The Effect of Antimalarial Campaigns on Child Mortality and Fertility in Sub-Saharan Africa

Joshua Wilde Bénédicte H. Apouey Joseph Coleman
Gabriel Picone *

June 13, 2019

Abstract: We examine the extent to which recent declines in mortality and fertility in Sub-Saharan Africa can be attributed to insecticide-treated bed nets (ITNs). Exploiting the rapid increase in ITNs during the mid-2000s, we employ a difference-in-differences estimation strategy to identify the causal effect of ITNs on mortality and fertility. We show that ITNs reduced all-cause child mortality, but surprisingly increased total fertility rates in spite of reduced desire for children and increased contraceptive use. We explain this paradox by showing evidence for an unexpected increase in fecundity and sexual activity due to the better health environment after the ITN distribution.

Keywords: Malaria, Bed nets, Child mortality, Fertility, Sub-saharan Africa.

*Joshua Wilde: Corresponding Author. University of South Florida. 4202 E. Fowler Ave. CMC 342, Tampa, FL 33620, USA. E-mail: jkwilde@usf.edu. Bénédicte H. Apouey: Paris School of Economics - CNRS. 48 Boulevard Jourdan, 75014 Paris, France. E-mail: benedicte.apouey@psemail.eu. Joseph Coleman: University of South Florida. 4202 E. Fowler Ave. CMC 342, Tampa, FL 33620, USA. E-mail: jscoleman@mail.usf.edu. Gabriel Picone: University of South Florida. 4202 E. Fowler Ave. CMC 342, Tampa, FL 33620, USA. E-mail: gpicone@usf.edu. The authors thank Martine Audibert, David Canning, Denis Cogneau, Pascaline Dupas, Andrew Foster, Marlène Guillon, Allan Hill, Patricia Jones, Maria Kuecken, Giulia La Mattina, David Lam, T. Paul Schultz, Josselin Thuilliez, Marie-Anne Valfort, David Weil, and participants at the CSAE 2014 conference, the 2014 "Journées de Microéconomie Appliquée," the 2014 PopPov Research Network conference, the 2014 Congress of the European Economic Association, and the 2017 PAA meeting as well as seminar participants at the University of South Florida and the University of North Dakota for helpful comments and encouragement. The authors are also grateful to Stacey Gelsheimer, Robyn Kibler, and Arseniy Yashkin for their research assistance. The authors thank the support of Grant Number R03TW009108 from the Fogarty International Center. The content is solely the responsibility of the authors and does not necessarily represent the official views of the Fogarty International Center or the National Institute of Health.

JEL codes: I15, J13, O10, O15.

Understanding the complex relationship between fertility and child mortality has been a major subject of investigation within economics and demography for at least the last century.[1] However, even after decades of study, both the theoretical and empirical relationships between these two variables remain unclear. There are several theoretical channels by which child mortality and fertility are linked, some of which predict a positive relationship between the two variables, while others predict a negative one. In addition, finding historical examples of exogenous changes in child mortality which do not also directly affect fertility is difficult. As a result, strong causal identification of the effect of child mortality on fertility remains elusive.

With the advent of the Millennium Development Goals (MDG) in 2000, there has been a massive increase in funding and support for programs reducing infant and child mortality in the developing world. In fact, the fourth MDG was solely focused on the reduction of infant and child mortality – specifically by two-thirds by 2015 from its 1990 level. Given the unclear relationship between child mortality and fertility, the effect of these programs on overall population growth is ambiguous. Many academics, policy groups, and several major international organizations, however, are promoting the idea that reducing child mortality will not only lead to smaller populations, but is actually a necessary condition for population slowing. For example, Hans Rosling of the Gapminder Foundation claimed in his 2010 Ted Talk that "It's only by [improving] child survival that we will stop population growth." (Rosling, 2010). In addition, in their 2014 annual letter, the Gates Foundation listed "Saving lives leads to overpopulation" – with an emphasis on child survival – as one of the three main

[1] Interest in the topic increased dramatically since the 1950s as two major demographic developments caused global population to explode: first, a baby boom in the developed world after World War II, and second, a huge reduction in mortality from infectious diseases in the developing world. Mass public health campaigns in the developing world – financed by the developed world – led to an international epidemiological transition in which the rates of malaria, tuberculosis, and trachoma fell sharply across the globe. As mortality rates fell and fertility rates rose, concerns about overpopulation and resource depletion increased – most famously exemplified by the book "The Population Bomb" written by Paul Ehrlich in 1968. As developed nations began pushing controversial population control programs in the 1970s and 1980s to deal with these concerns, the academic world began taking a closer look at disentangling the complex demographic relationships between infant mortality and fertility in particular. As the international community backed away from a heavy-handed approach to population control beginning in the 1990s, efforts to prevent further population explosions shifted towards reducing fertility by increasing reproductive freedom – including improved access to contraceptives, women's empowerment over their reproductive decisions, as well as increasing schooling opportunities for young girls.

myths that block progress for the poor (Gates Foundation, 2014).

In this paper, we contribute to the empirical literature on the relationship between infant mortality and fertility by analyzing the effect of an international program intended mainly to reduce infant and child mortality – but not fertility – on fertility itself. Specifically, we identify the effect of the large and rapid increase in the distribution of insecticide-treated bed nets (ITNs) in sub-Saharan Africa in the 2000s on child mortality and fertility rates. We employ a difference-in-differences approach, which exploits both differences in pre-intervention malaria prevalence rates and the timing of the ITN roll-out at the region level. Our model is estimated using a unique dataset that merges information on child mortality and fertility outcomes from the Demographic and Health Surveys (DHS) with a panel of malaria prevalence and antimalarial interventions from the Malaria Atlas Project (MAP) for 35 sub-Saharan countries between 2000 and 2014.

Worldwide, malaria is the second leading cause of death by infectious disease after pneumonia, responsible for 8 percent of all child deaths globally. Africa is especially hard hit – of the 660,000 deaths from malaria in 2010, 90% occurred in Africa. In addition, 16 percent of all deaths among children under 5 in Africa are from malaria (WHO, 2012). The disease is especially dangerous for young children who have not yet developed partial immunity against the disease. Particularly tragic is that using available technologies, malaria can be prevented, diagnosed, and cured quite easily. As a result, malaria was specifically targeted in the sixth Millennium Development Goal. In 1998 the Roll Back Malaria Partnership (RBM) was launched to coordinate global action against malaria. Between 2000 and 2015, the substantial expansion of malaria interventions – primarily the distribution of ITNs, but also indoor residual spraying (IRS) and the use of artemisinin-based combination therapies (ACT) – led to a 60 percent decline in malaria mortality rates globally, and 66 percent decline in Africa alone.[2] Since 2000, over 6.2 million deaths from malaria have been averted, primarily in children under five years of age in sub-Saharan Africa.[3]

We find that the introduction of ITNs reduces child mortality for children between 13-36 months old. Beyond 36 months, the effect is still significant in magnitude, but statistically insignificant. Specifically, for the mean region in

[2] See http://www.who.int/malaria/media/world-malaria-report-2015/en/

[3] See http://www.un.org/millenniumgoals/aids.shtml

our sample we find a 22.2 percent reduction in mortality for children aged 13-24 months, and a 40.6 percent reduction in mortality for children aged 25-36 months as a result of the ITN distribution campaigns. We also find a statistically insignificant 3.6 percent reduction in infant (0-12 months of age) mortality.[4] This is consistent with evidence from the biological literature which shows that children within the first six months of life generally gain malaria immunity from their mother's antibodies in breast milk. Mortality falls by 25.6 and 20.6 percent for 37-48 and 49-60 month-olds respectively, but this reduction is not significant at the 10 percent level.

We also find that the effect of bed nets on fertility is positive for older women in our overall sample. Specifically, we find that the distribution of bed nets in the average region increased the annual probability of having a child by 1.5 percent for women aged 15-19, and by 3.2, 3.6, 8.0, 3.6, and 14.6 percent for women aged 20-24, 25-29, 30-34, 35-39, and 40-44 respectively, but only statistically significant for women aged 30-34 and 40-44. However, this result masks significant heterogeneity in the effect of the ITN distribution campaign by socioeconomic status: we find large and significant increases in fertility among women with at least primary education for all age groups except for 15-19, whereas we find no increase in fertility among women without primary education for any age group except for 40-44. Overall, our estimates imply that the ITN campaigns raised the total fertility rate by 0.30 children in the overall sample (an increase of 5.3 percent), but by 0.45 and 0.15 children for the educated and uneducated respectively.

After showing the reduced form effect of the ITN distribution campaigns, we contribute to the theoretical literature on the effect of child mortality on fertility by providing evidence on the effects the campaigns had on various hypothesized mechanisms linking mortality and fertility. Beyond investigating heterogeneity in the effects of the ITN distribution by education level and gender, we also assess the impact on birth spacing and compositional effects of fertility. We analyze the effect of the distribution campaigns on all of the eight proximate determinants of fertility proposed by Bongaarts (1978) for which we have data. We find that the extensive margin of sexual activity increased for all women,

[4] An alternative way of interpreting our coefficients shows a statistically significant reduction in 0-12 month mortality of 29.8 percent, which will be discussed in detail in Section IV.A. However, we are not reporting that as our main interpretation in an attempt to be conservative.

yet only older uneducated women increased their contraceptive use, which may partially explain why fertility only rose for the educated group.

We also show that these increases in fertility occurred simultaneously with a fall in desired fertility, raising the possibility that the increase in fertility was, in fact, unintentional. We explore this possibility by hypothesizing three channels by which women are unable to perfectly control their fertility: low levels of empowerment, an unmet need for contraception, and an unexpected increase in fecundity. We show no evidence that increases in fertility were concentrated among women with less decision making power in the household or with an unmet need for contraception. However, we do find evidence for an increase in fecundity: fertility only increased for women who were not anemic, implying that since the ITN distribution campaigns lowered anemia rates (Apouey et al., 2017), this increase in health may have increased fecundity.

Our paper is related to the literature on malaria eradication and human capital outcomes generally. Barreca (2010) estimates the effect of malaria eradication in the United States, and finds that in utero exposure to malaria leads to lower levels of educational attainment. Bleakley (2010) studies the same eradication campaign in the United States as Barreca, and finds an positive effect on labor productivity later in life. Lucas (2010) identifies the effect of malaria eradication in Sri Lanka and Paraguay on years of schooling and literacy rates, and finds that after eradication there is an improvement in these variables for females. Cutler et al. (2010) finds similar results to Lucas (2010) in India, while Venkataramani (2012) explores the effects of declining malaria in Mexico. In sub-Saharan Africa, Barofsky et al. (2015) find that a malaria control program in Uganda increased years of schooling by 0.5 years, while Kuecken et al. (2016) find that country-level malaria distributions from the Roll Back Malaria initiative increased educational attainment across the continent.

Our study is most closely related to three papers in particular. First, Lucas (2013) studies the effect of malaria eradication in Sri Lanka in the 1950s on fertility directly. Her results mirror our own, in that she finds an increase in fertility rather than a decrease as most economic models would predict. Cogneau and Rossi (2016) find a non-causal correlation between bed net distribution and child survival in a large set of sub-Saharan African countries from 2000-2015. Similar to our results, they find that the preponderance of reduction in child mortality is concentrated among lower SES households. Finally, Pathania (2014)

uses the scale up of ITNs in Kenya to causally identify a 33 percent reduction in post-neonatal mortality due to the ITN distribution program in that country.

Our paper provides two very large contributions to the existing literature. The first is of policy interest: we provide the first causal, reduced form estimate of the efficacy of a very large, current international health intervention that attracts billions of dollars of funding on one of its main outcomes of interest: child mortality. Of particular importance is that this paper derives its estimates using extensive population data, rather than smaller, localized ITN distribution experiments which may suffer from questions of external validity at scale, or whether they can capture spillover or general equilibrium effects. In addition, we provide a reduced form estimate of a major unintended consequence of that program: fertility change. Importantly, we find that while the program achieved its main focus in reducing child deaths by malaria, the effect on fertility is exactly the opposite of the prevailing belief among aid and advocacy organizations, and many academics: rather than reducing fertility, the ITN distribution programs exacerbated fertility in an area already struggling with the highest fertility rates in the world. Our results suggest that the program's effect of reduced mortality and increased fertility will cause a temporary rise in the dependency ratio, impeding economic growth (Ashraf et al., 2009; Ashraf et al., 2013; Canning et al., 2017). It also provides a cautionary tale for other large scale health interventions, in that it suggests additional investments in education and family planning may be needed to offset unintended fertility effects if the interventions not only save lives, but also improve general health. It should be noted, however, that the nature of our analysis requires that our fertility estimates be interpreted as relatively short run effects – the long run effect of child mortality on fertility may in fact be negative, and will only be known in this context after the women in our sample fully complete their childbearing, which has not yet happened.

The second major contribution of our paper is that it pushes the boundary of the literature on the specific mechanisms by which a change in the mortality environment affects fertility. Our paper tests over ten different mechanisms and determinants of fertility change hypothesized in the literature in a quasi-experimental population-based setting, allowing us to provide evidence for the importance of some channels, while minimizing the importance of others. For example, Lucas (2013) showed that a malaria eradication program in Sri Lanka increased fertility and hypothesized a health effect, particularly for

firstborn children. We show the first evidence for that channel. In addition, we also provide evidence for a second, previously ignored channel related to better health: curing malaria may lead to increases in sexual activity by healthier individuals, which may in turn increase fertility.

The paper proceeds as follows. Section I gives background on the anti-malaria campaigns. Section II describes the data used in our analysis and Section III outlines our empirical methodology. Section IV presents our main results. In Section V, we test several mechanisms by which fertility increased after the ITN distribution and discuss their implications. Section VI discusses the paradox of increasing fertility in spite of decreasing stated desires for children, introduces a Beckerian rational choice model (included in Appendix A) and contrasts it with Bongaarts' proximate determinants model. Finally we test several channels by which women may be unable to perfectly control their fertility. Section VII concludes.

I Background

Malaria is one of the most important public health challenges worldwide, with 214 million cases and 438,000 deaths in 2015.[5] Sub-Saharan Africa carries a disproportionately high share of the global malaria burden, since 90% of these deaths occurred in that region.[6]

Malaria is caused by the bite of a female anopheline mosquito that is infected with protozoan parasites. Although there are several species of the parasite, the Plasmodium falciparum strain is the most common (responsible for 98% of infections) and the deadliest in Africa (RBM, 2012).[7] There are around 430 species of Anopheles mosquitoes (Crawley and Nahlen, 2004), and around 30 of them are malaria vectors. Following the bite by an infected mosquito, the parasites leave the skin and migrate to the liver. After release, the parasites penetrate red blood cells where they multiply, causing an infection. An infected individual with no previous immunity is almost certain to develop severe flu-like symptoms that may lead to death, depending on the age and general health

[5] See http://www.rollbackmalaria.org/about/about-malaria/key-facts

[6] See http://www.who.int/mediacentre/factsheets/fs094/en/

[7] In this study, we refer to "malaria episodes" and "malaria prevalence" as those caused by the P. falciparum parasite.

of the individual. Over years of exposure, individuals develop partial immunity to the infection. Children under 5 and pregnant women are at higher risk of contracting the disease (Crawley and Nahlen, 2004).[8] Importantly, while young children have not yet developed immunity, pregnant women also temporarily lose their immunity.

Because of the high morbidity and mortality associated with the infection, the Roll Back Malaria Partnership (RBM) was launched in 1998 to coordinate action against malaria. Preventive interventions against malaria include ITN coverage, IRS, intermittent preventive treatment uptake during pregnancy (IPTp), use of mosquito repellants, cleaning of drains, and treatment of standing water with larvicidal chemicals.[9] These interventions work by reducing the number of mosquitoes and/or by preventing bites – except for IPTp which is the administration of a dose of antimalarial treatment, regardless of whether the pregnant woman has malaria or not. Sleeping under an ITN is considered the most cost-effective intervention to prevent malaria (Lengeler, 2004). Specifically, anophelene mosquitoes tend to bite at night, and rest inside the house (RBM, 2012). When they come into contact with ITNs, they immediately die, which not only prevents infection but also reduces the vector population. Among these vector control measures, RBM recommends two core interventions: ITN usage and IRS. In parallel, the 2008 Millennium Development Goals Malaria Summit of the United Nations set a target of universal coverage with ITNs (that is, one ITN per two individuals) for all endemic areas in Africa (RBM, 2012).

International donors – such as the Global Fund, the President's Malaria Initiative, and the World Bank – provide ITNs and funding to perform IRS in each country. Then, the National Malaria Control Program (NMCP) for each country is responsible for the distribution of nets and the implementation of IRS with the help of non-governmental organizations. Most countries have a goal of universal coverage with nets.

Until recently, chloroquine was the most widely used antimalarial. However, as parasite resistance to chloroquine began to spread, ACT – which works by combining two active ingredients to avoid drug resistance – has become the most common curative intervention. Currently, both the RBM and WHO guide-

[8] See http://www.who.int/mediacentre/factsheets/fs094/en/.

[9] Larval source management only plays a minor role in malaria control in Africa.

lines suggest ACT as the first-line treatment for confirmed cases of malaria. Unfortunately, frequent supply shortages and stock-outs of the drug, poor access to rapid diagnostic testing, and demand-side impediments (such as cost, distance to health clinics, etc.) have led to malaria treatment rates which are far from universal (Apouey et al., 2018).

II Data

We derive household- and individual-level information on mortality, fertility, and socio-demographic characteristics from a set of surveys produced by the DHS Program between 2000 and 2014 for all countries in sub-Saharan Africa. The list of surveys used can be found in Table 1. Information on malaria prevalence and preventive behaviors are from the Malaria Atlas Project (MAP). Both of these datasets are described separately below.

II.A The DHS Program

The DHS Program assists hundreds of national survey programs to collect globally standardized data on population and health in low- and middle-income countries. Within this program, we use the standard Demographic Health Surveys (DHS), the Interim Demographic Health Survey (DHS-I), the Malaria Indicator Surveys (MIS), and the AIDS Indicator Surveys (AIS) to compile a detailed data set of birth histories for 833,246 women and 1,147,543 children. In addition, these data contain detailed information on health and preventive health behaviors for children, women, and men.

Information on child mortality and fertility are derived from these birth histories. In the DHS and DHS-I surveys, the women's questionnaire provides birth histories for all children born to women in the sample, including the date of birth for all children ever born and the date of death for deceased children. The MIS and AIS usually include a shorter birth history module that contains the date of birth for the last five children and data on whether the children are alive at the interview date.[10] Because malaria eradication was not a health policy priority before the creation of the Roll Back Malaria Partnership, we restrict our sample

[10]The only exception is the last two MIS surveys in Malawi (2012 and 2014) which only have data on the last four births.

to surveys carried out after 1999. Overall, the data set includes 91 surveys from 35 countries and 365 sub-national regions.

II.B The Malaria Atlas Project

Information on malaria prevalence and preventive behaviors come from MAP. Using the region of residence of households as given in the household data, we are able to merge the birth histories with data on malaria prevalence rates, ITN usage, and IRS coverage.[11] MAP uses parasite surveys as well as environmental data to calculate annual malaria prevalence rates on a 5x5 km grid using a geo-statistical model.[12] With shape files for each sub-national region given by the DHS program, we use GIS software to calculate a panel of malaria prevalence rates for each region in our survey. In instances where regional boundaries in the birth history data have changed between surveys, we combine regions using maps provided in the public report of each survey to get consistent regions over time.

We measure malaria prevalence as the Plasmodium falciparum parasite rate (PfPR). Specifically, our measure of PfPR is PfPR_{2-10}, which represents the percentage of children between the ages of two and 10 who have measurable levels of the P. falciparum parasite in their peripheral blood (Gething et al., 2011).[13] Figure 1 shows the parasite rate in the set of countries used in our sample both in 2000 and 2014 using the raw MAP data at the 5x5 km resolution. There are two striking features to this figure. First, there are large variations in PfPR across

[11] A region in our data is a sub-national geographic unit as defined by the DHS. Generally they correspond to the GEOLEV1 GIS regions of a country, which are usually the first-level administrative region. For example, the first-level administrative region in the United States is the state, while the second-level is the county. However, since these sub-Saharan African countries are generally much smaller than the US, the average region size in our sample is approximately the same size as a US county.

[12] For Africa, MAP uses total of 27,573 geo-referenced points from literature searches, personal communications, and household surveys across 43 African countries spanning a time range from 1995 to 2014. Useful pieces of parasite survey information are the location-time of each survey, the number of individuals tested for the parasite, and the number of individuals infected with the parasite. The environmental data include rainfall, temperature, land cover, and urban/rural status. In the geostatistical model, the imputed value of PfPR for a particular target location is a weighted average of the observed values of PfPR (from the nearby parasite surveys) and the predicted values of PfPR (computed using the environmental factors). The weights reflect the spatial and temporal proximity between the target location and the location of the parasite surveys. See Bhatt et al. (2015) and its supplemental material for more details about the MAP data.

[13] This is the age group for which the parasite is most easily detected

space – even in areas where malaria is considered endemic. Second, there has been a significant decline in malaria prevalence rates in a relatively short time span of 15 years. The WHO reports that between 2000 and 2015, malaria prevalence rates fell by 66 percent in Africa.[14] This rapid decline is due to the Roll Back Malaria program and its scale-up of ITN and IRS in sub-Saharan Africa.

In addition to prevalence data, MAP also provides data on ITN usage at the 5x5 km resolution, and we calculate average ITN usage as we did malaria prevalence rates. It also provides information on IRS and ACT uptake for each country-year. Figure 3 represents the evolution of ITN usage, IRS, and ACT uptake for the set of countries used in our analysis between 2000 and 2014. The figure shows a rapid scale-up of access to ITNs and ACT starting around 2005. IRS rates also increased between 2000 and 2010, but recently have declined slightly.

Not only is there temporal variation in the scale-up of the ITN distribution campaigns demonstrated in Figure 3, but there is also spatial variation. Figure 2 shows the geographic distribution of ITN usage across the countries in our sample in 2014 from the MAP data. We do not show the distribution of ITN usage in 2000 because it was zero everywhere – therefore, the change in the ITN usage rates from 2000-2014 can be seen by simply looking at the 2014 usage rates. Evident from the figure, there is a large variation in the intensity by which regions received ITNs – variation which does not always correlate with underlying malaria prevalence. It is this variation – both spatially in Figure 1 and Figure 2, and temporally in Figure 3 – from which our identification strategy is derived.

III Empirical Specification

III.A Models

We estimate the effect of antimalarial campaigns on child mortality and fertility by using a continuous difference-in-differences model that exploits variation in the timing and intensity of the campaigns in different regions, along with variation in pre-campaign malaria prevalence intensity. This estimation strategy is broadly similar to that used in Bleakley (2010), Barofsky et al. (2015), Lucas

[14] See http://www.who.int/malaria/media/world-malaria-report-2015/en/

(2010), Cutler et al. (2010), and Venkataramani (2012), among others in this literature. Specifically, we estimate the following mortality and fertility equations:

$$M^a_{irct} = \gamma^m_1 ITN_{rct} + \gamma^m_2 \overline{P_{rc}} \times ITN_{rct} + \Pi^m X^m_{irct} + \alpha^m_{rc} + \theta^m_t + \psi^m_c t + \epsilon^m_{irct} \quad (1)$$

and

$$F^b_{jrct} = \gamma^f_1 ITN_{rct-1} + \gamma^f_2 \overline{P_{rc}} \times ITN_{rct-1} + \Pi^f X^f_{jrct} + \alpha^f_{rc} + \theta^f_t + \psi^f_c t + \epsilon^f_{jrct} \quad (2)$$

where M^a_{irct} is a dummy variable for whether child i of age a who lives in region r in country c at time t died during her ath year of life. Similarly F^b_{jrct} is an indicator for whether a woman j in age group b who lives in region r in country c at time t had a child within the last 12 months. We estimate five mortality equations, one corresponding to each child's first five years of life (0-12 months, 13-24 months, 25-36 months, 37-48 months, and 49-60 months). Similarly, we estimate six fertility equations – one for each five year age group beginning with 15-19 and ending with 40-44 – corresponding with a woman's fertile years. It should be noted that since we have a yearly panel, our estimates imply annual hazards of birth or death. Therefore, in our fertility equation a single woman can enter an age group sample up to five times, one for each year she is in that age group, before moving on to the next age group sample.

ITN_{rct} is the fraction of individuals in region r in country c at time t who report sleeping under an ITN, given in the MAP data. For the fertility equation, we lag the ITN variable by one year, since if the decision to have a child is dependent on campaign intensity, the parents of a child born this year were deciding to have a child based on the conditions at least nine months previous, if not earlier. Also notice that the ITN variable is calculated at the region level – we are not measuring whether the specific child i or woman j slept under an ITN. Therefore, the interpretation of this variable should be as the intensity of the ITN distribution campaign in the region – *not* the effect of whether the individual actually slept under a bed net. This is intentional – our intent is to identify the effect of the *campaign* – including spillover effects – on mortality and fertility in a region, using regional ITN usage as a proxy for campaign intensity. Since the main effect of the campaigns was to reduce malaria, this is

likely a somewhat tedious distinction, but important to make since we cannot rule out additional effects of the campaigns on mortality and fertility through other channels.[15] However, this is precisely the methodology one should employ for the purposes of this paper: both policy makers wishing to conduct cost-benefit analyses – and many academics interested in estimating the effect of mortality on fertility – are concerned with the aggregate mortality effects of campaigns including general equilibrium and spillover effects, rather than isolating specific channels. In addition, the individual decision to sleep under an ITN is highly endogenous, since it is likely correlated with any number of other unobserved individual-level characteristics which may also explain mortality or fertility. Finally, while the DHS does contain data on whether an individual respondent slept under a bed net the night before the survey, we do not have an annual panel of such responses, and therefore would not be able to identify how this variable changed over time as the intervention became more intense.

$\overline{P_{rc}}$ is our measure of pre-intervention malaria prevalence, PfPR_{2-10} in the year 2000.[16] The main parameters of interest in these specifications are on the interaction between ITN_{rct} and $\overline{P_{rc}}$, or γ_2^m and γ_2^f. If antimalarial campaigns are effective in reducing malaria mortality among children, then we expect $\gamma_2^m < 0$. The sign on γ_2^f is ambiguous, since – as shown in our conceptual framework in Appendix A – the theoretical effect of antimalarial campaigns on fertility is itself ambiguous.

The control variables in the mortality equation, X_{irct}^m, include the mother's age at birth, mother's age at birth squared, birth order fixed effects, education level, child gender, and urban/rural status. The control variables in the fertility equation, X_{irct}^f, include the mother's age at birth, education, the birth interval, the birth interval squared, and urban/rural status. All regressions also control for the levels of two other malaria reduction interventions – anti-malarial drugs (Artemisinin-based Combination Therapy or ACT) and indoor residual spraying (IRS), which are given in the MAP data at the country-year level. Finally, we also control for region and year fixed effects (α_{rc}^m, α_{rc}^f, θ_t^m, and θ_t^f) and country

[15]For example, if the bed nets also prevented bites from the Tse-Tse fly, interpreting our results as solely the effect of malaria reduction would be incorrect since reductions in mortality from both sleeping sickness and malaria would be included in our estimates.

[16]Technically our measure of $\overline{P_{rc}}$ is demeaned from the average regional level of $\overline{P_{rc}}$ across our sample, since that allows us to interpret γ_1^m and γ_1^f as the average partial effect of ITNs on malaria prevalence. This change does not affect the interpretation of our coefficients of interest.

specific time trends ($\psi_c^m t$ and $\psi_c^f t$).

III.B Identification Assumptions

The main econometric concern of our study is the endogeneity of our interaction term. Notice that it is not necessary for our identification strategy that the intensity of the campaigns themselves be exogenous to the average levels of mortality, fertility, or even pre-existing levels of malaria prevalence in each region. In fact, it seems a priori unlikely that decisions about antimalarial campaigns were made without taking these differences into account. In addition, places which have higher levels of malaria may also be systematically different than those which do not, in that they may receive higher levels and more types of non-ITN aid because they are just worse places generally. We address these concerns in two ways. First, and fortunately for our identification strategy, since our econometric specification includes region fixed effects, we are implicitly controlling for the fact that regions with higher malaria prevalence may be systematically different, and hence attract more aid in general once the campaigns begin. Second, we provide evidence (presented in Section VI.B) that ITN distribution was relatively haphazard in three ways: (1) the timing of the ITN distribution campaigns at the country level was uncorrelated with observable country characteristics like the level of development or pre-intervention malaria prevalence, (2) once the campaigns began at the country level, the ITN distribution began in every region simultaneously, independent of observable characteristics, and (3) the amount of ITNs eventually received by each region is uncorrelated with the region's pre-intervention malaria prevalence.

A more realistic problem is if the policy makers systematically assigned more nets to those places where mortality or fertility were already changing faster – violating the parallel trends assumption. This is more difficult to test directly. Instead, we attempt to control for this problem in three ways. First, we include country-specific time trends in all our specifications. Second, we run placebo tests where we lead the ITN data by two and four years into the future. If it is true that our estimates were identifying off of pre-existing trends, then leading the data should produce similar effects. As discussed in the Results Section, we pass both of these placebo tests. Third, we plot the mortality and fertility rates for four groups of regions in Figure 4: those which even-

tually received above-median levels of ITNs and had above median levels of pre-intervention malaria prevalence, those with below median ITNs and above median pre-intervention malaria, as well as the corresponding two groups with below median pre-intervention malaria prevalence. For the period before any ITN distribution took place (2000-2005), there is no difference between the trends of mortality and fertility in the four sets of regions.[17]

Another concern is whether the bed nets were distributed in conjunction with other interventions which reduced mortality. This is especially important, since bed nets are in fact often distributed to women going to antenatal care or other health clinic visits, and therefore are likely to receive a bundle of health interventions at the same time. If these health clinic visits would have taken place regardless of the ITN campaigns, then the campaigns may not have increased the overall amount of these additional interventions. However, if people went to the clinics for bed nets, and then decided to get antenatal care and vaccinations they otherwise would not have gotten, then our estimated mortality and fertility effects may be driven by these additional health interventions, and not by ITNs per se. To test for this, we do two things. First, we control directly for antenatal care visits and vaccinations in the sub-sample for which those data exist. Second, we run our main specification using vaccinations and antenatal care visits as the dependent variable to test whether the increase in bed nets led to a higher level of these other public health interventions.[18] As discussed in the results below, we find no correlation between ITN usage and these other preventive health interventions.[19]

A final concern is whether ITN usage is purely measuring a supply shock.

[17]Traditionally one would compare the pre-intervention trends between regions which eventually received the intervention and those which did not. However, that requires a discrete treatment and control group, whereas in our analysis both the treatment and control groups are continuous, which is why we divide our sample into above- and below- median groups. We also divided the sample into quintiles of eventual treatment intensity and found the same results: there are no differential pre-existing trends in mortality and fertility across groups of regions with different eventual treatment intensities. Those results are available upon request.

[18]If this is the case, then it cannot be said that the bed net distribution campaigns alone had an effect of mortality. However, that is not to say that bed net campaigns are still not useful. If the campaigns caused more women to get vaccinations and antenatal care, which in turn reduced mortality, then that is part of the overall reduced form effect of the campaigns, which a policy maker evaluating the program would want to capture.

[19]While this may be because there is in fact no bundling of services, it may also be a net effect of bundling and crowding out – ITNs may be bundled with other interventions when they are received, but health clinics which receive more ITNs may receive less vaccines and other resources as policy makers try to distribute interventions fairly across clinics.

In this analysis, the underlying assumption is that ITN usage is an appropriate proxy for the intensity of the ITN distribution campaigns. However, it is possible that the increase in usage represented an increase in demand – access to the ITNs was always available, but different regions began demanding ITNs with different intensities at different times due to increases in income or other time-variant regional characteristic. We believe that this is not the case for two reasons. First, it is hard to believe that the sharp and simultaneous increase in ITN usage in all regions at the country level as illustrated in Figure B2 is a demand shock – if ITNs truly were available throughout the sample period, one would not expect ITN usage to be uniformly zero for all years until the intervention and then jump to 20-30 percent or more in a single year. Second, the influx of ITNs at the country level is well documented in the country Roll Back Malaria annual reports.

To further tease out demand from supply effects, as a robustness check we change our dependent variable from ITN usage to an indicator variable based on when the intervention began for a given region. Specifically, for each region-year we change the ITN variable to take a value of 1 if that region ever surpassed 10 percent ITN usage. By changing the ITN variable to an indicator, we are less likely to pick up differential changes in ITN demand, but rather only measure whether the supply intervention had begun. The results are so similar to our analysis using ITN usage that we decided not to report them, but they are available upon request. Similarly, we also explicitly tested for differential pre-trends using the indicator variable as the intervention timing, and found no evidence of non-parallel trends.

IV Results

IV.A The Effect of Campaigns on Mortality and Fertility

Table 3 Panel A reports our estimates of the effect of the campaigns on child mortality. The columns contain the results for the different age groups: children born 0-12, 13-24, 25-36, 37-48, and 49-60 months before the interview. Although not our main coefficient of interest, the coefficient on ITN usage gives the average partial effect of ITN usage on mortality. This effect is negative for the regressions representing infant mortality (Column 1) and mortality during

a child's second year of life (Column 2), and either insignificant or marginally significant thereafter. Although this coefficient cannot be interpreted causally, our result implies that regions which get more nets are correlated with lower levels of child mortality at young ages.

The interaction term between malaria prevalence and ITN usage (our coefficient of interest) is similar to the level effect of ITNs, with some important caveats. For infants, the coefficient is negative but not statistically significant. However, for children 13-24 and 25-36 months, there is a strong negative impact of ITN distribution on mortality. Our results are consistent with evidence from the biological literature which shows that children in their second year are most at risk for malaria, since children gain partial malaria immunity from the disease for approximately the first 6 months of life via maternal immunoglobulin G (IgG) antibodies acquired in utero. In addition, partial immunity during the first year may be gained through parasite growth-inhibitory factors such as lactoferrin and secretory IgA found in breast milk (Doolan et al., 2009).

Interpreting the magnitudes of our coefficients, we find that our estimates imply that the causal effect of the campaign to distribute ITNs in a region with average malaria prevalence (0.4117 from Table 2), and an increase in average ITN usage from 0 percent (approximately the 2000 level from Figure 3) to 50 percent (approximately the 2014 level) is -0.0108 $\cdot$ 0.5 $\cdot$ 0.4117 $\cdot$ 100 = -0.222 percentage points. From a base mortality rate of 7.13 percentage points, this is a 3.12 percent reduction in infant mortality – which is statistically insignificant. A similar calculation shows that the introduction of ITNs reduced mortality from 13-24 and 25-36 month-olds by 22.2 percent and 40.6 percent respectively, both of which are highly statistically significant at the one percent level. For 37-48 and 49-60 month old children, the ITN distribution reduced mortality by 25.6 and 20.6 percent respectively, however those estimates are not statistically significant due to the very low underlying mortality rates.[20]

[20]The correct method of interpreting our coefficient is somewhat unclear. On the one hand, the average partial effect of ITNs on mortality is technically the sum of the coefficient on the level effect of ITNs plus the coefficient on the interaction times the malaria prevalence in the region. However, this sum cannot be interpreted as being causal, since the level effect is unidentified. On the other hand, if we only use the coefficient on the interaction term, then we are only using the causally identified parameter to estimate the effect of the ITN distribution, but are then assuming that the level effect is zero. For the remainder of the paper, we opt for the second method, for two reasons: First, we are most concerned with only using properly identified coefficients. Second, this is the option which gives us more conservative results. For example, including the level effects now causes infant mortality to be significantly negatively impacted

Table 3 Panel B reports our estimates of the effect of ITNs on fertility. In the columns, we report the results by 5-year age groups, allowing us to interpret our results as changes in the age-specific fertility rate. As before, the coefficient on ITN usage gives the average partial effect of ITNs on age-specific fertility rates. We find no evidence that areas which received more nets have systematically higher fertility rates.

Just as with our results for mortality, our coefficient of interest is on the interaction term between malaria prevalence and ITN usage. This coefficient is positive for every age group, and but only significant at the 5 percent level older women aged 30-34 and 40-44. To interpret these coefficients, we take as an example the estimate in Column (1), for women ages 15-19. Doing the same calculation we did for mortality, we find that the distribution of ITNs in the average region increased the annual probability of having a child for women in this age group by 0.173 percentage points. With a base fertility rate of 11.71 percent annual hazard of having a child, this implies a 1.5 percent increase in the age-specific fertility rate. Similarly we find a 3.2, 3.6, 8.0, 3.6, and 14.6 percent increase in the age specific fertility rates for women aged 20-24, 25-29, 30-34, 35-39, and 40-44 respectively. Overall, this implies an increase in the total fertility rate of 0.3 children per woman, an increase of 5.3 percent on a base of 5.6 children.

IV.B Threats to Identification and Falsification Tests

As noted in the Empirical Methodology Section, there are several threats to the credibility of our mortality and fertility estimates just presented. However, it may be first beneficial to talk about common concerns which are actually *not* threats to identification.

First, one may be concerned that our estimates would be biased if policy-makers sent regions with worse malarial conditions more ITNs. However, since our regressions contain region fixed effects, this controls for the fact that certain regions have exogenously worse malarial conditions and – jointly with the regressor for the level effect of ITNs – simultaneously controls for the higher

by the ITN distribution, with a decrease of 25.1 percent – significant at the one percent level – as opposed to an insignificant 3.12 percent decline. 13-24 and 25-36 mortality still significantly drops by 46.5 and 26.5 percent respectively, while 37-48 and 49-60 mortality is insignificantly affected, even if the coefficients are still large at 15.6 and 39.0 percent reductions in mortality.

level of ITNs sent to such regions once the interventions began. Second, one may be concerned that regions with better institutional characteristics received more nets once the interventions began – since they are better able to manage and distribute the ITNs. Again, since our regressions contain region fixed effects and the level of ITNs directly, this controls for both institutional quality between regions and for the level of ITNs sent to the region.

In addition, even if the fixed effects did not control these region-specific characteristics, it appears that the timing or the intensity of the ITN distribution were not correlated with observable characteristics of the country or region. We provide four pieces of evidence that this is the case. First in Figure B1 we plot the eventual intensity of the ITN distribution (ITN usage in 2015) against the pre-intervention malaria prevalence (prevalence in the year 2000). The figure shows, surprisingly, that the ITN distribution intensity was completely uncorrelated with the level of malaria prevalence in a region. Second, Figure B2 shows the same data as Figure B1 in the panel on the left, but only for a single country Nigeria. [21] In the panel on the right, we add a graph showing the intensity of the ITN distribution, by region and year. There are three notable features to this figure. First, as we showed in Figure B1, the intensity of the regional ITN distribution is uncorrelated with the level of malaria in that region. Second, when the ITN distribution started in Nigeria, it began in all regions simultaneously. Third, in spite of beginning simultaneously in all regions, there is significant heterogeneity in the ITN distribution intensity among the regions.

The third piece of evidence that the timing and intensity of the ITN distribution was uncorrelated with observable characteristics is in Figure B3. We plot the timing of the beginning of the ITN distribution by region in our entire sample – defined as the first year ITN usage rose above 10% of the population – against the pre-intervention malaria prevalence. As can be seen from the figure, there is no correlation between the timing of the beginning of the ITN distribution campaigns and how malarious a region was. Finally, in Table B14 we regress the regional timing of the beginning of the ITN distribution on a series of nine observable characteristics related to the level of development in a region, and

[21] Ideally we would show this for every country, but that is impossible due to space constraints. We chose Nigeria because it has the highest malaria burden in sub-Saharan Africa. All other countries follow a similar pattern of distribution. This data for all countries are available on our website.

find there is no correlation between timing and any of these variables.[22]

What is a problem for identification, however, is if mortality or fertility were already changing differentially in regions which eventually received more ITNs. We attempt to control for the existence of different parallel trends by including a country-specific time trend in all of our regressions. Another method for testing for non-parallel trends is to run a placebo test where the intervention in the data takes place at a different time than it does in reality. In the case that the results are driven by a general trend, this placebo regression should show similar results to the regression with the "true" intervention, because the results are driven by the general trend and not the intervention itself.

We run this placebo test in Tables 4 for mortality and 5 for fertility. Each table contains two panels – Panel A in which we lead the intervention by two years, and Panel B in which we lead it by four. For the mortality placebo regression in Table 4, the interaction between malaria prevalence and ITNs are not significant in either the two- or four-lead specifications for any age group.

The fertility placebo regressions in Table 5, however, are significantly different from zero in 2 of the 10 specifications – specifically for older women with 4 year leads. While certainly far from a universal or robust result, it still may give one pause regarding our parallel trends assumption. However, since our main fertility results show a positive effect of ITNs on fertility, the fact that a few of the placebo estimates are negative suggests that if there are pre-existing trends, they are precisely in the opposite direction than would be driving our main results. If anything, this provides evidence that our main fertility results may be underestimated.

Another concern is that regions which received more ITNs also received more of other interventions which affected infant mortality or fertility directly, such as vaccines, antenatal care, visits by health workers, contraception, etc. In this case, the effect of these interventions would be picked up by our coefficients, overstating the effect of the malaria control programs. To test this, we re-estimate our main specification using as our dependent variables several health behaviors which are not directly affected by bed net usage. Specifically, we use a dummy for whether the child has received a visit from a health care

[22]The variables are fraction of the women which report living in an urban region, owning a radio, TV, refrigerator, car, having electricity, and improved source of water, improved toilet, and a quintile indicator for household wealth.

worker in the last 12 months, whether the child has been given full vaccination of BCG for tuberculosis, DPT (for diphtheria, pertussis, or tetanus), or either of those two vaccines. If the interaction term between malaria prevalence and the ITN variable is positively and significantly correlated with other health interventions, then we know that our main estimates are likely picking up the effect of these additional interventions.

The results of the falsification test are given in Table 6. We find no effect on the interaction term in each of our regressions. Therefore, these results suggest that instead of being complements, ITNs and other health interventions are either uncorrelated, or that the bundling effect was negated by the ITNs crowding out other resources to the health clinics.

Finally, in Figure 4 we plot the mortality and fertility rates for regions over time to show there are no preexisting trends in regions which eventually received above the median level of ITNs. For the period before any ITN distribution took place (2000-2005) there is no difference between the trends of mortality and fertility in the above-median ITN distribution regions compared to those regions which received a below-median level of ITNs.

V Mechanisms

Up to this point, our analysis has been limited to estimating the causal effect of ITN distribution campaigns on mortality and fertility. However, we have not yet identified the mechanisms driving this relationship. From our conceptual framework in Online Appendix A, we know that the effect of mortality on fertility can be ambiguous. And even if it were not, many social scientists critique the idea that the number of children a woman has is the product of a rational decision making process at all, especially in the context of sub-Saharan Africa where female empowerment is low and women are less able to control fertility compared to the developed world.

In this section, we attempt to elucidate the reasons why fertility is increasing due to the ITN distribution campaigns. We analyze heterogeneity by gender and education to see if certain subgroups were more affected by the malaria interventions than others. We also test whether the increases in fertility were along the intensive or extensive margins by exploring the effect on birth spacing. We then use Bongaarts' (1978) proximate determinants model to determine

the mechanics behind the change in fertility. Finally, we test whether fertility preferences changed, and end the section with some discussion of our results.

V.A Heterogeneity Results

In Table B4 (in the Online Appendix B), we divide our sample between female births and male births to see if the ITN distribution had heterogeneous effects on mortality by gender. In the overall sample, ITNs only had an effect on mortality in the second and third years of life. We find a similar effect for males – mortality falls for children between 13 and 36 months. However, infant mortality for females is only significantly reduced for girls aged 13-24 months, and that result is only significant at the 10 percent level. This is consistent with a number of theories, such as male preference (the ITNs are more likely to be used for male children than female) or the fragile male hypothesis (males are more likely to die than females, meaning the introduction of ITNs would save more male lives than female).

We now check to see whether the effects on both mortality and fertility differ by socioeconomic status. To do this, in each regression we add a triple interaction for whether the mother has not completed primary education. Our results are shown in Table B2. Starting with the mortality regressions, we find that the coefficient on the interaction of malaria prevalence, ITNs, and not having a primary education is large and negative for children of all age categories except for 49-60 months. At the same time, the coefficient on the interaction between malaria prevalence and ITNs has become a statistical zero for all ages except for 13-24 months, which is only significant at the 10 percent level. Specifically, our point estimates imply that, for the average region, child mortality between 13-24 months of uneducated women fell by 34.5 percent, compared with a 3.8 percent decline for women with at least a primary education. This compares with the 22.6 percent decline in the pooled sample in Table 3. This implies that our original result was driven solely by reductions in infant mortality among the children of uneducated women.

Conversely, however, we find that the increases in fertility are much stronger among women with at least a primary education than the uneducated. Adding a triple interaction with no education to our fertility regressions yields negative and highly significant coefficients in the ages of 20-34, which ages form the bulk

of child-bearing women. In addition, the coefficients on the interaction between ITN usage and malaria prevalence (which represents the effect for the educated) is now positive and significant for all age groups except 15-19. Among the uneducated, only older women (ages 40-44) increased their fertility significantly in response to the ITN distribution. Interpreting the coefficients in this table, we find that age specific fertility rates rose for women aged 15-19 by 4.1 percent for the educated and fell by 1.3 percent for the uneducated. Fertility rose by 6.0 percent and -0.5 percent for 20-24 year old women, by 6.4 and 0.5 percent for ages 25-29, by 11.9 and 3.6 percent for ages 30-34, by 6.7 and 1.1 percent for ages 35-39, and by 13.3 and 17.0 percent for ages 40-44, respectively for the educated and uneducated. All coefficients are significant at the 1 percent level for educated women except 15-19 years old, and are all insignificant for uneducated women except 40-44 years old.

Interestingly, since infant mortality only fell for the uneducated while fertility increased for the educated, this suggests that the set of women who experienced reductions in infant mortality were different from those who increased their fertility. This begs the question of who exactly received the ITNs: if only lower SES individuals received more nets, perhaps this explains why infant mortality disproportionately fell among this group. Unfortunately, the MAP data do not break out ITN usage by education level of the mother. However, the birth history data does have a household-level bed net usage variable which we can use to answer this question.

In Figure B4, we plot reported bed net usage rates from the DHS data by education group.[23] We see that households with women without primary education started with lower bed net usage rates than households with at least primary-educated women in 2000. However, less-educated households increased their bed net usage at a faster rate and essentially converged to educated households by 2010, with the largest scale-up in bed net usage beginning in 2005 corresponding with the main ITN scale-up shown in the MAP data in Figure 3. It is important to note, however, that the differences between the two groups are

[23] Since DHS surveys do not exist for every year for every country, we do not have a balanced panel of bed net usage at the region level as in the MAP data. Therefore, we use geometric interpolation between DHS survey years at the country level to derive this figure. Note also that the DHS data ask about any bed net usage – not ITNs specifically – which explains why bed net usage rates in 2000 are approximately 25 percent, even though Figure 3 reports ITN usage to be approximately 0 percent in 2000.

relatively small – only separated by less than 5 percentage points at the largest gap. Therefore, while there is some evidence suggesting the uneducated disproportionately benefited by the ITN distribution programs, we do not feel comfortable concluding these differences are the main driver of the heterogeneous results between education groups.

Finally, we test how a specific dynamic of fertility – birth spacing – changed after the ITN distribution. Knowing whether women are having more children due to changes in spacing will help elucidate whether fertility increases are along the intensive or extensive margins. For example, if overall fertility rises with no change in birth spacing, this is likely because a larger fraction of women are having children. In Table B3 we run the same fertility regressions with triple interactions as in Table B2 on each 5-year age group, except that the dependent variable is the number of months since a woman had her last child.

We find that younger educated women significantly intensify their births as a result of ITN distribution. For example, we find that educated women ages 15-19 in the average region reduced their birth spacing by 1.42 months on a base of 23.5, implying that fertility intensified along the intensive margin by 6.9 percent. Similarly, we find an increase in the intensive margin of fertility of 2.9 percent for educated women ages 20-24, and 3.1 percent for ages 25-29. All other age groups for older women are insignificant. Note that overall fertility rose for these three groups by 1.5, 3.2, and 3.6 percent respectively. This implies that the extensive margin of fertility must have fallen by 5.4 percent for 15-19 year olds, and risen by only 0.3 percent and 0.5 percent for 20-24 and 25-29 years old. As a result it appears that the increases in fertility for these age groups are only happening along the intensive margins rather than the extensive.

For uneducated women, the triple interaction term is almost always equal and opposite to the coefficient on the ITN * Malaria Prevalence term, which implies that there was no change in birth spacing for any group of uneducated women. Since there was no change in birth spacing for any older women, and both groups of older women had significantly higher fertility, this implies that – in contrast with younger women – that older women's fertility increased mainly on the extensive margin.

V.B Fertility and Bongaarts' Proximate Determinants

Bongaarts (1978) posited a proximate determinants model of fertility which famously proposed that irrespective of whether fertility is a choice, it should be directly affected by eight different channels: proportion married, contraception, induced abortion, lactational infecundability, frequency of intercourse, sterility, spontaneous intrauterine mortality, and duration of the fertile period. In this section, we test whether these proximate determinants of fertility changed in order to understand what is driving the increase in fertility after the ITN interventions. The DHS data provides direct measures for a number of these proximate determinants. Specifically, the DHS has data on marital status, contraception, pregnancy termination, and sexual activity. No comprehensive data exists on sterility, lactation, or duration of the fertile period.[24]

We begin with induced abortion and spontaneous intrauterine mortality. It is difficult to test for fetal loss directly in the DHS data for several reasons. First, pregnancies are particulary fragile during the first two weeks after conception, with approximately 75 percent of conceptions being terminated spontaneously even before the mother knows she is pregnant (Wilcox et al., 1988; Boklage, 1990; Wilde et al., 2017). Since a termination is only counted in the DHS birth history if a woman knows she is pregnant and then the pregnancy terminates, the vast majority of fetal loss will not be captured by the data. Second, even if the DHS did contain the true universe of terminations, it does not distinguish between spontaneous and induced abortions.

To overcome these challenges, we do two things. First, due to the fact that males are generally weaker in utero than females, a common indicator for excess intrauterine mortality is the gender ratio at birth. In Table B4, we run our main specification in equation (1), except that our dependent variable is an indicator for whether the birth is a male. We find there is no detectable effect on the gender ratio due to the ITN distribution campaigns. Second, in spite of the difficulties we run a similar analysis in regards to reported terminations in Table B5. We find no evidence for a reduction in terminations at any age for neither the educated nor the uneducated.[25] As a result, neither Table B4 nor B5 provide

[24]Questions regarding contraception sometimes include responses referring to sterility and lactational infecundability. However, there are no questions strictly related to these fertility determinants.

[25]There is one negative and statistically significant coefficient on the triple interaction for

consistent evidence that fertility increased due to reductions in spontaneous or induced abortion.

Next we look at sexual activity. In Table B6 we run our fertility regression with a triple interaction for primary education, except our dependent variable is an indicator variable for whether the woman reported being sexually active in the past four weeks. For educated women, we generally find positive coefficients on sexual activity, which are significant for women ages 25-29, 30-34, and 35-39. We also find that the effect for uneducated women is not statistically different from the educated. Therefore, one possible explanation for the increase in fertility is that after the ITN distribution campaigns, more women became sexually active. This makes sense – if sexual activity or libido is linked with personal comfort, then individuals who are sick with malaria may forgo sexual activity they would have otherwise engaged in in the absence of the disease.

Finally we ask whether there were changes in contraceptive usage.[26] In Table B7 we run a similar regression as before with an indicator variable for whether women reported using not contraception as the dependent variable. We find that uneducated older women (ages 35-39 and 40-44) have significantly higher contraceptive usage (lower rates of not using contraception) after the intervention than before in areas with higher ITN distribution compared with educated women. However, educated women of all ages see no change in their contraceptive usage. Combining the coefficients, uneducated women ages 30-34, 35-39, and 40-44 increased their overall contraceptive use, and all other groups saw no change, with the exception of uneducated women ages 15-19 who decreased their contraceptive use.

V.C Fertility Desires

While Bongaarts' proximate determinants model is useful to understand the mechanics of fertility decline, it does not address how a woman's fertility choice is determined. For example, a reduction in contraceptive use may be caused by a desire to reduce fertility, but it may also be due to contraceptive supply shocks, pressure from intimate partners or extended family, or social norms more broadly. As a result, in order to understand why fertility increased as a

educated women ages 25-29, but when combined with the double interaction yields a coefficient for the educated of essentially zero.

[26] We define contraceptive use to include both traditional and modern forms.

result of the ITN interventions, it may be useful to understand the extent to which the better health environment induced changes in the planned or desired number of children rather than the actual number of births.

Using the DHS data, we test whether women desired more children as a result of the ITN distribution programs. We analyze the responses to two different survey questions: an indicator variable for whether a woman wants another child, and another for whether she wants another child within the next two years. We run the same fertility regressions with triple interactions as in Table B2 on each 5-year age group. The results are reported in Tables B8 and B9. In Table B8 – which reports the effect of the ITN distribution on whether women want another child generally – we observe strong and universal declines in desired children, for all age groups except for the oldest women, and both for educated and uneducated women. In addition, there are no detectable differences in the effect of the ITN distribution on fertility preferences between the educated and uneducated. In Table B9 – which reports the effect of the ITN distribution on whether women want another child within the next two years – we also find declines for women 20-35, and no difference between the educated and uneducated. Interestingly, the percentage magnitude of the two questions are roughly the same for all age groups for both questions – 30 percent – yet it is harder to detect significance in Table B9 because there is less variation in the dependent variable.

V.D Discussion

So what does this all mean? To summarize our results, we found the only changes among Bongaarts' proximate fertility determinants were that sexual activity rose among both educated and uneducated women ages 25-40, but contraceptive use only rose for the uneducated. In addition, we found large and robust declines in desires for children across almost all groups of women. Comparing these findings with what actually happened to fertility, it appears that fertility rose among groups of women who 1. increased sexual activity, and 2. did not increase their usage of birth control, i.e. educated women, particularly at older ages.

These results leave us with a puzzle: if reductions in child mortality induce women to both want less children and to take action to prevent pregnancy, then

why did fertility rise for educated women, especially since their children's mortality rates were unchanged after the ITN distribution campaigns? This is particularly troublesome because these women strongly reported wanting less children, yet they increased their rates of sexual activity while not increasing contraceptive use. This disconnect between actions, desires, and outcomes seems to suggest fertility was not perfectly controlled by these women. If this is true, then that might lend credence to scholars who criticize the rational choice models of fertility – critics who contend that fertility cannot be controlled in this context, and therefore rational choice models should be substituted with models which focus more on the mechanics of fertility change.

VI Is Fertility a Choice?

In this section, we discuss the relative merits of two types of models of fertility decline, and how they can be used to explain our results. First, we quickly outline the history and differences between two schools of thought on fertility change. We then briefly introduce our economic model contained in Appendix A and sketch its main findings. Finally, we propose three reasons fertility desires and outcomes could diverge in the sub-Saharan African context, and test the extent to which these mechanisms are present in our results.

VI.A Becker vs. Bongaarts

Up until the 1950s, the decline in net fertility in Europe during the demographic transition of the 19th and early 20th centuries was generally explained by demographers and sociologists as the result of changing social norms and preferences. Fertility choice was widely considered to be outside the realm of economic analysis (Doepke, 2015). This changed with Gary Becker's seminal work on fertility, which modeled fertility decline not as a change in preferences, but rather as a tradeoff between quantity and quality (Becker, 1960). This Beckerian framework has formed the basis of most, if not all, economic models of fertility since.

On the other hand, demographers and sociologists have been less enthusiastic with this Beckerian approach, since they recognize there are many forces outside of a pure rational choice framework which affects fertility decisions.

For example, social norms, family influence, access to contraception, and the degree to which a woman actually has a say in the intra-household bargaining process can cause a woman's realized number of children to deviate from the number she would optimally bear as dictated by the Beckerian framework. As mentioned previously, Bongaarts (1978) seminal work outlined eight proximate determinants which affect fertility decline – only some over which the woman has any say.

In regards to the effects of child mortality on fertility decisions, both schools of thought treat the problem differently. In general, demographers gravitate towards models of fertility decline which are caused mechanically by falling infant mortality – if fertility preferences are fixed, then a falling infant mortality rate lowers fertility through reductions in replacement children. In a later iteration of Becker's model, Barro and Becker (1989) show that fertility should be completely independent of child mortality – unless it changed the overall cost of a surviving child. Interestingly, if falling child mortality lowers the cost of a surviving child, the Barro-Becker model actually predicts an increase in fertility, in contrast with the general trend of simultaneous declines in fertility and child mortality observed over the past 200 years. Later economic models based on the Beckerian framework corrected this apparent contradiction, by including mechanisms for replacement children and precautionary childbearing into their models.[27]

VI.B Conceptual Framework

We extend a version of a recent Beckerian model in our conceptual framework in Appendix A. The ideas contained in this model are not new. In fact, our model is essentially a modification of Kalemi-Ozcan (2003), in which we strip out the educational component and add in a variable time cost of childbearing which depends on the mortality environment. The model includes a quantity-quality tradeoff, uncertainty over the number of surviving children, and a cost of childbearing parameter which is assumed to be decreasing in mortality risk. The uncertainty over the number of surviving children encapsulates both the replacement child effect – since a higher mortality risk lowers the expected value of children – and the precautionary childbearing effect since we assume risk

[27] See Sah (1991), Kalemi-Ozcan (2003), and Doepke (2005) for examples.

averse agents.

Our model shows that the effect of a decline in child mortality is ambiguous. The quantity-quality, replacement, and precautionary childbearing mechanisms all imply that a decline in child mortality would reduce fertility. All of these mechanisms reduce fertility such that the desired number of births declines exactly by the number of children saved, and there is no change in net fertility, with the exception of precautionary childbearing, which reduces fertility more than one for one. The only mechanism that would increase fertility is the reduction in the cost of childbearing that comes with a better health environment when mortality declines. An implication of our model is that fertility should decline slower (or increase faster) for women whose cost of childbearing changes the most, which in the case of malaria is older women (Poespoprodjo et al., 2008; Hamer et al., 2009; Ayoola et al., 2012; Takem and D'Alessandro, 2013).

We make three observations linking the predictions of our model to our empirical results. First, if our model and results are correct, then this implies the ITN distribution campaigns significantly reduced the costs of childbearing – so much so that it more than offset the precautionary childbearing, replacement child, and quantity-quality mechanisms.[28] Second, our model predictions are consistent with the fact that the fertility effects are larger for older women. Third, it is important to note that our model is only useful in predicting what happens to a woman's optimal *desired* fertility. In this case, our results show a clear and universal decline in fertility preferences. As a result, it may be erroneous to assume a reduction in the cost of childbearing is the main driver of the increase in fertility: it may just be that woman's chosen fertility and realized fertility are not the same. We explore this idea in the following section.

[28] As mentioned in Appendix A, the costs of childbearing in this context can be quite large – they include not only lower direct time costs of caring for sick children or educating children which can learn faster, but also reduced risk of maternal death with each pregnancy, and a lower number of pregnancies needed to achieve a live birth. In addition, since time can be traded for income in our model through a wage, any reduction in pecuniary costs associated with healthier children (e.g. lower health care costs) or increase in the current or future earning capacity (e.g. child labor earnings, or an increase in old age assistance) of children would have the same effect. So although it may seem that a net positive fertility response of 5 percent necessitates an unrealistically large change in the cost of childbearing, in reality these costs may in fact be sufficiently large.

VI.C Women's Empowerment, Unmet Need, and Unexpected Fecundity

Our results show that the ITN distribution increased fertility in spite of reducing fertility preferences. In this section, we explore three potential reasons why women may not be able to lower their fertility in spite of wanting to do so: low empowerment or say in household decision making, unmet need for contraception, and unexpected effects of the ITN distribution campaigns on fecundity.

Our first hypothesis is low levels of say in household decision making. If a woman has little or no input in fertility decisions, then if the ITN distribution increased fecundity and the woman was exposed to a constant pregnancy risk by the husband, then fertility would increase. A testable implication of this theory is that fertility increases should be concentrated in women with low levels of empowerment. We test this hypothesis in Table B10. We estimate our baseline fertility regression, except we now include a triple interaction with a proxy indicator variable that takes a value of one if the woman ever reports having any say in any household decision reported in the DHS.[29] Interestingly, the coefficient on the interaction between ITNs and malaria prevalence is a statistical zero, while the triple interaction is generally positive and sometimes statistically significant – which implies the fertility effects are only present in women who reported having a say in decisions. This is the opposite of what our hypothesis proposed, and so we conclude there is no evidence that the divergence of fertility preferences and outcomes are caused by low levels of female input in decision making processes.

Our second hypothesis is unmet need for contraception.[30] Similar to our rationale behind the effect of women's empowerment of fertility, if a woman is exposed to a constant fertility risk in the presence of an increase in fecundity due

[29]For six different household decisions, the DHS records whether a woman reports having a joint say with a partner, joint say with another person, or complete say. The six decisions are: decisions on her own health care, making large household purchases, making household purchases for daily needs, visits to family or relatives, food to be cooked each day, or what to do with money her husband earns. We define our "Ever Say" variable to be one if a woman has at least partial say in any of the six decisions, and zero otherwise. This is a fairly low bar – however, only about 50 percent of the women in our sample report ever having any say.

[30]A woman is broadly defined as having an unmet need for contraception if she is sexually active, is not using contraception, yet doesn't want another child. We use definition two in the DHS for unmet need, which keeps women who never had sex, are infertile, or menopausal in the sample, but records them as not having an unmet need. The other alternative is to set those women equal to missing. Our results are robust to both definitions.

to the ITN distribution campaign, then we would expect the increase in fertility to be concentrated among women with an unmet need for contraception. We test this hypothesis in Table B11. The triple interaction between ITNs, malaria prevalence, and unmet need is uniformly negative and statistically significant for five of the six age groups, which implies that women who have unmet need actually had lower fertility after the ITN distribution campaign. Since there is no evidence of an increased fertility rate among women with an unmet need for contraception, we rule out this explanation.

Our last hypothesis is unexpected health effects on fecundity. The strongest evidence from the biological literature for this is through the channel of male fecundity: sperm quality decreases significantly during malaria episodes, presumably because the resulting fevers increase the temperature in the testes (Singer et al., 1987). When malaria is cured, male fecundity rebounds very rapidly. In addition, female fecundity may also be affected during malaria episodes. McReady et al. (2012) find that the probability of miscarriage for a woman with malaria is 50 percent, as opposed to a 20 percent risk for a healthy woman. If the ITN distribution campaigns allowed women or their partners to avoid malaria, then it may be the case that since they were less sick, they were more likely to become pregnant or retain their pregnancies. If this increase in fecundity was unexpected, then women would not change their behavior which could lead to higher pregnancy rates and a shorter birth spacing, consistent with our previous results. It is also important to note that this channel could interact with our other two hypotheses.

Unfortunately fecundity cannot be tested directly from our data. However, there are two indicators of health which are related to fecundity in the DHS: BMI and anemic status. We run similar regressions to those in Tables B10 and B11 except with underweight and anemic status as the dependent variables, to see if the fertility increases are concentrated among women who are underweight or anemic. We define underweight as a dichotomous variable which takes a value of one if a woman's BMI is under 18.5 (the standard threshold for being underweight), and anemic as a dichotomous variable which takes a value of one if a woman is reported to have either moderate or severe anemia in the DHS.

Our results for underweight women are found in Table B12. We find that the coefficient on the interaction between ITN usage and malaria prevalence is a statistical zero, implying that there was no increase in fertility among women

who were not underweight. In addition, the triple interaction is positive and significant for two of the six age groups, which implies that fertility rose faster for women in these age groups if they were underweight. This is suggestive that women who were unhealthy (proxied by being underweight) increased fertility faster than those who were healthy, implying there is a possibility that the ITN distribution campaign increased the fecundity of the least fertile women. In results not shown, we also tested whether there may have been an change in fertility due to a compositional effect: if fertility increased for the underweight, and the fraction of women who were underweight changed as a result of the ITN campaigns, then this could affect the aggregate fertility rate. We found no effect on the ITN distribution campaign on the number of women who reported being underweight.

Moving to our anemia results, in Table B13, the interaction between ITN usage and malaria prevalence is large and positive for four of the six age groups, while the triple interaction is negative for two of the six. This implies that increases in fertility were relatively higher among women who were not anemic. This is slightly the opposite of our previous results, where the women who were unhealthy (i.e. underweight) were more likely to have higher fertility after the ITN intervention. However, this result could also reinforce the idea that the ITN distribution could have increase fecundity unexpectedly if women moved from being anemic to not anemic as a result of the ITN distribution. In a previous paper, Apouey et al. (2017) performed a similar analysis using the same data as this paper on the effect of the ITN campaigns and anemic status, and found that anemia significantly fell as a result of the ITN distribution. Therefore, after the ITN distribution campaign, a larger fraction of the population consisted of the group of women who experienced an increase in fertility, which could have led to higher fertility in the overall population due to a composition effect.

VII Discussion

Of all the hypotheses tested in this paper which could explain the increased fertility after the ITN distribution campaigns, only three real explanations have not been ruled out. The first is the prediction of a standard Beckerian fertility model that the ITN distribution campaigns lowered the cost of childbearing enough to cause women to choose higher levels of fertility. Although possible, this answer

is somewhat problematic since we also found a large and uniform reduction in stated preferences for childbearing after the ITN campaigns.

The second explanation is an unexpected increase in fecundity due to the ITN campaigns. We find suggestive evidence that this is case, although a direct test of this hypothesis is impossible given our data. It is also important to note that Lucas (2013) also finds a positive effect of malaria eradication on fertility in Sri Lanka, and suggests that the fecundity effect is a primary mechanism through which this occurs. As evidence for her claim, she draws from the epidemiological literature on malaria which demonstrates the effects of malaria on stillborn births and miscarriage are higher for women experiencing their first pregnancy than higher order pregnancies. In her paper, she finds that malaria eradication increased survival among first-born children, suggesting malaria infections are an important channel by which reductions in malaria increases fertility. While this mechanism is a preferred explanation of our results, additional evidence is needed to confirm or refute this channel.

The final explanation is an increase in sexual activity – coupled with a constant rate of contraceptive use – after the ITN distribution campaigns. There are two possibilities as to why this may occur. First, there may have been an increase in fertility preferences after the campaigns, which led women to increase their sexual activity and not increase their contraceptive use in order to have more children. This possibility is ruled out by the fact that women reported wanting less children, not more. The other explanation is that the reduction in malaria after the campaigns led more individuals to engage in sexual activity simply because they felt less sick, and not because they were intending to have more children.

Taken together, these last two explanations suggest that the ITN distribution campaigns led to increased fertility which was not fully intended – and therefore both the Beckerian rational choice framework and Bongaarts' proximate determinate models could both be correct in their own way: women rationally chose to have less children, yet ended up having more children due to a factor outside their control – imperfect information about conception probabilities.

VIII Conclusion

Over the past decade, there has been a large international emphasis on malaria eradication in sub-Saharan Africa. According to the World Malaria Report 2012, just under $2 billion were spent on malaria eradication efforts in 2011 alone. Most of the effort to reduce malaria has come through the distribution of insecticide-treated bed nets, as they are considered the most cost-effective malaria control intervention. However, measuring the effectiveness of these nets is difficult. Child mortality has been falling in Sub-Saharan Africa before the rapid introduction of nets, mainly due to general improvements in the health environment. We observe mortality falling in sub-Saharan Africa as nets are distributed, but the extent to which we can attribute the decline in mortality to net usage remains unclear. Similarly, the impact of net usage on fertility is largely unknown.

Using a large data set of birth histories combined with information on ITN usage and malaria prevalence, we estimate the effect of the rapid increase in ITN usage in sub-Saharan Africa on child mortality and fertility. We find that bed nets have been effective in their goal of reducing child mortality, for children ages 1 to 3. For instance, the increase in ITN usage in an average region has led to a decrease of the probability of a child between 13 and 24 months dying by 22.2 percent, and a decrease in 25-36 month mortality by 40.6 percent. We also find that the introduction of ITNs has a positive impact on fertility for educated women. Specifically, we find than the introduction of ITNs increased the total fertility rate by 5.6 percent in the average region, or 0.3 children per woman.

Although our paper explores the reduced-form effect of bed nets on mortality and fertility, we cannot causally determine the effect of the reduction on child mortality on fertility directly. This relationship forms an integral part of many theories of fertility decline, especially within the demographic transition framework which has been very influential among demographers and economists alike. However, in contrast to the somewhat convincing evidence supporting a negative effect of fertility on infant mortality, conclusive evidence on the effect of child mortality on fertility has not been forthcoming, with different studies producing quite different results. We explore the mechanisms which drove the fertility increase after the mortality decline, by looking at the proximate determinants of fertility change. We find that women generally desired

less children, and some took measures to prevent childbirth – consistent with the theoretical Beckerian literature suggesting negative effects of mortality on fertility through reductions in precautionary childbearing and movements along the quantity-quality frontier. However, the fact that fertility increased in spite of falling fertility preferences suggests that fertility control was not perfect. Our results are consistent with the hypothesis that the distribution of ITNs increased fecundity, which led to higher levels of fertility. We also found that sexual activity increased after the distribution campaigns.

Our findings on the impact of ITNs on child mortality strengthen the arguments made by the WHO for an increase in funding for disbursements for malaria control. After rising from $100 million in 2000 to $1.71 billion in 2010, international donations for malaria control have stagnated over the past three years. There is a sense that donor fatigue may threaten the funding for the continued distribution of malaria control commodities. According to the World Malaria Report 2012, an estimated US$ 5.1 billion is needed every year to achieve universal coverage of malaria interventions including ITNs. However, only $2.3 billion is available, less than half of what is needed.

In contrast, our findings do not support the contention that erosion of international funding for malaria control, specifically of ITNs, would lead to higher fertility rates in the short run. In fact, we show the exact opposite. Inasmuch as higher fertility rates are associated with lower educational achievement, higher maternal mortality, and lower income per capita, it is essential that programs which aim to reduce child mortality be coupled with complementary programs to increase funding for health, education, and family planning services in order to blunt the possible deleterious effects of increased population growth on standards of living in the short run.

References

[1] Apouey, Bénédicte, Gabriel Picone, and Joshua Wilde. 2018. "The Economics of Malaria Prevention." Oxford Research Encyclopedia of Economics and Finance. New York: Oxford University Press.

[2] Apouey, Bénédicte H., Gabriel Picone, Joshua Wilde, Joseph Coleman, and Robyn Kibler. 2017. "Malaria and Anemia Among Children in Sub-Saharan Africa: The Effect of Mosquito Net Distribution." *Revue Economique*, 68(2), 163-197.

[3] Ashraf, Quamrul, Ashley Lester, and David Weil. 2009. "When Does Improving Health Raise GDP?" *NBER Macroeconomics Annual 2008*, University of Chicago Press.

[4] Ashraf, Quamrul, David Weil, and Joshua Wilde. 2013. "The Effect of Fertility Reduction on Economic Growth." *Population and Development Review*, 39(1): 97-130.

[5] Ayoola, Omolola, Andrew Whatmore, William Balogun, Olatokunbo Jarrett, John Cruickshank, and Peter Clayton. 2012. "Maternal malaria status and metabolic profiles in pregnancy and in cord blood: relationships with birth size in Nigerian infants." *Malaria Journal*, 75(11).

[6] Barofsky, Jeremy, Claire Chase, and Tobenna Anekwe. 2010. "Malaria Eradication and Economic Outcomes in Sub-Saharan Africa: Evidence from Uganda." *Journal of Health Economics*, 44:118-136.

[7] Barreca, Alan. 2010. "The Long-Term Economic Impact of In Utero and Postnatal Exposure to Malaria." *Journal of Human Resources*, 45(4): 865-892.

[8] Barro, Robert, and Gary Becker. 1989. "Fertility Choice in a Model of Economic Growth." *Econometrica* 57(2):481–501.

[9] Becker, Gary. 1960. "An Economic Analysis of Fertility." *Demographic and Economic Change in Developed Countries*. Princeton: Princeton University Press.

[10] Bhatt, S., D. J. Weiss, E. Cameron, D. Bisanzio, B. Mappin, U. Dalrymple, K. E. Battle, et al. 2015. "The effect of malaria control on Plasmodium falciparum in Africa between 2000 and 2015." *Nature*, 526, 207–211.

[11] Bleakley, Hoyt. 2010. "Malaria Eradication in the Americas: a Retrospective Analysis of Childhood Exposure." *American Economic Journal: Applied Economics* 2(2):1-45.

[12] Boklage, Charles. 1990. "Survival Probability of Human Conceptions from Fertilization to Term." *International Journal of Fertility*, 35(2):75-94.

[13] Bongaarts, John. 1978. "A Framework for Analyzing the Proximate Determinants of Fertility." *Population and Development Review* 4(1):105-132.

[14] Canning, David, Mahesh Karra, and Joshua Wilde. 2017. "The Effect of Fertility Decline on Economic Growth in Africa: A Macrosimulation Model." *Population and Development Review*, 43(S1): 237-263.

[15] Cogneau, Denis and Pauline Rossi. 2016. "Malaria Control and Infant Mortality in Africa." PSE Working Paper 2017-02.

[16] Crawley, Jane, and Bernard Nahlen. 2004. "Prevention and Treatment of Malaria in Young African Children." *Seminars in Pediatric Infectious Diseases* 15(3):169-80.

[17] 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*, 2(2):72-94.

[18] Doepke, Matthias. 2005. "Child Mortality and Fertility Decline: Does the Barro-Becker Model Fit the Facts?" *Journal of Population Economics*, 18(2):337-366.

[19] Doepke, Matthias. 2015. "Gary Becker on the Quantity and Quality of Children." *Journal of Demographic Economics*. 81(1):59-66.

[20] Doolan, Denise, Carlota Dobano, and J. Kevin Baird. 2009. "Acquired Immunity to Malaria." *Clinical Microbiology Reviews* 22(1):13-36.

[21] Gates Foundation. 2014. *Annual Letter 2014.* Available at: http://www.gatesfoundation.org/Who-We-Are/Resources-and-Media/Annual-Letters-List/Annual-Letter-2014. Accessed August 15, 2016.

[22] Gething, Peter, Anand Patil, David Smith, Carlos Guerra, Iqbal Elyazar, Geoffrey Johnston, Andrew Tatem, et al. 2011. "A new world malaria map: Plasmodium falciparum endemicity in 2010." *Malaria Journal* 10:378.

[23] Hamer, Davidson, Mrigendra Singh, Blair Wylie, Kojo Yeboah-Antwi, Jordan Tuchman, Meghna Desai, Venkatachalam Udhayakumar, et al. 2009. "Burden of malaria in pregnancy in Jharkhand State, India." *Malaria Journal*, 210(8).

[24] Kalemli-Ozcan, Sebnem. 2003. "A Stochastic Model of Mortality, Fertility, and Human Capital Investment." *Journal of Development Economics*, 70(1):103-118.

[25] Kuecken, Maria, Josselin Thuilliez, and Marie-Anne Valfort. 2016. "Disease and Human Capital Accumulation: Evidence from the Roll Back Malaria Partnership in Africa." Mimeo.

[26] Lengeler, Christian. 2004. "Insecticide-treated bed nets and curtains for preventing malaria." *Cochrane Database of Systematic Reviews* 2.

[27] Lucas, Adrienne. 2010. "Malaria Eradication and Educational Attainment: Evidence from Paraguay and Sri Lanka." *American Economic Journal: Applied Economics*, 2:46-71.

[28] Lucas, Adrienne. 2013. "The Impact of Malaria Eradication on Fertility." *Economic Development and Cultural Change*, 61(3):607-631.

[29] McGready, R., Lee, S.J., Wiladphaingern, J., Ashley, E.A., Rijken, M.J., Boel, M., Simpson, J.A., Paw, M.K., Pimanpanarak, M., Oh Mu, Singhasivanon, P., White, N.J., Nosten, F.H. 2011. "Adverse effects of falciparum and vivax malaria and the safety of antimalarial treatment in early pregnancy: a population-based study." *The Lancet Infectious Diseases*, 12(5): 388-396.

[30] Pathania, Vikram. 2014. "The Impact of Malaria Control on Infant Mortality in Kenya." *Economic Development and Cultural Change*, 62(3):459-487.

[31] Poespoprodjo, Jeanne, Wendy Fobia, Enny Kenangalem, Daniel Lampah, Noah Warikar, Andrew Seal, Rose McGready, et al. 2008. "Adverse Pregnancy Outcomes in an Area Where Multidrug-Resistant Plasmodium vivax and Plasmodium falciparum Infections are Endemic." *Clinical Infectious Diseases* 46(9):1374–81.

[32] Roll Back Malaria. 2012. *The Global Malaria Action Plan.* Available at: www.rollbackmalaria.org/gmap. Accessed September 10, 2013.

[33] Rosling, Hans. 2010, June. *Hans Rosling: Global population growth, box by box.* [Video file]. Retrieved from https://www.ted.com/talks/hans_rosling_on_global_population_growth/transcript/a?language=en

[34] Sah, Raaj. 1991. "The Effects of Child Mortality Changes on Fertility Choice and Parental Welfare." *Journal of Political Economy* 99, 582–606.

[35] Singer, R., Segenreich, E., Sagiv, M., Shohat, B., Livni, E., Bartoov, B., Zukerman, Z., Leiba, S. and Servadio, C. 1987. "Decreased semen quality in a male infected with malaria." *International Journal of Andrology*, 10: 685–689.

[36] Takem, Ebako Ndip and Umberto D'Alessandro. 2013. "Malaria in Pregnancy". *Mediterranean Journal of Hematology and Infectious Diseases*, 5(1).

[37] Venkataramani, Atheendar. 2012. "Early Life Exposure to Malaria and Cognition in Adulthood: Evidence from Mexico." *Journal of Health Economics* 31(5):767-780.

[38] Wilcox, Allen J., Donna Day Baird, and Clarice R. Weinberg. 1999. "Time of Implantation of the Conceptus and Loss of Pregnancy." *New England Journal of Medicine*, 340:1796-1799.

[39] Wilde, Joshua, Bénédicte H. Apouey, and Toni Jung. 2017. "The Effect of Ambient Temperature Shocks During Conception and Early Pregnancy on Later Life Outcomes." *European Economic Review*, 97C: 87-107.

[40] Wilson, Nicholas. 2015. "Child Mortality Risk and Fertility: Evidence from Prevention of Mother-to-Child Transmission of HIV" *Journal of Development Economics*, 116(September):74-88.

[41] World Health Organization. 2012. *World Malaria Report 2012*. Geneva: WHO.

Table 1: List of Surveys

Country	Years	Regions	Surveys
Angola	2000-2011	18	MIS06-07, MIS11
Benin	2000-2012	12	DHS06, DHS11-12
Burkina Faso	2000-2014	13	DHS03, DHS10, MIS14
Burundi	2000-2012	17	DHS10, MIS12
Cameroon	2000-2011	10	DHS04, DHS11
Chad	2000-2004	8	DHS04
Comoros	2000-2012	3	DHS12
Congo (Brazzaville)	2000-2012	11	DHS05, AIS09, DHS11-12
Congo (DRC)	2000-2014	9	DHS07, DHS13-14
Côte d'Ivoire	2000-2012	11	AIS05, DHS11-12
Ethiopia	2000-2003	11	DHS05, DHS11
Gabon	2000-2012	9	DHS12
Gambia	2000-2013	6	DHS13
Ghana	2000-2014	10	DHS03, DHS08, MICS11, DHS14
Guinea	2000-2012	8	DHS05, DHS12
Kenya	2000-2009	8	DHS03, DHS08-09, DHS14, MIS15
Lesotho	2000-2009	10	DHS04-05, DHS09-10
Liberia	2000-2013	15	DHS06-07, MIS08-09, MIS11, DHS13
Madagascar	2000-2013	6	DHS03-04, DHS08-09, MIS11, MIS13
Malawi	2000-2014	27	DHS04, DHS10, MIS12, MIS14
Mali	2000-2012	9	DHS01, DHS06, DHS12-13
Mozambique	2000-2011	11	DHS03-04, AIS09, DHS11
Namibia	2000-2013	13	DHS00, DHS06-07, DHS13
Niger	2000-2012	8	DHS06, DHS12
Nigeria	2000-2013	37	DHS03, DHS08, MIS10, DHS13
Rwanda	2000-2013	5	DHS05, DHS(I)07-08, DHS10, MIS13
Sao Tome	2000-2008	4	DHS08-09
Senegal	2000-2014	11	DHS05, MIS06, MIS08-09, DHS10-11, DHS12-13, DHS14
Sierra Leone	2000-2013	4	DHS08, DHS13
Swaziland	2000-2010	4	DHS06-07
Tanzania	2000-2012	9	DHS04-05, AIS07-08, DHS09-10, AIS11-12
Togo	2000-2013	5	DHS13
Uganda	2000-2014	4	DHS06, MIS09-10, DHS11, MIS14-15
Zambia	2000-2014	9	DHS01-02, DHS07, DHS13-14
Zimbabwe	2000-2014	10	DHS05-06, DHS10-11

Notes. AIS stands for AIDS Indicator Survey, DHS for Demographic and Health Survey, DHS(I) for Interim DHS, and MIS for Malaria Indicator Survey.

Table 2: Descriptive Statistics

Variable	Mean	S.d.	Min	Max
Mortality equation				
Malaria prevalence in 2000	0.4117	0.1986	0.0059	0.8821
ITN at t	0.1652	0.1941	0	0.9692
IRS at t	0.0433	0.1006	0	1
ACT at t	0.0501	0.0851	0	0.5089
Risk exposure	0.9510	0.1751	0	1
Male	0.5069	0.5001	0	9
Urban	0.2665	0.4421	0	1
Birth order	3.4980	2.3530	1	20
Mother's age at birth	26.3231	6.6325	14	50
Mother's primary edu	0.5371	0.4986	0	1
Mother's primary edu+	0.1803	0.3845	0	1
Fertility equation				
Malaria prevalence in 2000	0.3977	0.2048	0.0059	0.8821
ITN at t-1	0.1447	0.1846	0	0.9692
IRS at t-1	0.0389	0.0930	0	0.8059
ACT at t	0.0491	0.0841	0	0.5089
Urban	0.3642	0.4812	0	1
Age	26.6729	7.9031	15	44
Primary edu	0.6217	0.4849	0	1
Secondary edu+	0.2910	0.4542	0	1

Table 3: Malaria Control Effects on Mortality and Fertility

	(1)	(2)	(3)	(4)	(5)	(6)
Panel A. Child mortality						
Months since birth	0-12	13-24	25-36	37-48	49-60	
Mean mortality rate	0.0713	0.0224	0.0076	0.0041	0.0029	
	(0.2574)	(0.1480)	(0.0869)	(0.0641)	(0.0534)	
	Dead	Dead	Dead	Dead	Dead	
ITN	-0.0380***	-0.0132***	0.0026*	0.0010	-0.0013	
	(0.0045)	(0.0022)	(0.0015)	(0.0012)	(0.0011)	
Malaria prev. in 2000 * ITN	-0.0108	-0.0242**	-0.0150***	-0.0051	-0.0029	
	(0.0170)	(0.0109)	(0.0048)	(0.0035)	(0.0036)	
Observations	1,147,542	1,021,288	868,009	736,594	611,217	
R-squared	0.014	0.013	0.004	0.002	0.002	
Panel B. Fertility						
Woman's age	15-19	20-24	25-29	30-34	35-39	40-44
Mean fertility rate	0.1171	0.2354	0.2414	0.2251	0.1874	0.1208
	(0.3215)	(0.4243)	(0.4279)	(0.4177)	(0.3903)	(0.3259)
	Birth	Birth	Birth	Birth	Birth	Birth
ITN	0.0101	0.0072	-0.0008	0.0057	0.0127	0.0018
	(0.0100)	(0.0112)	(0.0146)	(0.0134)	(0.0116)	(0.0101)
Malaria prev. in 2000 * ITN	0.0087	0.0384	0.0440	0.0902**	0.0340	0.0886**
	(0.0278)	(0.0301)	(0.0367)	(0.0350)	(0.0349)	(0.0366)
Observations	1,354,134	1,290,249	1,117,627	849,769	612,839	332,852
R-squared	0.3782	0.1523	0.0551	0.0414	0.0372	0.0406

Notes: Standard errors clustered at the region level in parentheses. *** p<0.01, ** p<0.05, * p<0.1. For Panel A, the dependent variable is an indicator for whether a child was (if alive) or would have been (if dead) alive and in a given age range (in months) at the time of the survey. For Panel B, the dependent variable is an indicator for whether the woman had a live birth within the last 12 months for a given woman-year, for each age group. All regressions include birth year fixed effects, birth order fixed effects, region fixed effects, country time trends, and a set of individual controls as outlined in Section III.

Table 4: Mortality Falsification Tests: Leads

	(1)	(2)	(3)	(4)	(5)
	Months since birth				
	0-12	13-24	25-36	37-48	49-60
Panel A. Explanatory variables at t+2					
	Dead	Dead	Dead	Dead	Dead
ITN at t+2	-0.0171***	-0.0058**	-0.0029**	0.0006	-0.0004
	(0.0041)	(0.0026)	(0.0012)	(0.0011)	(0.0010)
Malaria prev. in 2000 * ITN at t+2	-0.0137	-0.0022	-0.0006	-0.0024	0.0024
	(0.0176)	(0.0107)	(0.0054)	(0.0036)	(0.0034)
Observations	1,120,236	1,000,382	847,837	715,788	592,170
R-squared	0.014	0.012	0.004	0.002	0.002
Panel B. Explanatory variables at t+4					
	Dead	Dead	Dead	Dead	Dead
ITN at t+4	0.0027	-0.0005	-0.0024*	0.0004	0.0011
	(0.0037)	(0.0024)	(0.0013)	(0.0011)	(0.0011)
Malaria prev. in 2000 * ITN at t+4	-0.0056	-0.0018	-0.0081	0.0032	0.0027
	(0.0175)	(0.0103)	(0.0056)	(0.0040)	(0.0036)
Observations	1,029,629	924,077	774,024	643,697	522,846
R-squared	0.014	0.012	0.004	0.002	0.002

Notes: Standard errors clustered at the region level in parentheses. *** p<0.01, ** p<0.05, * p<0.1. The dependent variable is an indicator for whether a child was (if alive) or would have been (if dead) alive and in a given age range (in months) at the time of the survey. All regressions include birth year fixed effects, birth order fixed effects, region fixed effects, country time trends, and a set of individual controls as outlined in Section III.

Table 5: Fertility Falsification Tests: Leads

	(1)	(2)	(3)	(4)	(5)	(6)
Woman's Age	15-19	20-24	25-29	30-34	35-39	40-44
Panel A. Explanatory variables at t+2						
	Birth	Birth	Birth	Birth	Birth	Birth
ITN at t+2	-0.0016	0.0003	0.0012	0.0075	0.0062	0.0033
	(0.0073)	(0.0082)	(0.0108)	(0.0093)	(0.0094)	(0.0093)
Malaria prev. in 2000 * ITN at t+2	-0.0232	-0.0031	0.0126	0.0240	-0.0056	0.0249
	(0.0237)	(0.0282)	(0.0348)	(0.0304)	(0.0313)	(0.0334)
Observations	1,336,890	1,273,313	1,101,171	837,287	603,031	325,988
R-squared	0.3785	0.1520	0.0544	0.0406	0.0361	0.0397
Panel B. Explanatory variables at t+4						
	Birth	Birth	Birth	Birth	Birth	Birth
ITN at t+4	0.0062	0.0223***	0.0392***	0.0347***	0.0297***	0.0404***
	(0.0054)	(0.0071)	(0.0091)	(0.0093)	(0.0087)	(0.0092)
Malaria prev. in 2000 * ITN at t+4	-0.0048	-0.0231	-0.0321	-0.0474	-0.0587**	-0.0974***
	(0.0227)	(0.0273)	(0.0323)	(0.0343)	(0.0272)	(0.0334)
Observations	1,229,394	1,177,928	1,008,801	766,538	544,625	286,668
R-squared	0.3788	0.1510	0.0536	0.0392	0.0345	0.0372

Notes: Standard errors clustered at the region level in parentheses. *** p<0.01, ** p<0.05, * p<0.1. The dependent variable is an indicator for whether the woman had a live birth within the last 12 months for a given woman-year, for each age group. All regressions include birth year fixed effects, region fixed effects, country time trends, and a set of individual controls as outlined in Section III.

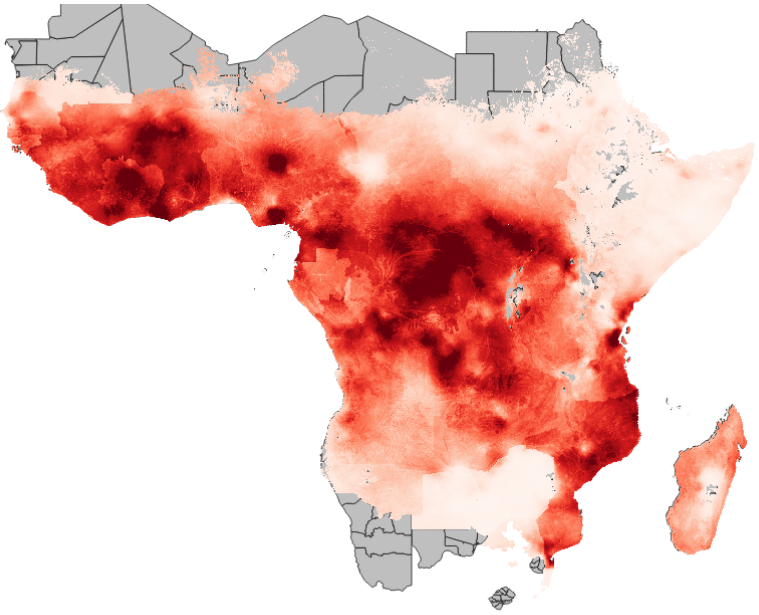
Table 6: Falsification Tests: Other Outcomes

	(1)	(2)	(3)	(4)
	BCG	DPT	Either vaccine	Worker's visit last 12 months
ITN	0.0193	-0.0540	-0.0102	0.0157
	(0.0248)	(0.0358)	(0.0230)	(0.0104)
Malaria prev. in 2000 * ITN	-0.0614	-0.1036	0.0040	-0.0097
	(0.0855)	(0.1167)	(0.0893)	(0.0522)
Observations	524,780	522,930	524,862	978,265
R-squared	0.2750	0.2896	0.2706	0.0567

Notes: Standard errors clustered at the region level in parentheses. *** p<0.01, ** p<0.05, * p<0.1. All regressions include year fixed effects, region fixed effects, country time trends, and the same set of individual controls as out main regression, as outlined in Section III.

Figure 1: Malaria Prevalence Rates (PFPR) in 2000 and 2014

2000



2014

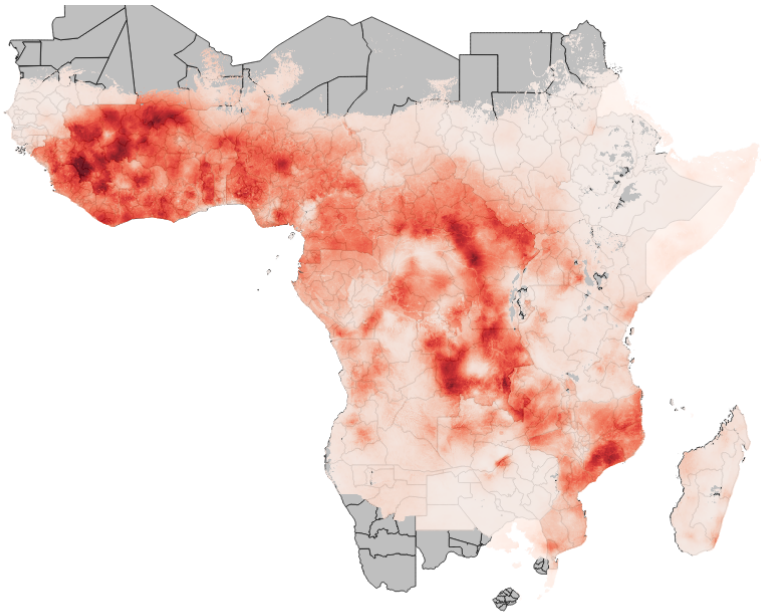


Figure 2: ITN Usage in 2014

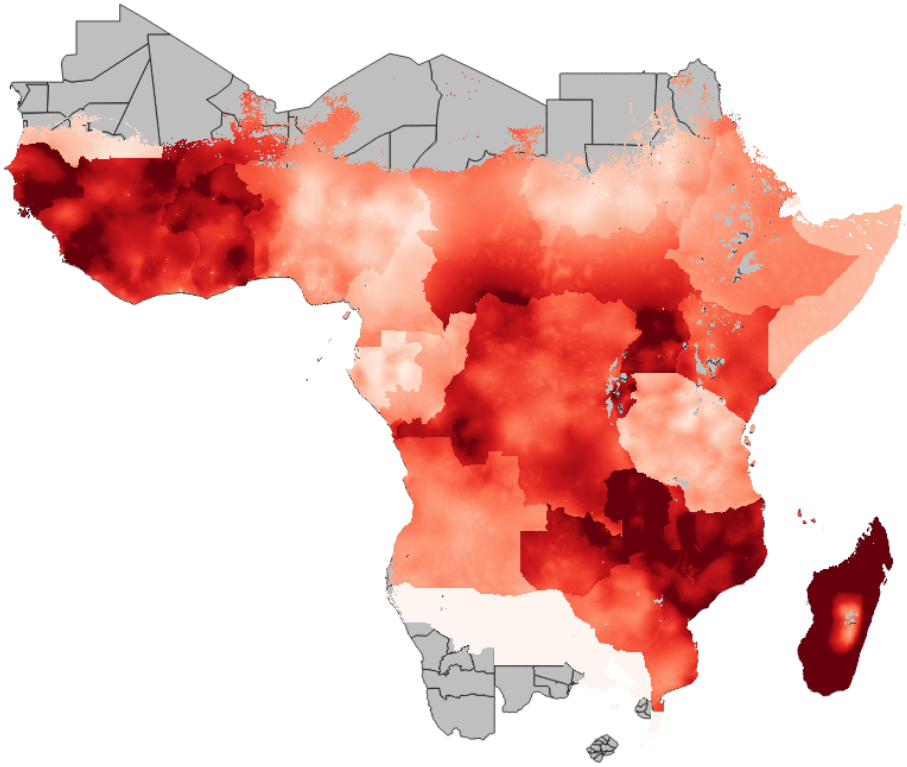
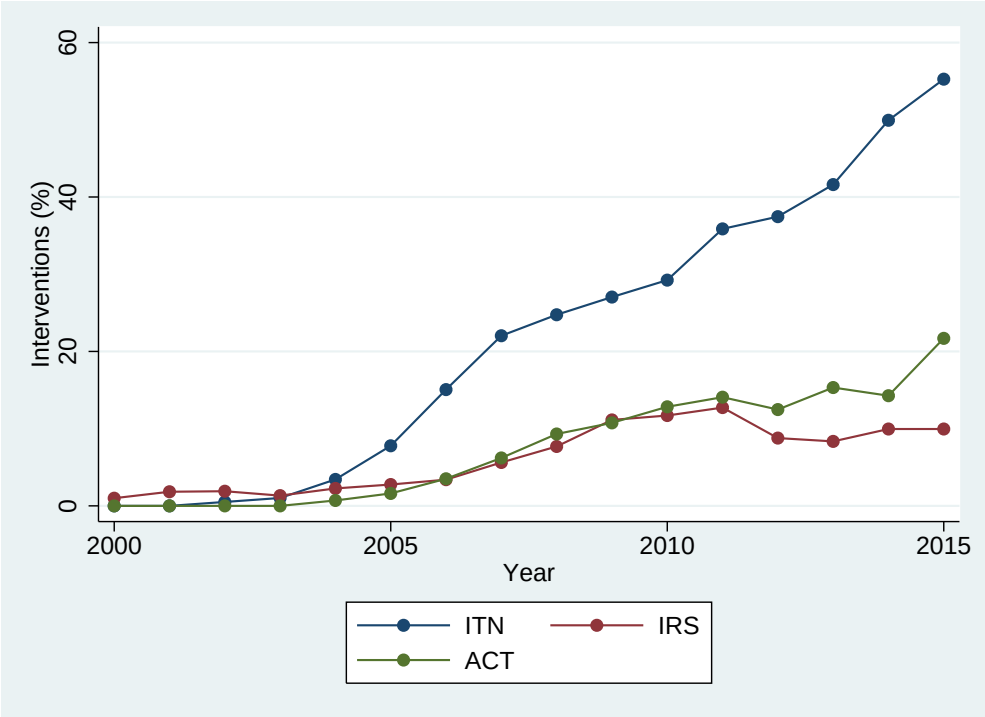
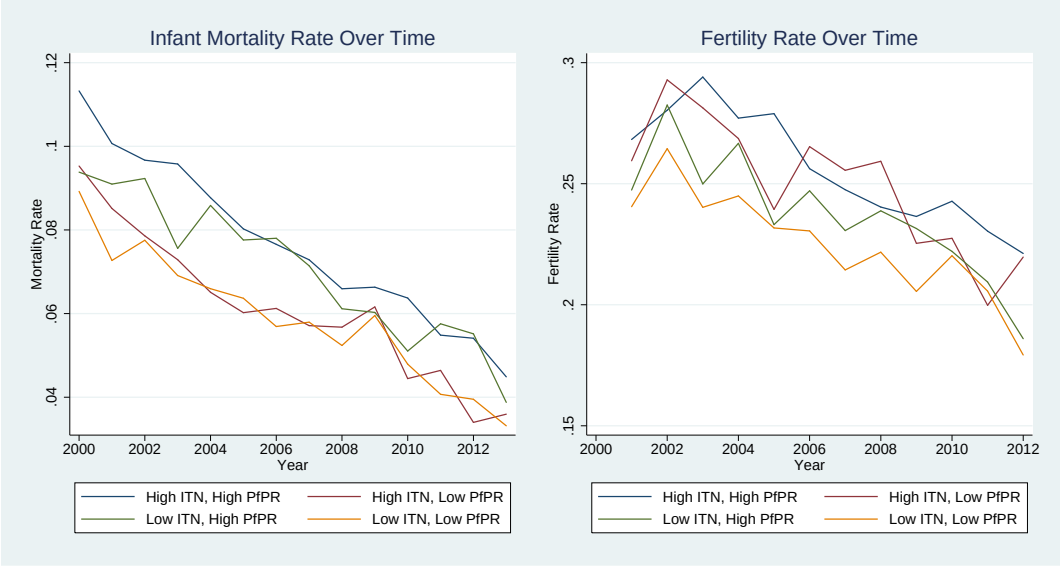


Figure 3: Evolution of ITN Usage, IRS, and ACT Uptake Since 2000



Notes: Using MAP data, we compute the average level in each region (for each of our variables of interest). Using these regional-level data, we then calculate the average level per year. Data are not weighted by population size.

Figure 4: Mortality and Fertility Rates by Treatment Intensity



Notes: Using MAP data, we compute the average level in each region (for each of our variables of interest). Using these regional-level data, we then calculate the average level per year. Data are not weighted by population size.

Appendix A. Conceptual Framework

That an increase in bed net usage should reduce child mortality is rather straightforward. In contrast, the channels through which net usage could affect fertility are less intuitive. There are several channels identified in the literature through which child mortality may influence fertility – all of which show that reductions in mortality should reduce fertility.

Most straightforward is the replacement child effect. If women target a certain number of surviving offspring, then each child who dies before reaching adulthood will be replaced, leading to a higher number of births. Related to this effect is the precautionary childbearing effect, which states than the reduction in mortality should be met with a larger than one-for-one fall in fertility as predicted by the replacement children effect. This is due to risk aversion – if a woman wants three surviving children and the mortality rate is 50 percent, she will need to have six children to get three in expectation, so will likely have more than six children to ensure she has at least 3 children survive. As child mortality approaches zero, the uncertainty concerning the number of surviving children falls, and therefore precautionary children are unnecessary.

Third, parents may not only derive utility from the number of surviving children they have, but also from their "quality". If the fraction of children which will die before reaching adulthood is high, then incentives to invest in those children is low, and therefore the parents are more likely to increase quantity of their children as opposed to their quality. As mortality is reduced, investment in children is relatively more attractive, as is the incentive to have a large quantity of children. This is generally referred to as moving along the quality-quantity frontier. There is one theoretical channel proposed in the literature by which there could be reverse causality between mortality and fertility. If higher fertility is associated with shorter birth intervals, and shorter birth intervals are associated with higher infant mortality, then reducing fertility may lead to healthier children who are less likely to die.

So far, we see that there is no theoretical reason to believe that decreases in mortality should increase fertility directly. However, in the context of the introduction of malaria control policies do not only affect child mortality, but also affect the cost of childbearing. This assumption can be justified in several ways. First, more incidents of malaria will directly increase the amount of time

parents need to care for children while they are sick (e.g. through increased visits to a clinic, caring for sick children at home, etc.) Second, since time and income are substitutes, if parents spend a portion of their income on remedies for malaria, this can be modeled as an increase in the time cost of raising children. Third, since malaria increases the probability of a miscarriage, higher malaria incidence increases the number of pregnancies needed to produce a live birth. Inasmuch as pregnancy is time intensive, this should lead to a higher time cost per child. Finally, there may be direct utility costs of higher malaria on bearing or raising children. For example, since maternal mortality is higher if there is more malaria, a woman may choose not to have an additional child if she values her own life.

Theoretical Model on Bed Nets and Fertility

Our model is a static model which incorporates all of the channels described above. It is mostly borrowed from Kalemi-Ozcan (2003) with a few slight modifications – specifically pertaining to the cost of childbearing. In this model, the prevalence of malaria affects a woman's fertility choice through three channels. The first three run directly through reductions in infant mortality – a higher probability of child survival to adulthood will reduce the need for both precautionary child-bearing and replacement children, as well as move the mother along the quantity-quality frontier. The fourth channel is that malaria increases the cost of having children, causing a reduction in malaria to have a positive effect on fertility since children are now less costly.[31] Consider a woman who derives utility from consumption and children in the following manner:

$$U = \gamma \ln (C) + (1 - \gamma) \ln (wN) \tag{3}$$

where C is consumption, N is the number of surviving adult children, and w is the prevailing wage rate. She optimizes over the number of children n she wishes to have, subject to a unit time constraint which is divided between raising children and working. The time cost of raising one child is $v(m)$, where m is the prevalence rate of malaria. We assume that $v'(m) > 0$, meaning that more malaria increases the time cost of raising children. As a result, the woman's

[31] As noted before, some of the additional costs to the mother may affect utility directly rather than increase the time cost of childbearing. While these utility costs are not time per se, modeling them as a time cost is functionally equivalent to introducing a direct disutility measure into the utility function since in our model time is traded for utility.

budget constraint is

$$C = w\left[1 - v(m)n\right] \tag{4}$$

Let $q(m)$ be the probability of survival of each child, where $q'(m) < 0$. The number of survivors N will be a random variable with a binomial distribution, meaning that the probability that N out of n children will live to adulthood is

$$f\left(N;\ n,\ q\right) = \left(\begin{array}{c} n \\ N \end{array} \right) q(m)^N \left[1 - q(m)\right]^{n-N} \tag{5}$$

for each integer N between 0 and n. Combining (3) and (4) and introducing this uncertainty into the model, the woman maximizes her expected utility

$$E\left(U\right) = \left\{\gamma \ln\left(w\left[1 - v(m)n\right]\right) + \left(1 - \gamma\right)\ln\left(wN\right)\right\} f\left(N;\ n,\ q(m)\right) \tag{6}$$

Since the mean of the binomial is nq,

$$U(N) = U\left[nq\left(m\right)\right] + \left[N - nq\left(m\right)\right] U_N\left[nq\left(m\right)\right] + \frac{\left[N - nq\left(m\right)\right]^2}{2!} U_{NN}\left[nq\left(m\right)\right] + \frac{\left[N - nq\left(m\right)\right]^3}{3!} U_{NNN}\left[nq\left(m\right)\right] \tag{7}$$

From log utility, the partial derivatives are:

$$U_N = \frac{\left(1 - \gamma\right)}{N},\ U_{NN} = -\frac{\left(1 - \gamma\right)}{N^2},\ U_{NNN} = \frac{2\left(1 - \gamma\right)}{N^3}$$

Substituting back into the above $U(N)$ equation and taking expectations we have:

$$E\left(U\right) = U\left[nq\left(m\right)\right] + E\left\{\left[N - nq\left(m\right)\right]\frac{\left(1 - \gamma\right)}{nq(m)}\right\} - E\left\{\frac{\left[N - nq\left(m\right)\right]^2}{2!}\frac{\left(1 - \gamma\right)}{\left[nq(m)\right]^2}\right\} + E\left\{\frac{\left[N - nq\left(m\right)\right]^3}{3!}\frac{2\left(1 - \gamma\right)}{\left[nq(m)\right]^3}\right\} \tag{8}$$

The second and fourth terms are zero since the first and third central mo-

ments of the binomial distribution are zero. The third term contains the second central moment of the binomial, which is $E\left[N - nq(m)\right]^2 = nq(m)\left[1 - q(m)\right]$. Therefore, (10) can be rewritten as

$$E\left(U\right) = U\left[nq\left(m\right)\right] - nq\left(m\right)\left(1 - q\right)\frac{\left(1-\gamma\right)}{2\left[nq(m)\right]^2},$$

which can also be rewritten as

$$E\left(U\right) = \gamma \ln\left(w\left(1 - v(m)n\right)\right) + \left(1-\gamma\right)\ln\left[wnq(m)\right] - \frac{\left(1-\gamma\right)\left[1-q(m)\right]}{2nq(m)}.$$

Therefore, we simplify this utility function by using a third-order Taylor expansion around the mean of N to get:

$$E\left(U\right) = \gamma \ln\left(w\left(1 - v(m)n\right)\right) + \left(1-\gamma\right)\ln\left[wnq(m)\right] - \frac{\left(1-\gamma\right)\left[1-q(m)\right]}{2nq(m)} \tag{9}$$

Taking the first order condition of (9) with respect to n and multiplying by n^2 for simplicity yields

$$G\left[n, m\right] = \frac{-\gamma v\left(m\right)n^2}{1 - v(m)n} + \left(1-\gamma\right)n + \frac{\left(1-\gamma\right)\left[1-q(m)\right]}{2q(m)} = 0 \tag{10}$$

This defines an implicit function from which we can calculate the effect of an increase in malaria prevalence m on fertility n, where

$$\frac{dn}{dm} = -\frac{G_m}{G_n}$$

In order to understand the mechanisms driving the results of our model, we now consider two cases: one where $\frac{dv}{dm} = 0$, and another where $\frac{dv}{dm} > 0$. First, consider the case where $\frac{dv}{dm} = 0$. In this case:

$$G_m = \frac{\gamma - 1}{2q(m)^2} \cdot \frac{dq}{dm} > 0 \text{ since } \gamma \in (0,1) \text{ and } \frac{dq}{dm} < 0$$

$$G_n = \frac{-\gamma vn\left(2\left(1 - vn\right) + vn\right)}{\left(1 - vn\right)^2} < 0 \text{ since } 1 - vn > 0$$

Since G_m is positive and G_n is negative, it follows that $\frac{dn}{dm} > 0$, implying that a reduction in malaria due to the introduction of bed nets should lead to a reduction in fertility. As mentioned previously, this is working through two channels. First, a decrease in malaria increases child survival to adulthood, meaning it will take less children born to reach a woman's target number of surviving children. This is the case even if there is no uncertainty in the model over how many of her children will die. However, the second channel – a reduction in precautionary child-bearing – is a direct result of the uncertainty in the model. A risk averse woman who faces a greater probability of losing children will opt to have more children than she otherwise would, simply to insure against the catastrophic case where most or all of her children die before reaching adulthood. If the probability of death falls due to a reduction in malaria, this case becomes less likely, meaning she will have less "safety" children.

Now consider the case where $\frac{dv}{dm} > 0$. In this case, G_n remains unchanged. However, an additional term is added to G_m to become

$$G_m = \frac{\gamma - 1}{2q(m)^2} \cdot \frac{dq}{dm} - \frac{\gamma n^2}{\left[1 - v\left(q\right)\right]^2} \cdot \frac{dv}{dm}$$

Since $\frac{dv}{dm}$ is positive, the second term will be positive. Therefore the sign of G_m now becomes ambiguous. As a result, the sign of $\frac{dn}{dm}$ becomes ambiguous as well. The intuition here is that if eliminating malaria causes children to be less costly to raise, women will choose to have more of them. This channel runs in the opposite direction as the two channels which run directly through decreases in mortality.

Bed Nets and Fertility in Sub-Saharan Africa

Which of these channels will dominate is an empirical question that only few studies have examined. The most notable among them is Lucas (2013) who focuses on the eradication of malaria in Sri Lanka and Paraguay in the 1950s, and finds that the elimination of malaria led to an increase in fertility. She hypothesizes that malaria posed a biological constraint on women's ability to conceive and carry children to full term, and that absent this constraint, more children were born. However, Sri Lanka in the 1950s is very different from Africa in the 2000s. For example, it is unlikely that women in Sri Lanka in the 1950s had much access to contraception. Therefore they had a limited ability to actually choose the number of children they had and fertility was mainly a byproduct of

sexual activity. The total fertility rate in Sri Lanka before 1950 was consistently high at approximately six children per women, and did not begin to decline until the mid 1960s (UN, 2013). As a result, Lucas' interpretation of malaria being a biological constraint on fertility is appropriate, and corresponds to the case in our model where the only channel which is operative is the (biological) cost of children, which implies that when malaria incidence is reduced, fertility increases.

In contrast, although contraception was far from universal in Africa in the 2000s, fertility rates had already begun to fall. Fertility in sub-Saharan Africa was constant at approximately 6.7 children per woman from 1950 to 1985, after which it fell by approximately 0.1 child per woman every year on average (UN, 2013). Therefore, the technology for fertility reduction seems to have been in place in the 2000s. This implies that – unlike Sri Lanka in the 1950s – the assumption that women have the ability to choose their own fertility in sub-Saharan Africa by the 2000 seems appropriate. For this reason, it is likely that the inclusion of the first two channels in our model – a reduction in precautionary child-bearing and replacement fertility – could cause the relationship between malaria and fertility in Africa in the 2000s to be substantially different from that in Sri Lanka in the 1950s.

Appendix B. Additional Tables and Figures

Table B1: Heterogeneous Effects of Malaria Control on Mortality by Gender

	(1)	(2)	(3)	(4)	(5)
	Months since birth				
	0-12	13-24	25-36	37-48	49-60
Panel A. Females					
	Dead	Dead	Dead	Dead	Dead
ITN	-0.0391***	-0.0122***	0.00176	0.00122	-0.000695
	(0.00540)	(0.00294)	(0.00178)	(0.00154)	(0.00169)
Malaria prev. in 2000 * ITN	-0.00687	-0.0251*	-0.00853	-0.00696	-0.00527
	(0.0208)	(0.0131)	(0.00700)	(0.00491)	(0.00602)
Observations	566,547	507,033	431,173	366,201	303,492
R-squared	0.014	0.013	0.005	0.003	0.003
Panel B. Males					
	Dead	Dead	Dead	Dead	Dead
ITN	-0.0367***	-0.0142***	0.00330	0.000809	-0.00183
	(0.00599)	(0.00274)	(0.00217)	(0.00155)	(0.00137)
Malaria prev. in 2000 * ITN	-0.0149	-0.0239**	-0.0212***	-0.00313	-0.000770
	(0.0201)	(0.0120)	(0.00592)	(0.00468)	(0.00468)
Observations	580,995	514,255	436,836	370,393	307,725
R-squared	0.014	0.013	0.005	0.003	0.002

Notes: Standard errors clustered at the region level in parentheses. *** p<0.01, ** p<0.05, * p<0.1. The dependent variable is an indicator for whether a child was (if alive) or would have been (if dead) alive and in a given age range (in months) at the time of the survey. All regressions include birth year fixed effects, birth order fixed effects, region fixed effects, country time trends, and a set of individual controls as outlined in Section III.

Table B2: Heterogeneous Fertility and Mortality Effects by Education

	(1)	(2)	(3)	(4)	(5)	(6)
Panel A. Child mortality						
Months since birth	0-12	13-24	25-36	37-48	49-60	
	Dead	Dead	Dead	Dead	Dead	
ITN	-0.0286***	-0.00852***	0.00479***	0.000920	-0.000196	
	(0.00460)	(0.00235)	(0.00157)	(0.00118)	(0.00112)	
Malaria prev. in 2000 * ITN	-0.00955	-0.00915*	-0.00156	-0.000783		
	(0.0181)	(0.0108)	(0.00512)	(0.00350)	(0.00355)	
Malaria prev. in 2000 * No primary	0.0215***	0.0102***	0.00565***	0.000762	0.00186***	
	(0.00224)	(0.00157)	(0.000902)	(0.000679)	(0.000683)	
ITN * No primary	-0.0193***	-0.00953***	-0.00454***	0.000273	-0.00212**	
	(0.00310)	(0.00206)	(0.00116)	(0.00101)	(0.000852)	
Malaria prev. in 2000 * ITN * No prim.	-0.0638***	-0.0278***	-0.0104*	-0.00774*	-0.00378	
	(0.0157)	(0.00909)	(0.00555)	(0.00416)	(0.00334)	
Observations	1,147,542	1,021,288	868,009	736,594	611,217	
R-squared	0.014	0.013	0.004	0.002	0.002	
Panel B. Fertility						
Woman's age	15-19	20-24	25-29	30-34	35-39	40-44
	Birth	Birth	Birth	Birth	Birth	Birth
ITN	0.0255**	0.0206*	0.0094	0.0137	0.0219*	0.0080
	(0.0102)	(0.0113)	(0.0147)	(0.0134)	(0.0117)	(0.0102)
Malaria prev. in 2000 * ITN	0.0242	0.0712**	0.0775**	0.1344***	0.0629*	0.0807**
	(0.0255)	(0.0302)	(0.0365)	(0.0371)	(0.0344)	(0.0372)
Malaria prev. in 2000 * No primary	-0.0001	0.0073	0.0072	0.0047	0.0032	0.0048
	(0.0099)	(0.0058)	(0.0093)	(0.0107)	(0.0100)	(0.0100)
ITN * No primary	-0.0584***	-0.0334***	-0.0202**	-0.0136*	-0.0168**	-0.0120*
	(0.0066)	(0.0064)	(0.0080)	(0.0075)	(0.0075)	(0.0072)
Malaria prev. in 2000 * ITN * No prim.	-0.0317	-0.0769**	-0.0713*	-0.0942**	-0.0526	0.0224
	(0.0330)	(0.0307)	(0.0367)	(0.0394)	(0.0404)	(0.0358)
Observations	1,354,134	1,290,249	1,117,627	849,769	612,839	332,852
R-squared	0.3784	0.1524	0.0551	0.0414	0.0372	0.0407

Notes: Standard errors clustered at the region level in parentheses. *** p<0.01, ** p<0.05, * p<0.1. For Panel A, the dependant variable is an indicator for whether a child was (if alive) or would have been (if dead) alive and in a given age range (in months) at the time of the survey. For Panel B, the dependent variable is an indicator for whether the woman had a live birth within the last 12 months for a given woman-year, for each age group. All regressions include birth year fixed effects, birth order fixed effects, region fixed effects, country time trends, and a set of individual controls as outlined in Section III.

Table B3: Malaria Control and Birth Intervals

Woman's age	(1) 15-19 Interval	(2) 20-24 Interval	(3) 25-29 Interval	(4) 30-34 Interval	(5) 35-39 Interval	(6) 40-44 Interval
ITN	-0.1264	0.4807	0.4195	-0.5493	0.2055	-2.9624
	(1.0143)	(0.7814)	(1.1059)	(1.4293)	(1.8773)	(3.1383)
Malaria prev. in 2000 * ITN	-6.9456*	-6.0829**	-7.3522*	-7.6456	-0.3745	-2.8149
	(3.7802)	(2.6846)	(4.0181)	(5.0471)	(7.6765)	(12.2891)
Malaria prev. in 2000 * No primary	-2.5396***	-1.5150**	-2.2830**	1.6807	2.5767	6.3034*
	(0.7658)	(0.6986)	(1.0083)	(1.7154)	(2.1428)	(3.4262)
ITN * No primary	0.1761	-0.2936	-1.0026	-0.4661	-1.9293	-7.0272***
	(0.7723)	(0.5653)	(0.7901)	(1.0517)	(1.5072)	(2.6717)
Malaria prev. in 2000 * ITN * No prim.	6.8737*	7.1690***	9.6531**	6.1066	-3.4073	8.1398
	(3.8851)	(2.4055)	(3.7573)	(4.6931)	(7.5147)	(13.0751)
Observations	49,810	213,042	240,239	181,060	110,672	38,757
R-squared	0.0404	0.0450	0.0619	0.0658	0.0653	0.0742

Notes: Standard errors clustered at the region level in parentheses. *** p<0.01, ** p<0.05, * p<0.1. The dependent variable is the number of months since the last birth for a given woman-year, for each age group. All regressions include year fixed effects, region fixed effects, country time trends, and a set of individual controls as outlined in Section III.

Table B4: Malaria Control and Gender Ratios

	(1) Male
ITN	-0.0034
	(0.0075)
Malaria prev. in 2000 * ITN	-0.0143
	(0.0252)
Observations	1,331,682
R-squared	0.0008

Notes: Standard errors clustered at the region level in parentheses. *** p<0.01, ** p<0.05, * p<0.1. The dependent variable is a dummy for whether the birth was a male. All regressions include birth year fixed effects, birth order fixed effects, region fixed effects, country time trends, and a set of individual controls as outlined in Section III.

Table B5: Malaria Control and Terminations

Woman's age	(1) 15-19 Terminated	(2) 20-24 Terminated	(3) 25-29 Terminated	(4) 30-34 Terminated	(5) 35-39 Terminated	(6) 40-44 Terminated
ITN	-0.0126	0.0051	0.0154*	0.0323***	0.0277	0.0526
	(0.0106)	(0.0096)	(0.0090)	(0.0119)	(0.0173)	(0.0342)
Malaria prev. in 2000 * ITN	0.0146	-0.0061	0.0709	-0.0021	0.0215	0.0485
	(0.0742)	(0.0440)	(0.0452)	(0.0549)	(0.0800)	(0.1501)
Malaria prev. in 2000 * No primary	-0.0186	-0.0072	0.0156	0.0076	0.0126	0.0083
	(0.0131)	(0.0094)	(0.0106)	(0.0116)	(0.0190)	(0.0317)
ITN * No primary	0.0051	-0.0026	-0.0040	-0.0325***	-0.0148	-0.0399*
	(0.0082)	(0.0068)	(0.0070)	(0.0083)	(0.0129)	(0.0235)
Malaria prev. in 2000 * ITN * No prim.	0.0715	-0.0337	-0.0754**	-0.0416	-0.0422	-0.1562
	(0.0476)	(0.0366)	(0.0376)	(0.0440)	(0.0643)	(0.1232)
Observations	94,789	167,633	151,260	104,486	62,838	24,700
R-squared	0.0123	0.0131	0.0114	0.0122	0.0164	0.0448

Notes: Standard errors clustered at the region level in parentheses. *** p<0.01, ** p<0.05, * p<0.1. The dependent variable is an indicator variable for whether the woman's pregnancy ended in termination. All regressions include year fixed effects, region fixed effects, country time trends, and a set of individual controls as outlined in Section III.

Table B6: Malaria Control and the Extensive Margin of Sexual Activity

Woman's age	(1) 15-19 Active	(2) 20-24 Active	(3) 25-29 Active	(4) 30-34 Active	(5) 35-39 Active	(6) 40-44 Active
ITN	-0.1420***	-0.4673***	-0.7669***	-0.7925***	-0.7993***	-0.7329***
	(0.0504)	(0.0876)	(0.1447)	(0.1498)	(0.1376)	(0.1313)
Malaria prev. in 2000 * ITN	-0.1049	0.1747	0.5329*	0.7155**	0.5455**	0.4240
	(0.0982)	(0.1401)	(0.2867)	(0.3041)	(0.2612)	(0.2581)
Malaria prev. in 2000 * No primary	-0.0013	-0.1052***	-0.1346***	-0.0677*	-0.0976***	-0.0124
	(0.0446)	(0.0344)	(0.0340)	(0.0359)	(0.0363)	(0.0385)
ITN * No primary	-0.1103***	-0.0055	0.0206	0.0459*	0.0313	0.0517**
	(0.0335)	(0.0285)	(0.0235)	(0.0234)	(0.0238)	(0.0216)
Malaria prev. in 2000 * ITN * No prim.	-0.1082	-0.0484	0.1479	-0.0314	0.1228	-0.0759
	(0.1214)	(0.1212)	(0.1066)	(0.1143)	(0.1073)	(0.1022)
Observations	193,988	169,741	158,060	125,148	105,925	80,999
R-squared	0.1230	0.1431	0.1525	0.1586	0.1550	0.1551

Notes: Standard errors clustered at the region level in parentheses. *** p<0.01, ** p<0.05, * p<0.1. The dependent variable is an indicator variable for whether the woman was sexually active in the four weeks previous to the interview. All regressions include year fixed effects, region fixed effects, country time trends, and a set of individual controls as outlined in Section III.

Table B7: Malaria Control and Contraceptive Use

Woman's age	(1) 15-19 Not Using	(2) 20-24 Not Using	(3) 25-29 Not Using	(4) 30-34 Not Using	(5) 35-39 Not Using	(6) 40-44 Not Using
ITN	-0.0474	-0.0464	0.0231	0.0172	0.0008	-0.0506
	(0.0311)	(0.0392)	(0.0408)	(0.0447)	(0.0438)	(0.0508)
Malaria prev. in 2000 * ITN	0.0064	-0.0468	-0.1526	-0.2759*	-0.0414	-0.0233
	(0.0775)	(0.1235)	(0.1568)	(0.1469)	(0.1387)	(0.1556)
Malaria prev. in 2000 * No primary	0.0845***	0.1377***	0.0334	-0.0180	0.0345	-0.0247
	(0.0214)	(0.0330)	(0.0360)	(0.0368)	(0.0379)	(0.0355)
ITN * No primary	0.0064	-0.0196	0.0268	0.0265	0.0193	0.0035
	(0.0148)	(0.0284)	(0.0387)	(0.0361)	(0.0347)	(0.0343)
Malaria prev. in 2000 * ITN * No prim.	0.1759**	-0.1626	-0.3431	-0.2148	-0.5686***	-0.4173**
	(0.0851)	(0.1640)	(0.2275)	(0.2080)	(0.1879)	(0.1847)
Observations	156,244	137,465	129,928	101,763	86,115	65,986
R-squared	0.0848	0.1252	0.1549	0.1644	0.1520	0.1488

Notes: Standard errors clustered at the region level in parentheses. *** p<0.01, ** p<0.05, * p<0.1. The dependent variable is an indicator variable which takes a value of 1 if the woman reports not using contraception, either modern or traditional. All regressions include year fixed effects, region fixed effects, country time trends, and a set of individual controls as outlined in Section III.

Table B8: Malaria Control and Fertility Desires: Whether Wants Another Child

Woman's age	(1) 15-19 Wants Child	(2) 20-24 Wants Child	(3) 25-29 Wants Child	(4) 30-34 Wants Child	(5) 35-39 Wants Child	(6) 40-44 Wants Child
ITN	0.3228	-0.0236	-0.3055**	-0.2972**	-0.2025*	-0.1037
	(0.2137)	(0.1838)	(0.1426)	(0.1266)	(0.1049)	(0.0700)
Malaria prev. in 2000 * ITN	-1.1968**	-1.3349***	-0.7928***	-0.6690***	-0.4302**	-0.2079
	(0.4621)	(0.3743)	(0.2431)	(0.2278)	(0.1879)	(0.1275)
Malaria prev. in 2000 * No primary	0.0452	0.0238	0.0464	0.0106	0.0460	0.0621
	(0.0324)	(0.0286)	(0.0358)	(0.0421)	(0.0365)	(0.0384)
ITN * No primary	-0.0415	-0.0279	-0.0259	-0.0494*	-0.0728***	-0.0657***
	(0.0294)	(0.0213)	(0.0232)	(0.0297)	(0.0225)	(0.0216)
Malaria prev. in 2000 * ITN * No prim.	-0.0287	-0.1010	-0.1616	0.0026	-0.0942	-0.1135
	(0.1402)	(0.1110)	(0.1236)	(0.1466)	(0.1232)	(0.1216)
Observations	193,988	169,741	158,060	125,148	105,925	80,999
R-squared	0.4471	0.4181	0.3302	0.2616	0.1924	0.1195

Notes: Standard errors clustered at the region level in parentheses. *** p<0.01, ** p<0.05, * p<0.1. The dependent variable is an indicator variable which takes a value of 1 if the woman reports wanting another child. All regressions include year fixed effects, region fixed effects, country time trends, and a set of individual controls as outlined in Section III.

Table B9: Malaria Control and Fertility Desires: Wants Child Within the Next Two Years

Woman's age	(1) 15-19 Wants Child	(2) 20-24 Wants Child	(3) 25-29 Wants Child	(4) 30-34 Wants Child	(5) 35-39 Wants Child	(6) 40-44 Wants Child
ITN	-0.0069	-0.0373	-0.0895*	-0.0987*	-0.0971*	-0.0463
	(0.0271)	(0.0431)	(0.0474)	(0.0506)	(0.0554)	(0.0456)
Malaria prev. in 2000 * ITN	-0.0600	-0.1481*	-0.2010**	-0.1734*	-0.1007	-0.1135
	(0.0564)	(0.0785)	(0.0894)	(0.0995)	(0.1084)	(0.0796)
Malaria prev. in 2000 * No primary	0.0432	0.0235	-0.0265	-0.0136	0.0021	0.0122
	(0.0340)	(0.0284)	(0.0293)	(0.0303)	(0.0243)	(0.0318)
ITN * No primary	-0.0780***	-0.0436**	-0.0354**	-0.0102	-0.0321**	-0.0501***
	(0.0228)	(0.0194)	(0.0165)	(0.0186)	(0.0153)	(0.0168)
Malaria prev. in 2000 * ITN * No prim.	0.0412	0.0728	0.1126	0.0284	-0.0043	-0.0277
	(0.0947)	(0.0875)	(0.0816)	(0.0889)	(0.0797)	(0.0854)
Observations	193,988	169,741	158,060	125,148	105,925	80,999
R-squared	0.1044	0.0860	0.0846	0.0878	0.0896	0.0757

Notes: Standard errors clustered at the region level in parentheses. *** p<0.01, ** p<0.05, * p<0.1. The dependent variable is an indicator variable which takes a value of 1 if the woman reports wanting another child within the next two years. All regressions include year fixed effects, region fixed effects, country time trends, and a set of individual controls as outlined in Section III.

Table B10: Heterogeneous Fertility Effects by Whether a Woman Ever Has a Say in Major Household Decisions

Woman's age	(1) 15-19 Birth	(2) 20-24 Birth	(3) 25-29 Birth	(4) 30-34 Birth	(5) 35-39 Birth	(6) 40-45 Birth
ITN	0.0483***	-0.0027	-0.0023	-0.0002	0.0060	-0.0002
	(0.0152)	(0.0174)	(0.0207)	(0.0188)	(0.0154)	(0.0138)
Malaria prev. in 2000 * ITN	0.0016	0.0246	-0.0145	0.0367	-0.0878	-0.0139
	(0.0532)	(0.0584)	(0.0646)	(0.0584)	(0.0604)	(0.0587)
Malaria prev. in 2000 * Ever say	-0.0093	-0.0177*	-0.0165*	-0.0002	0.0079	-0.0179
	(0.0093)	(0.0093)	(0.0091)	(0.0100)	(0.0117)	(0.0136)
ITN * Ever say	-0.0605***	-0.0002	-0.0060	0.0021	0.0056	0.0061
	(0.0082)	(0.0070)	(0.0084)	(0.0089)	(0.0090)	(0.0092)
Malaria prev. in 2000 * ITN * Ever say	0.0618	0.0612*	0.0459	0.0250	0.1066**	0.0983**
	(0.0461)	(0.0364)	(0.0430)	(0.0479)	(0.0439)	(0.0476)
Observations	577,433	813,279	789,620	616,498	444,805	239,201
R-squared	0.3519	0.0930	0.0408	0.0377	0.0346	0.0368

Notes: Standard errors clustered at the region level in parentheses. *** p<0.01, ** p<0.05, * p<0.1. The dependent variable is an indicator for whether the woman had a live birth within the last 12 months for a given woman-year, for each age group. "Ever say" is an indicator variable which takes the value of one if a woman reported having at least partial say on any of six household decisions, as outlined in a footnote in Section VI. All regressions include birth year fixed effects, region fixed effects, country time trends, and a set of individual controls as outlined in Section III.

Table B11: Heterogeneous Fertility Effects by Unmet Need

Woman's age	(1) 15-19 Birth	(2) 20-24 Birth	(3) 25-29 Birth	(4) 30-34 Birth	(5) 35-39 Birth	(6) 40-45 Birth
ITN	-0.0071	-0.0129	-0.0343**	-0.0382**	-0.0401***	-0.0405***
	(0.0117)	(0.0131)	(0.0169)	(0.0155)	(0.0121)	(0.0101)
Malaria prev. in 2000 * ITN	0.0395	0.0678**	0.0614	0.1153***	0.0501	0.1298***
	(0.0334)	(0.0340)	(0.0468)	(0.0398)	(0.0411)	(0.0380)
Malaria prev. in 2000 * Unmet need	0.0175*	0.0136	0.0214*	0.0361**	0.0446***	0.0660***
	(0.0093)	(0.0087)	(0.0119)	(0.0143)	(0.0171)	(0.0229)
ITN * Unmet need	0.0573***	0.0554***	0.1121***	0.1450***	0.1838***	0.1761***
	(0.0051)	(0.0045)	(0.0055)	(0.0066)	(0.0082)	(0.0086)
Malaria prev. in 2000 * ITN * Unmet need	-0.0964***	-0.0910***	-0.0566	-0.1416***	-0.1469***	-0.1956***
	(0.0339)	(0.0301)	(0.0404)	(0.0456)	(0.0558)	(0.0712)
Observations	1,068,779	1,067,172	943,692	722,695	524,523	286,832
R-squared	0.3744	0.1379	0.0546	0.0454	0.0413	0.0431

Notes: Standard errors clustered at the region level in parentheses. *** p<0.01, ** p<0.05, * p<0.1. The dependent variable is an indicator for whether the woman had a live birth within the last 12 months for a given woman-year, for each age group. "Unmet need" is an indicator variable which takes the value of one if a woman reports she is sexually active, not using birth control, and does not want more children. All regressions include birth year fixed effects, region fixed effects, country time trends, and a set of individual controls as outlined in Section III.

Table B12: Heterogeneous Fertility Effects by BMI

Woman's age	(1) 15-19 Birth	(2) 20-24 Birth	(3) 25-29 Birth	(4) 30-34 Birth	(5) 35-39 Birth	(6) 40-45 Birth
ITN	-0.0062	-0.0182	-0.0165	-0.0183	-0.0108	-0.0086
	(0.0128)	(0.0158)	(0.0205)	(0.0184)	(0.0137)	(0.0125)
Malaria prev. in 2000 * ITN	0.0350	0.0444	0.0059	0.0175	0.0306	0.0321
	(0.0323)	(0.0328)	(0.0456)	(0.0414)	(0.0445)	(0.0449)
Malaria prev. in 2000 * Underweight	-0.0148***	-0.0084	-0.0328***	-0.0302***	-0.0014	0.0063
	(0.0055)	(0.0063)	(0.0105)	(0.0115)	(0.0143)	(0.0138)
ITN * Underweight	0.0013	0.0063	0.0191	0.0159	0.0212*	0.0271**
	(0.0049)	(0.0066)	(0.0116)	(0.0121)	(0.0110)	(0.0113)
Malaria prev. in 2000 * ITN * Underweight	0.0630***	0.0041	0.1304*	0.0687	-0.0969	0.0328
	(0.0226)	(0.0393)	(0.0781)	(0.0666)	(0.0623)	(0.0602)
Observations	709,249	712,367	632,976	483,490	349,264	190,404
R-squared	0.3752	0.1343	0.0529	0.0433	0.0388	0.0401

Notes: Standard errors clustered at the region level in parentheses. *** p<0.01, ** p<0.05, * p<0.1. The dependent variable is an indicator for whether the woman had a live birth within the last 12 months for a given woman-year, for each age group. "Underweight" is an indicator variable which takes the value of one if a woman reports having a BMI of less than 18.5. All regressions include birth year fixed effects, region fixed effects, country time trends, and a set of individual controls as outlined in Section III.

Table B13: Heterogeneous Fertility Effects by Anemic Status

Woman's age	(1) 15-19 Birth	(2) 20-24 Birth	(3) 25-29 Birth	(4) 30-34 Birth	(5) 35-39 Birth	(6) 40-45 Birth
ITN	-0.0222	-0.0220	-0.0415*	-0.0229	-0.0190	-0.0113
	(0.0152)	(0.0173)	(0.0239)	(0.0206)	(0.0170)	(0.0150)
Malaria prev. in 2000 * ITN	0.1044**	0.1867***	0.2122***	0.1137	0.1950***	0.0756
	(0.0495)	(0.0533)	(0.0813)	(0.0714)	(0.0696)	(0.0628)
Malaria prev. in 2000 * Anemic	0.0002	0.0067	0.0017	0.0067	0.0026	-0.0038
	(0.0041)	(0.0045)	(0.0060)	(0.0085)	(0.0098)	(0.0109)
ITN * Anemic	0.0064	0.0080	0.0104	-0.0076	-0.0038	-0.0011
	(0.0048)	(0.0060)	(0.0070)	(0.0079)	(0.0106)	(0.0108)
Malaria prev. in 2000 * ITN * Anemic	-0.0292	-0.0835***	-0.0219	-0.0386	-0.0992*	-0.0257
	(0.0238)	(0.0306)	(0.0424)	(0.0400)	(0.0540)	(0.0508)
Observations	401,255	396,843	350,361	269,035	194,480	108,240
R-squared	0.3861	0.1369	0.0530	0.0447	0.0408	0.0391

Notes: Standard errors clustered at the region level in parentheses. *** p<0.01, ** p<0.05, * p<0.1. The dependent variable is an indicator for whether the woman had a live birth within the last 12 months for a given woman-year, for each age group. "Anemic" is an indicator variable which takes the value of one if a woman is either severely or moderately anemic. All regressions include birth year fixed effects, region fixed effects, country time trends, and a set of individual controls as outlined in Section III.

Table B14: Regional Timing of ITN Distribution

	(1) Int. Year	(2) Int. Year	(3) Int. Year	(4) Int. Year	(5) Int. Year	(6) Int. Year	(7) Int. Year	(8) Int. Year	(9) Int. Year	(10) Int. Year
Urban	0.743									0.375
	(0.534)									(0.592)
Radio		0.781								-0.361
		(0.807)								(0.758)
TV			1.052							3.331
			(0.853)							(2.645)
Fridge				1.681						-0.403
				(1.486)						(2.120)
Car					4.489*					4.521
					(2.630)					(3.678)
Electricity						0.504				-3.501**
						(0.760)				(1.577)
Wealth							0.0856			-0.138
							(0.172)			(0.190)
Imp. Water								0.728		0.850
								(0.526)		(0.802)
Imp. Toilet									0.726	-0.578
									(0.876)	(0.678)
Observations	344	344	344	344	344	332	251	344	344	239
R-squared	0.760	0.756	0.759	0.758	0.761	0.770	0.734	0.757	0.756	0.768

Notes: Standard errors clustered at the country level in parentheses. *** p<0.01, ** p<0.05, * p<0.1. The dependent variable is an indicator for whether the region has ever surpassed 10 percent ITN usage in a given year. Each regression includes country fixed effects.

Figure B1: Pre-Intervention Malaria Prevalence and Intervention Intensity

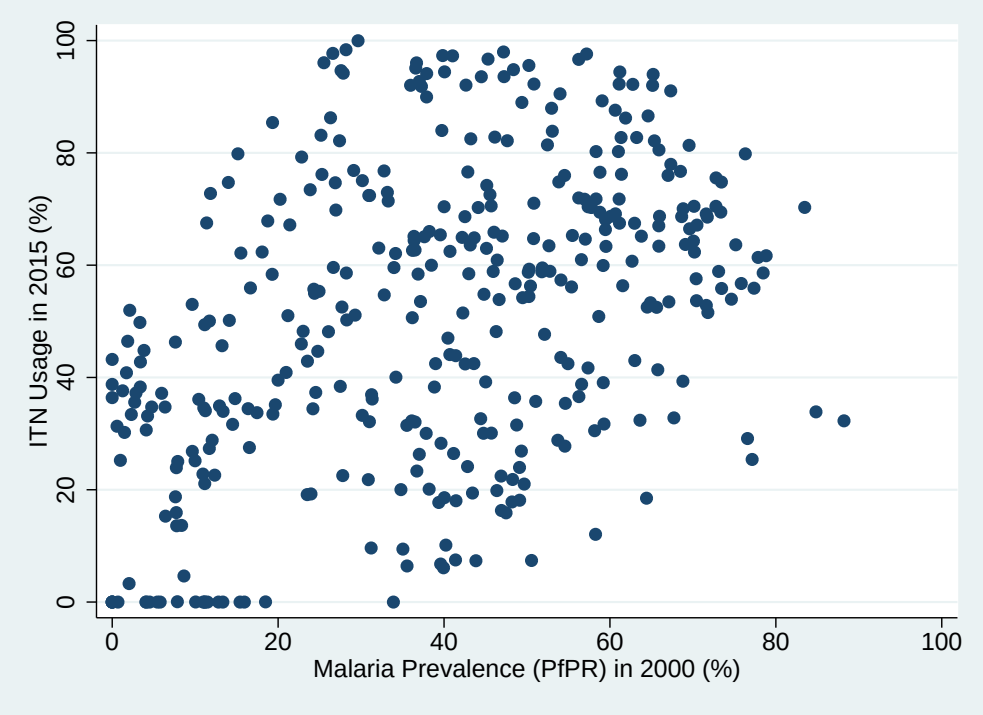


Figure B2: ITN Timing and Intensity in Nigeria

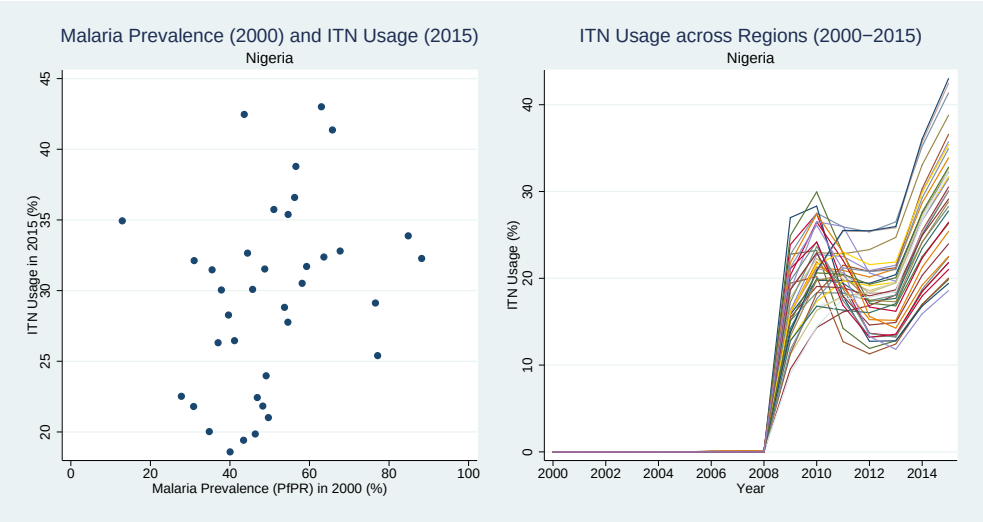
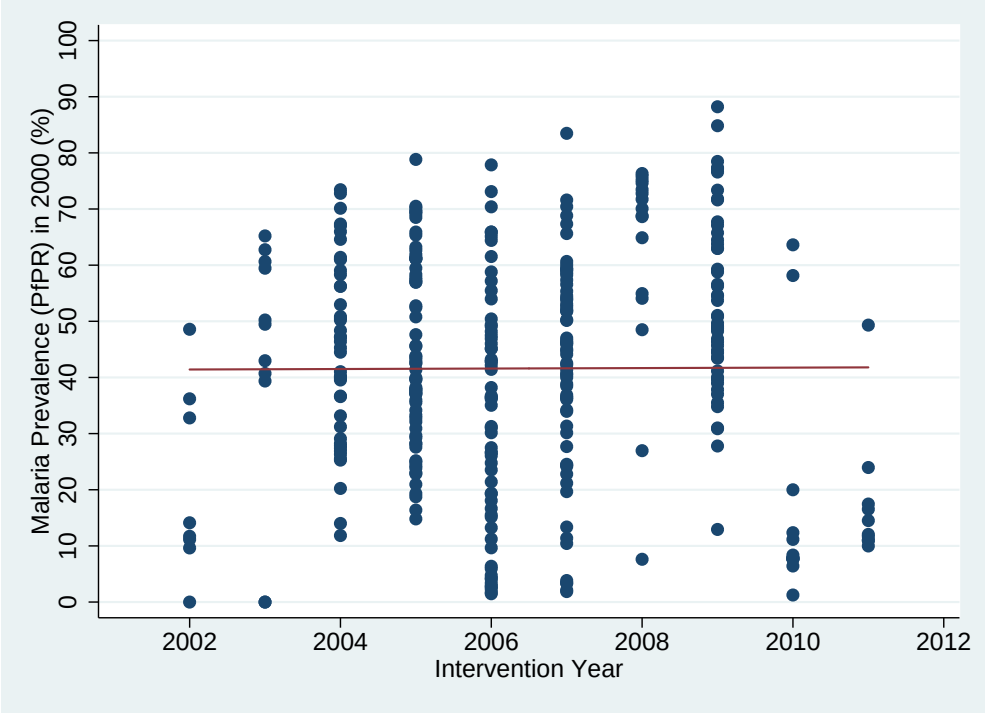
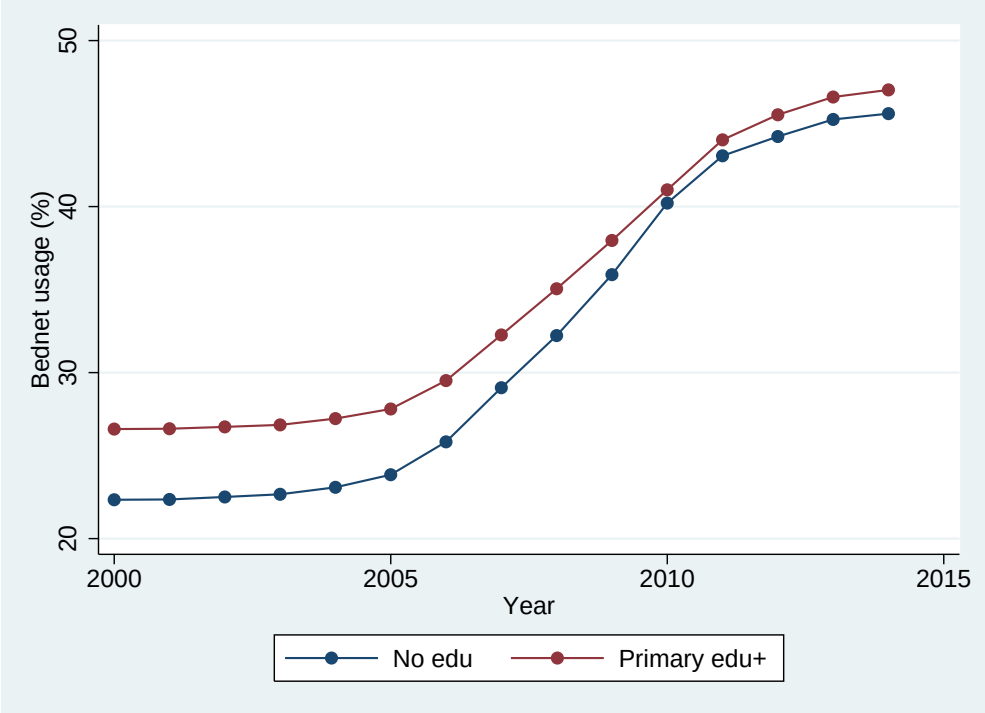


Figure B3: Intervention Year and Pre-Intervention Malaria Prevalence



Notes: Intervention Year is defined as the first year in a region in which ITN usage is reported to be greater than 10 percent.

Figure B4: Bed Net Usage by Education Level



Notes: Bed net usage numbers are reported from the DHS data, and also include non-ITN bed nets. Since usage numbers are only available for country-years in which there was a DHS survey, the non-DHS years are geometrically interpolated.