disbursements to the left, first by 20 years and then by 30 years. In other words, we artificially expose a different subset of individuals to RBM disbursements by pretending that campaigns began in 1983 or 1973. In all cases, we observe a negative or not significant relationship between RBM disbursements and our outcome variables. The effect of the RBM disbursements is, in other words, isolated to the post-2000 time frame.

## 5.4 Cost-effectiveness

Evaluating the cost-effectiveness of large-scale interventions is challenging. But it is an important exercise, especially considering the number of campaigns launched against preventable diseases. To the best of our knowledge, RBM rigorously evaluated five insecticide treated net programs (Eritrea, Malawi, Tanzania, Togo, Senegal) and two indoor residual spraying programs (KwaZulu-Natal, Mozambique). The cost per death averted by bednet programs ranged between $431-960. At $3,933-4,357, this figure is even higher for spraying

[24]Complementary analysis to Table A6 is available upon request.

programs.[25] By international standards, these costs are high. However, such a high cost-effectiveness is not surprising given that the proportion of deaths due to malaria represents only small part of the overall disease burden. For example, Bryce et al. (2005) find that 23 interventions aimed at eliminating 90% of global childhood deaths cost an average of $887 per child life saved. BenYishay and Kranker (2015) estimate that countrywide measles vaccination campaigns cost only $109 per child life saved.

The total cost of RBM campaigns in our 27 countries over our entire time period (proxied by GFATM, PMI and WB disbursements) is $8.17 billion or roughly $690 million per year. Given that the average population in our sample countries over this time period is just under 700 million per year, disbursements amount to approximately $1 per capita per year. With this disbursement rate, we use a back-of-the-envelope calculation to arrive at a cost-effectiveness estimate for infant mortality. We compute the benefit as the difference between the number of deaths of treated infants (those born during or after 2003) and untreated infants (those born prior to 2003). Our estimates in Table 3 show that, holding malaria risk in 2000 constant, an incremental increase in RBM spending reduces infant mortality by 18 percentage points. Multiplying this coefficient by the average level of malaria risk in 2000 (0.351 in Table A1) yields a treatment effect of 6.3 percentage points. We apply this treatment effect to averaged live birth and infant mortality rates in 2000[26] to obtain a cost of approximately $4,600 per additional life saved. This figure is not at odds with existing RBM estimates.

Computing the cost-effectiveness of educational outcomes requires more detailed assumptions about population distributions. We compute for all treated cohorts (individuals born in 1979 or later[27]) a treatment effect based on each cohort's time exposed to RBM campaigns and the estimated coefficient of years of education from Table 3 (2.4). We use a rough estimate of the population aged 0 to 24 averaged over 2003 to 2014 to compute the total years of education resulting from RBM per year. This leads us to a cost of $1.91 per each additional year of schooling. Restricting the population to ages 0 to 14 raises this estimate to $3.50.

Kremer and Holla (2009) review the cost-effectiveness of a wide range of targeted edu-

[25] See http://www.rollbackmalaria.org/files/files/partnership/wg/wg_itn/docs/rbmwin4ppt/3-8.pdf

[26] Data come from the United Nations Population Division World Population Prospects

[27] Consistent with DHS data, we assume that the maximum age possible for primary enrollment is 24.

cational interventions. Large-scale health campaigns are certainly less efficient compared to carefully controlled experiments. For instance, Miguel and Kremer (2004) found that each additional year of schooling attributable to mass school-based deworming treatments cost approximately $3.50. Even so, we use these interventions as a rough benchmark. RBM's educational cost effectiveness is low in absolute terms, and it is low relative to other health interventions aimed at improving education.

# 6 Conclusion

We document the effects of the RBM malaria control campaigns on human capital outcomes in Sub-Saharan Africa using microeconomic data from 27 countries. Consistent with other geographically-specific studies analyzing the effects of large-scale health interventions and policies, we find a positive impact of campaigns on human capital (Jayachandran and Lleras-Muney, 2009; Bleakley, 2010; Cutler et al., 2010; Lucas, 2010; Venkataramani, 2012). We show that exposure to RBM improves infant survival, reduces fertility, and improves adult labor force participation and children's educational attainment.

Our findings highlight the importance of considering other outcomes in addition to health when investing in large-scale health interventions. Furthermore, they fit to our theoretical framework which allows increases in both early childhood health and education if health and education are sufficiently complementary. Mass interventions can help to break intergenerational health-based poverty traps in which poor early childhood health impedes school participation and performance, lowers labor participation and earnings, and increases the need for health care. Sub-Saharan Africa is not only the last region to initiate the fertility transition, but it has also experienced a weaker rate of decline in fertility relative to other regions. Population growth due to lower mortality and sustained high birth rates threatens the well-being of individuals and communities across Sub-Saharan Africa.

Our study shows that health is a key piece of this puzzle and that large-scale public health programs have the potential to play a role in the transition to a modern demographic regime. Documenting the additional effects of such interventions is not a trivial exercise given the difficulty in estimating the medium-term effectiveness of programs aiming to *reduce* but not eliminate health challenges (Miguel and Kremer, 2004; Ashraf, Fink and Weil, 2014).

Certainly the educational benefits from malaria interventions will never be large enough to compete with the health benefits (Jamison et al., 2013), but they may be able to compete with or complement standard educational programs.

Our results do face some limitations. While we provide evidence that our effects may be persistent, a more general analysis of the long-run, general equilibrium impacts induced by RBM is left for further investigation. For example, population increases thanks to health interventions may put pressure on social service provision. Similarly, how the labor market reacts to rightward shifts in human capital has important implications for economic productivity and growth. Therefore, observing the net effect of the RBM on GDP per capita will take time to come to fruition, and our understanding is limited to a transitory phase. It is also important to note that we study a vertical health intervention that may have secondary effects on health care provision itself. Because we are not able to distinguish the extent to which RBM influences service delivery, we contribute instead to the body of evidence on how improving health outcomes may have significant economic returns.

Nonetheless, we believe our analysis can inform the debate on the effect of large-scale health programs in developing countries. Some question if policy-makers can promote education and economic development via public healthcare interventions (see Acemoglu and Johnson (2007, 2014) and Bloom, Canning and Fink (2014) for a discussion). We provide evidence that, at least in the case of malaria control efforts, the resulting improvements in human capital must not be overlooked.

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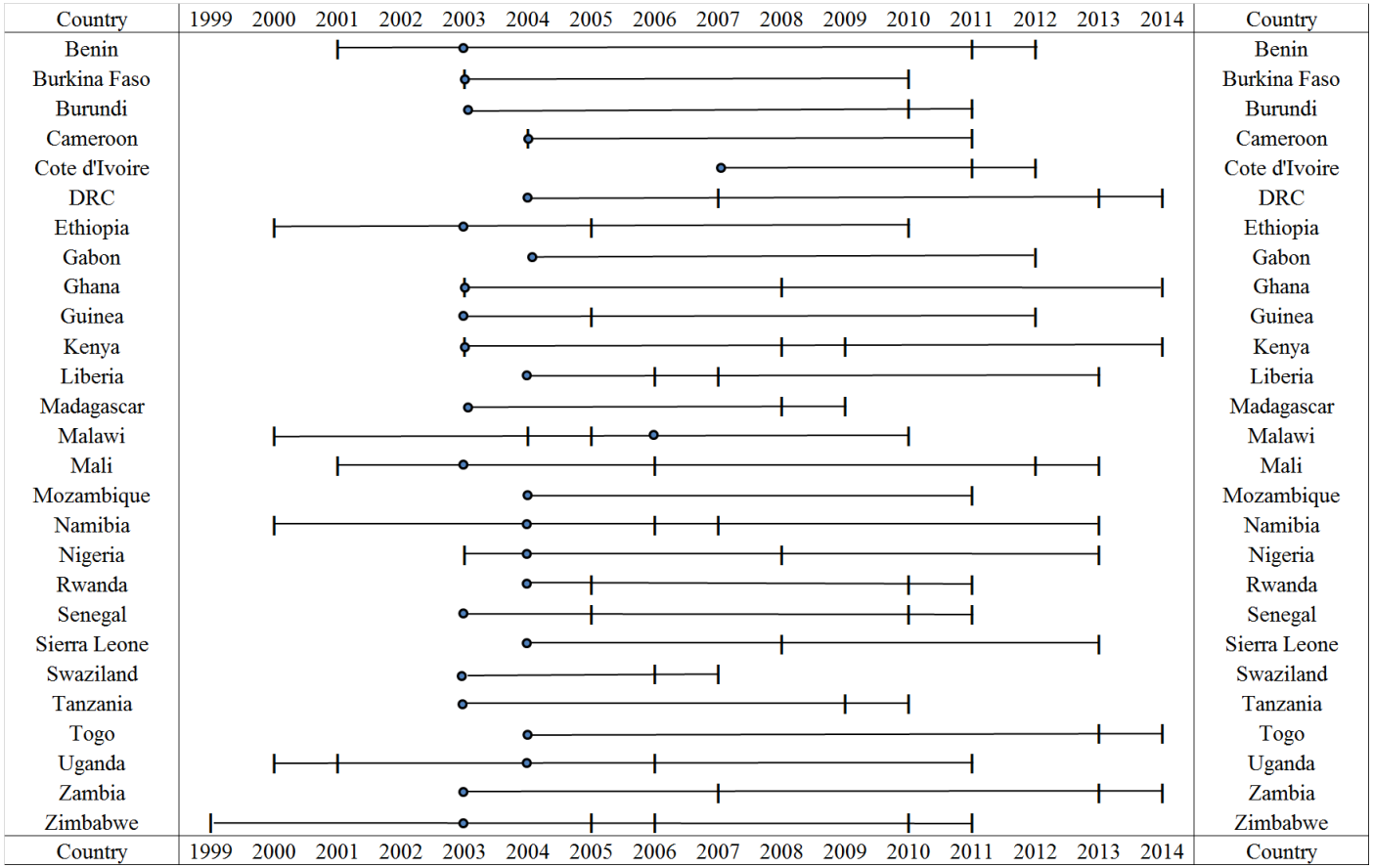
**Figure 1:** Demographic and Health survey years and Roll Back Malaria start dates in our 27 countries




*Notes:* Vertical bars show the Demographic and Health survey years available for each country, and points mark the start of Roll Back Malaria disbursements.

**Figure 2:** Spatial distribution of DHS clusters and initial malaria risk (*Plasmodium falciparum* parasite rate) from Bhatt et al. (2015) in our 27 countries

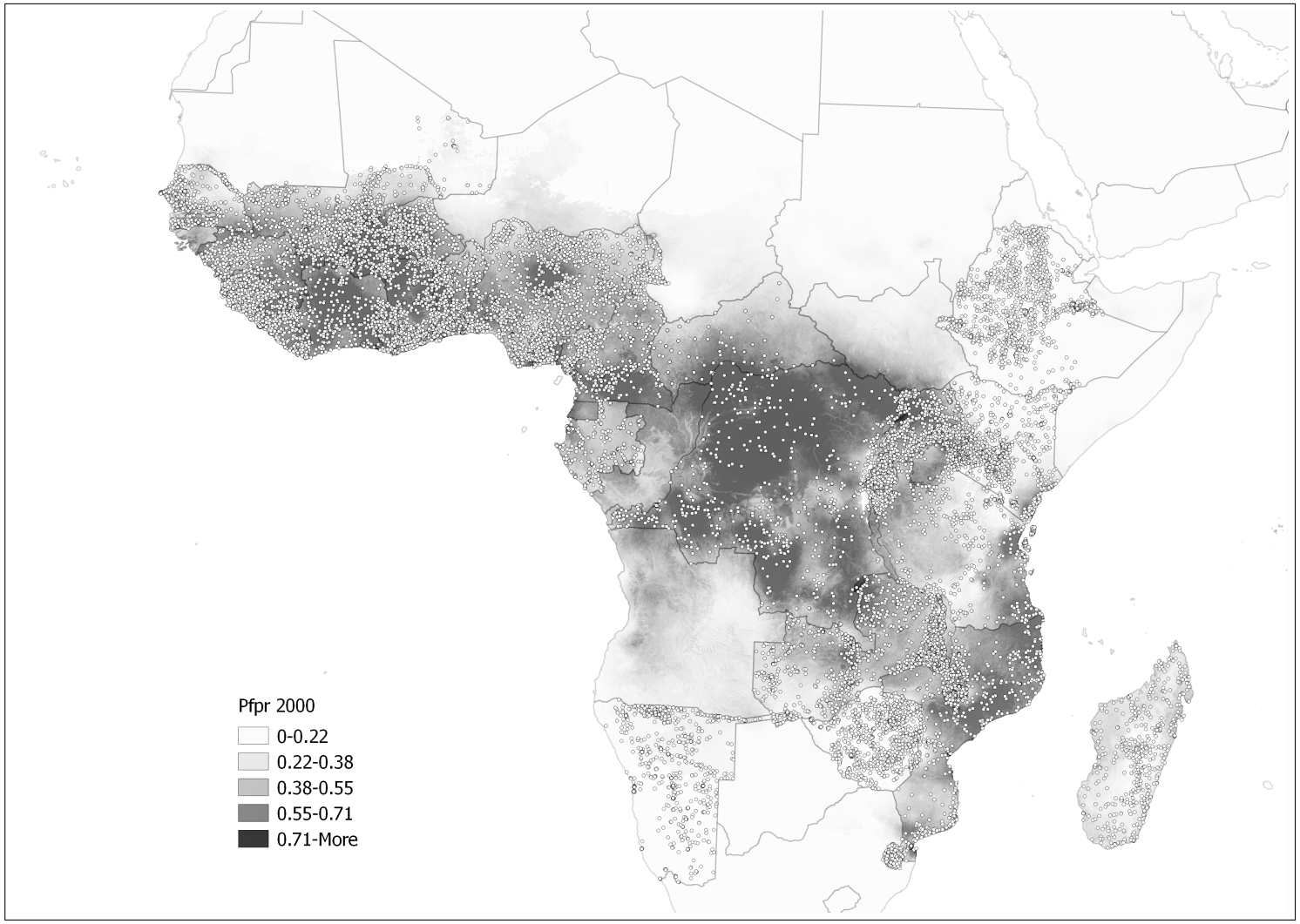
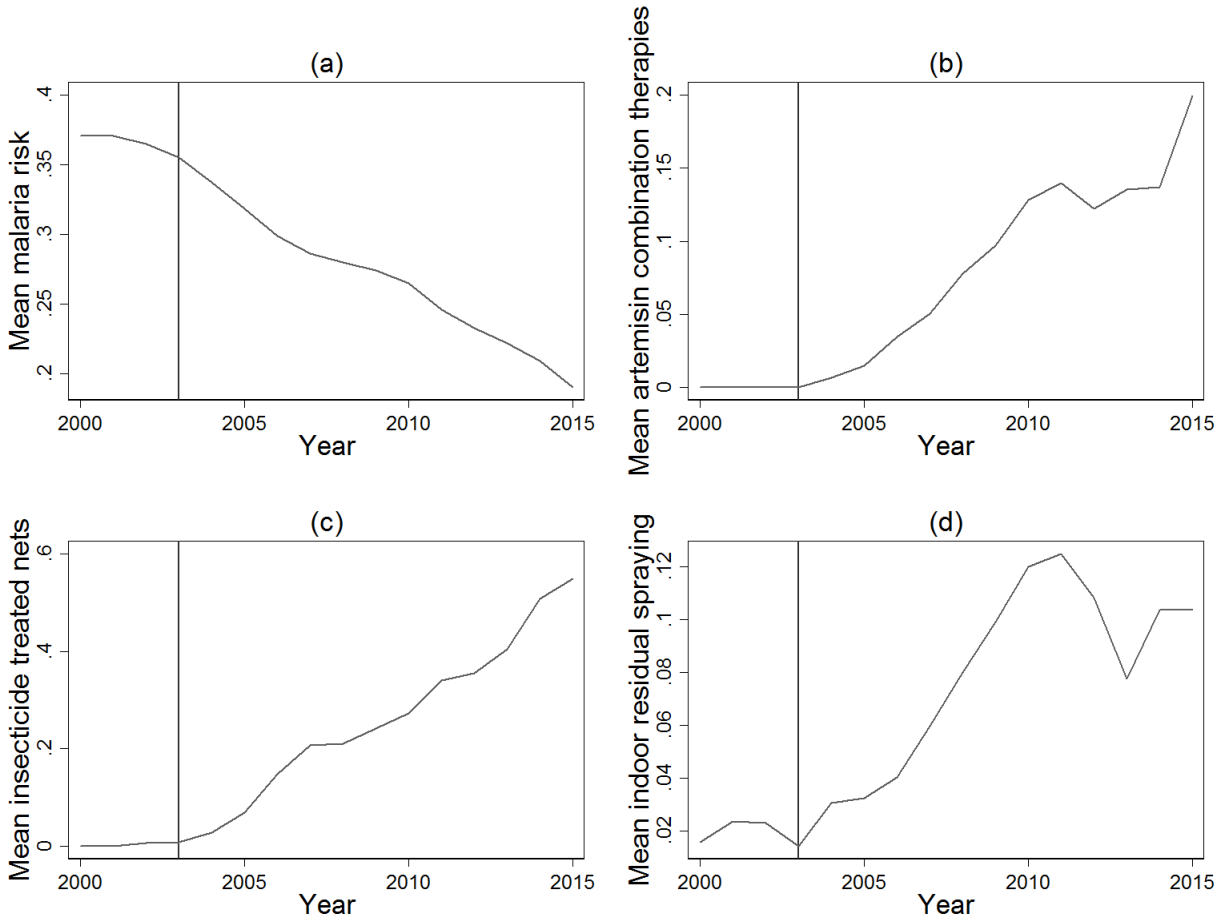
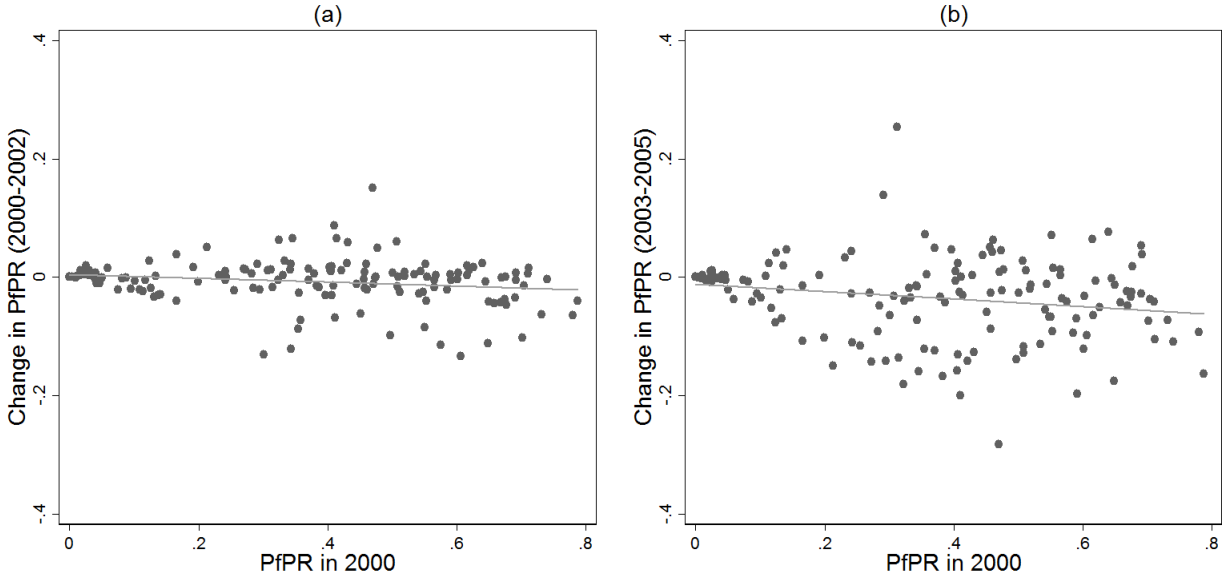


**Figure 3:** Evolution of malaria risk (*Plasmodium falciparum* parasite rate) and coverage by malaria control strategies in our 27 sample countries



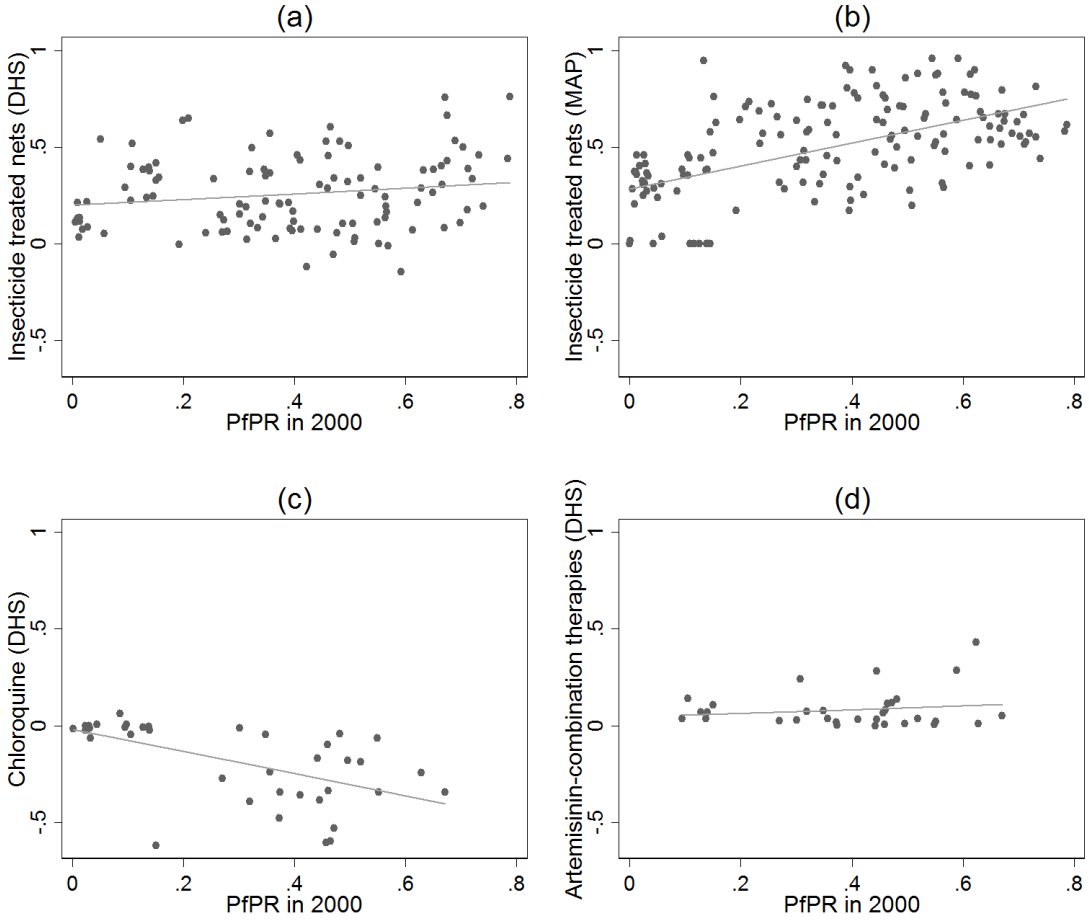

*Notes:* Each line plots annualized indicators from the Malaria Atlas Project, totaled for all countries in our sample, over time. The vertical bar denotes the scale-up of RBM disbursements. Figure 3a plots the mean malaria risk (*Plasmodium falciparum* parasite rate). Figure 3b plots the mean coverage of artemisinin combination therapies. Figure 3c plots the mean coverage of insecticide treated nets. Figure 3d plots the mean coverage of indoor residual spraying.

**Figure 4:** Evolution of regional malaria risk conditional on initial malaria risk (*Plasmodium falciparum* parasite rate)



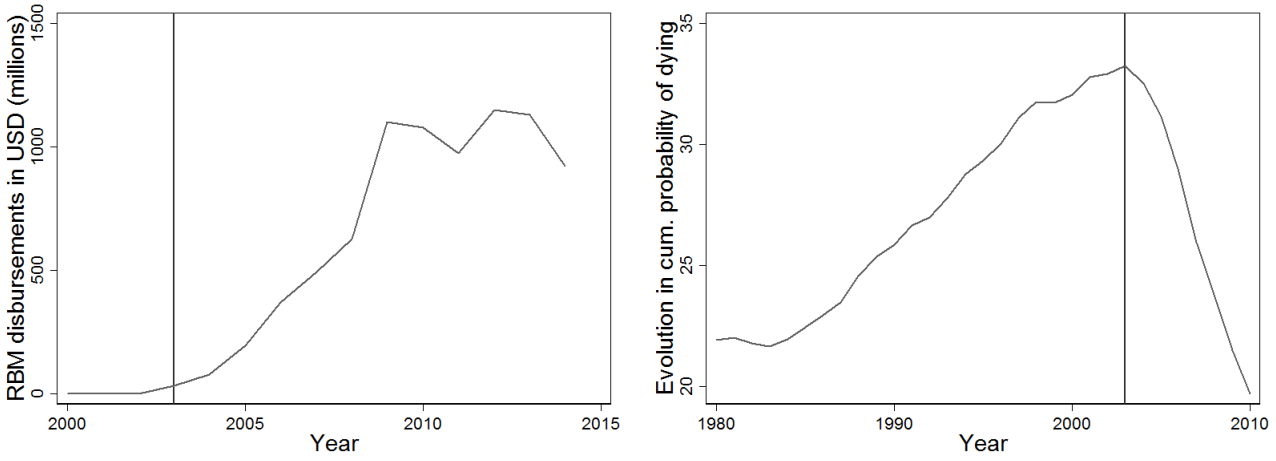

*Notes:* Each point represents a region. We obtain yearly malaria risk (PfPR) from the Malaria Atlas Project. In a univariate regression of the change in malaria risk between 2003 and 2005, the coefficient of initial malaria risk is -0.064 and is statistically significant at 0.1% (Figure 4b, N = 155).

**Figure 5:** Evolution of bednets and antimalarial use conditional on initial malaria risk (*Plasmodium falciparum* parasite rate)




*Notes:* Each point represents a region. We obtain yearly malaria risk from the Malaria Atlas Project. The change in Figures 5a, 5c, and 5d is the difference between the first and last DHS survey available in our sample. The change in Figure 5b is the difference between 2003 and 2015, the latest available year of MAP data. In a univariate regression of the change in household bednet use for children under 5 (from the DHS) on initial malaria risk (in 2000), the coefficient of initial malaria risk is 0.148 and is statistically significant at the 10% level (Figure 5a, N = 108). For the change in bednet use (from the Malaria Atlas Project), the coefficient of initial malaria risk is 0.586 and is statistically significant at the 0.1% level (Figure 5b, N = 155). For the change in chloroquine use for fever in children under 5, the coefficient of initial malaria risk is -0.554 and is statistically significant at the 0.1% level (Figure 5c, N = 39). For the change in artemisinin combination therapy use for fever in children under 5, the coefficient of initial malaria risk is 0.074 but it is not statistically significant due to the low number of observations (Figure 5d, N = 32).

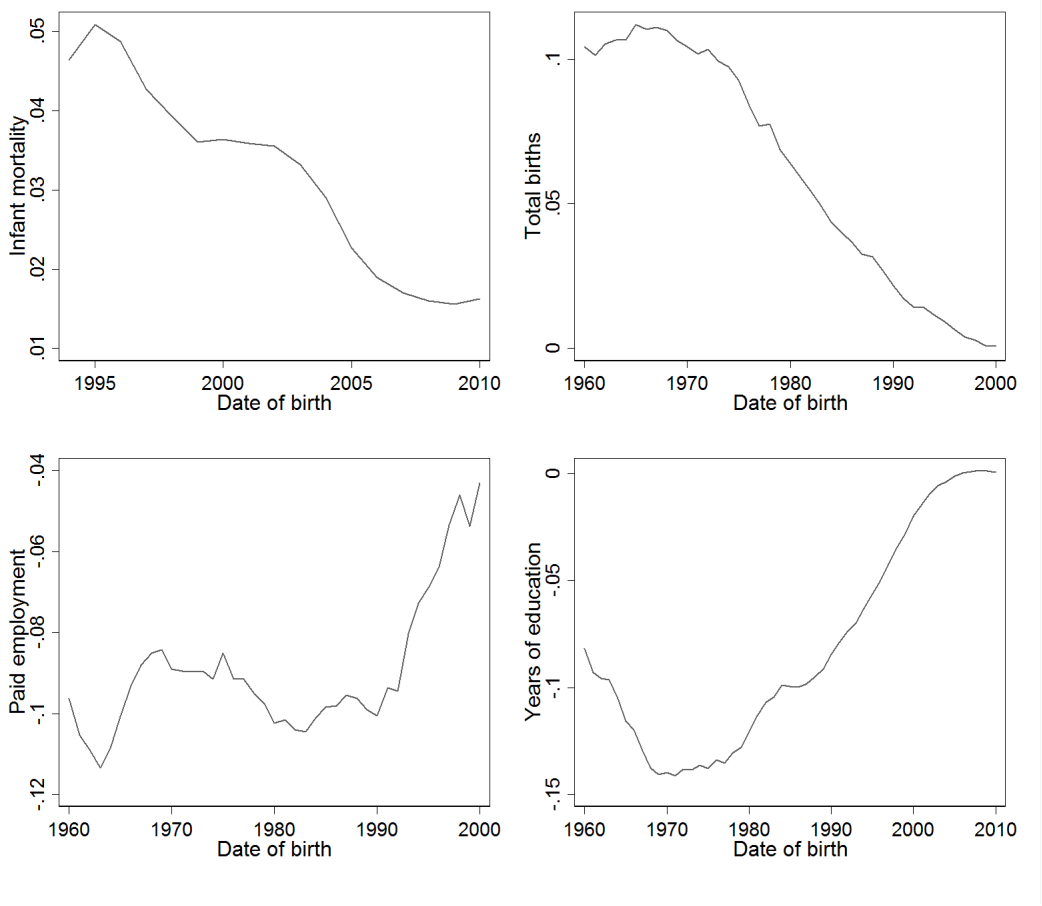
**Figure 6:** Increase in Roll Back Malaria disbursements vs. Cumulative probability of dying from malaria among children under five in Sub-Saharan Africa






*Notes:* The graph on the left plots Roll Back Malaria disbursements totaled over all countries in our sample for each year. The graph on the right plots the cumulative probability of dying among children under five in all of Sub-Saharan Africa. This measure refers to the total number of children under five out of 1,000 who are likely to die from malaria in the absence of all other causes from the Institute for Health Metrics and Evaluation. Murray et al. (2012) describe the estimation process which relies on all available data on sub-national malaria mortality. The vertical bar at 2003 marks the start of Roll Back Malaria scale-up.

**Figure 7:** Relationship of initial malaria risk with infant mortality, fertility, adult labor supply and education




*Notes:* These graphs display the standardized relationship of pre-campaign malaria risk with each outcome variable by cohort. For each cohort, we regress of the standard deviation of the dependent variable on the standard deviation of malaria risk in 2000, controlling for individual covariates and survey year.

**Table 1**: Ruling out pre-campaign catch-up effects

| | Coefficient of ($\text{malaria}_{2000j} \times \text{exposure}_{Nct}$) | | |
|---|---|---|---|
| Dep. var. | (1) | (2) | (3) |
| Height-For-Age z-scores | -8.202 | -19.649 | -26.920 |
| | (50.291) | (62.725) | (74.468) |
| $R^2$ | 0.257 | 0.258 | 0.259 |
| Observations | 276,860 | 276,860 | 276,860 |
| Years of education completed | -2.929ˆ | -4.235* | -4.189ˆ |
| | (1.546) | (1.929) | (2.227) |
| $R^2$ | 0.565 | 0.566 | 0.566 |
| Observations | 565,214 | 565,214 | 565,214 |
| Primary education completed | -0.161 | -0.262 | -0.488* |
| | (0.168) | (0.207) | (0.239) |
| $R^2$ | 0.476 | 0.476 | 0.477 |
| Observations | 565,214 | 565,214 | 565,214 |
| Age-by-region FE | no | yes | yes |
| Exposure-by-region FE | no | no | yes |

*Notes*: The unit of observation is the individual. For all dependent variables listed on the left, each cell reports the OLS estimate of coefficient $\beta$ in Equation (1). The regression controls for gender, wealth, and DHS cluster fixed effects as well as country-by-cohort-by-DHS year fixed effects and their subcomponents. Standard errors (in parentheses) are clustered at the DHS cluster level. ˆ, *, ** and *** indicate significance at the 10, 5, 1 and 0.1% levels.

**Table 2**: Difference in means analysis of human capital outcomes

| Dep. var. | (1) | (2) | (3) | Dep. var. | (1) | (2) | (3) |
|---|---|---|---|---|---|---|---|
| | Post-RBM | Pre-RBM | Difference | | Post-RBM | Pre-RBM | Difference |
| Infant mortality | | | | Employed in last 12 mo. (adult) | | | |
| Highly malarious | 0.066 | 1.000 | -0.034*** | Highly malarious | 0.706 | 0.649 | 0.057*** |
| Less malarious | 0.063 | 0.089 | -0.026*** | Less malarious | 0.671 | 0.631 | 0.040*** |
| | | | -0.008*** | | | | 0.017*** |
| Neonatal mortality | | | | Paid in cash when emp. (adult) | | | |
| Highly malarious | 0.033 | 0.047 | -0.014*** | Highly malarious | 0.647 | 0.578 | 0.069*** |
| Less malarious | 0.033 | 0.043 | -0.010*** | Less malarious | 0.719 | 0.715 | 0.004 |
| | | | -0.004*** | | | | 0.065*** |
| Postnatal mortality | | | | Years of ed. | | | |
| Highly malarious | 0.035 | 0.058 | -0.023*** | Highly malarious | 3.038 | 2.014 | 1.024*** |
| Less malarious | 0.032 | 0.050 | -0.018*** | Less malarious | 3.559 | 2.915 | 0.644*** |
| | | | -0.005*** | | | | 0.380*** |
| Tot. births | | | | Completed primary ed. | | | |
| Highly malarious | 2.935 | 2.637 | 0.299*** | Highly malarious | 0.245 | 0.138 | 0.107*** |
| Less malarious | 2.647 | 2.254 | 0.394*** | Less malarious | 0.296 | 0.227 | 0.069*** |
| | | | -0.095*** | | | | 0.380*** |

*Notes*: A highly (resp. less) malarious DHS cluster is defined as having an above (resp. below) median level of malaria risk in 2000. Pre-campaign surveys occur prior to 2003 and post-campaign surveys occur in 2003 or later.

**Table 3**: Effect of the RBM campaign on human capital outcomes

| | Coefficient of ($\text{malaria}_{2000j} \times \text{exposure}_{Nct}$) | | | | | |
|---|---|---|---|---|---|---|
| | (1) | (2) | (3) | (4) | (5) | (6) |
| | Infant | | Neonatal | | Postnatal | |
| | -0.514*** | -0.182*** | -0.458*** | -0.140* | -0.546*** | -0.135* |
| | (0.040) | (0.050) | (0.049) | (0.051) | (0.055) | (0.053) |
| $R^2$ | 0.126 | 0.323 | 0.117 | 0.323 | 0.126 | 0.320 |
| Observations | 341,542 | 341,542 | 329,786 | 329,786 | 330,171 | 330,171 |
| Exposure-by-region FE | no | yes | no | yes | no | yes |
| | Tot. births | | | | | |
| | -3.951*** | -3.242*** | -3.837*** | | | |
| | (0.213) | (0.241) | (0.291) | | | |
| $R^2$ | 0.659 | 0.665 | 0.666 | | | |
| Observations | 646,169 | 646,169 | 646,169 | | | |
| Age-by-region FE | no | yes | yes | | | |
| Exposure-by-region FE | no | no | yes | | | |
| | Employed last 12 mo. (adult) | | | Paid in cash for emp. (adult) | | |
| | 0.361*** | 0.316*** | 0.489*** | 0.215*** | 0.181* | 0.175* |
| | (0.040) | (0.048) | (0.057) | (0.049) | (0.059) | (0.068) |
| $R^2$ | 0.328 | 0.333 | 0.334 | 0.409 | 0.411 | 0.412 |
| Observations | 896,857 | 896,857 | 896,857 | 607,572 | 607,572 | 607,572 |
| Age-by-region FE | no | yes | yes | no | yes | yes |
| Exposure-by-region FE | no | no | yes | no | no | yes |
| | Years of ed. | | | Completed primary ed. | | |
| | 1.913*** | 1.776*** | 2.406*** | 0.178*** | 0.167*** | 0.224*** |
| | (0.087) | (0.082) | (0.112) | (0.008) | (0.008) | (0.011) |
| $R^2$ | 0.614 | 0.632 | 0.634 | 0.521 | 0.535 | 0.537 |
| Observations | 2,841,538 | 2,841,538 | 2,841,538 | 2,841,538 | 2,841,538 | 2,841,538 |
| Age-by-region FE | no | yes | yes | no | yes | yes |
| Exposure-by-region FE | no | no | yes | no | no | yes |

*Notes*: The unit of observation is the individual. For all dependent variables, each cell reports the OLS estimate of coefficient $\beta$ in Equation (1). The regression controls for gender, wealth, and DHS cluster fixed effects as well as country-by-cohort-by-DHS year fixed effects and their subcomponents. Standard errors (in parentheses) are clustered at the DHS cluster level. ˆ, *, ** and *** indicate significance at the 10, 5, 1 and 0.1% levels.

**Table 4**: Controlling for exposure to concurrent public policies interacted with malaria risk in 2000 in baseline equation

| | Coefficient of ($\text{malaria}_{2000j} \times$ exposure) | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) |
| | Infant | Neonatal | Postnatal | Tot. births | Employed | Paid in cash | Years of ed. | Completed primary ed. |
| Education | -0.082ˆ | -0.037 | -0.085ˆ | -2.940*** | 0.300*** | 0.207* | 1.937*** | 0.178*** |
| | (0.045) | (0.045) | (0.048) | (0.350) | (0.070) | (0.082) | (0.121) | (0.012) |
| $R^2$ | 0.551 | 0.553 | 0.546 | 0.667 | 0.335 | 0.413 | 0.634 | 0.538 |
| Observations | 341,542 | 329,786 | 330,171 | 646,169 | 896,857 | 607,572 | 2,841,538 | 2,841,538 |
| Health | -0.092* | -0.044 | -0.093* | -3.408*** | 0.364*** | 0.200* | 1.992*** | 0.192*** |
| | (0.034) | (0.034) | (0.035) | (0.407) | (0.084) | (0.101) | (0.134) | (0.013) |
| $R^2$ | 0.637 | 0.649 | 0.623 | 0.667 | 0.335 | 0.413 | 0.635 | 0.538 |
| Observations | 341,542 | 329,786 | 330,171 | 646,169 | 896,857 | 607,572 | 2,841,538 | 2,841,538 |
| Military | -0.059ˆ | -0.031 | -0.055 | -3.191*** | 0.350*** | 0.265* | 2.040*** | 0.188*** |
| | (0.034) | (0.033) | (0.037) | (0.346) | (0.071) | (0.086) | (0.112) | (0.011) |
| $R^2$ | 0.636 | 0.643 | 0.629 | 0.667 | 0.335 | 0.413 | 0.634 | 0.538 |
| Observations | 341,542 | 329,786 | 330,171 | 646,169 | 896,857 | 607,572 | 2,841,538 | 2,841,538 |
| All Govt. | -0.387*** | -0.350*** | -0.395*** | -2.514*** | 0.189* | 0.056 | 1.421*** | 0.143*** |
| | (0.024) | (0.033) | (0.029) | (0.439) | (0.091) | (0.106) | (0.104) | (0.010) |
| $R^2$ | 0.615 | 0.618 | 0.605 | 0.666 | 0.334 | 0.412 | 0.634 | 0.538 |
| Observations | 341,542 | 329,786 | 330,171 | 646,169 | 896,857 | 607,572 | 2,841,538 | 2,841,538 |
| FPE | | | | -2.093*** | 0.532*** | 0.181ˆ | 1.911*** | 0.190*** |
| | | | | (0.415) | (0.087) | (0.104) | (0.137) | (0.013) |
| $R^2$ | | | | 0.667 | 0.335 | 0.413 | 0.635 | 0.539 |
| Observations | | | | 646,169 | 896,857 | 607,572 | 2,841,538 | 2,841,538 |

*Notes*: The unit of observation is the individual. For all dependent variables, each cell reports the OLS estimate of coefficient $\beta$ in Equation (1). The regression controls for gender and wealth as well as fixed effects for DHS clusters, country-by-cohort-by-DHS year and subcomponents, age-by-region, and exposure-by-region. Standard errors (in parentheses) are clustered at the DHS cluster level. ˆ, *, ** and *** indicate significance at the 10, 5, 1 and 0.1% levels.

**Table 5a**: Substituting alternative treatment probabilities for initial malaria risk (*Plasmodium falciparum* parasite rate)

| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) | (9) |
|---|---|---|---|---|---|---|---|---|---|
| | Infant | | | Neonatal | | | Postnatal | | |
| Exposure*Δ Malaria | 0.399*** | | | 0.297* | | | 0.287* | | |
| | (0.112) | | | (0.109) | | | (0.114) | | |
| Exposure*Δ Antibiotics | | -2.453*** | | | -2.232*** | | | -2.638*** | |
| | | (0.156) | | | (0.205) | | | (0.207) | |
| Exposure*Δ Bednets | | | -0.890*** | | | -0.628* | | | -0.671* |
| | | | (0.257) | | | (0.236) | | | (0.270) |
| $R^2$ | 0.321 | 0.110 | 0.312 | 0.321 | 0.106 | 0.316 | 0.318 | 0.103 | 0.310 |
| Observations | 341,542 | 341,542 | 341,542 | 329,786 | 329,786 | 329,786 | 330,171 | 330,171 | 330,171 |

*Notes*: The unit of observation is the individual. For all dependent variables, each cell reports the OLS estimate of coefficient $\beta$ in Equation (1). The regression controls for gender and wealth as well as fixed effects for DHS clusters, country-by-cohort-by-DHS year and subcomponents, age-by-region, and exposure-by-region. Regional fixed effects are omitted for regressions with first-line antibiotics (artemisin combination therapies) measured at the national level. Standard errors (in parentheses) are clustered at the DHS cluster level. ˆ, *, ** and *** indicate significance at the 10, 5, 1 and 0.1% levels.

**Table 5b**: Substituting alternative treatment probabilities for initial malaria risk (*Plasmodium falciparum* parasite rate)

| | (1) | (2) | (3) | (4) | (5) | (6) |
|---|---|---|---|---|---|---|
| | Tot. births | | | | | |
| Exposure*Δ Malaria | 8.056*** | | | | | |
| | (0.640) | | | | | |
| Exposure*Δ Antibiotics | | -72.663*** | | | | |
| | | (5.507) | | | | |
| Exposure*Δ Bednets | | | -9.945*** | | | |
| | | | (0.826) | | | |
| $R^2$ | 0.666 | 0.651 | 0.665 | | | |
| Observations | 646,169 | 646,169 | 646,169 | | | |
| | Employed last 12 mo. | | | Paid in cash for emp. | | |
| Exposure*Δ Malaria | -1.027*** | | | -0.357* | | |
| | (0.123) | | | (0.140) | | |
| Exposure*Δ Antibiotics | | 6.837*** | | | 3.797*** | |
| | | (0.858) | | | (0.891) | |
| Exposure*Δ Bednets | | | 1.302*** | | | 0.412* |
| | | | (0.158) | | | (0.161) |
| $R^2$ | 0.334 | 0.325 | 0.333 | 0.412 | 0.409 | 0.412 |
| Observations | 896,857 | 896,857 | 896,857 | 607,572 | 607,572 | 607,572 |
| | Years of ed. | | | Completed primary ed. | | |
| Exposure*Δ Malaria | -4.717*** | | | -0.440*** | | |
| | (0.247) | | | (0.024) | | |
| Exposure*Δ Antibiotics | | 36.127*** | | | 3.370*** | |
| | | (2.531) | | | (0.239) | |
| Exposure*Δ Bednets | | | 7.304*** | | | 0.681*** |
| | | | (0.399) | | | (0.039) |
| $R^2$ | 0.630 | 0.596 | 0.630 | 0.534 | 0.507 | 0.534 |
| Observations | 2,841,538 | 2,841,538 | 2,841,538 | 2,841,538 | 2,841,538 | 2,841,538 |

*Notes*: The unit of observation is the individual. For all dependent variables, each cell reports the OLS estimate of coefficient $\beta$ in Equation (1). The regression controls for gender and wealth as well as fixed effects for DHS clusters, country-by-cohort-by-DHS year and subcomponents, age-by-region, and exposure-by-region. Standard errors (in parentheses) are clustered at the DHS cluster level. ˆ, *, ** and *** indicate significance at the 10, 5, 1 and 0.1% levels.

**Table 6**: Estimating baseline equation with restricted sub-samples

| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) |
|---|---|---|---|---|---|---|---|---|
| | Infant | Neonatal | Postnatal | Tot. births | Employed | Paid in cash | Years of ed. | Completed primary ed. |
| Residents[a] | -0.090 | -0.150 | -0.021 | -2.977*** | 0.594*** | -0.122 | 1.556*** | 0.152*** |
| | (0.117) | (0.115) | (0.117) | (0.824) | (0.118) | (0.177) | (0.231) | (0.027) |
| $R^2$ | 0.647 | 0.656 | 0.643 | 0.703 | 0.396 | 0.442 | 0.667 | 0.550 |
| Observations | 55,849 | 53,604 | 53,804 | 100,647 | 174,562 | 122,643 | 430,696 | 430696 |
| Born before 1960 | | | | -3.825*** | 0.476*** | 0.179* | 2.263*** | 0.206*** |
| | | | | (0.291) | (0.056) | (0.067) | (0.104) | (0.010) |
| $R^2$ | | | | 0.668 | 0.334 | 0.413 | 0.604 | 0.512 |
| Observations | | | | 670,113 | 950,792 | 651,475 | 3,235,297 | 3,235,297 |
| Above age 5 | | | | -3.837*** | 0.489*** | 0.175* | 3.140*** | 0.293*** |
| | | | | (0.291) | (0.057) | (0.068) | (0.169) | (0.017) |
| $R^2$ | | | | 0.666 | 0.334 | 0.412 | 0.597 | 0.512 |
| Observations | | | | 646,169 | 896,857 | 607,572 | 2,313,954 | 2,313,954 |

*Notes*: The unit of observation is the individual. For all dependent variables, each cell reports the OLS estimate of coefficient $\beta$ in Equation (1). The regression controls for gender, wealth, and DHS cluster fixed effects as well as country-by-cohort-by-DHS year fixed effects and their subcomponents. Standard errors (in parentheses) are clustered at the DHS cluster level. ˆ, *, ** and *** indicate significance at the 10, 5, 1 and 0.1% levels.

[a] When we drop exposure-by-region fixed effects (a demanding control given the small sample size), we obtain the following coefficients and standard errors: infant (-2.806*** [0.080]), neonatal (-2.710*** [0.096]), postnatal (-2.502*** [0.087]).

FOR ONLINE PUBLICATION

# Supplemental Appendix to Disease and Human Capital Accumulation: Evidence from the Roll Back Malaria Partnership in Africa

## 1 Model

### 1.1 Set-up

We consider a unitary household composed of one adult and her potential surviving offspring.[1] We assume that the household cares about its own consumption $c$ and leisure $\ell$ as well as about the number $n$ and human capital $h$ of the offspring. Though human capital in childhood is an important determinant of future earnings (Becker, 1975; Currie and Madrian, 1999; Currie, 2009; Hong, 2013), we focus only on contemporaneous effects of improved health on fertility, labor supply and education. We denote by $b$ the number of births and by $s$ the probability for a newborn to survive, such that $n = sb$ (with $0 < s < 1$).

The household's preferences are summarized by the following Cobb-Douglas utility function:

$$u(c, \ell, sb, h(e, H)) = (c\ell)^{1-\gamma}(sbh(e, H))^{\gamma}, \tag{1}$$

where

$$0 < \gamma < 1 \text{ and } h(e, H) = (e^{\rho} + H^{\rho})^{\frac{1}{\rho}} \text{ with } \rho < 1.$$

[1] The impact of children's exposure to malaria control campaigns on their adult longevity has not yet been documented (and cannot be investigated based on our data). Our analysis therefore relies on a one-period model, with no reference to adult longevity. The interplay between adult longevity and some of our outcomes is complex. For instance, increased longevity does not necessarily increase parents' incentives to invest in their offspring's education. On one hand, the longer the stream of payouts, the more valuable the investment. Increased longevity should therefore translate into more parental investment in education (Ben-Porath, 1967; Soares, 2005; Jayachandran and Lleras-Muney, 2009). On the other hand however, a higher life expectancy affects not only the returns to children's quality but also the returns to their quantity. Greater longevity might therefore result in no increase in the level of education chosen by the parents (Hazan and Zoabi, 2006) and could even reduce income per capita (Acemoglu and Johnson, 2007). As is apparent from the subsequent sections, the impact of improved children's survival rate and health on our various outcomes is not trivial either.

The human capital $h(e, H)$ of a surviving child is assumed to be a CES production function that depends on his/her education $e$ and his/her health $H$, with $H > 1$. We denote by $\sigma$ the elasticity of substitution between these two inputs. Therefore, $\sigma = \frac{1}{1-\rho}$. The lower (resp. the higher) $\rho$, the higher the complementarity (resp. the substitutability).[2] In particular, if $\rho < 0$, then the impact of one additional unit of education on human capital increases with health ($h_{eH} > 0$). If $0 < \rho < 1$ instead, the reverse is true ($h_{eH} < 0$).

For the sake of generality, we allow $\rho$ to take positive values. We assume that each member of the household is endowed with one unit of time. The adult allocates this unit between work, leisure and the time she spends on caring for her surviving child(ren). We denote by $\tau$ the time the parent dedicates to each surviving child, with $\tau > 0$.[3] As for each surviving child, she allocates her unit of time between education and child labor. Put differently, we assume that education and child labor are mutually exclusive.[4] The wage rates of the parent and of each child are denoted by $W$ and $w(H) = wH$ respectively, with $W > 0$ and $w > 0$.[5] Therefore, the budget constraint is

$$c \leq W(1 - \ell - \tau sb) + sbwH(1 - e). \tag{2}$$

The parameters of the model are chosen to yield an interior solution for the optimal number of births $b^*$, the optimal level of leisure $\ell^*$ and the optimal level of education $e^*$. More precisely, they ensure that (i) $0 < b^* < \frac{W(1-\ell^*)}{s(W\tau - wH(1-e^*))}$, that (ii) $0 < \ell^* < 1$ (iii) and that $0 < e^* < 1$. The first condition is obtained by assuming a positive consumption. This notably implies that $W\tau - wH(1 - e^*) > 0$ for all $e^*$ between 0 and 1. Put differently, a surviving child must cost more than the labor earnings she generates. Otherwise, the optimal number of births is infinite.

[2] If $\rho$ converges to 1, $h(e, H)$ is a linear production function; if $\rho$ converges to 0, $h(e, H)$ is a Cobb-Douglas production function; if $\rho$ converges to $-\infty$, $h(e, H)$ is a Leontief production function.

[3] For the sake of simplicity, but without loss of generality, $\tau$ depends on neither $e$ nor $H$.

[4] Evidence confirms that, on average, child labor and education exert a negative impact on one another (Beegle, Dehejia and Gatti, 2009; Edmonds and Shrestha, 2014), although exceptions obviously exist (Basu and Van, 1998; Basu, 1999; Psacharopoulos, 1997).

[5] Just as $w(H)$ depends positively on $H$, $W$ depends positively on the parent's health. However, this relationship is not made explicit since we focus on the impact of an increase in *children*'s survival rate and health.

## 1.2 Predictions

Solving the model yields the optimal level of education $e^*$, the optimal amount of time worked by the parent $\Lambda^*$ and the optimal number of births $b^*$:

$$e^* = (\frac{W\tau - wH}{wH^{\rho+1}})^{\frac{1}{1-\rho}}, \tag{3}$$

$$\Lambda^* = 1 - \ell^* - \tau s b^* = \frac{(1-\gamma)W\tau - wH(1-e^*)}{(2-\gamma)(W\tau - wH(1-e^*))}, \tag{4}$$

and

$$b^* = \frac{\gamma W}{(2-\gamma)s(W\tau - wH(1-e^*))}. \tag{5}$$

**Proof**:

Using monotonic transformation, the household's preferences can be represented by

$$U(c, \ell, sb, h(e, H)) = (1-\gamma)[\log c + \log \ell] + \gamma[\log sb + \log h(e, H)],$$

which yields the following maximization problem:

$$\max_{e,b,\ell}(1-\gamma)[\log(W(1-\ell-\tau sb) + w(H)sb(1-e)) + \log \ell] + \gamma[\log sb + \log h(e, H)].$$

The first-order conditions for the optimal level of education $e^*$, for the optimal number of births $b^*$ and for the optimal level of leisure $\ell^*$ are given by Equations (1) to (3) respectively:

$$\frac{\gamma e^{\rho-1}}{e^\rho + H^\rho} = \frac{(1-\gamma)sbwH}{W(1-\ell) - sb(W\tau - wH(1-e))}, \tag{6}$$

$$\frac{\gamma}{b} = \frac{(1-\gamma)s(W\tau - wH(1-e))}{W(1-\ell) - sb(W\tau - wH(1-e))}, \tag{7}$$

and

$$\frac{1}{\ell} = \frac{W}{W(1-\ell) - sb(W\tau - wH(1-e))}. \tag{8}$$

Dividing Equation (1) by Equation (2) and rearranging yields

$$e^* = (\frac{W\tau - wH}{wH^{\rho+1}})^{\frac{1}{1-\rho}}.$$

Solving the system of Equation (2) and Equation (3) yields

$$\ell^* = \frac{1-\gamma}{2-\gamma},$$

and

$$b^* = \frac{\gamma W}{(2-\gamma)s(W\tau - wH(1-e^*))}$$

□

We observe that $b^*$ declines in inverse proportion to the survival rate $s$. Indeed, since parents care about surviving children, this probability affects parents' fertility choices through the net cost of a surviving child (denoted by $W\tau - wH(1-e^*)$), which is independent of $s$. Consequently, parents choose the preferred number of surviving children irrespective of $s$, leading to a total number of births that is inversely proportional to this parameter. Therefore, since education and labor supply depend on the number of surviving children (not on the total number of births), they are not affected by $s$.

### 1.2.1 The optimal level of education

The optimal level of education $e^*$ increases with child health if $\rho < \overline{\rho_1}$, with $\overline{\rho_1} = -\frac{W\tau}{W\tau - wH}$; $e^*$ decreases with child health otherwise.

**Proof**:

Deriving $e^*$ with respect to $H$ yields

$$\frac{\partial e^*}{\partial H} = -(\frac{1}{1-\rho})(\frac{\rho(W\tau - wH) + W\tau}{wH^{\rho+2}})(\frac{W\tau - wH}{wH^{\rho+1}})^{\frac{\rho}{1-\rho}}.$$

We observe that $\frac{\partial e^*}{\partial H}$ is of the opposite sign of $\rho(W\tau - wH) + W\tau$. More precisely, $\frac{\partial e^*}{\partial H}$ is positive if $\rho < -\frac{W\tau}{W\tau - wH}$. It is negative otherwise. □

Consistent with Bleakley (2010), an increase in child health raises their wage rate ($wH$) and, hence, the opportunity cost of education. If education and health are substitutes ($0 < \rho < 1$), this negative effect is reinforced. If, however, education and health are complements ($\rho < 0$), an increase in health generates an additional impact that runs in the opposite direction: better health improves the returns to education. In this setting, if the complementarity between education and health is sufficiently high ($\rho < \overline{\rho_1}$), then the latter effect wins out, and $e^*$ increases with child health.

### 1.2.2 The optimal labor supply and number of births

The optimal labor supply $\Lambda^*$ increases with child health while the optimal number of births $b^*$ decreases with child health if $\rho < \overline{\rho_2}$, with $\overline{\rho_2} < \overline{\rho_1}$; $\Lambda^*$ (resp. $b^*$) decreases (resp. increases) with child health otherwise.

**Proof**:

Deriving $\Lambda^*$ and $b^*$ with respect to $H$ yields

$$\frac{\partial \Lambda^*}{\partial H} = -\frac{\gamma W \tau w (1 - e^* - \frac{\partial e^*}{\partial H} H)}{(2-\gamma)(W\tau - wH(1-e^*))^2},$$

and

$$\frac{\partial b^*}{\partial H} = \frac{\gamma W w (1 - e^* - \frac{\partial e^*}{\partial H} H)}{(2-\gamma)s(W\tau - wH(1-e^*))^2}$$

respectively.

We observe that $\Lambda^*$ (resp. $b^*$) increases (resp. decreases) with $H$ when $\frac{\partial e^*}{\partial H} > \frac{1-e^*}{H} > 0$. Yet, as we show below, $\frac{\partial e^*}{\partial H}$ decreases with $\rho$ when it is positive. Consequently, there exists a unique value of $\rho$, that we denote by $\overline{\rho_2}$, such that (i) $\frac{\partial e^*}{\partial H} = \frac{1-e^*}{H}$; (ii) $\frac{\partial e^*}{\partial H} > \frac{1-e^*}{H}$ when $\rho < \overline{\rho_2}$; (iii) $\frac{\partial e^*}{\partial H} < \frac{1-e^*}{H}$ when $\rho > \overline{\rho_2}$. Given that $\frac{1-e^*}{H}$ is strictly positive, $\overline{\rho_2}$ is necessarily lower than $\overline{\rho_1} = -\frac{W\tau}{W\tau - wH}$.

To show that $\frac{\partial e^*}{\partial H}$ decreases with $\rho$ when it is positive, one can rewrite $\frac{\partial e^*}{\partial H}$ as the product of two functions, denoted by$f(\rho)$ and $g(\rho)$, with

$$f(\rho) = -\frac{wH(\rho(W\tau - wH) + W\tau)}{1-\rho}$$

and

$$g(\rho) = (\frac{W\tau - wH}{wH^2})^{\frac{\rho}{1-\rho}}.$$

When $\frac{\partial e^*}{\partial H}$ is positive (i.e. $\rho < -\frac{W\tau}{W\tau - wH}$), $f(\rho)$ is a positive decreasing function of $\rho$. Moreover, using the fact that $e^*$ must be comprised between 0 and 1 and, hence, that $\frac{W\tau - wH}{wH^{\rho+1}} < 1$ for $\rho < 1$, it is easy to show that $g(\rho)$ is also a positive decreasing function of $\rho$. Therefore, $\frac{\partial e^*}{\partial H}$ is decreasing with $\rho$ when it is positive. □

We know that an increase in child health decreases each child's "price" by raising their wage rate $wH$. As a result, the adult's labor supply decreases, and her preferred number of births increases. If education also decreases with health, these effects are reinforced since better health increases children's working time. If, however, education increases with health, then a child's "price" increases. Provided that the complementarity between education and health is sufficiently high, this counter-effect wins out: the parent's labor supply increases and her preferred number of births decreases, following a well-documented quality-quantity trade-off (Becker and Lewis, 1973; Rosenzweig and Zhang, 2009; Bleakley and Lange, 2009).[6]

Note that the condition of $\overline{\rho_2} < \overline{\rho_1}$, emphasized in Proposition 3, is more binding than that of Proposition 2. For the labor supply (resp. the number of births) to increase (resp. decrease) with health, the impact of health on education does not simply need to be positive: it must be greater than a strictly positive value.

# 2 Data

### Human capital

**Source(s):** The Demographic and Health Surveys are nationally representative studies that collect detailed information on numerous population and health characteristics. We select 27 countries which were surveyed at least once post-campaign, which received RBM

[6]The variation in opposite directions of $\Lambda^*$ and $b^*$ is intuitive: a lower fertility allows the parent to dedicate more time to the labor market, while a lower labor supply allows the parent to raise more children.

disbursements, and which include geocoded clusters and all of our outcome variables. Figure 1 outlines the period of time covered for each country within the range of 1999 to 2014.

**Infant mortality (infants):** Because infant mortality may vary substantially within the first year of life, we follow Pathania (2014) to compute three binary indicators of mortality: neonatal (if death occurred within the first month of life); postnatal (if death occurred within months 1-11); infant (if death occurred within first year of life) from the Child Recode of children under five.

**Total births (women):** Total number of children ever born to eligible women (ages 15 to 45) in the Individual Recode.

**Total years of education (all):** Education in single years for all individuals in the DHS Person Recode.

**Completed primary education (all):** We use total years of education to compute an indicator for all respondents who have completed at least the full number of years of primary education (5, 6 or 7) in their country's educational system.

**Employed in last 12 mo. (adults):** Binary variable equal to one if respondent was employed within last 12 months for all eligible respondents in Individual and Male Recodes.

**Paid in cash for employment (adults):** Binary variable equal to one if respondent is paid at least partly in cash while employed for all eligible respondents in Individual and Male Recodes.

**Hours worked (children):** A subset of DHS surveys contain child labor modules (Benin, Burkina Faso, Burundi, Cote d'Ivoire, DRC, Cameroon, Gabon, Guinea, Mali, Malawi, Rwanda, Sierra Leone, Senegal, Togo, and Uganda). From these surveys, we rely on the number of hours worked by a child (ages 5 to 14) over the previous week.

### RBM disbursements

**Source(s):** According to Pigott et al. (2012), the three largest funders of anti-malaria campaigns to date, aside from governments, are the Global Fund to Fight AIDS, Tuberculosis and Malaria (since 2003), the President's Malaria Initiative (since 2005), and the World Bank Booster Program for Malaria Control in Africa (since 2006). We focus on external aid, using fixed effects and robustness checks to address the question of malaria-specific government expenditure. We extract GFATM disbursements on malaria, tuberculosis, and HIV/AIDS by

year from individual country files available at the International Aid Transparency Initiative website (http://iatiregistry.org/). We transcribe PMI expenditures by country and year from the PMI Ninth Annual Report to Congress (2005-2014). Finally, we rely on data kindly provided by David Pigott to compile disbursements from the World Bank Booster Program. We total RBM disbursements by country and year following studies on malaria expenditure (Snow et al., 2010; Pigott et al., 2012). Because these disbursements are at the country-year level, we merge them with each DHS recode by country-year. In a handful of cases, negative disbursements indicate corrupt or inefficient use of funds that donors requested for return.

**Exposure to RBM campaigns:** Exposure captures the yearly amount per capita (at the country level)[7] disbursed by the three main RBM funders during a respondent's lifetime. A respondent's lifetime is defined as the difference between the DHS survey year and this individual's year of birth, from which we subtract one year. We consider a respondent's exposure to begin in utero (though defining the beginning as the year after birth does not alter our results). To illustrate the construction of this variable, we take the example of Ethiopia. As reported in Figure 1, RBM campaigns in Ethiopia started in 2003. Moreover, three DHS surveys years are available (in 2000, 2005 and 2010). Let's consider an individual born in 1999. If this individual is surveyed in 2000, he experiences no exposure since the RBM disbursements were to begin only in 2003. If he is surveyed instead in 2005, he experiences three years of exposure to RBM disbursements. His exposure will therefore be equal to the sum of the RBM disbursements per capita during these three years, divided by his lifetime, hence 2005-(1999-1)=7 years. Similarly, if this individual is surveyed in 2010, he experiences eight years of exposure to RBM disbursements. His exposure will therefore be equal to the sum of RBM disbursements per capita during these eight years, divided by his lifetime, hence 2010-(1999-1)=12 years.

### Malaria risk and control programs

**Source(s):** We obtain data on malaria prevalence and control strategies from the Malaria Atlas Project (http://www.map.ox.ac.uk/). Data on ITN use and access to ACTs from over one million households were combined with national malaria control programme data

[7] Yearly population data come from the World Development Indicators.

on ITN, ACT and IRS provision to develop time-series models of coverage at the country level (Bhatt et al., 2015). We use the DHS to provide complementary measures of control strategies from microdata.

**Malaria risk (2000-2015):** We proxy for malaria risk by relying on the *P. falciparum* parasite rate (PfPR) computed for each DHS cluster. For a given year, PfPR describes the estimated proportion of individuals in the general population aged 2 to 10 years old who are infected with *P. falciparum* at any given time. These estimates are generated by a geostatistical model that relies on parasite rate surveys as well as bioclimatic and environmental characteristics. Gething et al. (2011) and Bhatt et al. (2015) describe the estimation process.

**Insecticide treated net coverage (MAP):** Calculated at the household level as the proportion of individuals who slept under a net.

**Artemisin combination therapies coverage (MAP):** Percentage of fever cases in children under five treated with ACT.

**Indoor residual spraying coverage (MAP):** Proportion of the population at risk of malaria who benefits from IRS.

**Insecticide treated net usage (DHS):** Proportion of households per region that always use ITN for children under 5 (DHS Household Recode).

**Artemisin combination therapies usage (DHS):** Proportion of children under five per region treated for fever with ACT during past 2 weeks (DHS Child Recode).

**Chloroquine usage (DHS):** Proportion of children under five per region treated for fever with chloroquine during past 2 weeks (DHS Child Recode).

### Public expenditure

We obtain expenditure on public education as a percentage of GDP from the World Bank EdStats, Education Statistics: Core Indicators. To compute total public expenditure per capita in each of these categories, we rely on GDP in current USD and total population (both from WDI).

# 3 Tables

**Table A1**: Descriptive statistics

| | Mean | SD | Min | Max | N |
|---|---|---|---|---|---|
| | (1) | (2) | (3) | (4) | (5) |
| **Explanatory variables** | | | | | |
| Malaria risk (PfPR in 2000) | 0.351 | 0.236 | 0.000 | 0.964 | 25,827 |
| Exposure to Roll Back Malaria | 0.570 | 0.787 | -0.162 | 8.918 | 2,857,253 |
| **Dependent variables** | | | | | |
| *Child survival and health* | | | | | |
| Infant mortality | 0.068 | 0.251 | 0 | 1 | 341,829 |
| Neonatal mortality | 0.034 | 0.182 | 0 | 1 | 330,094 |
| Postnatal mortality | 0.036 | 0.185 | 0 | 1 | 330,475 |
| *Fertility* | | | | | |
| Total number of births | 2.757 | 2.722 | 0 | 18 | 646,208 |
| *Adult labor supply* | | | | | |
| Employed in last 12 months | 0.684 | 0.465 | 0 | 1 | 896,887 |
| Paid in cash if employed | 0.680 | 0.466 | 0 | 1 | 607,668 |
| *Education* | | | | | |
| Number of years of education completed | 3.220 | 4.137 | 0 | 26 | 2,850,793 |
| Primary education completed | 0.262 | 0.440 | 0 | 1 | 2,850,793 |
| **Socioeconomic controls** | | | | | |
| Male | 0.489 | 0.500 | 0 | 1 | 2,866,372 |
| Age | 16.837 | 12.930 | 0 | 53 | 2,866,431 |
| Wealth | 2.969 | 1.427 | 1 | 5 | 2,866,431 |

*Notes*: The variables are measured at the individual level, with the exception of PfPR in 2000 which is computed at the DHS cluster level.

**Table A2**: Descriptive statistics for exposure to Roll Back Malaria disbursements

| Date of birth | Mean | SD | Min | Max | N | Age | Mean | SD | Min | Max | N |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | (1) | (2) | (3) | (4) | (5) | | (6) | (7) | (8) | (9) | (10) |
| 1965 | 0.119 | 0.148 | 0.000 | 0.705 | 25,475 | 0 | 1.190 | 1.093 | -0.162 | 4.251 | 112,882 |
| 1970 | 0.130 | 0.160 | 0.000 | 0.785 | 32,186 | 5 | 1.007 | 1.044 | 0.000 | 5.024 | 95,621 |
| 1975 | 0.154 | 0.185 | 0.000 | 0.885 | 39,127 | 10 | 0.604 | 0.655 | 0.000 | 3.139 | 107,579 |
| 1980 | 0.176 | 0.210 | 0.000 | 1.016 | 46,755 | 15 | 0.409 | 0.455 | 0.000 | 2.158 | 70,563 |
| 1985 | 0.211 | 0.250 | 0.000 | 1.191 | 54,222 | 20 | 0.293 | 0.336 | 0.000 | 1.644 | 70,973 |
| 1990 | 0.243 | 0.298 | 0.000 | 1.439 | 63,871 | 25 | 0.236 | 0.268 | 0.000 | 1.328 | 63,667 |
| 1995 | 0.331 | 0.377 | 0.000 | 1.817 | 77,621 | 30 | 0.202 | 0.225 | 0.000 | 1.114 | 63,188 |
| 2000 | 0.522 | 0.526 | 0.000 | 2.466 | 101,094 | 35 | 0.178 | 0.196 | 0.000 | 0.959 | 51,359 |
| 2005 | 1.108 | 0.768 | 0.000 | 3.793 | 88,740 | 40 | 0.162 | 0.174 | 0.000 | 0.842 | 43,139 |
| 2010 | 1.993 | 1.252 | 0.132 | 6.822 | 57,944 | 45 | 0.181 | 0.153 | 0.010 | 0.751 | 25,535 |
| 2014 | 1.498 | 0.558 | 0.000 | 1.835 | 8,280 | 50 | 0.246 | 0.152 | 0.091 | 0.677 | 12,095 |
| Survey year | | | | | | Survey year | | | | | |
| 1999 | 0.000 | 0.000 | 0.000 | 0.000 | 22,263 | 2007 | 0.391 | 0.505 | 0.016 | 3.263 | 130,321 |
| 2000 | 0.000 | 0.000 | 0.000 | 0.000 | 148,225 | 2008 | 0.219 | 0.242 | 0.026 | 1.604 | 242,097 |
| 2001 | 0.000 | 0.000 | 0.000 | 0.000 | 94,699 | 2009 | 0.563 | 0.430 | 0.108 | 2.287 | 81,118 |
| 2002 | . | . | . | . | . | 2010 | 0.786 | 0.741 | 0.087 | 4.566 | 385,477 |
| 2003 | 0.005 | 0.008 | 0.000 | 0.050 | 133,568 | 2011 | 0.868 | 0.774 | 0.091 | 4.199 | 258,130 |
| 2004 | 0.008 | 0.018 | 0.000 | 0.107 | 83,450 | 2012 | 0.949 | 0.811 | -0.162 | 3.519 | 219,140 |
| 2005 | 0.096 | 0.140 | 0.000 | 0.944 | 226,954 | 2013 | 1.026 | 1.171 | 0.000 | 8.918 | 453,016 |
| 2006 | 0.159 | 0.238 | 0.010 | 1.760 | 136,707 | 2014 | 0.971 | 0.609 | 0.000 | 2.598 | 251,266 |
| Country | | | | | | Country | | | | | |
| Benin | 1.145 | 1.063 | 0.000 | 4.067 | 105,223 | Mali | 0.334 | 0.578 | 0.000 | 2.539 | 170,726 |
| Burkina Faso | 0.380 | 0.622 | 0.001 | 3.128 | 118,737 | Mozambique | 0.770 | 0.532 | 0.167 | 2.024 | 56,122 |
| Burundi | 0.517 | 0.371 | 0.116 | 1.676 | 38,501 | Nambia | 1.078 | 1.642 | 0.000 | 8.918 | 97,664 |
| DRC | 0.515 | 0.509 | 0.016 | 1.830 | 123,167 | Nigeria | 0.272 | 0.258 | 0.000 | 0.891 | 325,292 |
| Cote d'Ivoire | 0.476 | 0.373 | 0.091 | 1.794 | 44,616 | Rwanda | 1.058 | 1.227 | 0.028 | 4.566 | 92,345 |
| Cameroon | 0.362 | 0.603 | 0.002 | 3.381 | 107,951 | Senegal | 0.648 | 0.707 | 0.024 | 2.504 | 123,800 |
| Ethiopia | 0.170 | 0.256 | 0.000 | 1.238 | 178,991 | Sierra Leone | 0.451 | 0.346 | 0.046 | 1.467 | 103,122 |
| Gabon | 0.605 | 0.402 | -0.162 | 1.387 | 34,729 | Swaziland | 0.089 | 0.076 | 0.020 | 0.352 | 18,776 |
| Ghana | 0.570 | 0.662 | 0.001 | 2.502 | 98,760 | Tanzania | 0.930 | 0.710 | 0.154 | 2.416 | 42,984 |
| Guinea | 0.396 | 0.541 | 0.011 | 2.630 | 70,528 | Togo | 0.493 | 0.315 | 0.000 | 1.154 | 42,056 |
| Kenya | 0.689 | 0.540 | 0.001 | 1.835 | 204,828 | Uganda | 0.432 | 0.490 | 0.000 | 1.740 | 107,516 |
| Liberia | 1.771 | 1.654 | 0.080 | 5.294 | 73,229 | Zambia | 1.335 | 0.889 | 0.130 | 3.670 | 108,573 |
| Madagascar | 0.540 | 0.419 | 0.096 | 1.604 | 75,111 | Zimbabwe | 0.184 | 0.298 | 0.000 | 1.588 | 94,030 |
| Malawi | 0.440 | 0.631 | 0.000 | 2.164 | 209,054 | | | | | | |

**Table A3**: Correlation of children's survival rate and health, fertility, adult labor market outcomes and education with variations in malaria prevalence, malaria incidence and coverage by antimalarial strategies

| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) |
|---|---|---|---|---|---|---|---|---|
| | Δ Risk | Δ Incid. | Δ Drugs (MAP) | Δ Drugs (DHS) | Δ Nets (MAP) | Δ Nets (DHS) | Δ Under 5 nets (DHS) | Δ Spraying (MAP) |
| Infant | 0.053*** | 0.077*** | -0.033*** | -0.194*** | -0.037*** | -0.046*** | -0.050*** | -0.086*** |
| | (0.003) | (0.003) | (0.003) | (0.009) | (0.002) | (0.002) | (0.003) | (0.004) |
| $R^2$ | 0.333 | 0.333 | 0.332 | 0.414 | 0.333 | 0.358 | 0.353 | 0.333 |
| Observations | 341,829 | 341,829 | 341,829 | 129,220 | 341,829 | 191,182 | 142,846 | 341,829 |
| Neonatal | 0.025*** | 0.037*** | -0.017*** | -0.112*** | -0.018*** | -0.024*** | -0.025*** | -0.047*** |
| | (0.002) | (0.003) | (0.003) | (0.007) | (0.001) | (0.002) | (0.003) | (0.003) |
| $R^2$ | 0.202 | 0.202 | 0.202 | 0.259 | 0.202 | 0.220 | 0.220 | 0.203 |
| Observations | 330,094 | 330,094 | 330,094 | 122,932 | 330,094 | 184,256 | 137,826 | 330,094 |
| Postnatal | 0.042*** | 0.060*** | -0.025*** | -0.148*** | -0.029*** | -0.037*** | -0.039*** | -0.062*** |
| | (0.002) | (0.003) | (0.003) | (0.007) | (0.001) | (0.002) | (0.003) | (0.003) |
| $R^2$ | 0.208 | 0.208 | 0.207 | 0.281 | 0.208 | 0.228 | 0.220 | 0.208 |
| Observations | 330,475 | 330,475 | 330,475 | 123,624 | 330,475 | 184,529 | 137,794 | 330,475 |
| Tot. births | 0.422*** | 0.595*** | 0.290*** | 0.052 | -0.099*** | -0.215*** | -0.575*** | -0.688*** |
| | (0.028) | (0.037) | (0.033) | (0.083) | (0.021) | (0.029) | (0.038) | (0.040) |
| $R^2$ | 0.575 | 0.575 | 0.574 | 0.568 | 0.574 | 0.569 | 0.571 | 0.575 |
| Observations | 646,208 | 646,208 | 646,208 | 228,972 | 646,208 | 357,848 | 263,657 | 646,208 |

**Table A3 (continued)**: Correlation of child survival rate and health, fertility, adult labor supply and education with variations in malaria prevalence, malaria incidence and coverage by antimalarial strategies

| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) |
|---|---|---|---|---|---|---|---|---|
| | Δ Risk | Δ Incid. | Δ Drugs (MAP) | Δ Drugs (DHS) | Δ Nets (MAP) | Δ Nets (DHS) | Δ Under 5 nets (DHS) | Δ Spraying (MAP) |
| Employed | -0.033*** | -0.013 | 0.112*** | 0.135*** | 0.083*** | 0.058*** | 0.099*** | -0.134*** |
| | (0.008) | (0.011) | (0.010) | (0.023) | (0.006) | (0.008) | (0.011) | (0.012) |
| $R^2$ | 0.125 | 0.125 | 0.126 | 0.111 | 0.126 | 0.118 | 0.131 | 0.126 |
| Observations | 896,887 | 896,887 | 896,887 | 293,084 | 896,887 | 475,397 | 352,660 | 896,887 |
| Paid in cash | -0.099*** | -0.123*** | -0.233*** | 0.403*** | -0.086*** | 0.118*** | -0.078*** | -0.104*** |
| | (0.012) | (0.016) | (0.015) | (0.030) | (0.008) | (0.012) | (0.016) | (0.015) |
| $R^2$ | 0.077 | 0.077 | 0.080 | 0.099 | 0.077 | 0.082 | 0.073 | 0.076 |
| Observations | 607,668 | 607,668 | 607,668 | 194,245 | 607,668 | 317,430 | 244,315 | 607,668 |
| Years of ed. | -1.227*** | -1.306*** | 0.901*** | 3.352*** | 0.152*** | 1.372*** | 0.706*** | 0.422*** |
| | (0.063) | (0.080) | (0.062) | (0.156) | (0.044) | (0.064) | (0.098) | (0.086) |
| $R^2$ | 0.311 | 0.311 | 0.310 | 0.299 | 0.309 | 0.326 | 0.319 | 0.309 |
| Observations | 2,850,735 | 2,850,735 | 2,850,735 | 1,041,303 | 2,850,735 | 1,600,922 | 1,183,236 | 2,850,735 |
| Primary ed. | -0.096*** | -0.099*** | 0.040*** | 0.268*** | 0.012* | 0.111*** | 0.048*** | -0.029*** |
| | (0.006) | (0.008) | (0.006) | (0.018) | (0.004) | (0.006) | (0.009) | (0.008) |
| $R^2$ | 0.246 | 0.245 | 0.245 | 0.231 | 0.245 | 0.265 | 0.266 | 0.245 |
| Observations | 2,850,735 | 2,850,735 | 2,850,735 | 1,041,303 | 2,850,735 | 1,600,922 | 1,183,236 | 2,850,735 |

*Notes*: The unit of observation is the individual. For each dependent variable listed on the left, each cell reports the OLS estimate of the coefficient of the explanatory variable specified in the left-hand column. Controls for gender, age and wealth are included. Standard errors (in parentheses) are clustered at the DHS cluster level. ˆ, *, ** and *** indicate significance at the 10, 5, 1 and 0.1% levels.

**Table A4**: Alternative infant health variables

| | (1) | (2) |
|---|---|---|
| | Acute respiratory infection | Diarrhea |
| ($\text{PfPR}_{2000j} \times$ exposure) | 0.036 | -0.004 |
| | (0.061) | (0.021) |
| R2 | 0.296 | 0.152 |
| Observations | 108,158 | 467,002 |

*Notes*: The unit of observation is the individual. For all dependent variables, each cell reports the OLS estimate of coefficient $\beta$ in Equation (1). The regression controls for gender and wealth as well as fixed effects for DHS clusters, country-by-cohort-by-DHS year and subcomponents, and exposure-by-region. Standard errors (in parentheses) are clustered at the DHS cluster level. ˆ, *, ** and *** indicate significance at the 10, 5, 1 and 0.1% levels.

**Table A5**: Estimation of artificial treatments

| | (1) | (2) | (3) | (4) | (5) |
|---|---|---|---|---|---|
| | Tot. births | Employed last 12mo. | Paid in cash for emp. | Years of ed. | Completed primary ed. |
| 20 years | 1.851*** | -0.306*** | 0.005 | -3.186*** | -0.223*** |
| | (0.344) | (0.086) | (0.097) | (0.551) | (0.063) |
| R2 | 0.595 | 0.296 | 0.419 | 0.593 | 0.507 |
| Observations | 521,530 | 717,674 | 531,953 | 1,274,398 | 1,274,398 |
| 30 years | 1.735* | -0.235* | -0.014 | 0.965 | 0.095 |
| | (0.695) | (0.108) | (0.110) | (0.837) | (0.093) |
| R2 | 0.539 | 0.332 | 0.456 | 0.603 | 0.515 |
| Observations | 311,351 | 431,628 | 335,523 | 861,112 | 861,112 |

*Notes*: The unit of observation is the individual. For all dependent variables, each cell reports the OLS estimate of coefficient $\beta$ in Equation (1). The regression controls for gender and wealth as well as fixed effects for DHS clusters, country-by-cohort-by-DHS year and subcomponents, age-by-region, and exposure-by-region. Standard errors (in parentheses) are clustered at the DHS cluster level. ˆ, *, ** and *** indicate significance at the 10, 5, 1 and 0.1% levels.

**Table A6**: Removal of potential outlying countries from baseline estimations

| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) | (9) |
|---|---|---|---|---|---|---|---|---|---|
| | No CI | No CD | No ET | No KE | No LB | No NG | No SL | No TZ | Mult. surveys |
| Infant mort. | -0.159*** | -0.161*** | -0.160*** | -0.135* | -0.211*** | -0.145*** | -0.163*** | -0.165*** | -0.220*** |
| | (0.044) | (0.044) | (0.043) | (0.044) | (0.056) | (0.039) | (0.044) | (0.045) | (0.059) |
| R2 | 0.327 | 0.320 | 0.326 | 0.332 | 0.328 | 0.361 | 0.334 | 0.319 | 0.287 |
| Observations | 333,292 | 325,281 | 320,362 | 316,511 | 329,208 | 296,277 | 327,696 | 332,993 | 283,232 |
| Tot. births | -3.806*** | -3.935*** | -3.839*** | -3.906*** | -4.137*** | -4.284*** | -3.638*** | -3.877*** | -3.715*** |
| | (0.291) | (0.298) | (0.291) | (0.312) | (0.316) | (0.297) | (0.291) | (0.296) | (0.317) |
| R2 | 0.667 | 0.667 | 0.664 | 0.666 | 0.667 | 0.668 | 0.667 | 0.666 | 0.672 |
| Observations | 636,453 | 619,683 | 604,825 | 599,616 | 630,320 | 568,333 | 622,339 | 636,438 | 537,894 |
| Employed in last 12 mo. | 0.470*** | 0.470*** | 0.488*** | 0.519*** | 0.509*** | 0.586*** | 0.430*** | 0.525*** | 0.497*** |
| | (0.057) | (0.059) | (0.057) | (0.062) | (0.062) | (0.060) | (0.057) | (0.059) | (0.062) |
| R2 | 0.335 | 0.335 | 0.337 | 0.330 | 0.334 | 0.336 | 0.335 | 0.333 | 0.330 |
| Observations | 882,587 | 859,110 | 836,870 | 847,802 | 871,433 | 786,627 | 863,378 | 884,704 | 742,553 |
| Years of ed. | 2.347*** | 2.267*** | 2.406*** | 2.554*** | 2.331*** | 2.539*** | 2.281*** | 2.416*** | 2.290*** |
| | (0.114) | (0.115) | (0.112) | (0.116) | (0.117) | (0.110) | (0.111) | (0.119) | (0.128) |
| R2 | 0.637 | 0.632 | 0.636 | 0.520 | 0.637 | 0.629 | 0.639 | 0.634 | 0.637 |
| Observations | 2,806,266 | 2,727,903 | 2,663,079 | 2,637,310 | 2,769,796 | 2,525,623 | 2,739,211 | 2,807,837 | 2,377,194 |

*Notes*: The unit of observation is the individual. For all dependent variables, each cell reports the OLS estimate of coefficient $\beta$ in Equation (1). The regression controls for gender and wealth as well as fixed effects for DHS clusters, country-by-cohort-by-DHS year and subcomponents, age-by-region, and exposure-by-region. Standard errors (in parentheses) are clustered at the DHS cluster level. ^, *, ** and *** indicate significance at the 10, 5, 1 and 0.1% levels. Our results are not driven by any country or subset of countries. Complementary analysis to Table A5 is available upon request.

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