

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

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Abstract

We study the effect of a large-scale anti-malaria campaign on human capital outcomes using microeconomic data from 27 countries in Sub-Saharan Africa. Using a difference-in-differences approach based on pre-campaign malaria risk and campaign timing and magnitude, we estimate the effect of the Roll Back Malaria Partnership 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

JEL: I15, I21, O15

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.

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

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

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

⁴These 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) and efficiency at completing domestic tasks. 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, 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 reductionist in this context. Treatment depends predominantly on the timing and intensity of RBM campaigns 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 5.3 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 more standard microempirical 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, due to our empirical strategy, 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 aim to 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 can improve future educational outcomes (Bharadwaj, Løken and Neilson, 2013) and 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 which, while not strictly novel, provide a structured framework for understanding our results.

We model a unitary household of one adult and his/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 his/her time between working and raising children. If each additional child costs less, a parent may reduce his/her own labor

supply and increase his/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 his/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 analysis takes advantage of newly available data on malaria risk from the Malaria Atlas Project (MAP). Our proxy for malaria risk is the *P. falciparum* parasite rate (PfPR) from the MAP⁶ (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 This

⁵The 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.

⁶See <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.

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

model is confronted by the MAP to a high number of reality checks. 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 5 km \times 5 km over the African continent, assigning a pre-campaign malaria risk measure to each geocoded DHS cluster.⁸ This procedure allows us to cover 25,827 DHS clusters scattered over 27 Sub-Saharan African 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 and 2003-2014), we plot the change in PfPR against the mean initial value of PfPR in 2000. Consistent with [Bhatt et al. \(2015\)](#), Figure 4 shows that initial PfPR and the change in malaria risk have a weakly negative correlation prior to 2002, a correlation which becomes more pronounced as the pre-RBM period progresses. Conditioning the use of malaria control techniques on initial malaria risk produces a similar

⁸Note that DHS displaces urban clusters up to two kilometers and rural clusters up to five kilometers. A further randomly selected 1% of clusters is displaced up to 10 kilometers. Since this displacement is white noise, it should not compromise our identification strategy. Regardless, results (available upon request) are robust to using different cluster radii (5 km, 10 km, 20 km) to calculate average malaria risk.

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).⁹ 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 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

⁹Malawi was the first African country to replace chloroquine in 1993, followed by Kenya in 1998 and Tanzania in 2000 (see Mohammed et al. (2013)).

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. Encouraged by the discovery of new and efficient first-line treatments – artemisinin-based combination therapies – 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.¹⁰ [Wilde et al. \(2014\)](#) use the rapid distribution of bednets to identify improvements in infant mortality and fertility.

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 Fund¹¹ (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 capita¹² 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

¹⁰The 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.”*

¹¹We show the funding process and steps, downloaded from the Global Fund’s website, in Figure A1.

¹²Yearly population data come from the World Development Indicators.

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 ¹³ 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. We also present frequency distributions of date of birth, age and exposure by survey year in Figure A2.

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.¹⁴ It is thus doubtful that the establishment of this Global Fund and its subsequent disbursements were anticipated by the general population of beneficiary countries.

¹³Negative values are possible in a small number of cases of young children when a country was required to return disbursed funds.

¹⁴A 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.

3.3 Outcomes of interest

The DHS provide our outcomes of interest. In the Supplemental Appendix, we report descriptive statistics in Table A1 as well as outline the DHS source data. Following [Pathania \(2014\)](#), we use infant mortality events (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 post-neonatal mortality which represent the probability of death within the first month and within months 1-11 respectively. The resulting data forms a pseudo-panel of birth cohorts.

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 under five.¹⁵ 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 two questions from the women’s questionnaire: the number of children ever born and the age of the respondent at first birth. While existing evidence emphasizes maternal health ([Lucas, 2013](#)) as a driving force behind fertility changes, additional mechanisms, such as opportunity costs, may be at play ([Soares, 2005](#)). 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 market-oriented rather than subsistence labor. Finally, for all individuals in the person-level recode, we compute education in single years. We also

¹⁵This 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.

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. As with infant mortality, we pool all surveys for each country in order to create pseudo-panels by year of birth cohorts.

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. A simplified version of our empirical strategy consists in computing the DD estimator from the following equation:

$$y_{ijct} = \beta_1 + \beta_2 \text{highmalaria}_{2000j} + \beta_3 \text{post}_{ct} + \beta_4 (\text{highmalaria}_{2000j} \times \text{post}_{ct}) + \epsilon_{ijct} \quad (1)$$

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 . The coefficient β_4 is the treatment effect. Low versus high malaria risk is defined within countries with respect to median pre-campaign risk, while post captures whether an individual’s year of survey falls before or after the start of major malaria control efforts. While we provide these simple differences when introducing our results, our preferred model uses continuous malaria risk and exposure to treatment. Relying on continuous measures allows us to move beyond a simple period model in order to exploit the full range of variability among cohorts in our data.

Our baseline specification thus 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 pre-campaign 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.¹⁶

$$\begin{aligned}
y_{ijct} &= \alpha + \beta \cdot (\text{malaria}_{2000j} \times \text{exposure}_{Nct}) + \mathbf{X}_{ijct}' \cdot \mathbf{\Gamma} \\
&+ \delta_{Nct} + \delta_{Nc} + \delta_{ct} + \delta_{Nt} \\
&+ \delta_j + \delta_c + \delta_t + \epsilon_{ijct}
\end{aligned} \tag{2}$$

As the coefficient of the interaction term between malaria_{2000j} and exposure_{Nct} , β identifies the treatment effect. The variable 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 malaria_{2000j} with 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 , exposure_{Nct} is defined by substantial variation to the extent that births, DHS surveys, and campaign start dates are difficult to predict relative to one another.

To control for each element of the interaction term and its correlates, we introduce DHS cluster fixed effects δ_j as well as country-by-cohort-by-DHS year fixed effects, δ_{Nct} , and their subcomponents: δ_{Nc} , δ_{ct} , δ_{Nt} , δ_N , δ_c and δ_t . While country fixed effects drop due to the concomitant control for DHS cluster fixed effects, others do not – for example, cohort-by-survey year and country-by-cohort-by-survey year fixed effects are not collinear because multiple countries may be surveyed in the same year. Finally, the vector \mathbf{X}_{ijct} includes individual covariates gender and wealth¹⁷ (age is already captured by δ_{ct}).

Adopting this restrictive parameterization isolates a treatment effect based on assignment

¹⁶To 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.

¹⁷Results are also robust when excluding the wealth index.

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).¹⁸ 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 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. \(2011\)](#)).¹⁹ 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 β (i) when Equation (2) is estimated; (ii) when the interactions between age_{ct} and region fixed effects are included;²⁰ (iii) and when, additionally, the interactions between exposure_{Nct} and region fixed effects are added.²¹

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 β in Equation (2) captures the impact of the RBM campaigns or if it simply reflects pre-campaign trends.

¹⁸Younger 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.

¹⁹See also [Kenkel \(1991\)](#) and [Dupas \(2011\)](#) for the relationship between education and health behavior.

²⁰We rely on region rather than cluster fixed effects to avoid multicollinearity. However, our results remain substantively unchanged if we rely instead on interactions between age_{ct} and cluster fixed effects or age_{ct} and malaria_{2000j} .

²¹We obviously cannot control for the interaction term between exposure_{Nct} and cluster fixed effects since this would drop the main variable of interest in our analysis, i.e. $(\text{malaria}_{2000j} \times \text{exposure}_{Nct})$.

To test for pre-campaign catch-up effects, we perform a falsification test. We estimate Equation (2) over individuals who were exposed to the campaign but whose outcomes could not be affected by the campaign. We examine the following outcomes: infant mortality for infants born prior to 2002, height-for-age z-scores based on WHO reference standards, a proxy for health conditions during childhood, among individuals who had completed their growth at the campaign’s start date (above age 25), total births (above age 40 at time of survey), and years of education completed as well as whether the respondent completed primary school (both above age 25 at the campaign’s start).

Table 1 reports the results of this test. We introduce both age-by-region and exposure-by-region fixed effects. For infant mortality, the coefficient β is negative but not significant. Height-for-age z-scores is also negative and not significant. Educational attainment variables are marginally significant but never positive. Finally, the coefficient of total births to older mothers is positive but not significant. 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. Supplemental Appendix Figure A3 provides a complementary graphical view using binary pre-/post-campaign malaria risk.

5 Results

Table 2 presents a preliminary view of the relationship between our outcomes and malaria risk. We regress these outcomes on regional variation in malaria prevalence and incidence as well as population coverage by standard control strategies – artemisinin combination therapies and insecticide treated nets. These changes over time may of course be endogenous. An increase in antibiotic treatments, for example, might serve as a bellwether of greater malaria risk, rather than the reverse. Bednet usage faces issues of household selection. Furthermore, the data face some limitations since MAP provides coverage with limited variation at the regional level (especially for ACTs) while DHS surveys do not always include questions related to malaria treatment. Nevertheless, a clear pattern emerges: variations in malaria

prevalence and incidence are positively (resp. negatively) correlated with infant mortality and total births (resp. adult labor supply, age at first birth, and education). The reverse is true for the variations in coverage by malaria control methods. These results are globally consistent with an impact of the RBM campaign that is conducive to human capital accumulation. There are some minor inconsistencies, whereby total births, age at first birth and probability of paid employment show opposite signs opposite of expectations when regressed on ACTs, though these coefficients are likely due to the challenges mentioned above.

Table 3 and Figure 7 support this interpretation. Table 3 reports the results of a difference in means analysis. It shows that the decrease (resp. increase) in infant mortality and total births (resp. adult labor supply, age at first birth, and education) between the post- and pre-campaign periods is greater in DHS clusters that show high rather than low pre-campaign malaria risk. In Figure 7, we plot the average of each dependent variable by age cohort at the time of survey (pre- and post-2003) between high and low levels of malaria risk. Prior to the start of anti-malaria campaigns, cohorts in highly malarious areas face a higher probability of infant mortality, higher birth rates, lesser probability of paid employment and lower number of years of education completed relative to their counterparts in less malarious areas. After the campaign start date, we observe a decrease in the distance between line plots in high and low malaria risk cohorts as well as a shifting of the curves - downward for infant mortality and total fertility and upward for the probability of paid employment and years of education completed. However, Table 3 and Figure 7 do not include any measure of exposure to campaign disbursements. We therefore further investigate these preliminary findings by estimating Equation (2) in the following section.

5.1 Infant mortality

Columns 1 through 6 of Table 4 report the OLS estimates of Equation (2) for infant mortality, without (odd columns) and with (even columns) exposure-by-region fixed effects. We omit controls for age (in months)-by-region in our baseline due to the fact that this information is missing for some infants and causes a reduction in sample size control. However, results are robust to their inclusion in Supplemental Appendix Table A3.

A marginal increase in the interaction term reduces the probability of infant mortality by 15 percentage points and postneonatal mortality by 10 percentage points. We observe

no effect on neonatal mortality, which is consistent with evidence that neonates possess some degree of clinical protection from malaria in the medical and economic literature (see [Pathania \(2014\)](#)). At first glance, these coefficients seem high, but they are not straightforward to interpret. Therefore, in Table 4, we also include a row to provide the treatment effect. In this row, we multiply the resulting coefficients by the mean level of malaria risk in 2000 (0.351). For an incremental increase in exposure to RBM at this average level of pre-campaign malaria risk in 2000, the resulting reduction in infant mortality is roughly 5 percentage points. As a comparison, ([BenYishay and Kranker, 2015](#)) find that measles campaigns raised the probability of a child’s survival to 60 months by approximately 2.4 percentage points for cohorts treated by the campaign. Hookworm disease has also been the subject of particular attention in the economic literature, but this disease is rarely lethal ([Bleakley and Lange, 2009](#)). We further interpret these results in Sections 5.4 and 5.5.

In the Supplemental Appendix, we subject this baseline specification to further tests. Table A3 presents mortality results when we control for additional mother-specific characteristics (age and length of preceding birth interval), mother fixed effects, age-by-region, and age-by-malaria_{2000j}.

5.2 Fertility, adult labor supply and education

The remainder of Table 4 reports the OLS estimates of Equation (2) for fertility, adult labor supply and education. We introduce age-by-region fixed effects and exposure-by-region fixed effects sequentially. Table 4 confirms the preliminary results from Table 3 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, at an average level of malaria risk in 2000, an incremental increase in RBM spending reduces the total number of live births by 1.3, and increases the probability of being employed and of being paid in cash by roughly 17 and 6 percentage points respectively. The effect on age at first birth is strongly significant and positive until the final column, in which we control for both age- and exposure-by-region. Exposure to RBM also improves educational outcomes, increasing both the probability of completing primary school by 8 percentage points and the number of years of education completed by 0.8. These results are consistent with a model of household production in which health and education operate as complementary inputs. They

are furthermore largely robust to specifications which include household fixed effects and an alternative age control (age-by-malaria). Adult labor outcomes lose significance, likely due to limited variation at the household level. We present these results in Supplemental Appendix Table A4. We further interpret the magnitude of Table 4’s baseline results in Section 5.5 by incorporating more specific assumptions as to the precise definition of an “incremental” increase in RBM exposure.

Our results on fertility are consistent with results from [Bleakley and Lange \(2009\)](#), but differ from other studies. For example, outside Africa, [Lucas \(2013\)](#) demonstrates that a decline in malaria caused an increase in fertility and a younger maternal age at first birth, while ([Fortson, 2009](#)) shows little impact of HIV/AIDS on fertility. The increase in education is consistent with the increased years of educational attainment and literacy found in several studies ([Lucas, 2010](#); [Barofsky, Anekwe and Chase, 2015](#); [Bleakley, 2010](#)). Though our results on labor participation differ from studies focusing on worms ([Bleakley and Lange, 2009](#)), they are consistent with studies on malaria ([Hong, 2013](#)). This divergence may be explained by the fact that malaria affects not only children but also adults, albeit less severely, in terms of mortality. These differences have important implications for external validity as our results may be limited to malaria in the African context. Note that we did not find any evidence of pre-trends in education or fertility outcomes.

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 data 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 U.S. dollars 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. Finally, to account for expenditure from non-governmental sources, we also draw data from the Global Fund’s disbursements allotted to HIV/AIDS.

In Table 5, we control for exposure to concurrent expenditure on education, health, military, and HIV/AIDS interacted with $malaria_{2000j}$ for all outcome variables. We then add all expenditures simultaneously. We also control for the percentage of a respondent’s life elapsed since the start of Free Primary Education. Our baseline results hold, though the magnitudes decrease slightly, as we net out the spillovers of government policies. Results controlling for HIV/AIDS expenditure are less robust for adult labor outcomes and age at first birth, likely due to the fact that adults are more likely to suffer from this disease (Azomahou, Boucekkine and Diene, 2016). (However, it is important to note that not necessarily all HIV/AIDS-burdened countries are highly malarious. For example, Mali faces a high incidence of malaria but a low incidence of HIV/AIDS, while the reverse is true for Namibia.) Otherwise, the effects of the RBM campaign are robust to controlling for significant concurrent policies.

5.3.2 Sample restrictions

In Table 6, we place various restrictions on our sample population beyond those tested in Tables A3 and A4. First, to account for the possibility of migration, we restrict our sample to individuals (or, in the case of infants, mothers) who lived in the same DHS cluster for at least five years. This information is available only for subset of DHS surveys which drastically reduces our sample size. Nevertheless, we observe that results hold for the majority of dependent variables, though age at first birth is positive but not significant and the probability of paid employment is negative but not significant. We then lift the restriction requiring all respondents to have been born post-1960. We also 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. Finally, we estimate our baseline equation over two major geographical regions represented in our sample, West and East Africa. Infant mortality is negative but not significant in West Africa, remaining negative and significant in East Africa even for neonatal mortality. The reduction in total births is slightly higher in West Africa, while the increase in age at first birth is substantially higher in

East Africa. Labor and educational outcomes are similar in both regions, with West Africa home to greater effects.

In a slightly different sub-sample check 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), those which experienced serious conflict (Cote d’Ivoire, Liberia, Sierra Leone), and one country nearing malaria eradication (Zambia). We also restrict our attention to countries with at least two DHS survey rounds available. Our results, presented in Table A5, are not driven by any country or subset of countries.²²

5.3.3 Alternative treatment probabilities and falsification tests

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 Supplemental Appendix Tables A6a and A6b, 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 since others, like indoor residual spraying, may be limited to specific geographic areas. 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. Artemisinin combination therapies vary primarily at the national level, leading to an increase in the estimated coefficients. The effect of artemisinin combination therapies thus appears to be globally higher on our outcomes but consistent with our results overall.

We run two different types of falsification tests in Tables A7 and A8 of the Supplemental

²²Complementary analysis is available upon request.

Appendix. For infants, we exchange mortality for two health outcomes which are the primary contributors to the under-five disease burden after malaria: acute respiratory infections and diarrhea. Estimating Equation (2) with these new dependent variables shows no effect of RBM exposure in Table A7. We then substitute four dependent variables that capture the probability of an infant receiving a full round of various vaccinations: Bacillus Calmette–Guérin (BCG), diphtheria, tetanus, and pertussis (DPT), polio, and measles. While all negative, only the BCG is significant at the 10% level. Routine childhood immunization has been one of the most successful and cost-effective public health interventions, responsible for a considerable reduction in infant morbidity in Africa (BenYishay and Kranker, 2015). But one perverse effect of vertical programs such as RBM could be a substitution for classical immunization programs, decreasing vaccination coverage.

Replicating this approach for adults is not feasible. Instead, in Table A8, 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 relationship that is either not significant or the opposite of what would be expected 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 Heterogeneous effects and non-linearity

5.4.1 Heterogeneous effects

In Table 7, we explore heterogeneous effects of the campaign on our outcomes. We look at two different cross-sections of our sample: female versus male and Fulani versus non-Fulani. The latter is an ethnic group that has been shown to exhibit partial immunity to malaria (see Thuilliez et al. (2017) for a discussion).

The effect of anti-malaria campaigns on infant mortality is stronger for girls and, except in the case of postneonatal mortality, not significant for boys. While the likelihood of being employed is greater for males than females, females are much more likely to be engaged in paid employment compared to males. Educational outcomes are roughly similar between genders, though marginally larger for females. If sex ratios favor men, women might antic-

ipate an easier marriage market and respond by supplying less labor and investing less in their education. Because sex ratios are lower on average in Africa, this may explain why we observe a comparatively larger decline in female mortality, one which could spur more investment in education and pursuit of paid employment.

The ethnicity tests should demonstrate no effect for the malaria-resistant group (Fulani), while those who are not partially immune (non-Fulani) should adhere closely to our baseline. This is what we observe. The only deviation is that the probability of employment for Fulani is negative and significant.

5.4.2 Non-linearity

Table 8 reports the OLS estimates for non-linear effects in campaign exposure. In order to compute non-linear effects, we first provide the results for human capital outcomes (fertility, labor and education) by age groups. For instance, for education, we expect that the earlier a respondent is exposed, the more he or she should benefit from the campaign. But the timing of survey, especially whether near or far from the start of the campaign, may also influence non-linear relationships. Note that we omit age at first birth which will, by definition, decline as age progresses.

We observe that the reduction in total births is slightly stronger for younger mothers, in the age groups 15-20 and 20-25. Probability of employment is similar in magnitude across all age groups but strongest in significance for younger cohorts. Finally, educational outcomes improve most substantially for younger cohorts, ages 5-10 and 10-15. These results fit to an explanation that earlier exposure leads to stronger effects.

5.5 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

programs.²³ 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 4 show that, for an average level of malaria risk in 2000, an incremental increase in RBM spending reduces infant mortality by 5.3 percentage points. We apply this treatment effect to averaged live birth and infant mortality rates in 2000²⁴ to obtain a cost of approximately \$5,526 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 later²⁵) a distinct treatment effect based on each cohort's time exposed to RBM campaigns and the average level of malaria risk in 2000. The average of these effects is 0.4. 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 educational interventions. Large-scale health campaigns are certainly less efficient compared to

²³See http://www.rollbackmalaria.org/files/files/partnership/wg/wg_itn/docs/rbmwin4ppt/3-8.pdf

²⁴Data come from the United Nations Population Division World Population Prospects

²⁵Consistent with DHS data, we assume that the maximum age possible for primary enrollment is 24.

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](#); [Baird et al., 2016](#)). 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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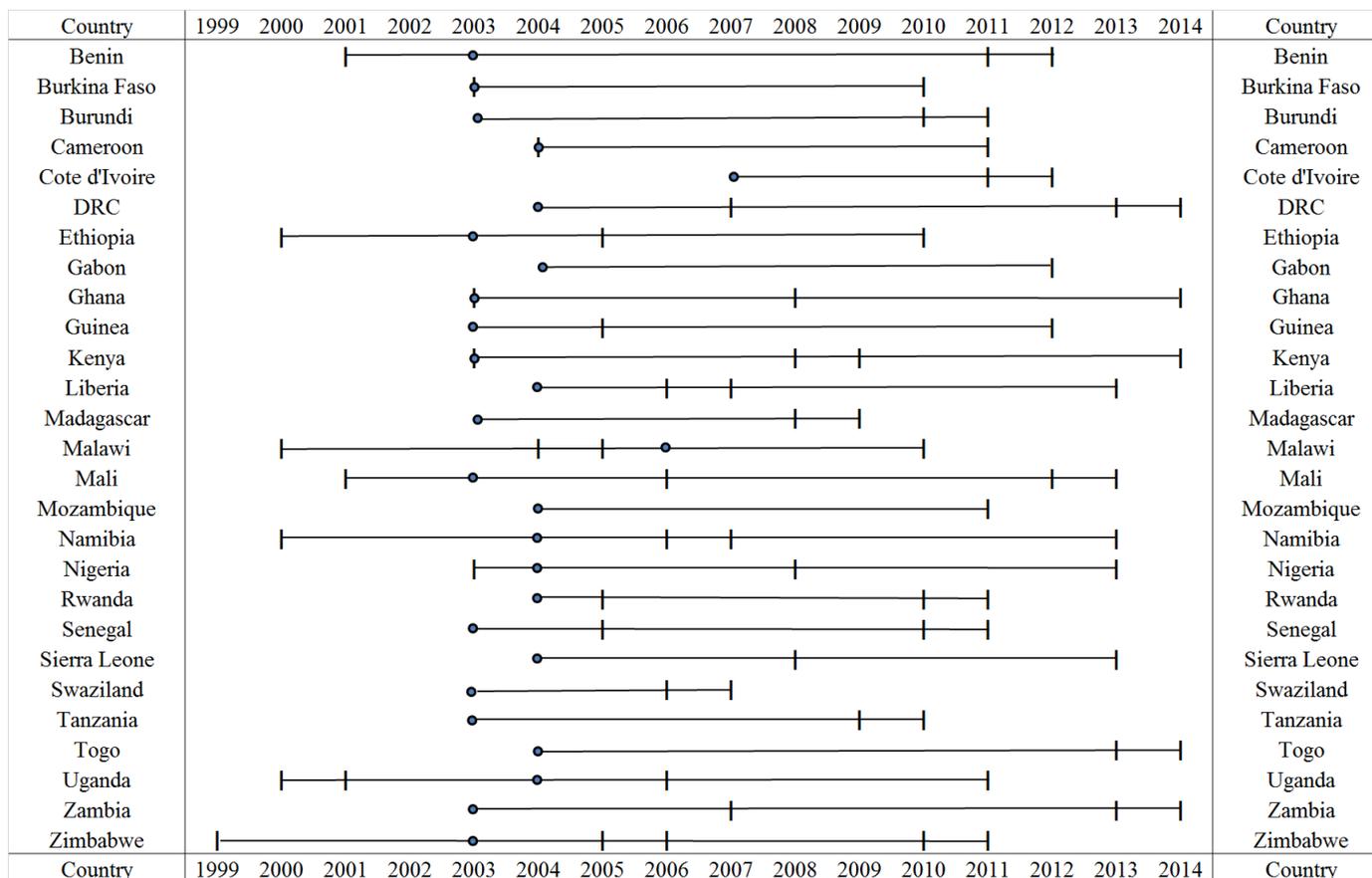
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Figure 1: Demographic and Health survey years and Roll Back Malaria start dates in our 27 countries



Notes: Vertical bars show the Demographic and Health survey years available for each country, and points mark the start of Roll Back Malaria disbursements. Individual exposure to anti-malaria programs is a function not only of disbursement starting dates but survey timing and years of birth.

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

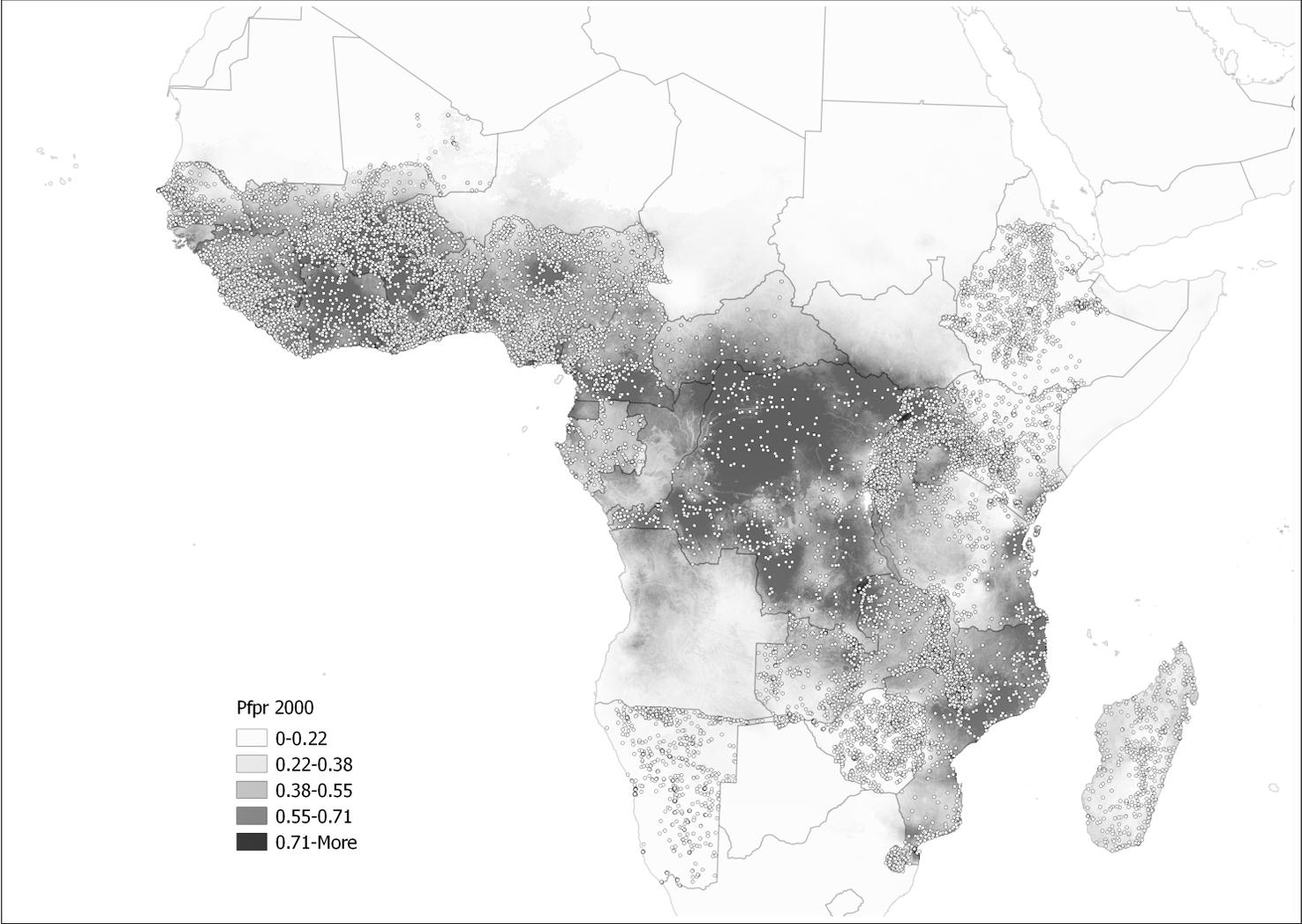
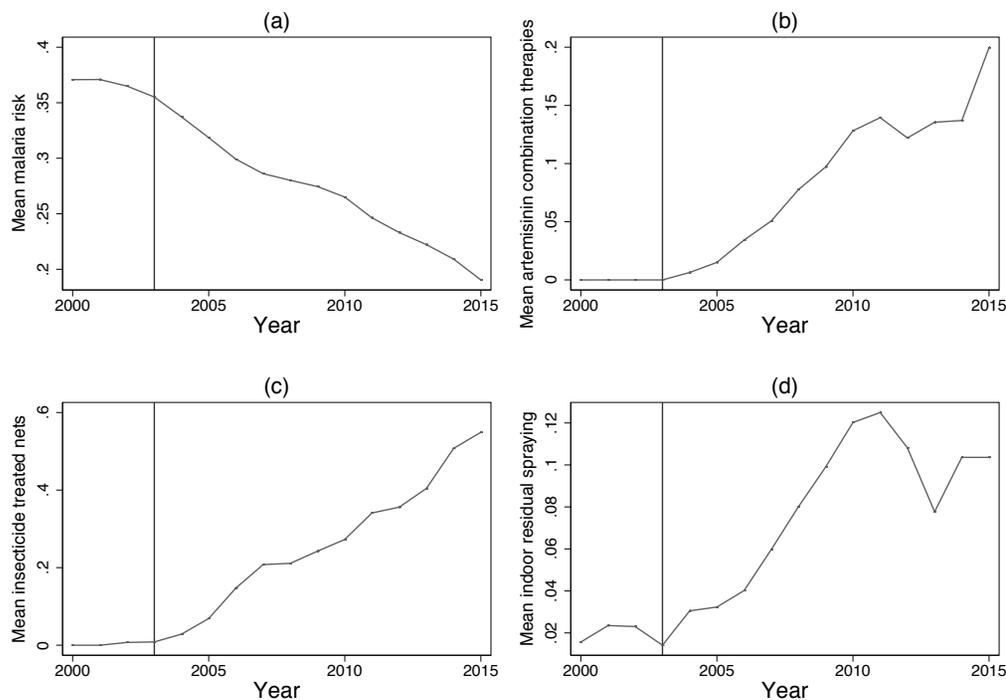
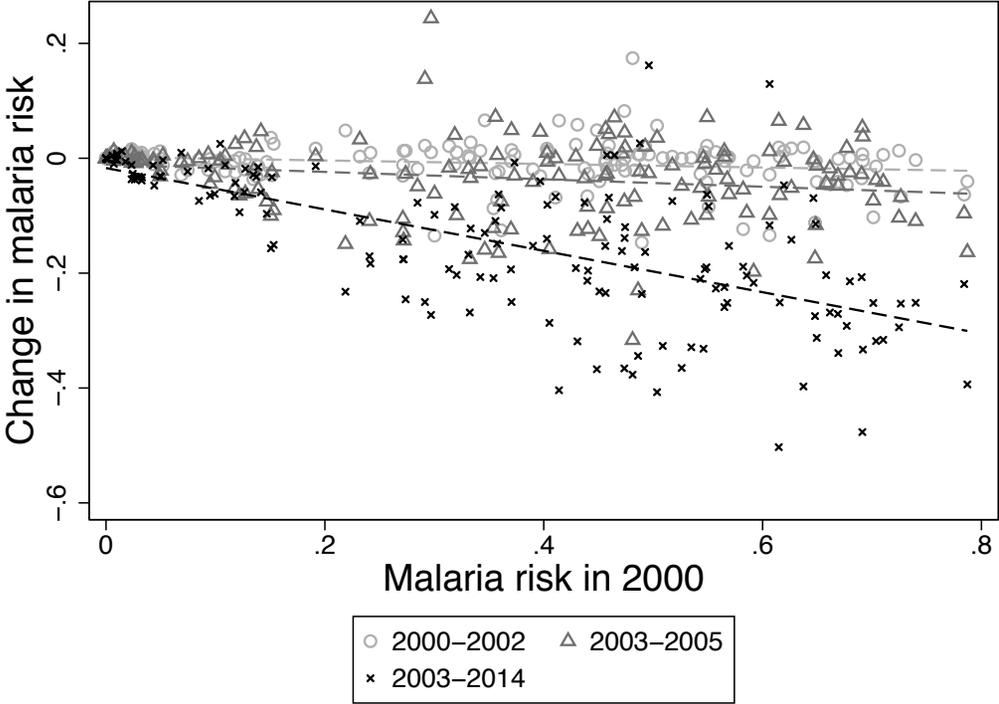


Figure 3: Evolution of malaria risk (*Plasmodium falciparum* parasite rate) and coverage by malaria control strategies in our 27 sample countries



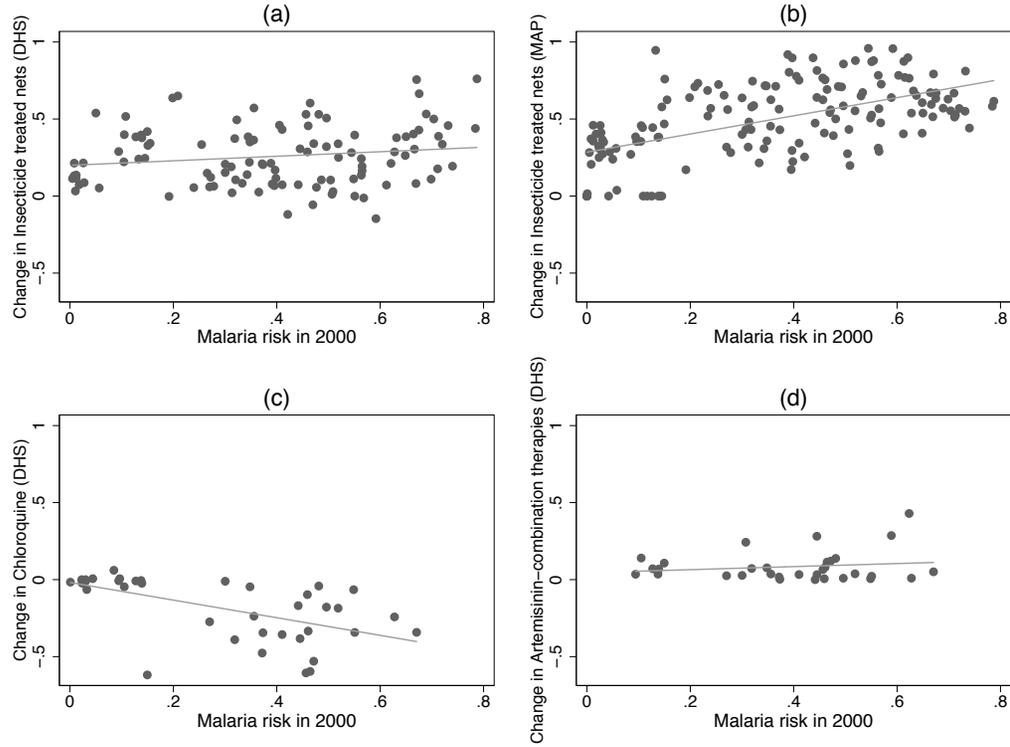
Notes: Each line plots annualized indicators from the Malaria Atlas Project, totaled for all countries in our sample, over time. The vertical bar denotes the scale-up of RBM disbursements. Figure 3a plots the mean malaria risk (*Plasmodium falciparum* parasite rate). Figure 3b plots the mean coverage 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. We obtain the dashed fitted lines by regressing the change in malaria risk over each period on initial malaria risk in 2000.

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, $N = 32$).

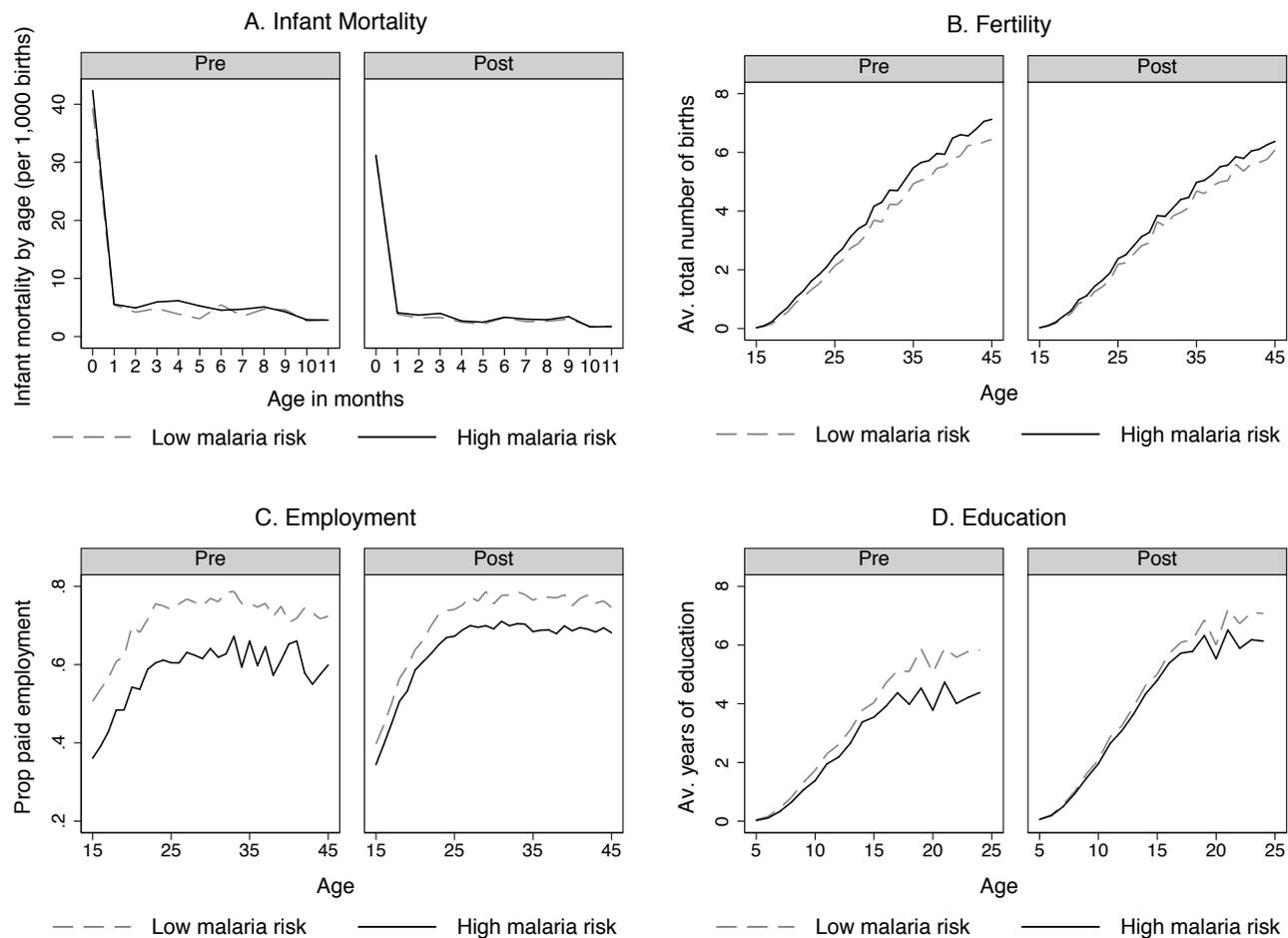
Figure 6: Increase in Roll Back Malaria disbursements vs. Cumulative probability of dying from malaria among children under five in Sub-Saharan Africa



41

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: We plot the average of each dependent variable by age cohort at the time of survey (pre- and post-2003) between high and low levels of malaria risk. A highly (resp. less) malarious cohort is defined as having an above (resp. below) median level of malaria risk in 2000. We note that the infant mortality variable has been rescaled in order to adhere to a more traditional definition of infant mortality (calculated over 1,000 births). The order of magnitude corresponds to key infant mortality figures in sub-Saharan Africa where the majority of mortality occurs during the first month of life.

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

Dep. var.	Coefficient of ($\text{malaria}_{2000j} \times \text{exposure}_{Nct}$)	
	(1)	(2)
Infant Mort. (born before 2002)	-0.466 (0.639)	Years of ed. (>25 y.o.) -4.189 [^] (2.227)
R ²	0.742	R ² 0.566
Observations	62,844	Observations 565,214
Height-For-Age z-scores (>25 y.o.)	-24.070 (136.370)	Primary ed. (>25 y.o.) -0.488* (0.239)
R ²	0.287	R ² 0.477
Observations	191293	Observations 565,214
Tot. births (>40 y.o.)	8.369 (7.329)	
R ²	0.414	
Observations	82,010	
Age-by-region	yes	yes
Exposure-by-region	yes	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 β in Equation (2). 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. The coefficient for infant mortality without age-by-region controls is 0.731 (standard error=0.682; N=73,382). Standard errors (in parentheses) are clustered at the DHS cluster level. [^], *, ** and *** indicate significance at the 10, 5, 1 and 0.1% levels.

Table 2: Regressions of main outcomes on change in malaria prevalence, change in malaria incidence and change in coverage by antimalarial strategies

	(1)	(2)	(3)	(4)	(5)	(6)
	Δ Risk	Δ Incid.	Δ Drugs (MAP)	Δ Drugs (DHS)	Δ Nets (MAP)	Δ Nets (DHS)
Infant Mort.	0.042*** (0.003)	0.062*** (0.003)	-0.025*** (0.003)	-0.165*** (0.008)	-0.029*** (0.002)	-0.038*** (0.002)
R ²	0.313	0.313	0.312	0.386	0.313	0.332
Observations	353,650	353,650	353,650	134,337	353,650	198,750
Neonatal Mort.	0.011*** (0.002)	0.017*** (0.003)	-0.008*** (0.002)	-0.060*** (0.006)	-0.008*** (0.001)	-0.011*** (0.002)
R ²	0.148	0.149	0.148	0.173	0.148	0.157
Observations	353,650	353,650	353,650	134,337	353,650	198,750
Postnatal Mort.	0.031*** (0.002)	0.045*** (0.002)	-0.017*** (0.003)	-0.105*** (0.006)	-0.021*** (0.001)	-0.027*** (0.002)
R ²	0.154	0.154	0.154	0.196	0.154	0.164
Observations	353,650	353,650	353,650	134,337	353,650	198,750
Tot. births	0.422*** (0.028)	0.595*** (0.037)	0.290*** (0.033)	0.052 (0.083)	-0.099*** (0.021)	-0.215*** (0.029)
R ²	0.575	0.575	0.574	0.568	0.574	0.569
Observations	646,208	646,208	646,208	228,972	646,208	357,848
Age first birth	-0.258*** (0.059)	-0.525*** (0.074)	-1.142*** (0.060)	-0.302* (0.153)	0.040 (0.039)	0.035 (0.054)
R ²	0.072	0.072	0.073	0.067	0.072	0.077
Observations	464,412	464,412	464,412	167,274	464,412	257,954

Table 2 (continued): Regressions of main outcomes on change in malaria prevalence, change in malaria incidence and change in coverage by antimalarial strategies

	(1)	(2)	(3)	(4)	(5)	(6)
	Δ Risk	Δ Incid.	Δ Drugs (MAP)	Δ Drugs (DHS)	Δ Nets (MAP)	Δ Nets (DHS)
Employed	-0.033*** (0.008)	-0.013 (0.011)	0.112*** (0.010)	0.135*** (0.023)	0.083*** (0.006)	0.058*** (0.008)
R ²	0.125	0.125	0.126	0.111	0.126	0.118
Observations	896,887	896,887	896,887	293,084	896,887	475,397
Paid in cash	-0.099*** (0.012)	-0.123*** (0.016)	-0.233*** (0.015)	0.403*** (0.030)	-0.086*** (0.008)	0.118*** (0.012)
R ²	0.077	0.077	0.080	0.099	0.077	0.082
Observations	607,668	607,668	607,668	194,245	607,668	317,430
Years of ed.	-1.227*** (0.063)	-1.306*** (0.080)	0.901*** (0.062)	3.352*** (0.156)	0.152*** (0.044)	1.372*** (0.064)
R ²	0.311	0.311	0.310	0.299	0.309	0.326
Observations	2,850,735	2,850,735	2,850,735	1,041,303	2,850,735	1,600,922
Primary ed.	-0.096*** (0.006)	-0.099*** (0.008)	0.040*** (0.006)	0.268*** (0.018)	0.012* (0.004)	0.111*** (0.006)
R ²	0.246	0.245	0.245	0.231	0.245	0.265
Observations	2,850,735	2,850,735	2,850,735	1,041,303	2,850,735	1,600,922

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. Changes in explanatory variables are at the regional level, except drugs (MAP) which vary primarily at the national level. Controls for gender, age and wealth are included. Standard errors (in parentheses) are clustered at the DHS cluster level. $\hat{\cdot}$, *, ** and *** indicate significance at the 10, 5, 1 and 0.1% levels.

Table 3: 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	0.064	0.095	-0.031***	Highly malarious	0.706	0.649	0.057***
Less malarious	0.061	0.085	-0.024***	Less malarious	0.671	0.631	0.040***
			-0.007***				0.017***
Neonatal mortality				Paid in cash when emp. (adult)			
Highly malarious	0.031	0.042	-0.011***	Highly malarious	0.647	0.578	0.069***
Less malarious	0.031	0.039	-0.008***	Less malarious	0.719	0.715	0.004
			-0.003***				0.065***
Post-neonatal mortality				Years of ed.			
Highly malarious	0.033	0.052	-0.019***	Highly malarious	3.038	2.014	1.024***
Less malarious	0.030	0.045	-0.015***	Less malarious	3.559	2.915	0.644***
			-0.004***				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***
Age at first birth							
Highly malarious	18.952	18.412	0.540***				
Less malarious	19.271	18.770	0.501***				
			0.039***				

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 4: Effect of the RBM campaign on human capital outcomes

	(1)	(2)	(3)	(4)	(5)	(6)
	Infant		Neonatal		Post-neonatal	
malaria _{2000j} × exposure _{Nct}	-0.453*** (0.034)	-0.149** (0.047)	-0.208*** (0.018)	-0.048 (0.039)	-0.245*** (0.019)	-0.101** (0.035)
Treatment effect		-0.052*		-0.017		-0.035*
R ²	0.116	0.279	0.094	0.183	0.096	0.176
Observations	353,379	353,379	353,379	353,379	353,379	353,379
Age-by-region	no	no	no	no	no	no
Exposure-by-region	no	yes	no	yes	no	yes
	Tot. births			Age at first birth		
malaria _{2000j} × exposure _{Nct}	-3.951*** (0.213)	-3.242*** (0.241)	-3.837*** (0.291)	1.895*** (0.464)	1.979*** (0.568)	0.954 (0.651)
Treatment effect			-1.347***			0.335
R ²	0.659	0.665	0.666	0.227	0.229	0.230
Observations	646,169	646,169	646,169	464,336	464,336	464,336
Age-by-region	no	yes	yes	no	yes	yes
Exposure-by-region	no	no	yes	no	no	yes
	Employed last 12 mo. (adult)			Paid in cash for emp. (adult)		
malaria _{2000j} × exposure _{Nct}	0.361*** (0.040)	0.316*** (0.048)	0.489*** (0.057)	0.215*** (0.049)	0.181* (0.059)	0.175* (0.068)
Treatment effect			0.172***			0.061*
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	no	yes	yes	no	yes	yes
Exposure-by-region	no	no	yes	no	no	yes
	Years of ed.			Completed primary ed.		
malaria _{2000j} × exposure _{Nct}	1.913*** (0.087)	1.776*** (0.082)	2.406*** (0.112)	0.178*** (0.008)	0.167*** (0.008)	0.224*** (0.011)
Treatment effect			0.845***			0.079***
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	no	yes	yes	no	yes	yes
Exposure-by-region	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 β in Equation (2). 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. $\hat{\cdot}$, *, ** and *** indicate significance at the 10, 5, 1 and 0.1% levels. The treatment effect multiplies the estimated coefficient by the mean level of malaria risk in 2000 (0.351).

Table 5: Controlling for exposure to concurrent public policies interacted with malaria risk in 2000

	Coefficient of (malaria _{2000j} × exposure)								
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
	Infant	Neonatal	Post-neonatal	Tot. births	Age at first birth	Employed	Paid in cash	Years of ed.	Completed primary ed.
Education	-0.711*** (0.030)	-0.333*** (0.018)	-0.377*** (0.017)	-2.554*** (0.343)	-0.194 (0.771)	0.290*** (0.069)	0.193* (0.081)	1.428*** (0.100)	0.133*** (0.010)
R ²	0.214	0.144	0.145	0.666	0.229	0.334	0.412	0.634	0.537
Observations	353,379	353,379	353,379	646,169	464,336	896,857	607,572	2,850,716	2,850,716
Health	-0.574*** (0.027)	-0.274*** (0.017)	-0.300*** (0.016)	-3.289*** (0.405)	0.701 (0.906)	0.346*** (0.083)	0.203* (0.100)	1.587*** (0.102)	0.154*** (0.010)
R ²	0.351	0.218	0.206	0.666	0.230	0.334	0.412	0.634	0.538
Observations	353,379	353,379	353,379	646,169	464,336	896,857	607,572	2,850,716	2,850,716
Military	-0.727*** (0.030)	-0.340*** (0.018)	-0.388*** (0.017)	-2.963*** (0.322)	0.390 (0.747)	0.240*** (0.067)	0.278*** (0.082)	1.505*** (0.088)	0.140*** (0.009)
R ²	0.162	0.114	0.121	0.666	0.229	0.334	0.412	0.633	0.537
Observations	353,379	353,379	353,379	646,169	464,336	896,857	607,572	2,850,716	2,850,716
HIV	-0.700*** (0.030)	-0.326*** (0.018)	-0.374*** (0.018)	-3.445*** (0.539)	0.179 (1.221)	0.329* (0.104)	0.177 (0.126)	1.716*** (0.106)	0.162*** (0.010)
R ²	0.147	0.109	0.113	0.666	0.229	0.330	0.414	0.627	0.525
Observations	350,004	350,004	350,004	606,996	435,362	858,180	580,924	2,717,679	2,717,679
All	-0.378*** (0.026)	-0.164*** (0.017)	-0.214*** (0.016)	-1.761* (0.660)	0.635 (1.529)	-0.154 (0.139)	-0.081 (0.170)	1.417*** (0.105)	0.144*** (0.010)
R ²	0.334	0.204	0.201	0.667	0.229	0.330	0.415	0.629	0.526
Observations	350,004	350,004	350,004	606,996	435,362	858,180	580,924	2,717,679	2,717,679
FPE	-0.147* (0.048)	-0.047 (0.039)	-0.099* (0.037)	-2.093*** (0.415)	-1.145 (0.900)	0.532*** (0.087)	0.181 [^] (0.104)	1.908*** (0.137)	0.190*** (0.013)
R ²	0.280	0.183	0.176	0.667	0.231	0.335	0.413	0.635	0.539
Observations	353,379	353,379	353,379	646,169	464,336	896,857	607,572	2,850,716	2,850,716
Age-by-Region	no	no	no	yes	yes	yes	yes	yes	yes
Exposure-by-region	yes	yes	yes	yes	yes	yes	yes	yes	yes

Notes: The unit of observation is the individual. For all dependent variables, each cell reports the OLS estimate of coefficient β in Equation (2). We compute exposure to various expenditures (education, health, military, HIV/AIDS) with the same method as our main RBM exposure variable, using per capita expenditure during a respondent's lifetime. 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 (exposure, in this table, incorporating the newly introduced expenditures). Standard errors (in parentheses) are clustered at the DHS cluster level.

[^], *, ** and *** indicate significance at the 10, 5, 1 and 0.1% levels.

Table 6: Restricted sub-samples

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
	Infant	Neonatal	Post-neonatal	Tot. births	Age at first birth	Employed	Paid in cash	Years of ed.	Completed primary ed.
Residents	-0.129 [^]	-0.014	-0.115	-4.017***	1.951	0.674***	-0.033	1.913***	0.185***
	(0.074)	(0.072)	(0.078)	(0.788)	(1.668)	(0.113)	(0.167)	(0.224)	(0.026)
R ²	0.434	0.250	0.275	0.692	0.311	0.384	0.436	0.668	0.553
Observations	144,280	144,280	144,280	120,562	90,536	208,617	147,012	511,647	511,647
Born before 1960				-3.825***	0.914	0.476***	0.179*	2.244***	0.204***
				(0.291)	(0.651)	(0.056)	(0.067)	(0.105)	(0.010)
R ²				0.668	0.222	0.334	0.413	0.605	0.512
Observations				670,113	487,633	950,792	651,475	3,244,475	3,244,475
Above age 5								3.140***	0.293***
								(0.169)	(0.017)
R ²								0.597	0.512
Observations								2,313,954	2,313,954
West Africa	-0.119	-0.027	-0.092	-4.007***	0.100	0.613***	0.382***	3.080***	0.264***
	(0.079)	(0.062)	(0.058)	(0.464)	(1.044)	(0.091)	(0.111)	(0.211)	(0.019)
R ²	0.208	0.145	0.130	0.660	0.222	0.323	0.412	0.571	0.505
Observations	162,719	162,719	162,719	284,825	207,671	395,641	275,633	1,267,680	1,267,680
East Africa	-0.240***	-0.147*	-0.093 [^]	-3.797***	2.562*	0.278***	0.042	1.632***	0.173***
	(0.061)	(0.054)	(0.054)	(0.408)	(0.879)	(0.082)	(0.095)	(0.148)	(0.015)
R ²	0.341	0.220	0.210	0.682	0.239	0.336	0.404	0.671	0.546
Observations	144,623	144,623	144,623	272,662	193,844	371,280	253,332	1,202,972	1,202,972
Age-by-region	no	no	no	yes	yes	yes	yes	yes	yes
Exposure-by-region	yes	yes	yes	yes	yes	yes	yes	yes	yes

Notes: The unit of observation is the individual. For all dependent variables, each cell reports the OLS estimate of coefficient β in Equation (2). 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. [^], *, ** and *** indicate significance at the 10, 5, 1 and 0.1% levels. Note that the tests in panels two and three do not apply to infant mortality variables, which are computed for the first year of life.

Table 7: Heterogeneous effects in sub-populations

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
	Infant	Neonatal	Post-neonatal	Tot. births	Age at first birth	Employed	Paid in cash	Years of ed.	Completed primary ed.
Full sample	-0.149*	-0.048	-0.101*	-3.837***	0.954	0.489***	0.175*	2.385***	0.222***
	(0.047)	(0.039)	(0.036)	(0.291)	(0.651)	(0.057)	(0.068)	(0.113)	(0.011)
R ²	0.279	0.183	0.176	0.666	0.230	0.334	0.412	0.634	0.537
Observations	353,379	353,379	353,379	646,169	464,336	896,857	607,572	2,850,716	2,850,716
Female	-0.200*	-0.092 [^]	-0.108*			0.367***	0.303***	2.463***	0.251***
	(0.065)	(0.053)	(0.051)			(0.066)	(0.086)	(0.119)	(0.012)
R ²	0.324	0.235	0.234			0.352	0.481	0.642	0.538
Observations	173,494	173,494	173,494			629,082	397,783	1,457,314	1,457,314
Male	-0.102	-0.014	-0.088 [^]			0.644***	0.086	2.264***	0.189***
	(0.064)	(0.053)	(0.048)			(0.088)	(0.098)	(0.118)	(0.011)
R ²	0.334	0.247	0.231			0.487	0.536	0.663	0.569
Observations	177,633	177,633	177,633			267,408	208,783	1,393,363	1,393,363
Fulani	-0.050	-0.043	-0.007	0.321	0.584	-0.416*	-0.094	-0.415	-0.121
	(0.192)	(0.184)	(0.186)	(1.123)	(3.006)	(0.200)	(0.260)	(0.943)	(0.079)
R ²	0.367	0.273	0.240	0.662	0.283	0.350	0.444	0.647	0.603
Observations	18,518	18,518	18,518	62,748	45,180	70,214	42,634	172,457	172,457
Non-Fulani	-0.151*	-0.046	-0.104*	-4.299***	1.146 [^]	0.549***	0.188*	2.522***	0.239***
	(0.048)	(0.039)	(0.037)	(0.300)	(0.659)	(0.059)	(0.070)	(0.110)	(0.011)
R ²	0.279	0.183	0.178	0.669	0.228	0.339	0.415	0.637	0.535
Observations	334,065	334,065	334,065	582,712	418,386	825,975	564,187	2,677,659	2,677,659
Age-by-Region	no	no	no	yes	yes	yes	yes	yes	yes
Exposure-by-region	yes	yes	yes	yes	yes	yes	yes	yes	

Notes: The unit of observation is the individual. For all dependent variables, each cell reports the OLS estimate of coefficient β in Equation (2). 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. [^], *, ** and *** indicate significance at the 10, 5, 1 and 0.1% levels.

Table 8: Non-linear effects by five-year age groups

	(1)	(2)	(3)	(4)	(5)
Non-linear age at survey	Tot. births	Employed	Paid in cash	Years of ed.	Completed primary ed.
(malaria _{2000j} × exposure) x DV(5-10)				0.973*** (0.125)	0.128*** (0.013)
(malaria _{2000j} × exposure) x DV(10-15)				0.646*** (0.165)	0.101*** (0.018)
(malaria _{2000j} × exposure) x DV(15-20)	-2.621*** (0.511)	0.361*** (0.109)	0.234 [^] (0.138)	0.028 (0.262)	0.016 (0.032)
(malaria _{2000j} × exposure) x DV(20-25)	-2.655*** (0.623)	0.331* (0.134)	0.194 (0.166)	-0.594 (0.396)	0.032 (0.041)
(malaria _{2000j} × exposure) x DV(25-30)	-2.310* (0.751)	0.334* (0.163)	0.262 (0.195)	-1.634* (-0.539)	-0.058 (0.053)
(malaria _{2000j} × exposure) x DV(30-35)	-2.529* (0.884)	0.356 [^] (0.191)	0.304 (0.227)	-0.869 (0.676)	0.023 (0.066)
(malaria _{2000j} × exposure) x DV(35-40)	-2.325* (1.050)	0.359 [^] (0.218)	0.312 (0.258)	-0.687 (0.795)	0.052 (0.078)
(malaria _{2000j} × exposure) x DV(above 40)	-3.904* (1.241)	0.462 [^] (0.256)	0.473 (0.300)	1.464 (0.987)	0.235* (0.095)
R ²	0.666	0.334	0.412	0.635	0.538
Observations	646,169	896,857	607,572	2,850,716	2,850,716
Age-by-Region	yes	yes	yes	yes	yes
Exposure-by-region	yes	yes	yes	yes	yes

Notes: The unit of observation is the individual. For all dependent variables, each cell reports the OLS estimate of coefficient β in Equation (2). 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. The regression also controls for exposure × DV(Age group), malaria_{2000j} × DV(Age group) and DV(Age group). Standard errors (in parentheses) are clustered at the DHS cluster level. [^], *, ** and *** indicate significance at the 10, 5, 1 and 0.1% levels.

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 the adult's surviving offspring.¹ We assume that the household cares about its own consumption c and leisure ℓ 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:

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

where

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

¹The 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). Adult and child mortality shocks can also bring about opposing influences on fertility (Boucekkine, Desbordes and Latzer, 2009). 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 σ the elasticity of substitution between these two inputs. Therefore, $\sigma = \frac{1}{1-\rho}$. The lower (resp. the higher) ρ , 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 ρ 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 time spent caring for surviving child(ren). We denote by τ the time the parent dedicates to each surviving child, with $\tau > 0$.³ As for each surviving child, the parent allocates time between education and child labor. Put differently, we assume that education and child labor are mutually exclusive.⁴ (Child labor may also extend to non-remunerative domestic tasks and other productive activities which we abstract from here.) 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

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

The parameters of the model are chosen to yield an interior solution for the optimal number of births b^* , the optimal level of leisure ℓ^* 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 he/she generates. Otherwise, the optimal number of births is infinite.

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

³For the sake of simplicity, but without loss of generality, τ depends on neither e nor H .

⁴Evidence 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).

⁵Just 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 Λ^* and the optimal number of births b^* :

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

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

and

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

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 ℓ^* are given by Equations (1) to (3) respectively:

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

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

and

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

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

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

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 $\rho < \bar{\rho}_1$, with $\bar{\rho}_1 = -\frac{W\tau}{W\tau - wH}$; e^* decreases with child health otherwise.

Proof:

Deriving e^* with respect to H yields

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

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 the child's 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 ($\rho < \bar{\rho}_1$), 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 Λ^* increases with child health while the optimal number of births b^* decreases with child health if $\rho < \bar{\rho}_2$, with $\bar{\rho}_2 < \bar{\rho}_1$; Λ^* (resp. b^*) decreases (resp. increases) with child health otherwise.

Proof:

Deriving Λ^* 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 Λ^* (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 ρ when it is positive. Consequently, there exists a unique value of ρ , that we denote by $\bar{\rho}_2$, such that (i) $\frac{\partial e^*}{\partial H} = \frac{1 - e^*}{H}$; (ii) $\frac{\partial e^*}{\partial H} > \frac{1 - e^*}{H}$ when $\rho < \bar{\rho}_2$; (iii) $\frac{\partial e^*}{\partial H} < \frac{1 - e^*}{H}$ when $\rho > \bar{\rho}_2$. Given that $\frac{1 - e^*}{H}$ is strictly positive, $\bar{\rho}_2$ is necessarily lower than $\bar{\rho}_1 = -\frac{W \tau}{W \tau - wH}$.

To show that $\frac{\partial e^*}{\partial H}$ decreases with ρ 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

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

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 ρ . 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 ρ . Therefore, $\frac{\partial e^*}{\partial H}$ is decreasing with ρ when it is positive. \square

We know that an increase in child health decreases each child’s “price” by raising the child’s 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 while the 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 The extent to which other aspects of fertility, such as age at first birth, may be influenced by this dynamic in the medium-run is unclear. Female age at first birth in Sub-Saharan Africa appears to follow a relatively stable trajectory (Field et al., 2016). Therefore, though we include this outcome in our empirical study, we do not augment our simple theoretical framework with more detailed fertility outcomes.

Note that the condition of $\bar{\rho}_2 < \bar{\rho}_1$, 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.

⁶The variation in opposite directions of Λ^* 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.

2 Data

2.1 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 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); post-neonatal (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.

Age at first birth (women): Age at first birth of 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): From the DHS Person Recode, 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 (five, six or seven) 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.

2.2 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). 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. If he was exposed, for example, to \$1 per capita each year, he

⁷Yearly population data come from the World Development Indicators.

would therefore receive $\$3/7=\0.43 per capita per year over his lifetime. 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. In this case, if he received \$1 per capita for each year of exposure, he would therefore receive $\$8/12=\0.66 per capita per year over his lifetime.

2.3 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 insecticide treated net (ITN) use and access to artemisinin combination therapies (ACTs) from over one million households were combined with national malaria control programme data on ITN and ACT 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. The PfPR is a commonly reported index of malaria transmission intensity. PfPR reaches a peak after about two years and remains fairly constant in older children until age ten before declining throughout adolescence and adulthood. The 2-10 age group has been suggested as the more informative to describe malaria risk and is therefore used. 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.

Insecticide treated net usage (DHS): Proportion of households per region that had an ITN for sleeping and that always use an ITN for children under five (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).

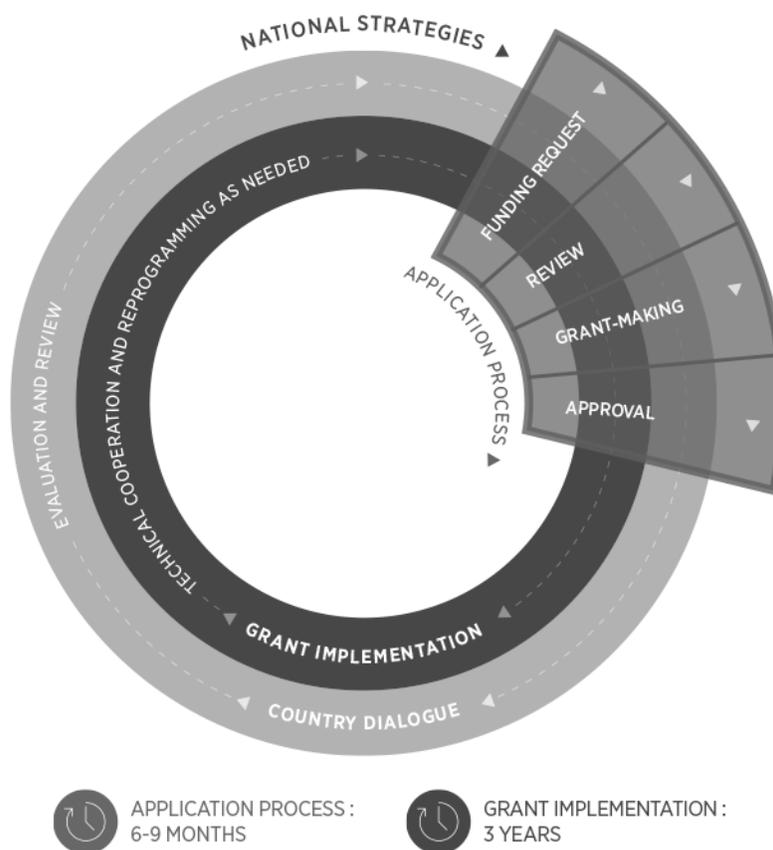
Chloroquine usage (DHS): Proportion of children under five per region treated for fever with chloroquine during past 2 weeks (DHS Child Recode).

2.4 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).

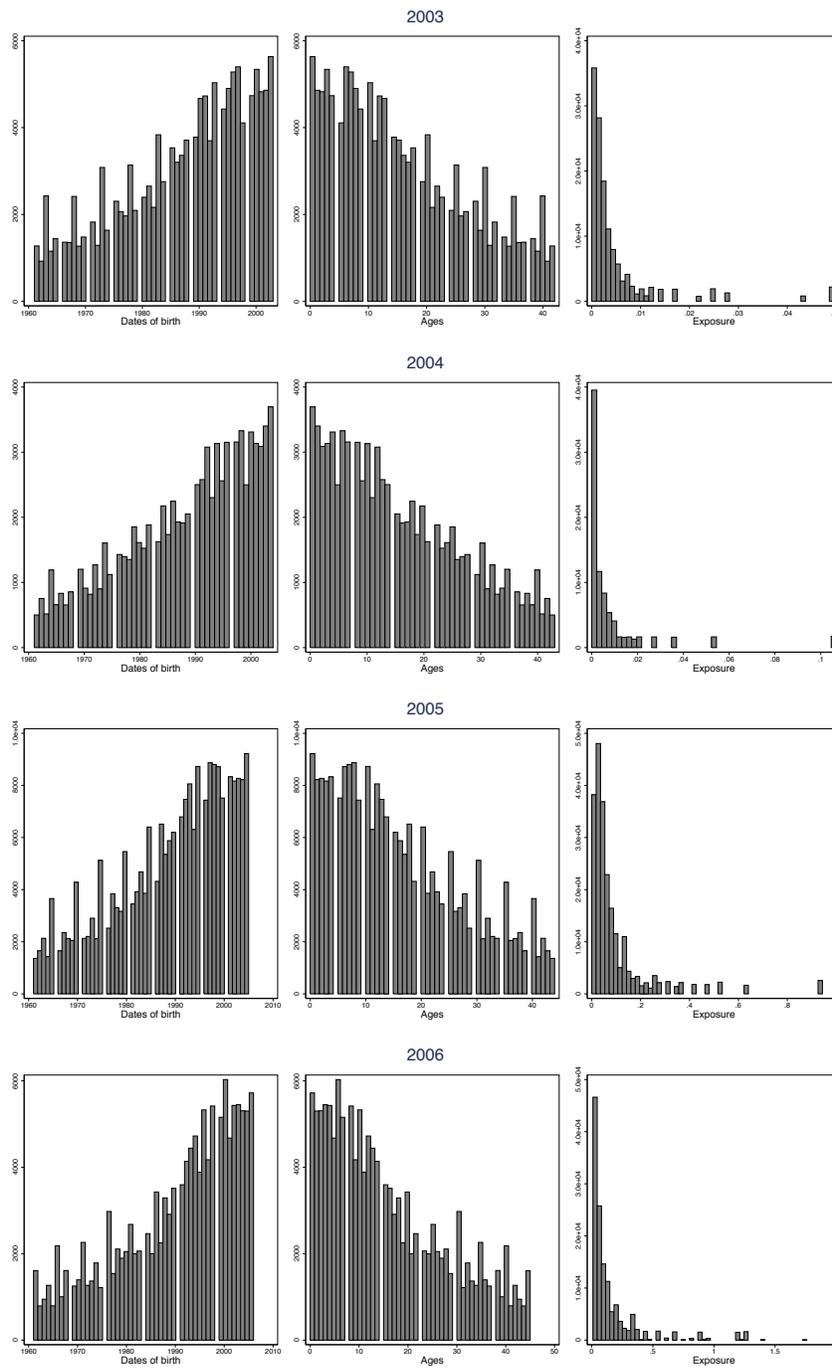
3 Figures

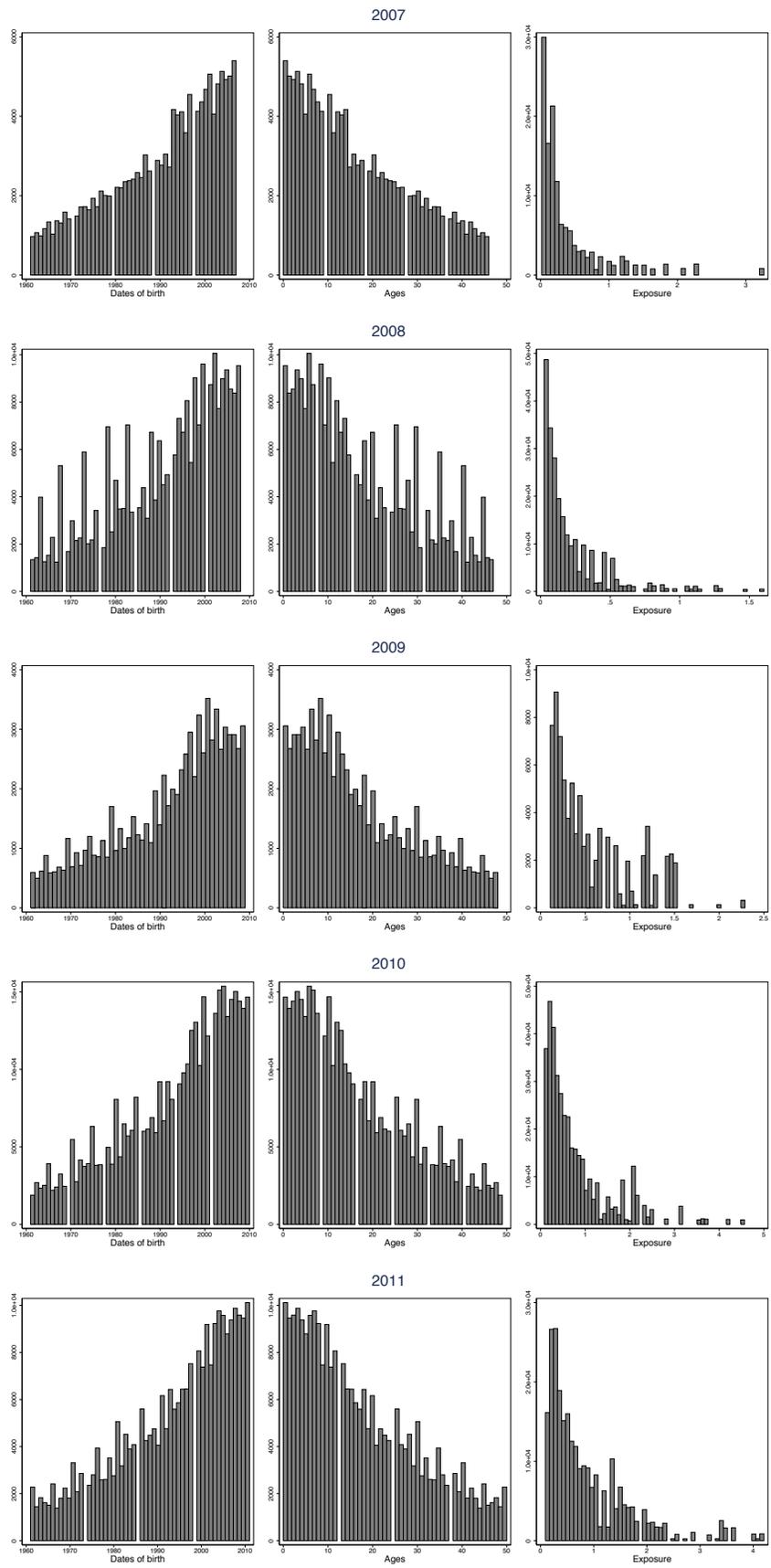
Figure A1: Example - The Global Fund funding process and steps



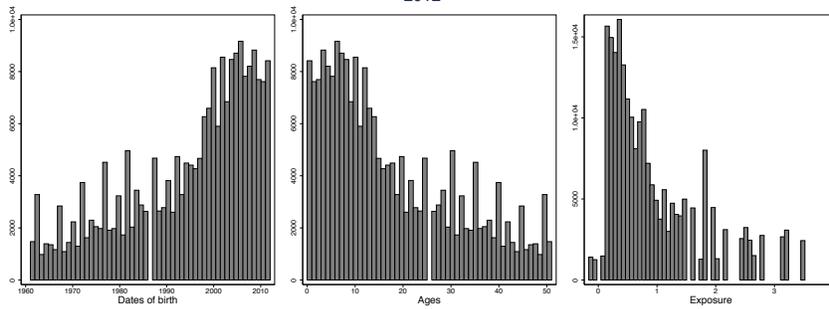
Notes: We obtained this figure from <http://www.theglobalfund.org/en/fundingmodel/process/>.

Figure A2: Frequency distributions of date of birth, age and Roll Back Malaria exposure by year of survey

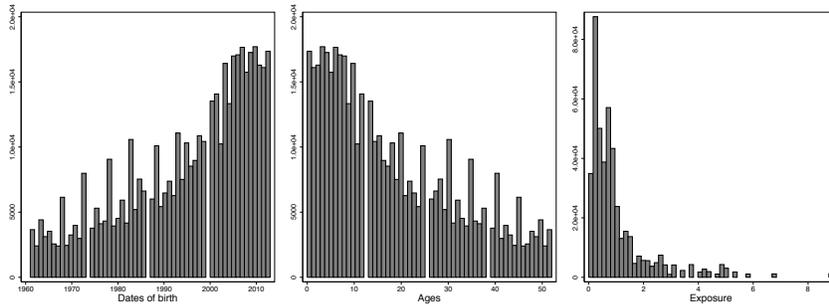




2012



2013



2014

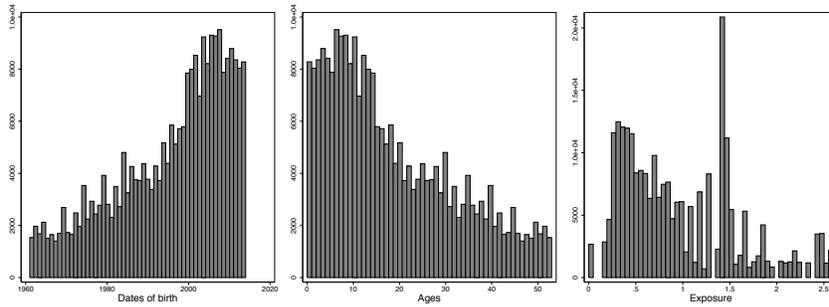
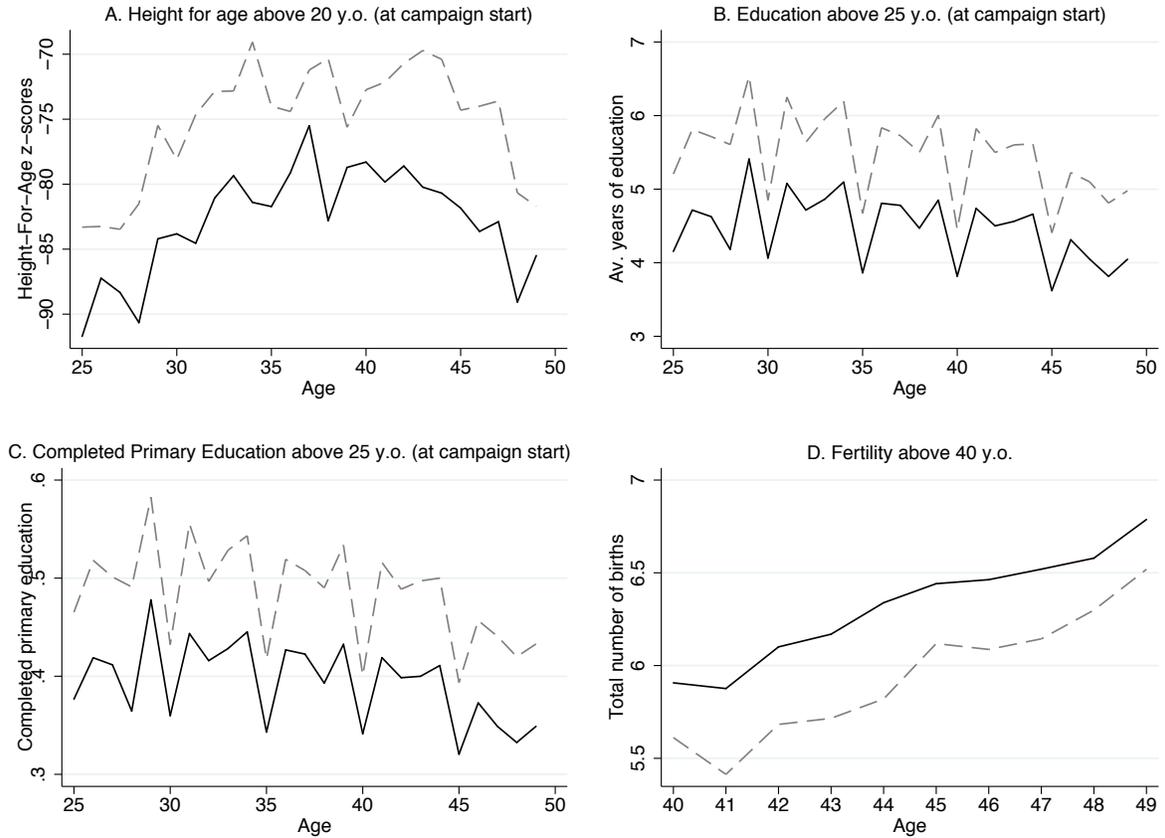


Figure A3: Ruling out pre-campaign catch-up effects



Notes: This figure considers individuals belonging to two groups: those living in clusters where malaria risk was above the median level in 2000 (solid line) versus where malaria risk was below the median level in 2000 (dashed line). We plot outcome variables against age for respondents above the age of 25 (above 40 in the case of plot D) at the start of anti-malaria campaigns to demonstrate parallel trends in high versus low exposure to malaria risk.

4 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					
<i>Child survival and health</i>					
Infant mortality	0.065	0.247	0	1	353,650
Neonatal mortality	0.032	0.176	0	1	353,650
Post-neonatal mortality	0.033	0.179	0	1	353,650
<i>Fertility</i>					
Total number of births	2.757	2.722	0	18	646,208
Age at first birth	19.095	3.779	12	45	487,700
<i>Adult labor supply</i>					
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
<i>Education</i>					
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: Controlling for mother fixed effects and additional controls

	(1)	(2)	(3)	(4)	(5)	(6)
Dep. var.	Infant		Neonatal		Post-neonatal	
Panel A: Mother FE						
malaria _{2000j} × exposure _{Nct}	-0.414*** (0.041)	-0.049 (0.037)	-0.195*** (0.024)	0.013 (0.047)	-0.219*** (0.023)	-0.061 (0.050)
R ²	0.564	0.875	0.565	0.755	0.536	0.676
Observations	129,636	90,813	129,636	90,813	129,636	90,813
Age-by-Region	no	yes	no	yes	no	yes
Exposure-by-Region	no	yes	no	yes	no	yes
Mother FE	yes	yes	yes	yes	yes	yes
Panel B: Same sub-sample without mother FE						
malaria _{2000j} × exposure _{Nct}	-0.450*** (0.042)	-0.085* (0.034)	-0.213*** (0.024)	-0.021 (0.052)	-0.237*** (0.023)	-0.064 (0.057)
R ²	0.230	0.792	0.213	0.569	0.200	0.453
Observations	129,636	90,813	129,636	90,813	129,636	90,813
Age-by-Region	no	yes	no	yes	no	yes
Exposure-by-Region	no	yes	no	yes	no	yes
Mother FE	no	no	no	no	no	no
Panel C: Full sample with additional controls						
malaria _{2000j} × exposure _{Nct}	-0.470*** (0.037)	-0.065* (0.029)	-0.196*** (0.020)	0.018 (0.037)	-0.274*** (0.022)	-0.083* (0.041)
R ²	0.137	0.739	0.112	0.468	0.117	0.404
Observations	276,898	204,936	276,898	204,936	276,898	204,936
Age-by-Region	no	yes	no	yes	no	yes
Exposure-by-Region	no	yes	no	yes	no	yes
Mother FE	no	no	no	no	no	no
Panel D: Controlling for Age-by-Region						
malaria _{2000j} × exposure _{Nct}	-0.453*** (0.034)	-0.061* (0.023)	-0.208*** (0.018)	0.005 (0.034)	-0.245*** (0.019)	-0.066 ^a (0.036)
R ²	0.116	0.742	0.094	0.467	0.096	0.381
Observations	353,379	258,809	353,379	258,809	353,379	258,809
Age-by-Region	no	yes	no	yes	no	yes
Exposure-by-Region	no	yes	no	yes	no	yes
Mother FE	no	no	no	no	no	no
Panel E: Replacing Age-by-Region by Age-by-Malaria						
malaria _{2000j} × exposure _{Nct}	-0.414*** (0.041)	-0.332*** (0.053)	-0.195*** (0.024)	-0.147* (0.052)	-0.219*** (0.023)	-0.185*** (0.052)
R ²	0.564	0.799	0.565	0.707	0.536	0.645
Observations	129,636	90,813	129,636	90,813	129,636	90,813
Age-by-Region	no	yes	no	yes	no	yes
Exposure-by-Region	no	yes	no	yes	no	yes
Mother FE	no	no	no	no	no	no

Notes: Each cell reports the OLS estimate of coefficient β in Equation (2). 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. The additional controls in Panel C are mother's age and length of preceding birth interval. Standard errors (in parentheses) are clustered at the DHS cluster level. ^a, *, ** and *** indicate significance at the 10, 5, 1 and 0.1% levels.

Table A4: Controlling for household fixed effects

Dep. var.	(1)	(2)	(3)	(4)	(5)	(6)
	Tot. births	Age at first birth	Employed	Paid in cash	Years of ed.	Completed primary ed.
Panel A: Household FE						
$\text{malaria}_{2000j} \times \text{exposure}_{Nct}$	-4.669*** (0.383)	0.507 (1.245)	0.544*** (0.067)	0.033 (0.094)	2.237*** (0.112)	0.213*** (0.011)
R ²	0.847	0.588	0.614	0.675	0.743	0.653
Observations	302,473	112,936	636,443	352,967	2,773,967	2,773,967
Age-by-Region	yes	yes	yes	yes	yes	yes
Exposure-by-Region	yes	yes	yes	yes	yes	yes
Household FE	yes	yes	yes	yes	yes	yes
Panel B: Same sub-sample without Household FE						
$\text{malaria}_{2000j} \times \text{exposure}_{Nct}$	-4.616*** (0.037)	0.477 (1.126)	0.517*** (0.062)	0.110 (0.082)	2.358*** (0.113)	0.222*** (0.011)
R ²	0.741	0.309	0.356	0.426	0.633	0.534
Observations	302,473	112,936	636,443	352,967	2,773,967	2,773,967
Age-by-Region	yes	yes	yes	yes	yes	yes
Exposure-by-Region	yes	yes	yes	yes	yes	yes
Household FE	no	no	no	no	no	no
Panel C: Replacing Age-by-Region by Age-by-Malaria						
$\text{malaria}_{2000j} \times \text{exposure}_{Nct}$	-1.800*** (0.366)	1.606* (0.810)	0.245*** (0.072)	0.068 (0.090)	1.331*** (0.120)	0.153*** (0.012)
R ²	0.664	0.228	0.333	0.412	0.628	0.532
Observations	646,169	464,336	896,857	607,572	2,850,716	2,850,716
Age-by-Malaria	yes	yes	yes	yes	yes	yes
Exposure-by-Region	yes	yes	yes	yes	yes	yes
Household FE	no	no	no	no	no	no

Notes: Each cell reports the OLS estimate of coefficient β in Equation (2). 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. $\hat{\cdot}$, *, ** and *** indicate significance at the 10, 5, 1 and 0.1% levels.

Table A5: Removal of potential outlying countries

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
	No CI	No CD	No ET	No KE	No LB	No NG	No SL	No TZ	No ZM	Mult. surveys
Infant	-0.148*	-0.164***	-0.149*	-0.122*	-0.165*	-0.133*	-0.149*	-0.156*	-0.149*	-0.183*
	(0.048)	(0.049)	(0.047)	(0.049)	(0.053)	(0.044)	(0.047)	(0.050)	(0.048)	(0.056)
R ²	0.281	0.277	0.280	0.285	0.283	0.310	0.286	0.274	0.268	0.244
Observations	347,929	336,529	334,539	331,016	343,760	308,887	342,072	347,697	339,754	293,595
Tot. births	-3.806***	-3.935***	-3.839***	-3.906***	-4.137***	-4.284***	-3.638***	-3.877***	-3.757***	-3.715***
	(0.291)	(0.298)	(0.291)	(0.312)	(0.316)	(0.297)	(0.291)	(0.296)	(0.309)	(0.317)
R ²	0.667	0.667	0.664	0.666	0.667	0.668	0.667	0.666	0.665	0.672
Observations	636,453	619,683	604,825	599,616	630,320	568,333	622,339	636,438	622,933	537,894
Age at first birth	0.927	1.388*	0.944	0.202	1.227	1.110 [^] a	1.076	0.936	0.947	1.394*
	(0.654)	(0.666)	(0.651)	(0.702)	(0.757)	(0.656)	(0.655)	(0.664)	(0.721)	(0.706)
R ²	0.231	0.232	0.233	0.229	0.231	0.224	0.232	0.230	0.230	0.232
Observations	457,099	444,924	438,131	430,300	451,552	410,038	446,421	457,308	446,828	385,247
Employed in last 12 mo.	0.470***	0.470***	0.488***	0.519***	0.509***	0.586***	0.430***	0.525***	0.422***	0.497***
	(0.057)	(0.059)	(0.057)	(0.062)	(0.062)	(0.060)	(0.057)	(0.059)	(0.062)	(0.062)
R ²	0.335	0.335	0.337	0.330	0.334	0.336	0.335	0.333	0.333	0.330
Observations	882,587	859,110	836,870	847,802	871,433	786,627	863,378	884,704	853,881	742,553
Years of ed.	2.347***	2.267***	2.406***	2.554***	2.331***	2.539***	2.281***	2.416***	2.310***	2.290***
	(0.114)	(0.115)	(0.112)	(0.116)	(0.117)	(0.110)	(0.111)	(0.119)	(0.118)	(0.128)
R ²	0.637	0.632	0.636	0.520	0.637	0.629	0.639	0.634	0.630	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	2,742,652	2,377,194

Notes: The unit of observation is the individual. For all dependent variables, each cell reports the OLS estimate of coefficient β in Equation (2). 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 (except infant mortality), 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.

Table A6a: Infant mortality estimates when substituting alternative treatment probabilities for initial malaria risk (*Plasmodium falciparum* parasite rate)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
	Infant			Neonatal			Post-neonatal		
Exposure* Δ Malaria	0.330*			0.106			0.224*		
	(0.106)			(0.086)			(0.175)		
Exposure* Δ ACTs		-2.430*			-0.782			-1.647*	
		(0.854)			(0.647)			(0.617)	
Exposure* Δ Bednets			-0.737*			-0.237			-0.500*
			(0.244)			(0.195)			(0.178)
R ²	0.278	0.266	0.270	0.182	0.180	0.180	0.175	0.164	0.169
Observations	353,379	353,379	353,379	353,379	353,379	353,379	353,379	353,379	353,379
Age-by-Region	no	no	no	no	no	no	no	no	no
Exposure-by-region	yes	yes	yes	yes	yes	yes	yes	yes	yes

Notes: The unit of observation is the individual. For all dependent variables, each cell reports the OLS estimate of coefficient β in Equation (2). Malaria risk and bednets vary at the regional level, while ACTs vary primarily at the national level. 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. $\hat{\cdot}$, *, ** and *** indicate significance at the 10, 5, 1 and 0.1% levels.

Table A6b: Fertility, employment and education estimates when substituting alternative treatment probabilities for initial malaria risk (*Plasmodium falciparum* parasite rate)

	(1)	(2)	(3)	(4)	(5)	(6)
	Tot. births			Age at first birth		
Exposure* Δ Malaria	8.056*** (0.640)			-2.069 (1.415)		
Exposure* Δ ACTs		-70.710*** (7.992)			24.560 (17.010)	
Exposure* Δ Bednets			-9.945*** (0.826)			3.102 (2.118)
R ²	0.666	0.661	0.665	0.230	0.229	0.230
Observations	646,169	646,169	646,169	464,336	464,336	464,336
	Employed last 12 mo.			Paid in cash for emp.		
Exposure* Δ Malaria	-1.027*** (0.123)			-0.357* (0.140)		
Exposure* Δ ACTs		9.326*** (1.384)			2.641* (1.048)	
Exposure* Δ Bednets			1.302*** (0.158)			0.412* (0.161)
R ²	0.334	0.330	0.333	0.412	0.412	0.412
Observations	896,857	896,857	896,857	607,572	607,572	607,572
	Years of ed.			Completed primary ed.		
Exposure* Δ Malaria	-4.683*** (0.250)			-0.437*** (0.024)		
Exposure* Δ ACT		43.723*** (3.926)			4.076*** (0.369)	
Exposure* Δ Bednets			7.288*** (0.404)			0.679*** (0.039)
R ²	0.631	0.615	0.631	0.535	0.522	0.535
Observations	2,850,716	2,850,716	2,850,716	2,850,716	2,850,716	2,850,716
Age-by-region	yes	yes	yes	yes	yes	yes
Exposure-by-region	yes	yes	yes	yes	yes	yes

Notes: The unit of observation is the individual. For all dependent variables, each cell reports the OLS estimate of coefficient β in Equation (2). Malaria risk and bednets vary at the regional level, while ACTs vary primarily at the national level. 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. \wedge , *, ** and *** indicate significance at the 10, 5, 1 and 0.1% levels.

Table A7: Alternative infant health variables

	(1)	(2)	(3)	(4)	(5)	(6)
	Acute respiratory infection	Diarrhea	BCG	DPT	Polio	Measles
$\text{malaria}_{2000j} \times \text{exposure}_{Nct}$	0.044	-0.003	-0.039 [^]	-0.023	-0.030	-0.012
	(0.063)	(0.022)	(0.021)	(0.026)	(0.028)	(0.022)
R ²	0.296	0.152	0.433	0.436	0.313	0.479
Observations	108,254	469,021	478,155	476,563	477,797	475,255
Age-by-Region	no	no	no	no	no	no
Exposure-by-Region	yes	yes	yes	yes	yes	yes

Notes: The unit of observation is the individual. For all dependent variables, each cell reports the OLS estimate of coefficient β in Equation (2). 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. [^], *, ** and *** indicate significance at the 10, 5, 1 and 0.1% levels.

Table A8: Estimation of artificial treatments

	(1)	(2)	(3)	(4)	(5)	(6)
	Tot. births	Age at first birth	Employed	Paid in cash	Years of ed.	Completed primary ed.
20 years	1.851***	-5.111***	-0.306***	0.005	-3.186***	-0.223***
	(0.344)	(0.820)	(0.086)	(0.097)	(0.551)	(0.063)
R ²	0.595	0.211	0.296	0.419	0.593	0.507
Observations	521,530	441,686	717,674	531,953	1,274,398	1,274,398
30 years	1.735*	1.326	-0.235*	-0.014	0.965	0.095
	(0.695)	(1.380)	(0.108)	(0.110)	(0.837)	(0.093)
R ²	0.539	0.231	0.332	0.456	0.603	0.515
Observations	311,351	278,524	431,628	335,523	861,112	861,112
Age-by-Region	yes	yes	yes	yes	yes	yes
Exposure-by-Region	yes	yes	yes	yes	yes	yes

Notes: The unit of observation is the individual. For all dependent variables, each cell reports the OLS estimate of coefficient β in Equation (2). 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. $\hat{\cdot}$, *, ** and *** indicate significance at the 10, 5, 1 and 0.1% levels.

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