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 an unprecedentedly rich set of human capital outcomes in 27 countries in Sub-Saharan Africa. Using pre-campaign malaria risk as a proxy for treatment probability, we first run a standard difference-in-differences strategy that exploits and synthesizes the period and cohort dimensions of our survey data. We then develop a more sophisticated empirical strategy that takes advantage of individuals' continuous exposure to campaign timing and disbursements. The estimates reveal a globally positive impact of health aid. A conservative interpretation shows that the campaign reduces infant mortality (5.2 percentage points) and fertility (0.4 births) and increases adult labor supply (5.3 percentage points) and educational attainment (0.5 years), although this average treatment effect hides variation by demographic characteristics and age groups. Our results underscore the importance of understanding how the effects of large-scale efforts to reduce disease extend beyond health outcomes.

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). This paper is the first to study the response of an unprecedentedly rich set of human capital outcomes to malaria control efforts in 27 Sub-Saharan African countries.

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.

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

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

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

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

This massive reduction in malaria-related mortality may have effects that reach beyond health. Improving early childhood health paves the way for greater educational attainment. But it also raises the opportunity cost of education by increasing a child's potential wages on the labor market (Bleakley, 2010) and efficiency at completing domestic tasks. This, in turn, can influence adult fertility and labor decisions by modifying 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 proceeds stepwise. Using pre-campaign malaria risk as a proxy for treatment probability, we first run a standard difference-in-differences strategy that exploits the period and cohort dimensions of our survey data. Doing so allows us to synthesize econometric approaches previously restricted to single dimensions. We then develop a more sophisticated empirical strategy that takes advantage of individuals' continuous exposure to campaign timing and disbursements. More precisely, we compare the outcomes of individuals with a continuous combination of time, cohort and country characteristics that command (or would command) high and low treatment intensities in the treated (highly malarious) and untreated (less malarious) groups. 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 climatic characteristics. This measure differs from previous studies relying on similar household data which tend to use possibly endogenous 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

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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 malaria risk would be reductionist in this context. Treatment depends predominantly on the timing and intensity of RBM campaigns across cohorts in a given country. This implies that two treated individuals surveyed in two different years in the same country receive 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, conditional on being treated, 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 rich identifying variation in assignment to treatment at the cluster level and intensity of treatment at the country-cohort-survey year level.

A conservative interpretation of our results, which are not driven by pre-campaign catch-up effects between treated and untreated populations, show that RBM campaigns reduce infant mortality (by a probability of 5.2 percentage points) and fertility (by 0.4 births), while increasing adult labor supply (by a probability of 5.3 percentage points) and educational attainment (by 0.5 years). 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 educa-

tional 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). In this way, we complement the existing literature on the role of vertical, single-disease health aid in reducing the burden from tropical diseases. As one of the few studies to identify aid effects at a sub-national level, this paper also contributes to the literature on health aid and economic development. 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.[5] 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.

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[5]We test fertility outcomes with our preferred empirical strategy as well as with a mother-year panel of birth events.

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

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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 data

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 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.[6] DHS uses a stratified two-stage cluster design. This design first draws enumeration areas from national census files, followed by a sample of households drawn from each enumeration area.

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.

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, evidence is consistent with the assumption that campaigns targeted regions with the highest pre-campaign malaria

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

risk.[7]

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 MAP (Bhatt et al., 2015).[8] 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.[9] The MAP subjects this model to a high number of site-date specific reality checks.[10] 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 ×5 km over the African continent, assigning a pre-campaign malaria risk measure to each geocoded DHS cluster.[11] 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 began. Figure 3a shows a precipitous decrease in mean malaria risk, particularly from 2003

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[7]We could equally assume that the greatest effects of the campaign occurred in the highest risk areas, either due to targeting or due to the fact that the most malarious areas stood to gain the most from anti-malaria campaigns.

[8]We sincerely thank Peter Gething for providing the yearly data (from 2000 to 2012) through personal communication for an earlier version of this paper.

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

[10]See http://www.map.ox.ac.uk/explorer/.

[11]Note 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.

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 passes and the post-RBM period begins. Conditioning the use of malaria control techniques on initial malaria risk produces a similar result. The higher the level of malaria risk in 2000, the greater the increase in insecticide treated net usage in Figures 5a and 5b (which use two different bednet measures from the DHS and MAP, respectively). In Figures 5c and 5d, we examine the trade-off between drugs administered for fever to children under five. Due to its lower effectiveness, chloroquine waned in popularity as a first-line treatment, and the prescription of ACTs increased instead (Flegg et al., 2013).[12] 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

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

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 grow-ing 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, gen-eral weaknesses in the health care infrastructure, and economic shocks that reduced government spending per capita on health care. The malaria situation worsened, and fatalism and resignation towards the disease became widespread."

The RBM Partnership formed in reaction to the deteriorating state of malaria control efforts. 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) es-timate 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 combi-

nation therapies, and indoor residual spraying, respectively.[13] Studying countries impacted by the President's Malaria Initiative, Jakubowski et al. (2017) estimate that the under-5 mortality rate decreased from 28.9 to 24.3 (per 1,000 person-years) within the period of 1995 to 2014. Finally, 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 Fund[14] (since 2003), the President's Malaria Initiative (since 2006), and the World Bank Booster Program for Malaria Control in Africa (since 2006). We observe disbursements at the level of the country-year. We use this information to compute a respondent's exposure as the yearly amount per capita[15] disbursed at the country level during an individual's lifetime by these three primary funders. An individual's lifetime is defined as the difference between the DHS survey year and his or her year of birth, from which we subtract one year. We consider exposure to begin in utero (though defining the start of exposure with the year after birth does not alter our results). (See the Supplemental Appendix for further details.)

An individual's exposure depends on his or her date of birth which is difficult to predict. Furthermore, we use in many cases multiple surveys per country, the timing of which is also difficult to systematically anticipate with respect to high-level DHS, organizational, and national priorities. This produces an exposure to treatment which varies from -0.162[16] 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 include the latter table to illustrate the variability of exposure, which is substantial, across these dimensions. We also present frequency distributions of date of birth, age and exposure by survey year in Figure A2.

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

[14]We show the funding process and steps, downloaded from the Global Fund's website, in Figure A1.

[15]Yearly population data come from the World Development Indicators. Using disbursement per capita may reduce the magnitude of the impact since anti-malaria campaigns aim to target certain segments of the population.

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

It is important that the timing and intensity of the RBM disbursements were not anticipated by the target population. If households anticipated better child health outcomes, for example, they could have modified decisions on fertility, labor supply or educational investments prior to the campaign's start. But the likelihood that the average citizen would have predicted the scale-up of RBM campaigns is low. The establishment of the Global Fund in 2002 marked RBM scale-up. The Global Fund itself evolved out of a series of high-level discussions between donors and multilateral agencies that began toward the end of 1999. These discussions notably culminated with the sixth of the eight Millennium Development Goals established following the Millennium Summit of the United Nations in 2000: "To combat HIV/AIDS, malaria, and other diseases." Moreover, it was only in 2011 that the Global Fund began to advertise its activities in countries of operation.[17] It is thus doubtful that the establishment of this Global Fund and its subsequent disbursements were anticipated by the general population of beneficiary countries.

3.3   Outcomes of interest

The DHS provide our outcomes of interest. 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 death events for each year of birth cohort.

To measure fertility, we rely on two questions from the women's questionnaire: the

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

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). As we show in our theoretical framework, mothers face a trade-off between working and raising children depending on the cost of each additional child. 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 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. For all dependent variables, we pool surveys for each country to create pseudo-panels by year of birth cohort. Additionally, for a follow-on analysis, we create a panel of birth events by mother-birth year. In this panel, the dependent variable is a binary indicator for a birth event in a given year, which is similar to our panel of infant mortality events.

4 Empirical strategy

4.1 Econometric specifications

We aim to isolate the treatment effect by comparing the outcomes of individuals with characteristics that 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. We build our strategy stepwise. First, we use the period dimension of our survey data, relying on pre-campaign malaria risk as a proxy for treatment probability. We then incorporate binary and continuous cohort intensity of treatment that takes advantage of individuals' exposure to anti-malaria campaign timing and disbursements. In this way, we synthesize the period and cohort dimensions of our dataset.

4.1.1 Period model

We begin with a simple period model, similar in spirit to Lucas (2013), which computes the difference-in-differences estimator from Equation 1:

$$y_{ijt} = \beta_1 + \beta_2 \text{highmalaria}_{2000j} + \beta_3 \text{post}_t + \beta_4(\text{highmalaria}_{2000j} \times \text{post}_t) + \epsilon_{ijt} \qquad (1)$$

where y_{ijt} is an outcome of individual i in DHS cluster j surveyed in year t. The variable post_t is a binary indicator denoting a respondent *interviewed* after the start of the campaign. The coefficient β_4 is the treatment effect.[18] Low versus high malaria risk is defined within countries with respect to median pre-campaign risk, while post_t 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 models incorporate 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.

4.1.2 Cohort model

One of the simplest cohort versions of Equation (1) changes the definition of post_t to account for a respondent's year of birth cohort c. Instead of basing post_t on a respondent's date of survey, this approach defines a new post_{Nc} as an indicator for *being born* after the start of the campaign in a given country. Equation (2) describes this model:

$$y_{ijct} = \beta_1 + \beta_2 \text{post}_{Nc} + \beta_3(\text{malaria}_{2000j} \times \text{post}_{Nc}) + \mathbf{X_{ijct}}'.\mathbf{\Gamma} + \delta_{Nc} + \delta_j + \epsilon_{ijct} \qquad (2)$$

We regress a respondent's individual outcome on an interaction term between the probability of belonging to the treated group and the treatment intensity (post_{Nc}). Instead of a binary indicator, the former assigns a continuous probability of pre-campaign malaria risk (from 0

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[18]In this equation, the time dimension, the date of interview, is similar to a period analysis. In this case, the coefficient β_4 = [(highmalaria$_1$, post$_1$) - (highmalaria$_0$, post$_0$)] - [(highmalaria$_1$, post$_1$) - (highmalaria$_0$, post$_0$)].

to 1) to individuals at a localized geographic level. More precisely, malaria$_{2000j}$ measures the level of the PfPR in 2000 in DHS cluster j, hence its pre-campaign malaria risk. Finally, the vector $\mathbf{X_{ijct}}$ includes individual covariates gender and wealth[19] 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 fixed effects, δ_{Nc}.

However, this approach has two drawbacks: First, we can only test this model on dependent variables measured in cohorts encompassing respondents born both before and after the campaign start date. In other words, this approach is relevant only for infant mortality and child educational outcomes. Indeed, we observe fertility outcomes only among women ages 15 to 45. If the campaign's start date is 2003 at the earliest and DHS surveys reach 2014 at the latest, a woman born in the post-RBM period will be too young to have given birth by 2014. This limitation does not apply to a panel of birth events by mother-birth year. The same reasoning applies to employment variables measured among adults ages 15 to 59. Second, a simple binary measure of campaign exposure does not account for the wide variation in respondents' experience with anti-malaria programs. In other words, this simple approach captures the extensive margin of anti-malaria campaigns, and we move to the intensive margin with the following specifications.

4.1.3 Duration model (period-cohort)

We can therefore build on Equation 2 by comparing the outcomes of individuals with a continuous combination of time, cohort and country characteristics that command high and low treatment intensities in the treated (highly malarious) and untreated (less malarious) groups. As before, malaria$_{2000j}$ represents a respondent's probability of belonging to the treated group. However, treatment intensity now exploits the variation in the timing of RBM disbursements relative to respondents' birth cohorts (c), DHS survey year (t), and country (N). Interacting these variables allows us to identify a causal pathway from RBM campaigns to human capital outcomes.[20] We present this new specification below:

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[19]Results from this equation and those that follow are also robust when excluding the wealth index.

[20]To focus on a post-colonization time frame, and therefore avoid concurrent shocks to health and educational policies, we restrict respondents to those born after 1960. However, we show in Table 7 that our baseline results hold when this restriction is lifted.

$$y_{ijct} = \alpha + \beta.(\text{malaria}_{2000j} \times \text{duration}_{Nct}) + \mathbf{X_{ijct}}'.\mathbf{\Gamma} + \delta_{Nct} + \delta_j + \epsilon_{ijct} \qquad (3)$$

where y_{ijct} is an outcome of individual i in DHS cluster j, belonging to cohort c and surveyed in year t. In this model, duration_{Nct} measures in a given country N the proportion of an individual's life spent post-campaign. As a function of country N, year of birth cohort c, and DHS survey year t, duration_{Nct} is defined by substantial variability. Relative to one another, births, DHS surveys, and campaign start dates are difficult to predict or anticipate. As the coefficient of the interaction term between malaria_{2000j} and duration_{Nct}, β identifies the treatment effect. As before, we include covariates $\mathbf{X_{ijct}}$, country-by-cohort-by-DHS year fixed effects δ_{Nct}, and DHS cluster fixed effects δ_j.

Yet duration_{Nct} fails to account for the intensity of RBM disbursements. As described in Section (3), RBM funding streams varied significantly within and between countries as well as over time. Our preferred specification replaces duration_{Nct} with exposure_{Nct}. This new measure of treatment intensity accounts for the yearly amount per capita disbursed during an individual's lifetime by the three primary external funders of the RBM campaigns.

4.1.4 Full exposure model (period-cohort-disbursements)

Our preferred specification thus makes full use of period, cohort and disbursement dimensions of our survey data, as well as continuous measures of probability and intensity of treatment:

$$y_{ijct} = \alpha + \beta.(\text{malaria}_{2000j} \times \text{exposure}_{Nct}) + \mathbf{X_{ijct}}'.\mathbf{\Gamma} + \delta_{Nct} + \delta_j + \epsilon_{ijct} \qquad (4)$$

Adopting this restrictive parameterization isolates a treatment effect based on assignment to treatment at the cluster level and a rich identifying variation in treatment intensity by a respondent's country, cohort and survey year. We further amend Equation (4) 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).[21] 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)).[22] Similarly, an area's pre-campaign malaria risk may induce mothers to postpone or anticipate birth decisions. Therefore, the impact of exposure to malaria control campaigns may vary depending on pre-campaign characteristics.

To mitigate these potential omitted variables biases, our tables display three columns of results per dependent variable. They report coefficient β (i) when Equation (4) is estimated; (ii) when the interactions between age_{ct} and region fixed effects are included;[23] (iii) and when, additionally, the interactions between exposure_{Nct} and region fixed effects are added.[24]

4.2.2   Pre-campaign trends

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 (4) captures the impact of the RBM campaigns or if it simply reflects pre-campaign trends.

To test for pre-campaign catch-up effects, we perform a falsification test. We estimate Equation (4) 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

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

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

[23]We 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}.

[24]We 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. $(\text{malaria}_{2000j} \times \text{exposure}_{Nct})$.

infants born prior to 2002 (where exposure is zero for those who die before the age of one and positive for those who die after the age of one or are still alive at the time of survey), 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 the campaign start date), 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. 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. Figure 7 provides a complementary graphical view using binary pre-/post-campaign malaria risk.

5 Results

5.1 Preliminary results

5.1.1 Correlations

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 correlations face several limitations. First, changes over time may be endogenous. An increase in antibiotic treatments, for example, might serve as a bellwether of greater malaria risk, rather than the reverse. Second, bednet usage faces potential issues of household selection. Third, the MAP provides coverage estimates with limited variation at the regional level (especially for ACTs) while DHS surveys do

not always include questions related to malaria treatment. This restricts the data available for identification. Despite these challenges, 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 the story that the RBM campaign is conducive to human capital accumulation. There are some minor inconsistencies — total births, age at first birth and probability of paid employment show signs opposite of expectations when regressed on ACTs, though these coefficients are likely due to the challenges mentioned above.

5.1.2 Period, cohort and period-cohort models

Our period model from Equation (1) supports this interpretation. Table 3 reports the results of this analysis, comparing the changes in outcomes in highly or less malarious DHS clusters between pre- and post-campaign time periods. 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.[25] Table 3 shows that the decrease (resp. increase) in infant mortality (resp. adult labor supply and education) between the post- and pre-campaign periods is greater in DHS clusters that show high rather than low pre-campaign malaria risk. For the total number of births, there is an increase in both treated and untreated areas but the increase in treated areas is more modest, as expected. Similarly, for age at first birth, there is a decrease in both treated and untreated areas but the decrease in the treated areas is more modest.

In Figure 8, 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

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[25]Our results are also robust to defining pre- and post-campaign relative to when the campaign began in each individual country.

infant mortality and total fertility and upward for the probability of paid employment and years of education completed. However, Table 3 and Figure 8 do not account for individual exposure to campaign disbursements.

We further investigate these preliminary findings by estimating Equations (2) and (3). The former is a cohort model with continuous treatment probability while the latter combines both period and cohort dimensions by additionally incorporating continuous intensity of treatment. Table 4 reports these results. Equation (2), due to its dependence on respondents born after the campaign, presents results only for infants and children. Being born after the initiation of RBM leads to a decrease in the probability of infant, neonatal and post-neonatal mortality and an increase in both the years of education completed and the probability of completing primary education. Regarding Equation (3), we observe in columns 1 through 3 that a marginal increase in the lifetime duration of anti-malaria campaigns is negatively related to the likelihood of infant, neonatal and post-neonatal mortality, but not significantly so for the two latter measures. However, this type of specification is not necessarily well-suited to investigating infant mortality, for which there can be only limited variation in lifetime RBM duration. Moving to adult outcomes, we observe a negative and significant relationship between lifetime campaign duration and a woman's total number of births. Consistent with this finding, an increase in the relative duration of RBM campaign produces a positive effect on age at first birth. Moreover, the effect is also positive for the remaining dependent variables: the probability of adult employment and being remunerated in cash, and the number of years of education and probability of completing primary education. These results suggest that increasing the proportion of a respondent's life under anti-malaria campaigns brings about an overall improvement in human capital outcomes. Nevertheless, we can be more precise about the effect of an individual's exposure to RBM by taking into account country- and year-specific disbursements and interpreting the magnitude in the following section.

5.2 Main results

Our main results rely on Equation (4), which supplements the period and cohort dimensions of the previous specifications with information on RBM disbursement intensity throughout a respondent's lifetime.

5.2.1 Infant mortality

For children, their dates of birth relative to the start of the campaign as well as the timing and intensity of disbursements over the period of their lives exposed to RBM contribute to identify the effect. These characteristics depend on the date of DHS interview and the program timing per country. Columns 1 through 6 of Table 5 report the OLS estimates of Equation (4) 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 Panel D of 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 5, 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[26] 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 the probability of a child's survival to 60 months increased by approximately 2.4 percentage points for cohorts treated by the Measles Initiative campaigns in Sub-Saharan Africa. Hookworm disease has also been the subject of particular attention in the economic literature, but this disease is rarely lethal (Bleakley and Lange, 2009). In Kenya, Pathania (2014) finds that malaria control induced a decrease in post-neonatal mortality of 33%. In their analysis of the effects of the President's Malaria Initiative, Jakubowski et al. (2017) estimate a 14-16% reduction in under-five mortality. While similar in magnitude to our results prior to the treatment effect transformation, this study uses coarser treatment measures: an indicator for whether a country received funding in a given year and per-capita disbursements at the

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[26]We show in subsequent sections that this incremental increase is equivalent to one additional dollar per capita, per year of RBM disbursements.

country-year level. Effectiveness could run not only through the reduction in malaria burden but through additional channels, such as health systems strengthening or better childhood resilience to other diseases, thereby increasing the magnitude of the observed effect. 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$_{2000j}$. We can control for mother fixed effects given that a mother typically has more than one child in the 5 years preceding the survey. Our results are robust to these alternative specifications.

5.2.2 Fertility, adult labor supply and education

The remainder of Table 5 reports the OLS estimates of Equation (4) for fertility, adult labor supply and education. In our identification strategy, if the rationale is the same for adults as for children, the variation in the duration of exposure for adults will be relatively smaller within their longer lifetimes. Moreover, we note that, as shown in Table 1, the effects will be driven by the younger cohort of adults. We introduce age-by-region fixed effects and exposure-by-region fixed effects sequentially. Table 5 confirms the preliminary results from Tables 3 and 4 and Figure 8: 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 the probability of completing primary school by 8 percentage points and the number of years of education completed by 0.8. An alternative, and more conservative, approach to the treatment effects consists in computing the treatment effect individually for each year of birth cohort exposed to RBM, since cohorts have varying degrees of exposure to RBM, and taking the average of these effects. Averaging is more conservative because it weights all cohorts equally, even those minimally exposed

to the campaign, whereas most of the effect is likely concentrated in those cohorts most exposed to the campaign. With this approach, exposure to RBM reduces total births by 0.4, increases the probability of employment by 5.3 percentage points, increases the probability of paid employment by 1.9 percentage points, increases educational attainment by 0.5 years and increases the probability of completing primary school by 4.4 percentage points.

These results are consistent with a model of household production in which health and education operate as complementary inputs. In Supplemental Appendix Table A4, they are furthermore largely robust to specifications which include household fixed effects (possible because respondents are nested in households) and an alternative age control (age-by-malaria). Adult labor outcomes lose significance, likely due to limited variation at the household level. We further interpret the magnitude of Table 5'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 education are comparable to similar studies. For example, using a difference-in-differences strategy based individuals born in pre- and post-eradication areas in Uganda, Barofsky, Anekwe and Chase (2015) show that malaria eradication led to an increase in educational attainment by 0.5 years. Barreca (2010) finds that schooling decreases by 0.4 years with just ten more malaria deaths per 100,000 individuals. Similarly, Bleakley (2010), Lucas (2010), and Venkataramani (2012) find positive effects of malaria reduction or eradication on educational outcomes.

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). In Appendix Table A5, we also test a panel of mother birth events similar to our mortality estimations. This panel captures whether a birth occurred or did not occur for each woman-year for all eligible women in our sample as well as for those women with at least one child. The former allows us to examine fertility choices while the latter may capture mother health. For both samples of women, the analysis shows that the RBM campaign decreases the probability of

a birth event.

Though our results on labor participation differ from evidence 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 there exist stronger complementarities between education and health when it comes to malaria. Our results are lower in magnitude compared to Barofsky, Anekwe and Chase (2015), who find an increase in the probability of male wage employment by 40%. However, we study a contemporary time period and multi-country sample while Barofsky, Anekwe and Chase (2015) present historical evidence from Uganda. These differences may influence the divergence of magnitude.

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 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.[27]

In Table 6, 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

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[27]We exclude PEPFAR disbursements, for which records begin relatively late in our period of interest.

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 7, 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. A further concern is that migration can be endogenous to initial malaria conditions. Nevertheless, we observe that results hold for the majority of dependent variables, though 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, in case regional differences in malaria patterns have implications for our results. Results in both regions are globally consistent. 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 erad-

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ication (Zambia). We also restrict our attention to countries with at least two DHS survey rounds available. Our results, presented in Table A6, are not driven by any country or subset of countries.[28]

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 A7a and A7b, 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 between malaria risk and exposure 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. However, the point estimates relying on artemisinin combination therapies are implausibly high. This is possibly due to the fact that the artemisinin combination therapy coverage varies almost entirely at the national level, and it is thus not ideally suited to serve as a proxy in our estimation.

We run two different types of falsification tests in Tables A8 and A9 of the Supplemental Appendix which serve as an additional check on pre-trends. 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 (4) with these new dependent variables shows no effect of RBM exposure in Table A8. 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,

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[28]Complementary analysis is available upon request.

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 A9, 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 8, 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 anticipate an easier marriage market and respond by supplying less labor and investing less in their education. The fact that sex ratios are, on average, more favorable to males in Africa may explain why we observe catch-up effect. The comparatively larger decline in female mortality could spur more investment in education and pursuit of paid employment.

The ethnicity tests should demonstrate no effect for the malaria-resistant group (Fulani),

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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 9 reports the OLS estimates for non-linear effects in campaign exposure by age group. We interact (malaria$_{2000j}$ × exposure$_{Nct}$) with dummies for five-year age groups. We report each coefficient as the sum of the average effect of (malaria$_{2000j}$ × exposure$_{Nct}$) and the marginal effect by each age group.

We provide the results for human capital outcomes (fertility, labor and education), omitting age at first birth which will, by definition, decline as age progresses. For education, for example, 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.

We observe that the reduction in total births is relatively consistent across all age groups but strongest for those women above age 40. The likelihood 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.[29] 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

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

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 5 show that, for an average level of malaria risk in 2000, an incremental increase in RBM spending reduces infant mortality by 5.2 percentage points. We apply this treatment effect to averaged live birth and infant mortality rates in 2000[30] 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 later[31]) a distinct treatment effect based on each cohort's time exposed to RBM campaigns and the average level of malaria risk in 2000. 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. This leads us to a cost of $1.63 per each additional year of schooling. Restricting the population to ages 0 to 14 raises this estimate to $2.25.

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 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

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

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

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). Moreover, these results fit into the emerging literature on the successes of vertical health programs in reducing the disease burden. This distinction is all the more important given the poor results typically attributed to health aid at the macroeconomic level. Certainly the secondary benefits from malaria interventions will never be large enough to compete with the direct 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. Finally, we use RBM as a catch-all for funding increases. However, a key component of RBM is its coordination efforts. This institutional dimension of global health programs is difficult to assess but should be the subject of careful evaluation in the future.

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.

References

Acemoglu, Daron, and Simon Johnson. 2007. "Disease and Development: The Effect of Life Expectancy on Economic Growth." *Journal of Political Economy*, 115(6): 925–985.

Acemoglu, Daron, and Simon Johnson. 2014. "Disease and Development: A Reply to Bloom, Canning, and Fink." *Journal of Political Economy*, 122(6): 1367–1375.

Alilio, Martin S., Ib C. Bygbjerg, and Joel G. Breman. 2004. "Are multilateral malaria research and control programs the most successful? Lessons from the past 100

years in Africa." *The American journal of tropical medicine and hygiene*, 71(2 suppl): 268–278.

Ashraf, Nava, Günther Fink, and David N Weil. 2014. "Evaluating the effects of large scale health interventions in developing countries: The Zambian Malaria Initiative." In *African Successes: Human Capital*. University of Chicago Press.

Azomahou, Théophile T, Raouf Boucekkine, and Bity Diene. 2016. "HIV/AIDS and Development: A Reappraisal of the Productivity and Factor Accumulation Effects." *The American Economic Review*, 106(5): 472–477.

Baird, Sarah, Joan Hamory Hicks, Michael Kremer, and Edward Miguel. 2016. "Worms at work: Long-run impacts of a child health investment." *The Quarterly Journal of Economics*, 131(4): 1637–1680.

Barofsky, Jeremy, Tobenna D. Anekwe, and Claire Chase. 2015. "Malaria eradication and economic outcomes in sub-Saharan Africa: Evidence from Uganda." *Journal of Health Economics*, 44: 118–136.

Barreca, Alan I. 2010. "The long-term economic impact of in utero and postnatal exposure to malaria." *Journal of Human Resources*, 45(4): 865–892.

Becker, Gary S. 1975. "Investment in Human Capital: Effects on Earnings." In *Human Capital: A Theoretical and Empirical Analysis, with Special Reference to Education, Second Edition*. NBER.

Becker, Gary Stanley, and Harold Gregg Lewis. 1973. "On the interaction between the quantity and quality of children." *Journal of Political Economy*, 81: S279–S288.

BenYishay, Ariel, and Keith Kranker. 2015. "All-Cause Mortality Reductions from Measles Catchup Campaigns in Africa." *Journal of Human Resources*, 50(2): 516–547.

Bharadwaj, Prashant, Katrine Vellesen Løken, and Christopher Neilson. 2013. "Early life health interventions and academic achievement." *The American Economic Review*, 103(5): 1862–1891.

Bhattarai, Achuyt, Abdullah S Ali, S. Patrick Kachur, Andreas Mårtensson, Ali K Abbas, Rashid Khatib, Abdul-wahiyd Al-mafazy, Mahdi Ramsan, Guida Rotllant, Jan F Gerstenmaier, Fabrizio Molteni, Salim Abdulla, Scott M Montgomery, Akira Kaneko, and Anders Björkman. 2007. "Impact of Artemisinin-Based Combination Therapy and Insecticide-Treated Nets on Malaria Burden in Zanzibar." *PLoS Medicine*, 4(11): e309.

Bhatt, S., D. J. Weiss, E. Cameron, D. Bisanzio, B. Mappin, U. Dalrymple, K. E. Battle, C. L. Moyes, A. Henry, P. A. Eckhoff, and others. 2015. "The effect of malaria control on Plasmodium falciparum in Africa between 2000 and 2015." *Nature*, 526(7572): 207–211.

Bleakley, Hoyt. 2010. "Malaria eradication in the Americas: A retrospective analysis of childhood exposure." *American Economic Journal: Applied Economics*, 1–45.

Bleakley, Hoyt, and Fabian Lange. 2009. "Chronic disease burden and the interaction of education, fertility, and growth." *The review of economics and statistics*, 91(1): 52–65.

Bloom, David E., David Canning, and Günther Fink. 2014. "Disease and Development Revisited." *Journal of Political Economy*, 122(6): 1355–1366.

Bryce, Jennifer, Robert E Black, Neff Walker, Zulfiqar A Bhutta, Joy E Lawn, and Richard W Steketee. 2005. "Can the world afford to save the lives of 6 million children each year?" *The Lancet*, 365(9478): 2193–2200.

Burlando, Alfredo. 2015. "The Disease Environment, Schooling, and Development Outcomes: Evidence from Ethiopia." *The Journal of Development Studies*, 51(12): 1563–1584.

Clarke, Sian E., Matthew CH Jukes, J. Kiambo Njagi, Lincoln Khasakhala, Bonnie Cundill, Julius Otido, Christopher Crudder, Benson Estambale, and Simon Brooker. 2008. "Effect of intermittent preventive treatment of malaria on health and education in schoolchildren: a cluster-randomised, double-blind, placebo-controlled trial." *The Lancet*, 372(9633): 127–138.

Cohen, Jessica, and Pascaline Dupas. 2010. "Free Distribution or Cost-Sharing? Evidence from a Randomized Malaria Prevention Experiment." *The Quarterly Journal of Economics*, 125(1): 1–45.

Cohen, Jessica, Pascaline Dupas, and Simone Schaner. 2015. "Price Subsidies, Diagnostic Tests, and Targeting of Malaria Treatment: Evidence from a Randomized Controlled Trial." *The American Economic Review*, 105(2): 609–645.

Currie, Janet. 2009. "Healthy, Wealthy, and Wise: Socioeconomic Status, Poor Health in Childhood, and Human Capital Development." *Journal of Economic Literature*, 47(1): 87–122.

Currie, Janet, and Brigitte C. Madrian. 1999. "Chapter 50 Health, health insurance and the labor market." In . Vol. 3, Part C, , ed. BT Handbook of Labor Economics, 3309–3416. Elsevier.

Cutler, David, Winnie Fung, Michael Kremer, Monica Singhal, and Tom Vogl. 2010. "Early-life malaria exposure and adult outcomes: Evidence from malaria eradication in India." *American Economic Journal: Applied Economics*, 72–94.

Dupas, Pascaline. 2011. "Do Teenagers Respond to HIV Risk Information? Evidence from a Field Experiment in Kenya." *American Economic Journal: Applied Economics*, 3(1): 1–34.

Dupas, Pascaline. 2014. "Getting essential health products to their end users: Subsidize, but how much?" *Science*, 345(6202): 1279–1281.

Flegg, Jennifer A., Charlotte J. E. Metcalf, Myriam Gharbi, Meera Venkatesan, Tanya Shewchuk, Carol Hopkins Sibley, and Philippe J. Guerin. 2013. "Trends in Antimalarial Drug Use in Africa." *The American Journal of Tropical Medicine and Hygiene*, 89(5): 857–865.

Fortson, Jane G. 2009. "HIV/AIDS and Fertility." *American Economic Journal: Applied Economics*, 1(3): 170–194.

Gething, Peter W., Anand P. Patil, David L. Smith, Carlos A. Guerra, I. R. Elyazar, Geoffrey L. Johnston, Andrew J. Tatem, and Simon I. Hay. 2011. "A new world malaria map: Plasmodium falciparum endemicity in 2010." *Malaria Journal*, 10(378): 1475–2875.

Graves, Patricia M., Jeremiah M. Ngondi, Jimee Hwang, Asefaw Getachew, Teshome Gebre, Aryc W. Mosher, Amy E. Patterson, Estifanos B. Shargie, Zerihun Tadesse, Adam Wolkon, and others. 2011. "Factors associated with mosquito net use by individuals in households owning nets in Ethiopia." *Malaria Journal*, 10(354): 10–1186.

Hazan, Moshe, and Hosny Zoabi. 2006. "Does longevity cause growth? A theoretical critique." *Journal of Economic Growth*, 11(4): 363–376.

Hong, Sok Chul. 2013. "Malaria: An early indicator of later disease and work level." *Journal of Health Economics*, 32(3): 612–632.

Hwang, Jimee, Patricia M. Graves, Daddi Jima, Richard Reithinger, S. Patrick Kachur, Ethiopia MIS Working Group, and others. 2010. "Knowledge of malaria and its association with malaria-related behaviors—results from the Malaria Indicator Survey, Ethiopia, 2007." *PLoS One*, 5(7): e11692.

Jakubowski, Aleksandra, Sally C Stearns, Margaret E Kruk, Gustavo Angeles, and Harsha Thirumurthy. 2017. "The US President's Malaria Initiative and under-5 child mortality in sub-Saharan Africa: A difference-in-differences analysis." *PLoS medicine*, 14(6): e1002319.

Jamison, Dean T., Lawrence H. Summers, George Alleyne, Kenneth J. Arrow, Seth Berkley, Agnes Binagwaho, Flavia Bustreo, David Evans, Richard GA Feachem, Julio Frenk, and others. 2013. "Global health 2035: a world converging within a generation." *The Lancet*, 382(9908): 1898–1955.

Jayachandran, Seema, and Adriana Lleras-Muney. 2009. "Life Expectancy and Human Capital Investments: Evidence from Maternal Mortality Declines." *The Quarterly Journal of Economics*, 124(1): 349–397.

Kenkel, Donald S. 1991. "Health behavior, health knowledge, and schooling." *Journal of Political Economy*, 287–305.

Kremer, Michael, and Alaka Holla. 2009. "Improving education in the developing world: what have we learned from randomized evaluations?" *Annual Review of Economics*, 1: 513.

Lucas, Adrienne M. 2010. "Malaria eradication and educational attainment: evidence from Paraguay and Sri Lanka." *American Economic Journal: Applied Economics*, 2(2): 46–71.

Lucas, Adrienne M. 2013. "The impact of malaria eradication on fertility." *Economic Development and Cultural Change*, 61(3): 607–631.

Miguel, Edward, and Michael Kremer. 2004. "Worms: identifying impacts on education and health in the presence of treatment externalities." *Econometrica*, 72(1): 159–217.

Mohammed, Asia, Arnold Ndaro, Akili Kalinga, Alphaxard Manjurano, Jackline Mosha, Dominick Mosha, Marco van Zwetselaar, Jan Koenderink, Frank Mosha, Michael Alifrangis, Hugh Reyburn, Cally Roper, and Reginald Kavishe. 2013. "Trends in chloroquine resistance marker, Pfcrt-K76T mutation ten years after chloroquine withdrawal in Tanzania." *Malaria Journal*, 12(1): 415.

Mung'ala-Odera, Victor, Robert W. Snow, and Charles R. J. C. Newton. 2004. "The Burden of the Neurocognitive Impairment Associated with Plasmodium Falciparum Malaria in Sub-Saharan Africa." *The American Journal of Tropical Medicine and Hygiene*, 71(2 suppl): 64–70.

Nabarro, David N., and Elizabeth M. Tayler. 1998. "The" roll back malaria" campaign." *Science*, 280(5372): 2067–2068.

Nankabirwa, Joaniter, Bonnie Wandera, Noah Kiwanuka, Sarah G. Staedke, Moses R. Kamya, and Simon J. Brooker. 2013. "Asymptomatic Plasmodium Infection and Cognition among Primary Schoolchildren in a High Malaria Transmission Setting in Uganda." *The American Journal of Tropical Medicine and Hygiene*, 88(6): 1102–1108.

Nganda, Rhoida Y., Chris Drakeley, Hugh Reyburn, and Tanya Marchant. 2004. "Knowledge of malaria influences the use of insecticide treated nets but not intermittent presumptive treatment by pregnant women in Tanzania." *Malaria Journal*, 3(8).

Pathania, Vikram. 2014. "The impact of malaria control on infant mortality in Kenya." *Economic Development and Cultural Change*, 62(3): 459–487.

Pigott, David M., Rifat Atun, Catherine L. Moyes, Simon I. Hay, and Peter W. Gething. 2012. "Funding for malaria control 2006–2010: a comprehensive global assessment." *Malaria Journal*, 11: 246.

Rhee, Michelle, Mahamadou Sissoko, Sharon Perry, Willi McFarland, Julie Parsonnet, and Ogobara Doumbo. 2005. "Use of insecticide-treated nets (ITNs) following a malaria education intervention in Piron, Mali: a control trial with systematic allocation of households." *Malaria Journal*, 4(1): 35.

Rosenzweig, Mark R., and Junsen Zhang. 2009. "Do population control policies induce more human capital investment? twins, birth weight and china's "one-child" policy." *The Review of Economic Studies*, 76(3): 1149–1174.

Soares, Rodrigo R. 2005. "Mortality reductions, educational attainment, and fertility choice." *American Economic Review*, 580–601.

Tanaka, Shinsuke. 2014. "Does Abolishing User Fees Lead to Improved Health Status? Evidence from Post-apartheid South Africa." *American Economic Journal: Economic Policy*, 6(3): 282–312.

Thuilliez, Josselin, Hippolyte d'Albis, Hamidou Niangaly, and Ogobara Doumbo. 2017. "Malaria and Education: Evidence from Mali." *Journal of African Economies*, 1–27.

Thuilliez, Josselin, Mahamadou S. Sissoko, Ousmane B. Toure, Paul Kamate, Jean-Claude Berthélemy, and Ogobara K. Doumbo. 2010. "Malaria and primary education in Mali: A longitudinal study in the village of Donéguébougou." *Social Science & Medicine*, 71(2): 324–334.

Venkataramani, Atheendar S. 2012. "Early life exposure to malaria and cognition in adulthood: Evidence from Mexico." *Journal of Health Economics*, 31: 767–780.

Vogl, Tom S. 2014. "Education and Health in Developing Economies." In . Vol. Encyclopedia of Health Economics, , ed. A.J. Cuyler. Elsevier.

WHO. 2015. *World malaria report 2015. Geneva: WHO; 2015.*

Wilde, Joshua, Bénédicte Apouey, Stacey Gelsheimer, and Gabriel Picone. 2014. "Infant mortality, fertility, and malaria: the effect of mosquito net distribution in sub-Saharan Africa." Working Paper.

Figure 1: Demographic and Health survey years and Roll Back Malaria start dates in our 27 countries
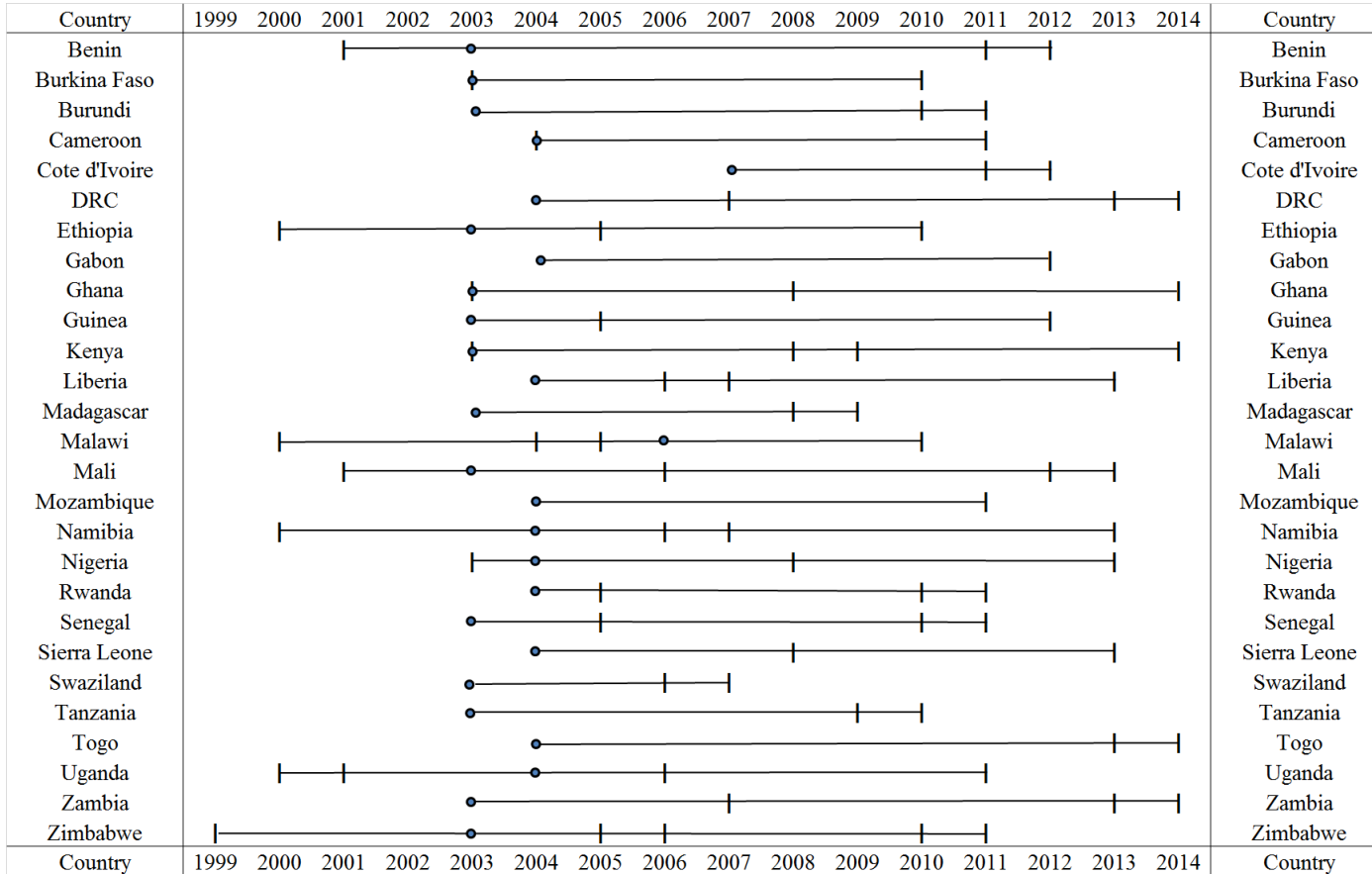
Notes: Vertical bars show the Demographic and Health survey years available for each country, and points mark the start of Roll Back Malaria disbursements. Individual exposure to anti-malaria programs is a function not only of disbursement starting dates but survey timing and years of birth.

Figure 2: Spatial distribution of DHS clusters and initial malaria risk (*Plasmodium falciparum* parasite rate) from Bhatt et al. (2015) in our 27 countries
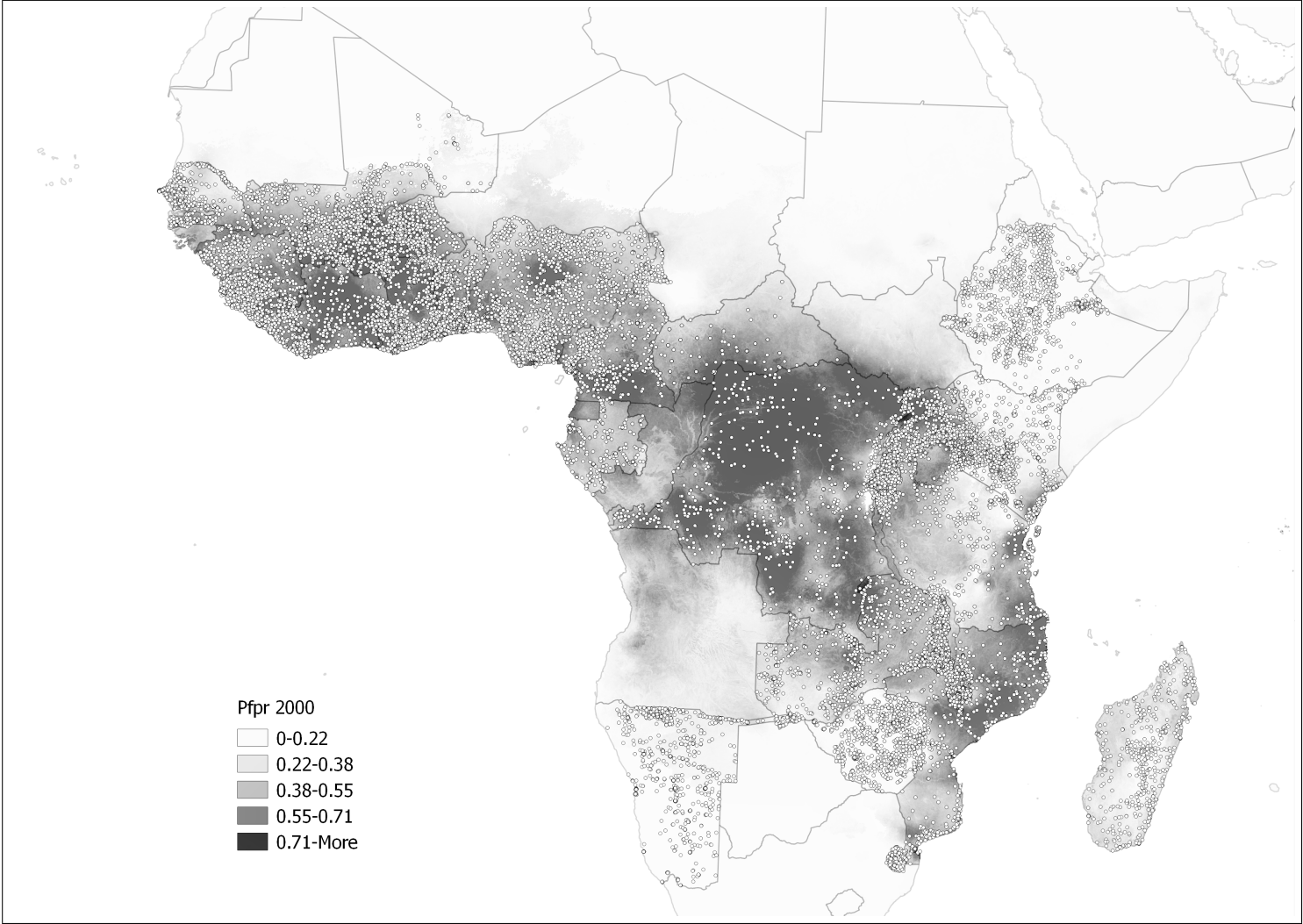
Figure 3: Evolution of malaria risk (*Plasmodium falciparum* parasite rate) and coverage by malaria control strategies in our 27 sample countries
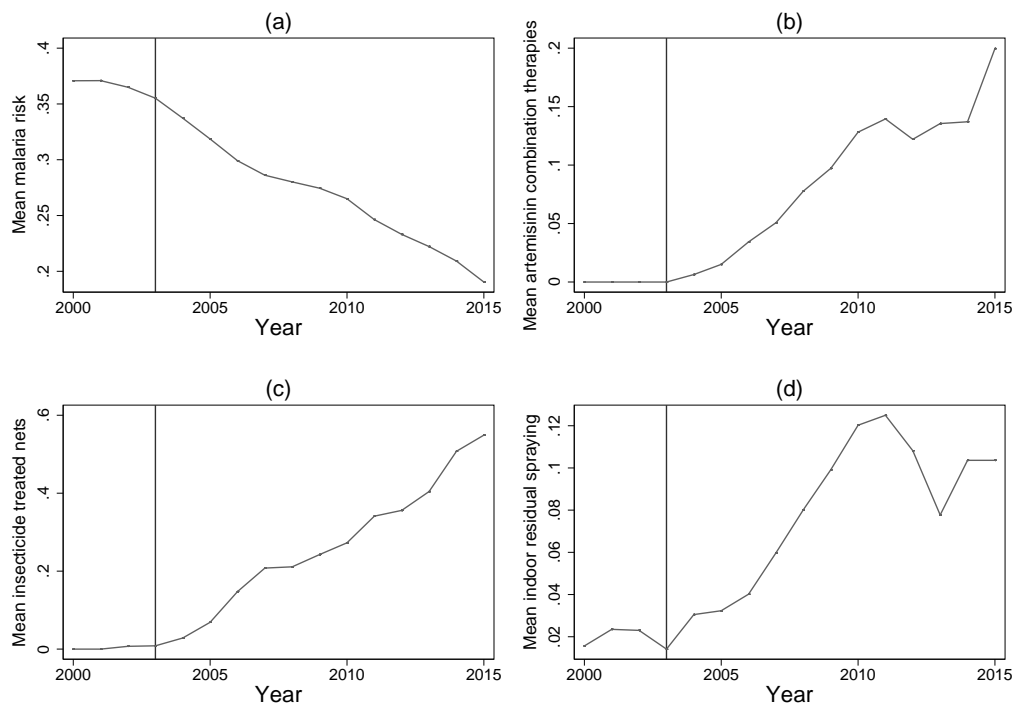
Notes: Each line plots annualized indicators from the Malaria Atlas Project, totaled for all countries in our sample, over time. The vertical bar denotes the scale-up of RBM disbursements. Figure 3a plots the mean malaria risk (*Plasmodium falciparum* parasite rate). Figure 3b plots the mean coverage 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)
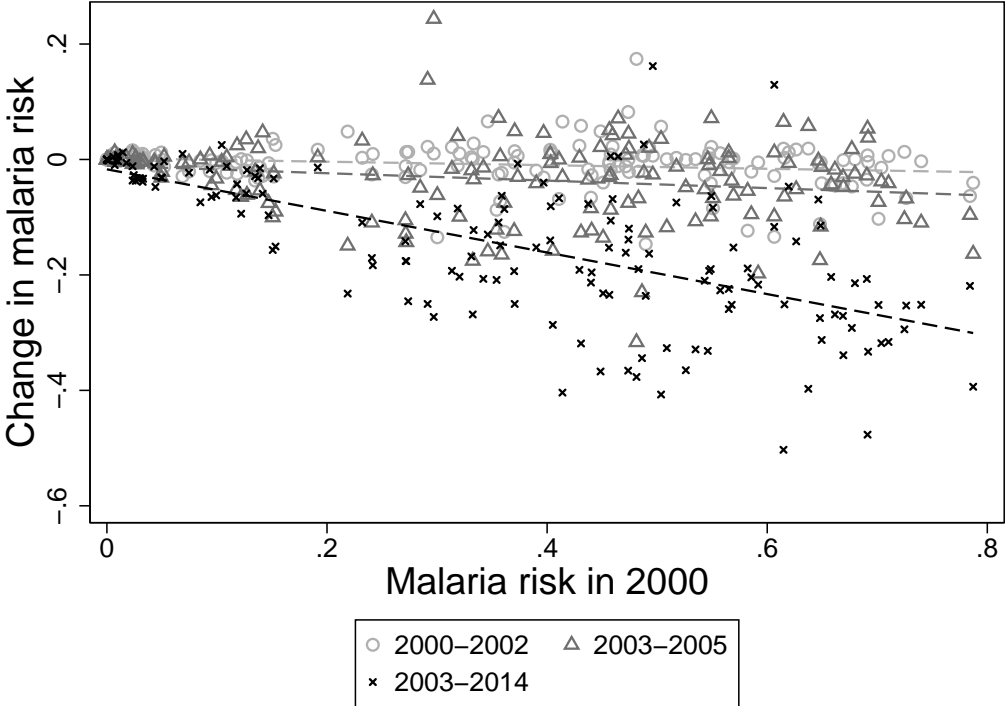
Notes: Each point represents a region. We obtain yearly malaria risk (PfPR) from the Malaria Atlas Project. We obtain the dashed fitted lines by regressing the change in malaria risk over each period on initial malaria risk in 2000.

Figure 5: Evolution of bednets and antimalarial use conditional on initial malaria risk (*Plasmodium falciparum* parasite rate)
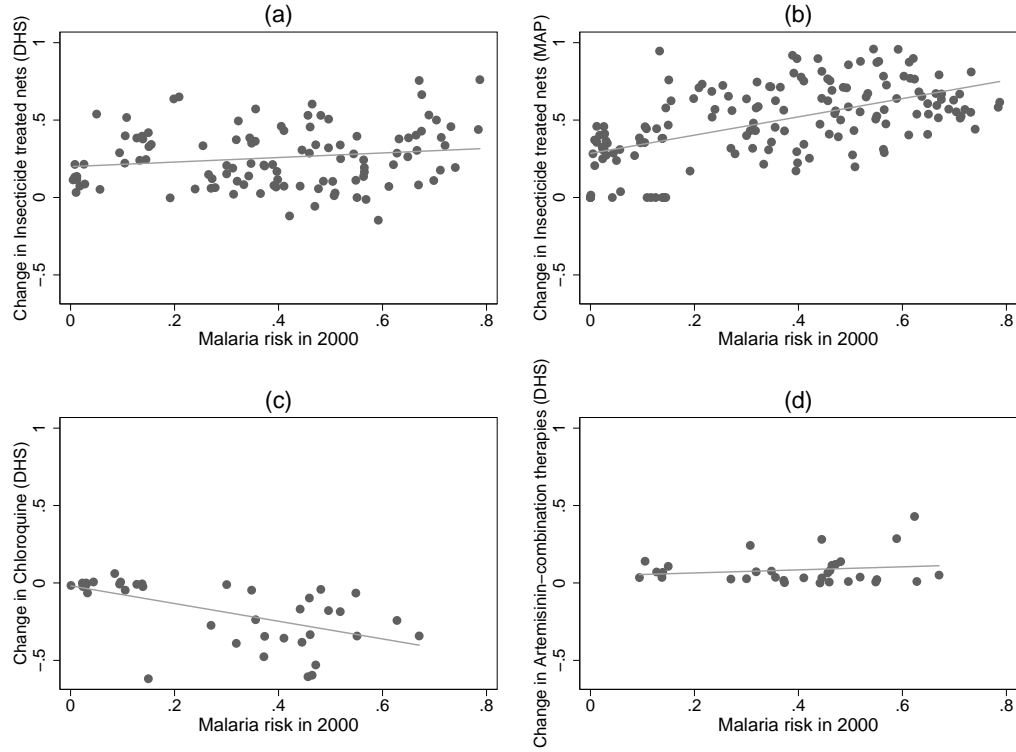
Notes: Each point represents a region. We obtain yearly malaria risk from the Malaria Atlas Project. The change in Figures 5a, 5c, and 5d is the difference between the first and last DHS survey available in our sample. The change in Figure 5b is the difference between 2003 and 2015, the latest available 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 presumably due to the low number of observations (Figure 5d, N = 32).

Figure 6: Total Roll Back Malaria disbursements over time
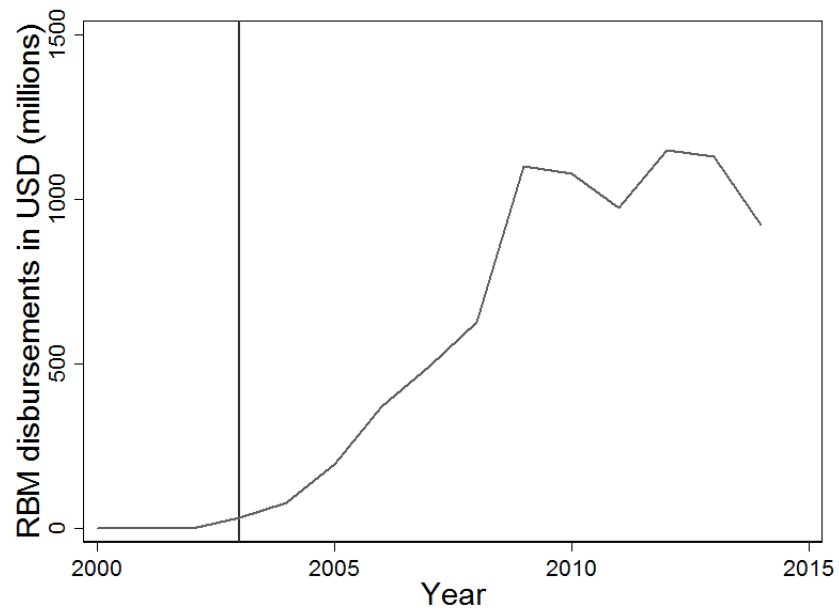
Notes: This graph plots Roll Back Malaria disbursements totaled over all countries in our sample for each year. The vertical bar at 2003 marks the start of Roll Back Malaria scale-up.

Figure 7: Ruling out pre-campaign catch-up effects

A. Height for age above 20 y.o. (at campaign start)

B. Education above 25 y.o. (at campaign start)

C. Completed Primary Education above 25 y.o. (at campaign start)

D. Fertility above 40 y.o.
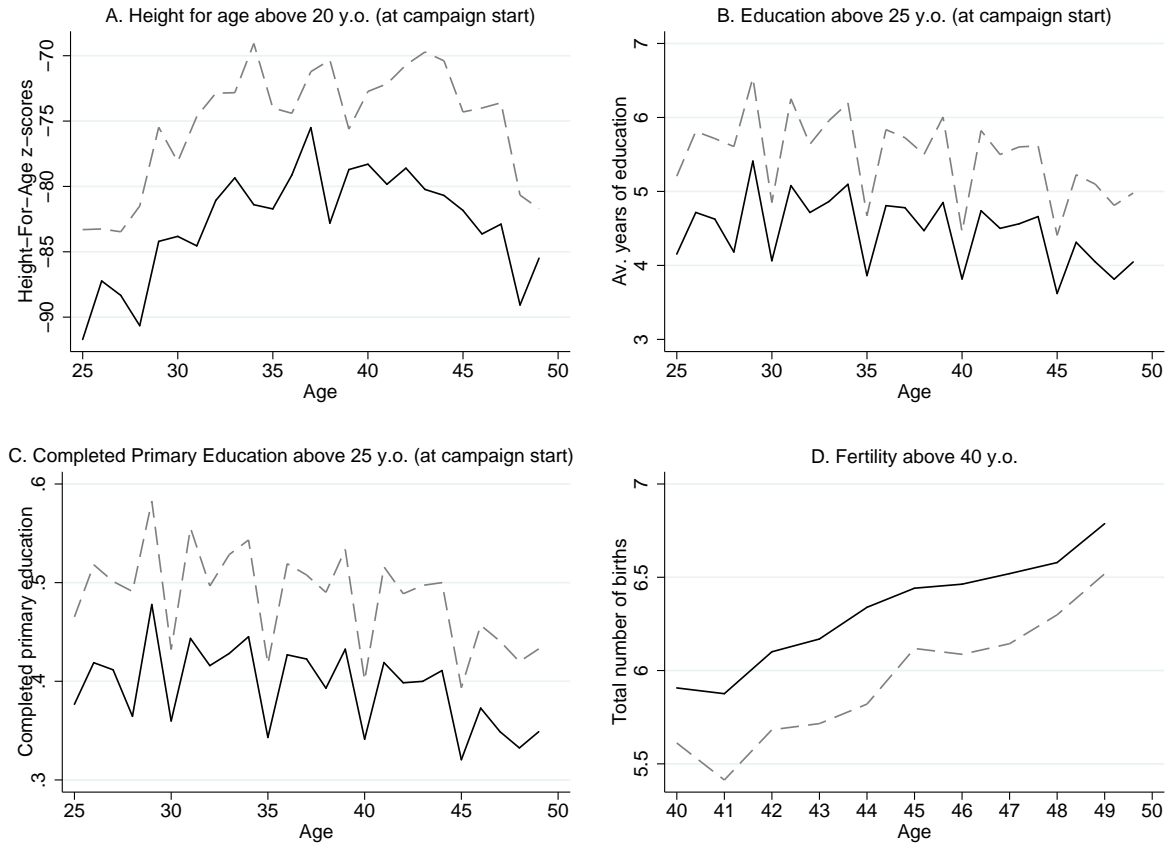
Notes: This figure considers individuals belonging to two groups: those living in clusters where malaria risk was above the median level in 2000 (solid line) versus where malaria risk was below the median level in 2000 (dashed line). We plot outcome variables against age for respondents above the age 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.

Figure 8: Relationship of initial malaria risk with infant mortality, fertility, adult labor supply and education
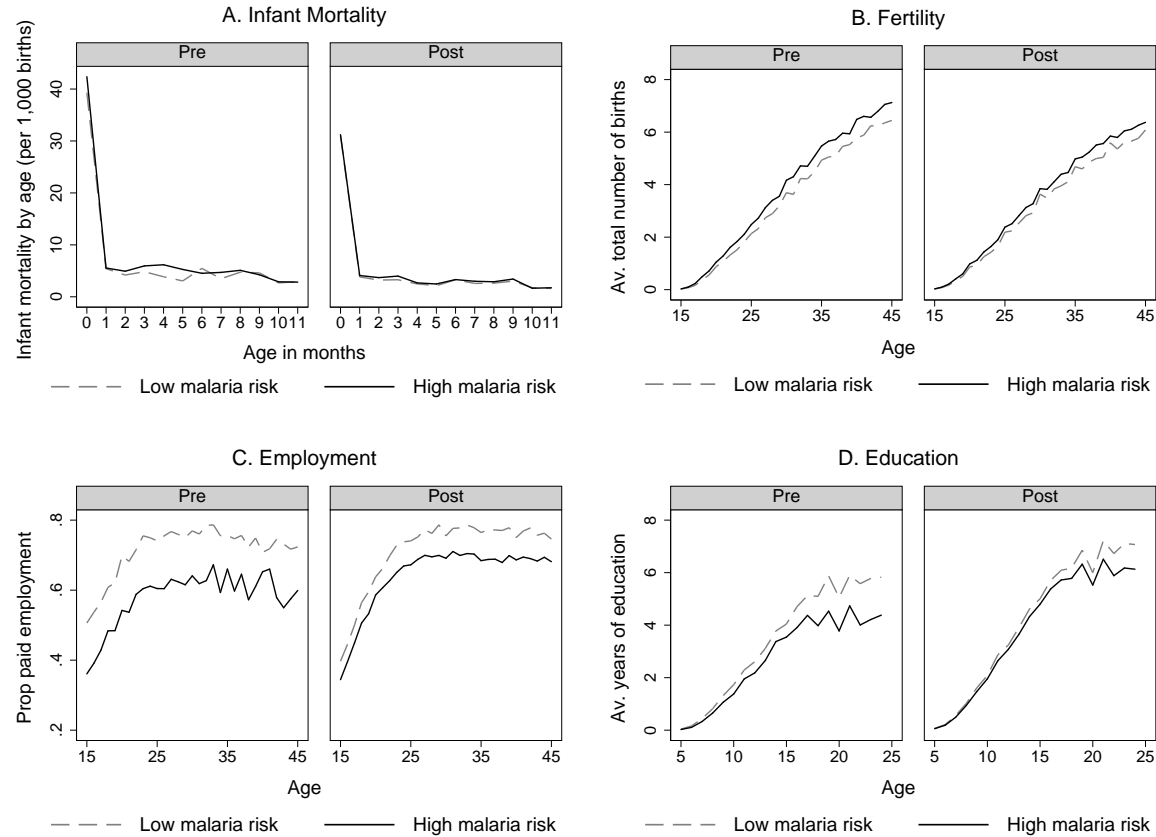
Notes: We plot the average of each dependent variable by age cohort at the time of survey (pre- and post-2003) between high and low levels of malaria risk. A highly (resp. less) malarious area is defined as having an above (resp. below) median level of malaria risk in 2000. We note that the infant mortality 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

	Coefficient of ($\text{malaria}_{2000j} \times \text{exposure}_{Nct}$)		
Dep. var.	(1)		(2)
Infant Mort. (born before 2002)	-0.466	Years of ed. (>25 y.o.)	-4.189ˆ
	(0.639)		(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	Primary ed. (>25 y.o.)	-0.488*
	(136.370)		(0.239)
R^2	0.287	R^2	0.477
Observations	191,293	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, each cell reports the OLS estimate of coefficient β in Equation (4). The regression controls for gender, wealth, and DHS cluster fixed effects as well as country-by-cohort-by-DHS year fixed effects. 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. ˆ, *, ** 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***	0.062***	-0.025***	-0.165***	-0.029***	-0.038***
	(0.003)	(0.003)	(0.003)	(0.008)	(0.002)	(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***	0.017***	-0.008***	-0.060***	-0.008***	-0.011***
	(0.002)	(0.003)	(0.002)	(0.006)	(0.001)	(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***	0.045***	-0.017***	-0.105***	-0.021***	-0.027***
	(0.002)	(0.002)	(0.003)	(0.006)	(0.001)	(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***	0.595***	0.290***	0.052	-0.099***	-0.215***
	(0.028)	(0.037)	(0.033)	(0.083)	(0.021)	(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***	-0.525***	-1.142***	-0.302*	0.040	0.035
	(0.059)	(0.074)	(0.060)	(0.153)	(0.039)	(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***	-0.013	0.112***	0.135***	0.083***	0.058***
	(0.008)	(0.011)	(0.010)	(0.023)	(0.006)	(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***	-0.123***	-0.233***	0.403***	-0.086***	0.118***
	(0.012)	(0.016)	(0.015)	(0.030)	(0.008)	(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***	-1.306***	0.901***	3.352***	0.152***	1.372***
	(0.063)	(0.080)	(0.062)	(0.156)	(0.044)	(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***	-0.099***	0.040***	0.268***	0.012*	0.111***
	(0.006)	(0.008)	(0.006)	(0.018)	(0.004)	(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 from the Demographic and Health Surveys and Malaria Atlas Project are at the regional level. However, drugs (MAP) varies primarily at the national level. Controls for gender, age and wealth are included. Standard errors (in parentheses) are clustered at the DHS cluster level. ˆ, *, ** and *** indicate significance at the 10, 5, 1 and 0.1% levels.

Table 3: Period 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. Though we present the simplest results here, they are robust to the inclusion of additional control variables.

Table 4: Cohort and period-cohort analyses of the effect of the RBM campaign on human capital outcomes

	(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.
					Panel A: Cohort model				
$\text{malaria}_{2000j} \times \text{post}_{Nc}$	-0.036*	-0.018^	-0.018^					1.457***	0.142***
	(0.014)	(0.010)	(0.011)					(0.116)	(0.011)
Treatment effect	-0.013*	-0.006^	-0.006^					0.511***	0.050***
R^2	0.087	0.082	0.080					0.590	0.501
Observations	353,379	353,379	353,379					3,244,475	3,244,475
					Panel B: Period-cohort model				
$\text{malaria}_{2000j} \times \text{duration}_{Nct}$	-0.003^	-0.001	-0.002	-3.898***	1.628*	0.271***	0.292***	0.088***	0.008***
	(0.002)	(0.001)	(0.001)	(0.301)	(0.646)	(0.052)	(0.060)	(0.008)	(0.001)
Treatment effect	-0.001^	0.000	0.000	-1.368***	0.571*	0.095***	0.102***	0.031***	0.003***
R^2	0.087	0.082	0.080	0.660	0.219	0.329	0.410	0.586	0.498
Observations	353,379	353,379	353,379	670,113	487,633	950,792	651,475	3,153,218	3,153,218

Notes: The unit of observation is the individual. For all dependent variables, each cell reports the OLS estimate of coefficient β in Equations (2) and (3). The treatment effect is obtained by multiplying the estimated coefficient by the mean level of malaria risk in 2000 (0.351). The regression controls for gender and wealth as well as fixed effects for DHS clusters and country-by-cohort-by-DHS year. Standard errors (in parentheses) are clustered at the DHS cluster level. ^, *, ** and *** indicate significance at the 10, 5, 1 and 0.1% levels.

Table 5: Effect of the RBM campaign on human capital outcomes

	(1)	(2)	(3)	(4)	(5)	(6)
	Infant		Neonatal		Post-neonatal	
$\text{malaria}_{2000j} \times \text{exposure}_{Nct}$	-0.453***	-0.149**	-0.208***	-0.048	-0.245***	-0.101**
	(0.034)	(0.047)	(0.018)	(0.039)	(0.019)	(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		
$\text{malaria}_{2000j} \times \text{exposure}_{Nct}$	-3.951***	-3.242***	-3.837***	1.895***	1.979***	0.954
	(0.213)	(0.241)	(0.291)	(0.464)	(0.568)	(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)		
$\text{malaria}_{2000j} \times \text{exposure}_{Nct}$	0.361***	0.316***	0.489***	0.215***	0.181*	0.175*
	(0.040)	(0.048)	(0.057)	(0.049)	(0.059)	(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.		
$\text{malaria}_{2000j} \times \text{exposure}_{Nct}$	1.913***	1.776***	2.406***	0.178***	0.167***	0.224***
	(0.087)	(0.082)	(0.112)	(0.008)	(0.008)	(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 (4). The treatment effect is obtained by multiplying the estimated coefficient by the mean level of malaria risk in 2000 (0.351). The regression controls for gender, wealth, and DHS cluster fixed effects as well as country-by-cohort-by-DHS year fixed effects. Standard errors (in parentheses) are clustered at the DHS cluster level. ˆ, *, ** and *** indicate significance at the 10, 5, 1 and 0.1% levels.

Table 6: 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***	-0.333***	-0.377***	-2.554***	-0.194	0.290***	0.193*	1.428***	0.133***
	(0.030)	(0.018)	(0.017)	(0.343)	(0.771)	(0.069)	(0.081)	(0.100)	(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***	-0.274***	-0.300***	-3.289***	0.701	0.346***	0.203*	1.587***	0.154***
	(0.027)	(0.017)	(0.016)	(0.405)	(0.906)	(0.083)	(0.100)	(0.102)	(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***	-0.340***	-0.388***	-2.963***	0.390	0.240***	0.278***	1.505***	0.140***
	(0.030)	(0.018)	(0.017)	(0.322)	(0.747)	(0.067)	(0.082)	(0.088)	(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***	-0.326***	-0.374***	-3.445***	0.179	0.329*	0.177	1.716***	0.162***
	(0.030)	(0.018)	(0.018)	(0.539)	(1.221)	(0.104)	(0.126)	(0.106)	(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***	-0.164***	-0.214***	-1.761*	0.635	-0.154	-0.081	1.417***	0.144***
	(0.026)	(0.017)	(0.016)	(0.660)	(1.529)	(0.139)	(0.170)	(0.105)	(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*	-0.047	-0.099*	-2.093***	-1.145	0.532***	0.181ˆ	1.908***	0.190***
	(0.048)	(0.039)	(0.037)	(0.415)	(0.900)	(0.087)	(0.104)	(0.137)	(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 (4). We compute exposure to 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, age-by-region, and exposure-by-region. Exposure incorporates the newly introduced expenditures. Standard errors (in parentheses) are clustered at the DHS cluster level. ˆ, *, ** and *** indicate significance at the 10, 5, 1 and 0.1% levels.

Table 7: 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 (4). 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. The regression controls for gender, wealth, and DHS cluster fixed effects as well as country-by-cohort-by-DHS year fixed effects. 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: 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)
R²	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)
R²	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)
R²	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)
R2	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)
R²	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 (4). The regression controls for gender, wealth, and DHS cluster fixed effects as well as country-by-cohort-by-DHS year fixed effects. Standard errors (in parentheses) are clustered at the DHS cluster level. ^, *, ** and *** indicate significance at the 10, 5, 1 and 0.1% levels.

Table 9: Non-linear effects by five-year age groups

Non-linear age at survey	(1) Tot. births	(2) Employed	(3) Paid in cash	(4) Years of ed.	(5) Completed primary ed.
Ages 5 to 10				0.973***	0.128***
				(0.125)	(0.013)
Ages 10 to 15				0.646***	0.101***
				(0.165)	(0.018)
Ages 15 to 20	-2.621***	0.361***	0.234^	0.028	0.016
	(0.511)	(0.109)	(0.138)	(0.262)	(0.032)
Ages 20 to 25	-2.655***	0.331*	0.194	-0.594	0.032
	(0.623)	(0.134)	(0.166)	(0.396)	(0.041)
Ages 25 to 30	-2.310*	0.334*	0.262	-1.634*	-0.058
	(0.751)	(0.163)	(0.195)	-(0.539)	(0.053)
Ages 30 to 35	-2.529*	0.356^	0.304	-0.869	0.023
	(0.884)	(0.191)	(0.227)	(0.676)	(0.066)
Ages 35 to 40	-2.325*	0.359^	0.312	-0.687	0.052
	(1.050)	(0.218)	(0.258)	(0.795)	(0.078)
Ages above 40	-3.904*	0.462^	0.473	1.464	0.235*
	(1.241)	(0.256)	(0.300)	(0.987)	(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 the sum of the average effect of (malaria$_{2000j}$ × exposure) and the marginal effect for each age group from a triple interaction term. The regression controls for gender, wealth, and DHS cluster fixed effects as well as country-by-cohort-by-DHS year fixed effects and subcomponents of the triple interaction (including malaria risk-by-age group and exposure-by-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.