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

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Abstract: We examine to what extent recent declines in mortality and fertility in sub-Saharan Africa can be attributed to the distribution of insecticide-treated bed nets (ITNs). Exploiting the rapid increase in the distribution of ITNs in the mid-2000s, we employ a difference-in-differences estimation strategy to identify the causal effect of ITNs on mortality and fertility in 37 sub-Saharan countries between 2000 and 2014. We show that ITNs reduced all-cause mortality by 9.8 percent for infants, 18.6 percent for one year-olds, and increased total fertility rates by 9 percent in spite of reduced desire for children and increased action to prevent pregnancy.

Keywords: Malaria, Bed nets, Child mortality, Fertility, Sub-Saharan Africa.
JEL codes: I15, J13, O10.

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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 relationship remains 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 relationship. 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 remains elusive.

With the advent of the Millennium Development Goals (MDG) in 2000, there has been a massive increase in funding and support for 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. Several major international organizations, however, are promoting the idea that reducing child mortality will not only lead to smaller populations, but in fact is 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 over-population" – with an emphasis on child survival – as one of the three main myths that block progress for the poor (Gates Foundation 2014).

[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.

Worldwide, malaria is the 2nd 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 particularly 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 artemisinin-based combination therapies (ACT) – led to a 60 per cent 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]

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 solely intended to reduce infant and child mortality – but not fertility – on fertility. 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 rollout 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), Malaria Indicator Surveys (MIS), and Multiple Indicator Cluster Surveys (MICS), with a panel of malaria prevalence and antimalarial interventions from the Malaria Atlas Project (MAP) for 37 sub-Saharan countries between 1999 and 2014.

We find that the introduction of bed nets reduces child mortality for children under two years old. Beyond 24 months, however, the effect is insignificant. Specifically, we find an 8.86 percent reduction in infant (0-12 months) mortal-

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

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

ity and a 25.8 percent reduction in mortality for children from 13-24 months. This is consistent with evidence from the biological literature which shows that children within the first year of life generally gain malaria immunity from their mother's antibodies in breastmilk. We also find that the effect of bed nets on fertility is positive for every 5-year age group of women of childbearing age. Specifically, we find that the distribution of bed nets in the average region increased the annual probability of having a child by 10.0 percent for women aged 15-19, and a 5.1, 5.6, 9.7, 8.9, and 17.8 percent increase in the age specific fertility rates for women aged 20-24, 25-29, 30-34, 35-39, and 40-44 respectively. This U-shaped life-cycle effect of bed nets on fertility by age is consistent with a theoretical model modified from Kalemi-Ozcan (2003) – included in the online Appendix A – which assumes the cost of childbearing is lowest for women of prime childbearing ages.

After showing the reduced form effect of the ITN distribution campaigns on mortality and fertility, we provide evidence on the effect the ITN distribution campaigns had on theoretical mechanisms which explain the relationship between mortality and fertility. In this way, we attempt to shed light on the theoretical literature on the effect of child mortality and fertility. We investigate heterogeneity in the effects of the ITN distribution by education level and gender, and assess the impact on birth spacing and compositional effects of fertility. We also 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 almost all the increases in fertility were concentrated among for educated women, in spite of no change in their rates of sexual activity or contraceptive use.

We also show that these increases in fertility occurred simultaneously with a fall in desired fertility, raising the possibility that the increase infertility was, in fact, unintentional. We explore this possibility by hypothesizing three channels by which women are unable to control their fertility: low levels of empowerment, an unmet need for contraception, and increases in fecundity. We show no evidence that increases in fertility we concentrated among women with less decision making power in the household or with an unmet need for contraception, suggesting there was an unexpected increase in fecundity among these women.

Our paper provides two very large contributions. 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 annually on its main outcome of interest, child mortality. 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 by approximately 50 percent, the effect on fertility is exactly the opposite of the prevailing belief among aid and advocacy organizations: rather than reducing fertility, the ITN distribution programs exacerbated fertility in a region 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.

The second major contribution of our paper is that it pushes the boundary of what we know about the relationship between child mortality and fertility. Our paper tests over ten different mechanisms and determinants of fertility change hypothesized in the literature in a quasi-experimental real world setting, allowing us to provide evidence for the importance of some channels, while minimizing the importance of others.

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 Sti 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 programs in Uganda increased years of schooling by 0.5 years, while Keucken et al (2016) find that country-level malaria distributions from the Roll Back Malaria initiative increased educational attainment across the continent.

Our study more closely related to two studies 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. Rossi and Cogneau (2016) find a non-causal correlation between bed net distribution and child survival in a large set of sub-Saharan Africa countries from 2000-2015. Similar to our results, they find that the preponderance of reduction in child mortality is concentrated among lower SES households.

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, 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, and tests 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.[4] Sub-Saharan Africa carries a disproportionately high share of the global malaria burden, since 90% of these deaths occurred in that region.[5]

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).[6] 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

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

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

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

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 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).[7]. Indeed, while young children have not yet developed immunity, pregnant women 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.[8] 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 women 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 then 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 Malaria Initiative, and the World Bank – provide ITNs and funding to perform IRS in each country. Then, the National Malaria Control Programs (NMCP) for each country is responsible for the distribution of nets and of the implementation of IRS with the help of non-governmental organizations. Most countries have a goal of universal coverage with nets. Moreover, a few districts are targeted for IRS.

Until recently, chloroquine was the most widely antimalarial. However, as

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

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

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 guidelines 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 malaria to treatment rates which are far from universal.

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 and Multiple Indicator Cluster Surveys (MICS) between 2000 and 2014 for all countries in sub-Saharan Africa. This encompasses 37 countries and 100 surveys, which can be found listed in Table 1. Information on malaria prevalence and preventive behaviors 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 surveys which collect data on population and health in low- and middle-income countries worldwide. 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 hundreds of thousands of women. In addition, these data contain detailed information on health and preventive health behaviors for children, women, and men. The MICS are a similar data collection program funded by UNICEF which also provides the same basic information as the DHS program surveys.

Information on child mortality and fertility are derived from these birth histories. In the DHS program 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 DHS-

I, MIS, and AIS usually include a shorter birth history module that may contain the date of birth for the last three children and data on whether the children are alive at the interview date. Similarly, while the MICS birth history modules can be either long or short, most surveys use the short birth history module only. Because malaria eradication was not a health policy priority before the creation of the Roll Back Malaria Partnership, we restrict our sample to surveys carried out after 1999. Overall, the data set includes 100 surveys from 37 countries and 377 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.[9] MAP uses parasite surveys as well as environmental data to calculate annual malaria prevalence rates on a five by five km grid using a geostatistical model. [10] With shape files for each sub-national region given by the DHS program, we use ArcGIS 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 per-

[9] A region in our data is a sub-national geographic unit as defined by the DHS. Generally they correspond to the GEOLVL1 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. As noted previously, there are 377 such regions in our sample.

[10] For Africa, MAP employs XXX parasite surveys carried out between 1985 and XXXX. 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.

centage 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).[11] 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 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.[12] This rapid decline is due to the Roll Back Malaria program and its scale-up of ITN and IRS in sub-Saharan Africa.

MAP also provides data on ITN usage at the 5x5 km resolution, and we calculate average ITN usage as we did malaria prevalence rates. MAP 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. It is this variation – both spatially in Figure 1 and temporally in Figure 3 – from which our identification strategy is derived.

III Empirical Specification

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 the estimation strategy used in Bleakley (2010), Barofsky et. al. (2011), Lucas (2010), Cutler et al. (2010), and Venkataramani (2012), among others. Specifically, we estimate the following mortality and fertility equations:

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

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

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

and

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

where M^a_{irt} is a dummy variable for whether child i of age a who lives in region r at time t died before her next birthday. Similarly F^b_{jrt} is an indicator for whether a woman j in age group b who lives in region r at time t had a child within the 12 months before her next birthday. We estimate 5 mortality equations, one corresponding which each of their five first years of life (0-12 months, 13-24 months, 25-36 months, 47-48 months, and 49-60 months). Similarly, we estimate 6 fertility equations – one for each five year age group beginning with 15-19 and ending with 40-44 – corresponding with her 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 5 times, one for each year she is in that age group, before moving on to the next age group sample.

ITN_{rt} is the fraction of women interviewed in region r at time t who report sleeping under a bed net. For the fertility equation, we use the ITN variable the year before, 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 specifically the child i or woman j slept under a bed net. 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* – spillovers and all – on mortality and fertility in a region. In addition, the individual decision to sleep under a bed net is highly endogenous, since it is likely correlated with any number of other 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 a 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.

P_{rt} is our measure of malaria prevalence, PfPR_{2-10}. The main parameters of

interest in these specifications are on the interaction between ITN_{rt} and P_{rt}, 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_{irt}^m, include the mother's age at birth, mother's age at birth squared, urban, education level, and child gender. The control variables in the fertility equation, X_{irt}^f, include the mother's age at birth, education, urban, the birth interval and the birth interval squared. All regressions also control for the levels and interactions with malaria prevalence of two other malaria reduction interventions – anti-malarial drugs (Artemisinin-based Combination Therapy or ACT) and indoor residual spraying (IRS). Finally, we also control for region and time fixed effects (α_r^m, α_r^f, θ_t^m, and θ_t^m) and country specific time trends ($\psi_c^m t$ and $\psi_c^f t$).

Currently, the preponderance of the literature on the effects of malaria on outcomes relies on the assumption that malaria rates go to zero after the campaigns. This assumption allows these studies to claim that their estimated effects are the causal effect of declines in malaria on their outcome of choice. However, since malaria was not eradicated completely in our case, our estimates must be interpreted as the effect of the campaigns on outcomes, not the effect of malaria itself. Since the main effect of the campaigns was to reduce malaria, this is likely a somewhat semantic distinction, but important to make since we cannot rule out additional effects of the campaigns on mortality and fertility through other channels.[13] 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 academics interested in estimating the effect of mortality on fertility – are more concerned with the aggregate mortality effects of campaigns, rather than isolating specific channels.

III.A Identification Assumptions

The main econometric concern of our study is the endogeneity of our main interaction term. Notice that it is not necessary for our identification strategy that

[13]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.

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 is highly 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. 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 began.

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 two ways. First, we include region-specific time trends in all our specifications to account for this possibility. 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.

Another concern is whether the bed nets were distributed in conjunction with some other intervention 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 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.[14] As discussed in the results below, we find either no correlation between these other preventive health interventions, or exactly the opposite: women in areas which received more ITNs actually reported less of some vaccinations, suggesting that these public health interventions may be substitutes rather than compliments. This further implies that our coefficients may in fact be underestimated – we are capturing the gross mortality effects of both more ITNs, and the possible crowd-out in public health services which occurs due to the distribution of those ITNs.

IV Results

IV.A The Effect of Campaigns on Mortality

Table 3 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 net usage gives the average partial effect of net 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 insignificant thereafter. Although this coefficient cannot be interpreted causally, our result imply that regions which get more nets do not seem to be correlated with lower levels of child mortality at later ages.

The interaction term between malaria prevalence and ITN usage (our coefficient of interest) follows the same patterns as the level effect of ITNs – significant and negative for children under 24 months, and insignificant thereafter. For infant mortality, our estimated coefficient implies that the causal effect of the campaign to distribute bed nets in a region with average malaria prevalence (0.4116 from Table 1), and an increase in average bed net use from 0 percent (approximately the 2000 level from Figure 3) to 50 percent (approximately the 2014 level) is $-0.034 * 0.5 * .4116 * 100 = -0.6997$ percentage points. From a base mortality rate of 7.1 percentage points, this is an 9.86 percent reduction

[14] 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.

in infant mortality. A similar calculation shows that the introduction of ITNS reduced mortality from 13-24 months by 18.6 percent. 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).

Note that the estimates above refer to declines in all-cause mortality. Another interesting question is what fraction of the decline in malaria-specific infant mortality was due to the introduction of bed nets. Malaria mortality was likely falling over this period for reasons besides the introduction of ITNs – general health improvements such as better water, sanitation, or nutrition could also have an effect. However, annual cause-specific infant mortality rates in Africa are not available. Some good estimates come from the Global Burden of Disease published by the WHO, but they are not for all years and never break out the mortality data specifically for infants.

Using a few admittedly crude statistics, however, we can back out a rough estimate of the fraction of malaria-specific mortality decline which can be attributed to the introduction of ITNs. Estimates of the fraction of total infant mortality in Nigeria due to malaria deaths range from 25 percent to 50 percent. Taking an intermediate value of 1/3, if infant mortality was 114 per 1000 in 2003, then approximately 38 per 1000 were due to malaria. The World Malaria Report 2011 reports that malaria-specific mortality had fallen by 33 percent since 2000 in the WHO African region. Using this number for the decline in malaria specific mortality in Nigeria from 2003 to 2010, malaria mortality in 2010 would be 25 per 1000, or a decline of 13 per 1000. Since we estimated the introduction of bed nets reduced child mortality by approximately 6.997 per 1000, this implies that a little over half (53.8%) of the decline in malaria was solely due to the introduction of bed nets.

For all specifications, the estimates on our control variables are as expected. In particular, we find that being in an urban area, having an educated mother, and the age of the mother are all negatively associated with mortality, while being male and the duration of exposure are positively associated with mortality.

IV.B The Effect of Campaigns on Fertility

Table 4 reports our estimates of the effect of bed nets 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 net usage gives the average partial effect of net usage on age-specific fertility rates. We find no evidence that areas which received more nets are not systematically correlated with 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 significant at least at the 10 percent level for all age groups except for 25-29 year old women (which is significant at the 10.8 percent level). To interpret this coefficient, 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 dissemination of bed nets in the average region increased the annual probability of having a child for women in this age group by 1.178 percentage points. With a base fertility rate of 11.71 percent annual hazard of having a child, this implies a 10.0 percent increase in the age-specific fertility rate. Similarly we find a 5.1, 5.6, 9.7, 8.9, and 17.8 percent increase in the age specific fertility rates for women aged 20-24, 25-29, 30-34, 35-39, and 40-44 respectively.

IV.C 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 nets. 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 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 were more 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.

What is a problem for identification, however, is if mortality or fertility were already changing differentially in regions which received more nets. We attempt to control for the existence of different parallel trends by including a region-specific time trend in all of our regressions. Another method for testing for non-parallel trends is to run a placebo test where the 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 5 and 6. Each table contains two panels – Panel A in which we lead the intervention year by 2 years, and Panel B in which we lead the intervention year by 4. For the mortality placebo regression in Table 5, the interaction between malaria prevalence and ITNs are not significant in either the two- or four-lead specifications with the exception of children in their fourth year of life. Yet this result is only significant at the 10 percent level and economically very small – consistent with the fact that on average one out of ten coefficients will be significant at the 10 percent level just by chance.

The fertility placebo regressions in Table 6, however, are significantly different from zero in 3 of the 10 specifications, each time at the 1 percent level. 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, over stating 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 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 health behaviors, 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 7. We find either no effect or negative effects 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 substitutes – areas which received more ITNs were either no more likely or less likely to receive other treatments. Consequently, if anything, our main estimates underestimate the effect of ITNs on mortality and fertility.

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, 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 direct mechanics of fertility change. We then test to see how fertility preferences changed, and end the section with some discussion of our results.

V.A Heterogeneity Results

In Table 8, we divide our sample between female births and male births and see if the effects of the ITN distribution had heterogeneous effects on mortality by gender. In the overall sample, ITNs only had an effect on mortality in the first two years of life. We find a similar effect for males – mortality falls for infants and children in their second year. However, infant mortality for females is not significantly reduced. 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 9. 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 0-12 months, and insignificant for later ages. At the same time, the coefficient on malaria prevalence and ITNs has become a statistical zero for all ages. Specifically, our point estimates imply that, for the average region, infant mortality of uneducated women fell by 15.4 percent, compared with a statistically insignificant 3.6 percent decline for women with at least a primary education. This compares with the 9.8 percent decline in the pooled sample in Table 3. This implies that our original result was driven solely by reductions in infant mortality by uneducated women.

Conversely, however, we find that the increases in fertility are much stronger for educated women than the uneducated. Adding a triple interaction with no education to our fertility regressions yields negative and highly significant coefficients in the ages of 20-35, which form the bulk of the child-bearing women. Among the uneducated, only the very young (ages 15-19) and older women (ages 35-44) increased their fertility in response to the ITN distribution. Adding and interpreting the coefficients in the Results section, we find that age specific fertility rose for women aged 15-19 by 17.4 percent for the educated and 13.2 percent for the uneducated. Fertility rose by 11.0 percent and 3.1 percent for 20-24 year old women, by 13.6 and 3.2 percent for ages 25-29, by 21.8 and 6.8 percent for ages 30-34, by 16.6 and 10.0 percent for ages 35-39, and by 23.9 and

25.6 percent for ages 40-44, respectively for the educated and uneducated. All coefficients are significant at the 1 percent level except for uneducated women 20-35 years old.

Interestingly, these results seem to suggest that the mix of women who saw reductions in infant mortality were different from those who increased their fertility – the children of the uneducated were saved, while the educated had more children. This begs the question of who exactly received the ITNs. One hypothesis is that if lower SES individuals received more nets, this could explain why the mortality of their children fell disproportionately. Unfortunately, the MAP data does not break out ITN usage by education level of the mother. However, the birth history data does have a household-level bed net usage question which we can use to answer this question.

In Figure 4 we plot reported bed net usage rates from the DHS data by education group.[15][16] We see that households with women without primary education started at a lower bed net usage rate than households with 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 as also shown in the MAP in Figure 3. It is important to note, however, than the differences between the two groups are relatively small – only separated by less than 5 percentage points at the largest gap. Therefore, while there is some evidence suggesting the uneducated were disproportionately benefited by the ITN distribution programs, we do 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 the fertility increase 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 11 we run the same fertility regressions with triple

[15] Since we don't have DHS surveys in every year for every country, we don't have a balanced panel of bed net usage at the region level like we do using the MAP data. Therefore, we used geometric interpolation between years at the country level to derive this figure.

[16] The DHS data asks 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.

interactions Table 9 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 2.78 months on a base of 24XXXX, implying that fertility intensified along the intensive margin by 13.1XXXX percent. Since the overall change in fertility for that group was 17.4XXXX percent, we can back out that the extensive margin would have needed to grow by 4.3XXXX percent, implying that increases in the intensive margin accounts for approximately three-fourths of the total increase in fertility in this age group. Similarly, we find an increase in the intensive margin of fertility of XXXX for educated women ages 20-24, XXXX for ages 25-29, and XXXX for ages 30-34. There are no detectable changes in birth spacing for educated women older than 35, implying that it is likely that all of the increase in childbearing for older women was due to higher fraction of women choosing to have children later in life. For uneducated women, we find no effect on birth spacing for any age. Since we only found overall fertility effects for very young (ages 15-19) and older women (ages 35-44), it appears that the increases in fertility among these groups of women are due to increases in the extensive rather than the intensive margins of fertility.

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.[17]

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 and Apouey 2017). Since a termination is only counted in the 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 miscarriages 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 10, we run our main specification in equation (1), except with an indicator variable for whether or not the birth is a male. We find that there was 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 16. The evidence on the effect of the ITN distributions on reported terminations is mixed: we detect no change for all women from ages 15-24 and 30-34, a positive effect only for educated women aged 25-29, and marginally negative effects for all women aged 35-44. As a result, neither Table 10 nor 16 provide consistent evidence that fertility increased due to reductions in spontaneous or induced abortion.

Next we look at sexual activity. In Table 14 we run our fertility regression with triple interactions 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 but insignificant coefficients on sexual activity, with the exception of 25-29 year old women, which is significant at the 10 percent level. For uneducated women, we find lower – but still statistically insignificant effects. The triple interaction with no primary education is significant for two of the 6 age groups – 20-24 and 40-44

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

year old women. Combining the coefficients, we only find one group of women whose sexual activity fell as a result of the ITN distribution – 20-24 uneducated women. So while there is some evidence sexual activity is decreasing for lower SES women and increasing for high SES women, those results are not robust or consistent, and we conclude that there is no evidence the ITN distribution changed the fraction of sexually active women in our sample.

Finally we ask whether there were changes in contraceptive usage.[18] In Table 15 we run a similar regression as before with an indicator variable for whether the women reported using contraception as the dependent variable. We find that uneducated older women (ages 30-44) have significantly lower contraceptive usage 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, women 30-39 reduced their overall contraceptive use, as the results for uneducated women 40-44 are not statistically different from zero.

V.C Fertility Desires

While Bongaarts' proximate proximate determinants model is useful to understand the mechanics of fertility decline, it doesn't 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 result of the ITN interventions, it may be useful to understand the extent to which the better health environment induced changed 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 9 on each 5-year age group. The results are reported in Tables 12 and 13. We observe strong and universal declines in desired children, for all age groups, and

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

both for educated and uneducated women. In addition, there are no detectable differences in the effect of ITN distribution on fertility preferences between the educated and uneducated.

V.D Discussion

So what does this all mean? To summarize our results for educated and uneducated women, we found that for women with a primary education had virtually no increase in sexual activity, no changes in contraceptive use, and reported desiring fewer children as a result of the anti-malaria campaigns. For women without a primary education, we found a weak decrease in sexual activity, increase in contraception at older ages, and a decrease in desires for more children. Taken together, it seems as if both groups desired less children, especially in the short run, but uneducated women were more likely to take action to prevent pregnancy, such as using birth control and reducing their likelihood of being sexually active. Comparing this with what happened to fertility, it appears that while increases in fertility rates for both groups were concentrated among the young and old, groups which attempted to control their fertility more – uneducated women – had either no increase or a smaller increase in fertility that those which did not.

These results leave us with a puzzle: if as our results suggest the reduction in child mortality induced women to want less children – and to take action to prevent pregnancy, especially among the uneducated – then how come fertility rose for both educated women and some uneducated women? This disconnect between actions, desires, and outcomes seem to suggest fertility was not perfectly controlled by these women. If this were true, then that might lend credence to scholars who criticize the rational choice models of fertility – since if fertility cannot be controlled in this context, then perhaps we should forgo rational choice models in favor of 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 – which we crudely dub "Beckerian" and Bongaartsian". We then quickly 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 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 century 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 all affect a woman's realized number of children away 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 though 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 child mortality should be completely independent of fertility – 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.[19]

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 in add 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 averse agents.

Our model shows that the effect of a decline in child mortality is ambiguous. The quality-quality, replacement, and precautionary childbearing mechanisms all imply that a decline in child mortality would reduce fertility. All of these mechanisms reduce mortality 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 the child what 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 our malaria is younger and older women (Poespoprodjo et al 2008, Hamer et al 2009, Ayoola et al 2012, Takem and DAlessandro 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

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

ITN distribution campaigns significantly reduced the costs of childbearing – so much so that it more than offset the precautionary childbearing, replacement child, and quality-quality mechanisms.[20] Second, our model predictions are consistent with the fact that the fertility effects were larger for older and younger 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 cost of childbearing is the main driver of the increase in fertility: it may just be that women's chosen fertility and realized fertility aren't the same. We we explore this idea in the following section.

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: unexpected effects of the ITN distribution campaigns on fecundity, low empowerment or say in household decision making, and unmet need for contraception.

A first hypothesis is unexpected health effects on fecundity. If the ITN distribution campaigns allowed women to avert malaria, then it may be the case that since they were less sick, they were more likely to become pregnant. If these increases in fecundity were unexpected, then this could lead to higher pregnancy rates and a shorter birth spacing, consistent with our results. Unfortunately, we cannot test this hypothesis directly, and therefore we consider this mechanism as a residual explanation of our results. It is also important to note that this channel could interact with our other two hypotheses, as explained below.

A second hypothesis is low levels of say in household decision making. If

[20] 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 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 (through child labor) or future earning capacity (via an increase in old age assistance) of children would have the same effect. So although it may seem that the a net positive fertility response of 9 percent could only be realized in the face of an unrealistically large change in the cost of childbearing, in reality these costs may be quite large.

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 those increases would be concentrated in women with low levels of empowerment. We test this hypothesis in Table 18. 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 a say in any household decision reported in the DHS.[21] 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 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 last hypothesis is unmet need for contraception.[22] Similar to our rationale women's empowerment, if a woman is exposed to a constant fertility risk in the presence of an increase in fecundity due to the ITN distribution campaign, then we would expect an increase in fertility among women with an unmet need for contraception. We test this hypothesis in Table 17. The triple interaction between ITNs, malaria prevalence, and unmet need is uniformly zero, which implies there is no evidence of an increased fertility rate among women with an unmet need for contraception.

VI.D Final Summary on Channels

In summary, we found no evidence that the increases in fertility after the ITN distribution campaigns were caused by two specific factors which caused a wedge between desired and realized fertility – low levels of female input in decision making, and unmet need for contraception. As a result, only two real explanations of the increased fertility result remain – both of which are problematic. The first is that the ITN distribution campaigns lowered the cost of childbearing enough to cause women to choose higher levels of fertility. This

[21] XXXX List Questions Here, talk about partial say vs. complete say, and explain that we use as our indicator if a woman ever has partial say on any decision.

[22] XXX Define unmet as found in the DHS here XXXX

answer is problematic since we also found a uniform reduction in stated preferences for childbearing after the ITN campaigns, although this result depends on the reliability of self-reported fertility desires.

The second explanation is an unexpected increase in fecundity due to the ITN campaigns. This answer is problematic not in the sense that we find evidence to contradict this hypothesis, but rather because there is no way for us to prove it using the data we have. 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 our preferred explanation of our results, additional evidence is needed to confirm or refute this channel.

VII 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 on birth histories and net usage combined with information on malaria ecology and climatic factors, we estimate the effect of the rapid increase in net 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 0 to 2 – for instance, the increase in bed net usage in an average country has led to a decrease of the probability of an infant dying by 9.8 percent, and a decrease in 13-24 month mortality by 25.8 percent. We also find that the introduction of bed nets has a positive impact on fertility for all women, with relatively larger increases for younger and older women. Specifically, we find than the introduction of ITNs increased TFR by 9 percent in the average region, or half a child 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 itself 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.

Beyond looking just at mortality and fertility, we also explored the proximate determinants of fertility change. We found 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 is not perfect. Our results are consistent with the hypothesis that the distribution of ITNs increased fecundity, which led to higher levels of fertility.

Our findings on the impact of nets 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 to achieve

universal coverage.

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 for younger and older women. 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.

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Table 1: List of surveys

Country	Years	Regions	Survey sources
Angola	2000-2011	18	MIS06-07, MIS11
Benin	2000-2012	12	DHS06-07, 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 (Brazza)	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	DHS03, 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	4	DHS04-05, DHS09-10
Liberia	2000-2013	15	DHS06-07, MIS08-09, MIS11, DHS13
Madagascar	2000-2013	6	DHS03-04, DHS08-09, MIS11, MICS12, MIS13
Malawi	2000-2014	27	DHS04, MICS06, DHS10, MIS12, MICS13-14, MIS14
Mali	2000-2012	9	DHS01, DHS06, DHS12-13
Mauritania	2000-2012	9	MICS11
Mozambique	2000-2011	11	DHS03-04, AIS09, DHS11
Namibia	2000-2013	12	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
South Sudan	2000-2010	10	MICS10
Swaziland	2000-2010	4	DHS06-07, MICS10
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,MICS09,DHS10-11,MICS14

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

Table 2: Descriptive statistics for mortality equations

Variable	Mean	S.d.	Min	Max
Mortality equation				
Malaria prevalence at t	0.4029	0.1993	0.0058	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 at t-1	0.3298	0.2174	0	0.9036
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 and mortality

	(1)	(2)	(3)	(4)	(5)
	Months when the child was born before the survey				
	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)
Malaria prevalence	0.0079	0.0163***	0.0051**	-0.0003	0.0035
	(0.0074)	(0.0046)	(0.0025)	(0.0023)	(0.0026)
ITN	-0.0359***	-0.0104***	0.0026	0.0009	-0.0011
	(0.0048)	(0.0025)	(0.0017)	(0.0012)	(0.0011)
Malaria prevalence * ITN	-0.0340*	-0.0204*	-0.0029	0.0035	0.0013
	(0.0189)	(0.0106)	(0.0052)	(0.0048)	(0.0039)
Observations	1,147,543	1,021,289	868,010	736,595	611,218

Notes. ITN, IRS, ACT and malaria prevalence are measured at the region level. The models include birth order fixed effects, birth year fixed effects, region fixed effects, and region fixed effects interacted with a time trend. Standard errors, clustered at the region level, are in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 4: Malaria control and fertility

	(1)	(2)	(3)	(4)	(5)	(6)
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)
Mean malaria prevalence	0.3220	0.3299	0.3331	0.3372	0.3373	0.3373
	(0.2157)	(0.2174)	(0.2174)	(0.2177)	(0.2173)	(0.2176)
Malaria prevalence	-0.0342***	-0.0074	-0.0189	-0.0155	-0.0231	-0.0416**
	(0.0131)	(0.0143)	(0.0184)	(0.0191)	(0.0188)	(0.0198)
ITN	0.0156	0.0173	0.0037	0.0104	0.0170	0.0037
	(0.0101)	(0.0119)	(0.0150)	(0.0141)	(0.0122)	(0.0107)
Malaria prevalence * ITN	0.0712**	0.0728*	0.0823	0.1327**	0.1013**	0.1305***
	(0.0344)	(0.0432)	(0.0512)	(0.0519)	(0.0501)	(0.0465)
Observations	1,354,134	1,290,249	1,117,627	849,769	612,839	332,852

Notes. ITN, IRS, ACT and malaria prevalence are measured at the region level. The models include region trends, year fixed effects, and region fixed effects. Standard errors, clustered at the region level, are in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 5: Mortality Falsification Tests: Leads

	(1)	(2)	(3)	(4)	(5)
	Months when the child was born before the survey				
	0-12	13-24	25-36	37-48	49-60
Panel A. Explanatory variables at t+2					
Malaria prevalence at t+2	0.0408***	0.0180***	0.0045	0.0034	-0.0018
	(0.0089)	(0.0062)	(0.0031)	(0.0031)	(0.0023)
ITN at t+2	0.0079**	0.0015	-0.0019*	-0.0009	-0.0013
	(0.0039)	(0.0027)	(0.0011)	(0.0009)	(0.0009)
Malaria prevalence * ITN at t+2	-0.0212	0.0076	0.0017	-0.0078*	0.0024
	(0.0149)	(0.0086)	(0.0055)	(0.0041)	(0.0039)
Observations	1,120,237	1,000,383	847,838	715,789	592,171
Panel B. Explanatory variables at t+4					
Malaria prevalence at t+4	0.0002	-0.0042	0.0040	0.0055	-0.0031
	(0.0108)	(0.0069)	(0.0040)	(0.0038)	(0.0043)
ITN at t+4	0.0018	0.0014	-0.0004	0.0011	0.0006
	(0.0037)	(0.0022)	(0.0012)	(0.0011)	(0.0010)
Malaria prevalence * ITN at t+4	0.0074	0.0125	-0.0030	0.0014	-0.0030
	(0.0150)	(0.0091)	(0.0052)	(0.0051)	(0.0041)
Observations	1,029,630	924,078	774,025	643,698	522,847

Notes. Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 6: Fertility Falsification Tests: Leads

	(1)	(2)	(3)	(4)	(5)	(6)
			Age of the Mother			
	15-19	20-24	25-29	30-34	35-39	40-44
Panel A. Explanatory variables at t+2						
Malaria prevalence	-0.0149	-0.0079	-0.0346**	-0.0190	-0.0267	-0.0271
	(0.0101)	(0.0120)	(0.0150)	(0.0145)	(0.0175)	(0.0203)
ITN at t+2	-0.0088	0.0014	-0.0026	0.0033	0.0019	-0.0047
	(0.0056)	(0.0079)	(0.0104)	(0.0088)	(0.0088)	(0.0094)
Malaria prevalence * ITN at t+2	-0.0989***	-0.0136	-0.0260	-0.0281	-0.0344	-0.0409
	(0.0273)	(0.0351)	(0.0430)	(0.0389)	(0.0370)	(0.0425)
Observations	1,437,937	1,382,662	1,191,481	902,776	638,789	334,831
Panel B. Explanatory variables at t+4						
Malaria prevalence	-0.0165	-0.0153	-0.0280	-0.0003	0.0320	0.0630***
	(0.0140)	(0.0148)	(0.0188)	(0.0203)	(0.0196)	(0.0231)
ITN at t+4	-0.0059	0.0173**	0.0249***	0.0142*	0.0115	0.0199**
	(0.0072)	(0.0072)	(0.0086)	(0.0081)	(0.0078)	(0.0091)
Malaria prevalence * ITN at t+4	0.0101	0.0376	-0.0031	-0.0296	-0.1078***	-0.1114***
	(0.0248)	(0.0297)	(0.0424)	(0.0387)	(0.0340)	(0.0402)
Observations	1,330,441	1,287,277	1,099,111	832,027	580,383	295,511

Notes. Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 7: Falsification tests: Other outcomes

	(1)	(2)	(3)	(4)
	BCG	DPT	Either vaccine	Worker's visit last 12 months
Malaria prevalence	0.0746**	0.4399***	0.0921***	0.0021
	(0.0338)	(0.0929)	(0.0349)	(0.0130)
ITN	0.0072	-0.0362	-0.0170	0.0179*
	(0.0247)	(0.0369)	(0.0226)	(0.0095)
Malaria prevalence * ITN	0.0235	-0.4703***	0.0061	0.0058
	(0.0816)	(0.1138)	(0.0833)	(0.0412)
Observations	524,780	522,930	524,862	978,265

Notes. Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 8: Mortality by gender. RGTREND Tables

	(1)	(2)	(3)	(4)	(5)
	Months when the child was born before the survey				
	0-12	13-24	25-36	37-48	49-60
Panel A. Females					
Malaria prevalence	0.0099	0.0185***	0.0056	0.0021	0.0049
	(0.0089)	(0.0057)	(0.0038)	(0.0031)	(0.0035)
ITN	-0.0364***	-0.0084***	0.0026	0.0013	-0.0011
	(0.0058)	(0.0031)	(0.0021)	(0.0017)	(0.0018)
Malaria prevalence * ITN	-0.0273	-0.0131	-0.0029	0.0017	-0.0024
	(0.0248)	(0.0125)	(0.0080)	(0.0070)	(0.0058)
Observations	566,547	507,033	431,173	366,201	303,492
R-squared	0.0149	0.0142	0.0057	0.0039	0.0037
Panel B. Males					
Malaria prevalence	0.0057	0.0143**	0.0046	-0.0024	0.0025
	(0.0100)	(0.0062)	(0.0033)	(0.0035)	(0.0034)
ITN	-0.0352***	-0.0124***	0.0026	0.0004	-0.0011
	(0.0063)	(0.0031)	(0.0024)	(0.0017)	(0.0014)
Malaria prevalence * ITN	-0.0410*	-0.0271**	-0.0022	0.0049	0.0047
	(0.0236)	(0.0136)	(0.0081)	(0.0075)	(0.0066)
Observations	580,995	514,255	436,836	370,393	307,725
R-squared	0.0148	0.0138	0.0056	0.0036	0.0034

Notes. The models include birth order fixed effects, birth year fixed effects, region fixed effects, and region fixed effects interacted with a time trend. Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 9: Education, child mortality, and fertility

	(1)	(2)	(3)	(4)	(5)	(6)
Panel A. Child mortality						
Month of birth	0-12	13-24	25-36	37-48	49-60	
Malaria prevalence	0.0015	0.0136***	0.0036	-0.0002	0.0033	
	(0.0075)	(0.0046)	(0.0026)	(0.0024)	(0.0027)	
ITN	-0.0310***	-0.0083***	0.0042**	0.0008	0.0001	
	(0.0055)	(0.0026)	(0.0019)	(0.0014)	(0.0012)	
Malaria prevalence * ITN	-0.0123	-0.0170	0.0018	0.0061	0.0038	
	(0.0213)	(0.0109)	(0.0066)	(0.0058)	(0.0046)	
Malaria prevalence * No edu	0.0120**	0.0047	0.0023	-0.0002	-0.0000	
	(0.0053)	(0.0031)	(0.0019)	(0.0015)	(0.0017)	
ITN * No edu	-0.0063	-0.0037	-0.0030*	0.0005	-0.0025**	
	(0.0043)	(0.0024)	(0.0016)	(0.0014)	(0.0010)	
Malaria prev. * ITN * No edu	-0.0407**	-0.0025	-0.0059	-0.0059	-0.0023	
	(0.0192)	(0.0119)	(0.0074)	(0.0055)	(0.0052)	
Observations	1,147,543	1,021,289	868,010	736,595	611,218	
Panel B. Fertility						
Woman's age	15-19	20-24	25-29	30-34	35-39	40-44
Malaria prevalence	-0.0352***	-0.0111	-0.0219	-0.0186	-0.0248	-0.0423**
	(0.0129)	(0.0140)	(0.0190)	(0.0196)	(0.0191)	(0.0206)
ITN	0.0273***	0.0304**	0.0207	0.0313**	0.0320***	0.0157
	(0.0100)	(0.0118)	(0.0148)	(0.0133)	(0.0122)	(0.0109)
Malaria prevalence * ITN	0.0989***	0.1263***	0.1592***	0.2382***	0.1513***	0.1404***
	(0.0336)	(0.0429)	(0.0501)	(0.0502)	(0.0519)	(0.0502)
Malaria prevalence * Low edu	-0.0011	0.0083	0.0084	0.0091	0.0034	0.0022
	(0.0090)	(0.0055)	(0.0092)	(0.0106)	(0.0091)	(0.0098)
ITN * No edu	-0.0443***	-0.0279***	-0.0282***	-0.0303***	-0.0256***	-0.0228***
	(0.0086)	(0.0081)	(0.0102)	(0.0092)	(0.0090)	(0.0084)
Malaria prev. * ITN * No edu	-0.0241	-0.0904***	-0.1218***	-0.1641***	-0.0599	0.0099
	(0.0353)	(0.0328)	(0.0416)	(0.0394)	(0.0404)	(0.0389)
Observations	1,354,134	1,290,249	1,117,627	849,769	612,839	332,852

Notes. Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 10: Selection in utero

	(1)
	0-12
	Male
Malaria prevalence	-0.0020
	(0.0124)
ITN	-0.0070
	(0.0078)
Malaria prevalence * ITN	-0.0458
	(0.0303)
Observations	1,197,291

Notes. The model includes birth order fixed effects, birth year fixed effects, region fixed effects, and region fixed effects interacted with a time trend. Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 11: Birth Intervals

VARIABLES	(1) 15-19 Interval	(2) 20-24 Interval	(3) 25-29 Interval	(4) 30-34 Interval	(5) 35-39 Interval	(6) 40-44 Interval
Malaria prevalence	1.8155	2.5482**	0.9226	2.1292	-0.1248	-6.9595
	(1.8149)	(1.2130)	(1.4272)	(2.0979)	(2.6700)	(4.9900)
ITN	-1.3662	0.4935	0.6214	-0.3103	1.1636	-4.2545
	(1.2603)	(0.9901)	(1.2645)	(1.8197)	(2.2455)	(3.9822)
Malaria prevalence * ITN	-13.4981***	-11.4593***	-16.3407***	-14.3779*	0.1650	-20.1817
	(5.0635)	(4.0219)	(5.0281)	(7.4856)	(9.8706)	(17.1768)
Malaria prevalence * No primary	-2.4686***	-1.8879***	-1.7341*	1.9580	2.6167	6.8104**
	(0.8098)	(0.7105)	(0.9956)	(1.7593)	(2.1069)	(3.3898)
ITN * No primary	-0.0514	0.0176	-0.9022	-1.4026	-3.1659	-6.2867*
	(1.0751)	(0.7692)	(1.1028)	(1.5368)	(2.1420)	(3.6645)
Malaria prev. * ITN * No primary	13.2696***	5.4770	7.8703	6.5073	-9.7124	19.7860
	(4.3688)	(3.6798)	(5.6602)	(7.5669)	(10.2385)	(18.5779)
Observations	47,342	202,000	226,849	170,575	104,093	36,621
R-squared	0.0472	0.0484	0.0659	0.0713	0.0733	0.0845

Notes. Interval refers to the time since the last birth, measured in months. *** p<0.01, ** p<0.05, * p<0.1.

Table 12: Wants child

VARIABLES	(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
Malaria prevalence	1.2887***	1.7585***	1.5744***	1.4672***	1.1152***	0.6023***
	(0.4245)	(0.3683)	(0.3044)	(0.2704)	(0.2212)	(0.1460)
ITN	0.5032**	0.0390	-0.2893*	-0.2108	-0.1203	-0.0549
	(0.2131)	(0.1891)	(0.1659)	(0.1484)	(0.1199)	(0.0823)
Malaria prevalence * ITN	-2.3141***	-3.0188***	-2.1704***	-1.7061***	-1.1492***	-0.7921***
	(0.8318)	(0.7443)	(0.5950)	(0.5433)	(0.4243)	(0.2925)
Malaria prevalence * No primary	0.1138***	0.0574*	0.0607*	0.0218	0.0471	0.0491
	(0.0389)	(0.0303)	(0.0348)	(0.0420)	(0.0390)	(0.0374)
ITN * No primary	0.0028	0.0129	0.0110	-0.0066	-0.0438**	-0.0420*
	(0.0271)	(0.0218)	(0.0229)	(0.0258)	(0.0218)	(0.0232)
Malaria prev. * ITN * No primary	-0.1349	-0.0840	-0.1195	0.0314	-0.1288	-0.0340
	(0.1168)	(0.1036)	(0.1148)	(0.1231)	(0.1238)	(0.1179)
Observations	193,988	169,741	158,060	125,148	105,925	80,999
R-squared	0.4749	0.4411	0.3476	0.2762	0.2043	0.1292

Notes. The dependent variable is an indicator variable which takes a value of 1 if the woman reports wanting another child. *** p<0.01, ** p<0.05, * p<0.1.

Table 13: Wants child within the next two years

VARIABLES	(1) 15-19 wants_now	(2) 20-24 wants_now	(3) 25-29 wants_now	(4) 30-34 wants_now	(5) 35-39 wants_now	(6) 40-44 wants_now
Malaria prevalence	0.1556**	0.3065***	0.4752***	0.5265***	0.5283***	0.3937***
	(0.0608)	(0.0957)	(0.1198)	(0.1222)	(0.1290)	(0.1099)
ITN	-0.0161	-0.0721	-0.1065*	-0.0998	-0.0825	-0.0220
	(0.0368)	(0.0575)	(0.0615)	(0.0609)	(0.0656)	(0.0567)
Malaria prevalence * ITN	-0.2836**	-0.6409***	-0.6313***	-0.5800**	-0.5464**	-0.4454**
	(0.1105)	(0.1970)	(0.2162)	(0.2306)	(0.2524)	(0.2124)
Malaria prevalence * No primary	0.0316	0.0086	-0.0348	-0.0437	-0.0156	0.0110
	(0.0309)	(0.0250)	(0.0266)	(0.0304)	(0.0248)	(0.0311)
ITN * No primary	-0.0533**	-0.0023	-0.0201	0.0095	-0.0171	-0.0362**
	(0.0214)	(0.0184)	(0.0146)	(0.0175)	(0.0160)	(0.0167)
Malaria prev. * ITN * No primary	0.0099	-0.0066	0.0591	-0.0125	-0.1215	0.0245
	(0.0935)	(0.0830)	(0.0751)	(0.0879)	(0.0969)	(0.0881)
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

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Notes. 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. *** p<0.01, ** p<0.05, * p<0.1.

Table 14: Sexually Active

VARIABLES	(1) 15-19 sex_active	(2) 20-24 sex_active	(3) 25-29 sex_active	(4) 30-34 sex_active	(5) 35-39 sex_active	(6) 40-44 sex_active
Malaria prevalence	0.3905***	0.3888*	0.3599	0.2859	0.4195	0.3358
	(0.1039)	(0.2124)	(0.2653)	(0.2750)	(0.2721)	(0.2631)
ITN	-0.1010*	-0.5202***	-0.8637***	-0.8851***	-0.8548***	-0.8280***
	(0.0605)	(0.1210)	(0.1781)	(0.1919)	(0.1788)	(0.1717)
Malaria prevalence * ITN	-0.2067	0.0215	0.8426*	0.8208	0.6323	0.4134
	(0.1728)	(0.3475)	(0.4928)	(0.5303)	(0.4747)	(0.4715)
Malaria prevalence * No primary	0.0648	-0.0660*	-0.1106***	-0.0782**	-0.0841**	0.0205
	(0.0436)	(0.0346)	(0.0359)	(0.0377)	(0.0396)	(0.0425)
ITN * No primary	-0.0976***	0.0319	0.0163	0.0457*	0.0100	0.0246
	(0.0354)	(0.0279)	(0.0253)	(0.0256)	(0.0268)	(0.0263)
Malaria prev. * ITN * No primary	-0.1825	-0.2809**	-0.0859	-0.1462	-0.1573	-0.3021**
	(0.1434)	(0.1295)	(0.1182)	(0.1263)	(0.1349)	(0.1369)
Observations	193,988	169,741	158,060	125,148	105,925	80,999
R-squared	0.1295	0.1528	0.1633	0.1693	0.1673	0.1666

Notes. The dependent variable is an indicator variable for whether the woman was sexually active in the four weeks previous to the interview. *** p<0.01, ** p<0.05, * p<0.1.

Table 15: Contraceptive Use

VARIABLES	(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
Malaria prevalence	0.0037	-0.1008	0.0970	0.0097	-0.0461	0.0115
	(0.0567)	(0.0912)	(0.0996)	(0.0967)	(0.1016)	(0.0892)
ITN	-0.0516	-0.0213	0.0414	-0.0239	-0.0036	-0.0357
	(0.0341)	(0.0432)	(0.0510)	(0.0520)	(0.0555)	(0.0627)
Malaria prevalence * ITN	0.0705	0.2585	-0.0683	0.1432	0.2632	0.4010
	(0.1353)	(0.1762)	(0.2180)	(0.1927)	(0.2308)	(0.2746)
Malaria prevalence * No primary	0.0837***	0.1507***	0.0332	-0.0097	0.0500	-0.0264
	(0.0212)	(0.0306)	(0.0365)	(0.0380)	(0.0407)	(0.0367)
ITN * No primary	0.0096	-0.0233	0.0314	0.0401	0.0470	0.0134
	(0.0173)	(0.0313)	(0.0449)	(0.0380)	(0.0379)	(0.0399)
Malaria prev. * ITN * No primary	0.0780	-0.0913	-0.2111	-0.3279*	-0.6078***	-0.4163**
	(0.0845)	(0.1426)	(0.2153)	(0.1868)	(0.1898)	(0.1946)
Observations	156,244	137,465	129,928	101,763	86,115	65,986
R-squared	0.0895	0.1308	0.1598	0.1699	0.1572	0.1553

Notes. The dependent variable is an indicator variable which takes a value of 1 if the woman reports not using contraception, either modern or traditional. *** p<0.01, ** p<0.05, * p<0.1.

Table 16: Terminations

VARIABLES	(1) Terminated	(2) Terminated	(3) Terminated	(4) Terminated	(5) Terminated	(6) Terminated
Malaria prevalence	0.0380	0.0410**	-0.0024	0.0079	0.1140***	0.0649
	(0.0238)	(0.0170)	(0.0178)	(0.0225)	(0.0345)	(0.0675)
ITN	-0.0033	0.0074	0.0237**	0.0372***	0.0301*	0.0591
	(0.0118)	(0.0089)	(0.0096)	(0.0123)	(0.0183)	(0.0393)
Malaria prevalence * ITN	-0.0352	-0.0179	0.1018**	-0.0288	-0.1430*	-0.3561*
	(0.0528)	(0.0411)	(0.0439)	(0.0564)	(0.0847)	(0.1877)
Malaria prevalence * No primary	-0.0005	-0.0142*	0.0172*	0.0075	0.0095	-0.0295
	(0.0126)	(0.0086)	(0.0093)	(0.0116)	(0.0184)	(0.0346)
ITN * No primary edu	-0.0010	0.0053	-0.0049	-0.0337***	-0.0001	-0.0437
	(0.0099)	(0.0074)	(0.0083)	(0.0105)	(0.0161)	(0.0354)
Malaria prev. * ITN * No primary	-0.0263	-0.0147	-0.0995**	-0.0009	0.0426	-0.0076
	(0.0503)	(0.0367)	(0.0410)	(0.0506)	(0.0791)	(0.1794)
Observations	94,789	167,633	151,260	104,486	62,838	24,700
R-squared	0.0164	0.0155	0.0140	0.0152	0.0207	0.0555

Notes. The dependent variable is an indicator variable for whether the woman's pregnancy ended in termination. Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 17: Unmet Need

	(1)	(2)	(3)	(4)	(5)	(6)
Woman's age	15-19	20-24	25-29	30-34	35-39	40-45
Malaria ecology	-0.0317***	-0.0080	-0.0215	-0.0188	-0.0290	-0.0481**
	(0.0107)	(0.0144)	(0.0184)	(0.0192)	(0.0190)	(0.0191)
ITN	0.0254***	0.0217*	0.0039	0.0064	0.0076	-0.0003
	(0.0075)	(0.0113)	(0.0145)	(0.0136)	(0.0122)	(0.0106)
Malaria ecology * ITN	0.0335	0.0733*	0.0838*	0.1435***	0.1116**	0.1348***
	(0.0283)	(0.0412)	(0.0489)	(0.0501)	(0.0497)	(0.0468)
Malaria ecology * Unmet need	-0.0302**	-0.0044	0.0018	0.0129	0.0182*	0.0386***
	(0.0120)	(0.0060)	(0.0080)	(0.0082)	(0.0103)	(0.0141)
ITN * Unmet need	0.0116	-0.0085	0.0240**	0.0359***	0.0566***	0.0254*
	(0.0130)	(0.0088)	(0.0108)	(0.0108)	(0.0129)	(0.0135)
Malaria ecology * ITN * Unmet need	0.0106	0.0291	0.0121	-0.0381	-0.0395	-0.0853
	(0.0584)	(0.0456)	(0.0526)	(0.0460)	(0.0562)	(0.0625)
Observations	1,351,267	1,285,480	1,113,193	846,788	611,129	332,168
R-squared	0.1430	0.1534	0.0566	0.0435	0.0397	0.0430

Notes. The dependent variable is an indicator variable which takes a value of 1 if the woman reports having an unmet need (definition 2 in the DHS), either modern or traditional. Includes year and region trend FE. *** p<0.01, ** p<0.05, * p<0.1.

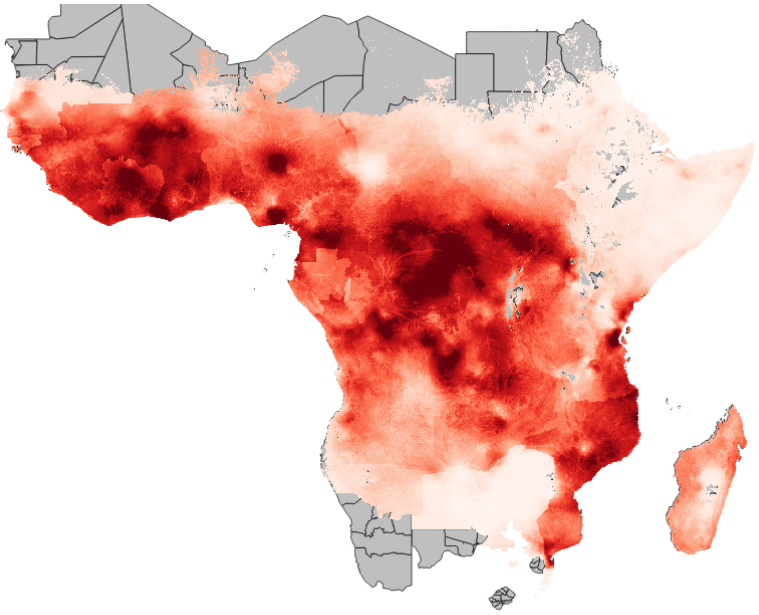
Table 18: Ever Say

	(1)	(2)	(3)	(4)	(5)	(6)
Woman's age	15-19	20-24	25-29	30-34	35-39	40-45
Malaria ecology	-0.0618**	-0.0215	-0.0278	-0.0535**	-0.0579***	-0.0545**
	(0.0271)	(0.0229)	(0.0245)	(0.0236)	(0.0202)	(0.0212)
ITN	0.0909***	0.0061	0.0011	-0.0017	0.0064	-0.0055
	(0.0172)	(0.0201)	(0.0223)	(0.0200)	(0.0169)	(0.0153)
Malaria ecology * ITN	0.0719	-0.0099	0.0023	0.0634	-0.0551	0.0180
	(0.0724)	(0.0770)	(0.0842)	(0.0789)	(0.0666)	(0.0666)
Malaria ecology * Ever say	-0.0140	-0.0188**	-0.0089	0.0061	0.0099	-0.0169
	(0.0155)	(0.0092)	(0.0087)	(0.0097)	(0.0105)	(0.0130)
ITN * Ever say	-0.0783***	0.0040	-0.0017	0.0017	0.0000	0.0015
	(0.0130)	(0.0094)	(0.0109)	(0.0102)	(0.0102)	(0.0115)
Malaria ecology * ITN * Ever say	-0.0330	0.1097***	0.0093	0.0382	0.1114***	0.0702
	(0.0614)	(0.0411)	(0.0485)	(0.0493)	(0.0420)	(0.0499)
Observations	576,436	812,128	788,642	615,802	444,352	238,944
R-squared	0.1784	0.0933	0.0414	0.0381	0.0344	0.0348

Notes. The dependent variable is an indicator variable which takes a value of 1 if the woman reports having an unmet need (definition 2 in the DHS), either modern or traditional. Includes year and region trend FE. *** p<0.01, ** p<0.05, * p<0.1.

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

2000



2014

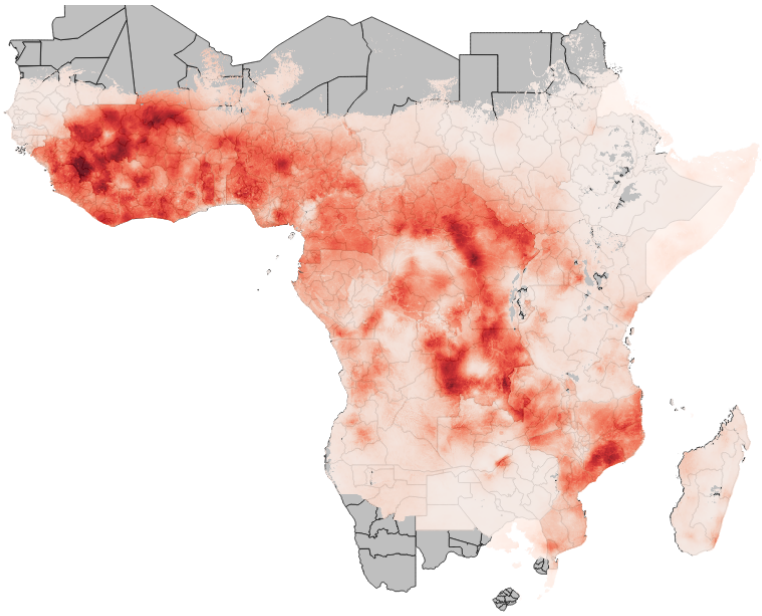


Figure 2: ITN Usage in 2014

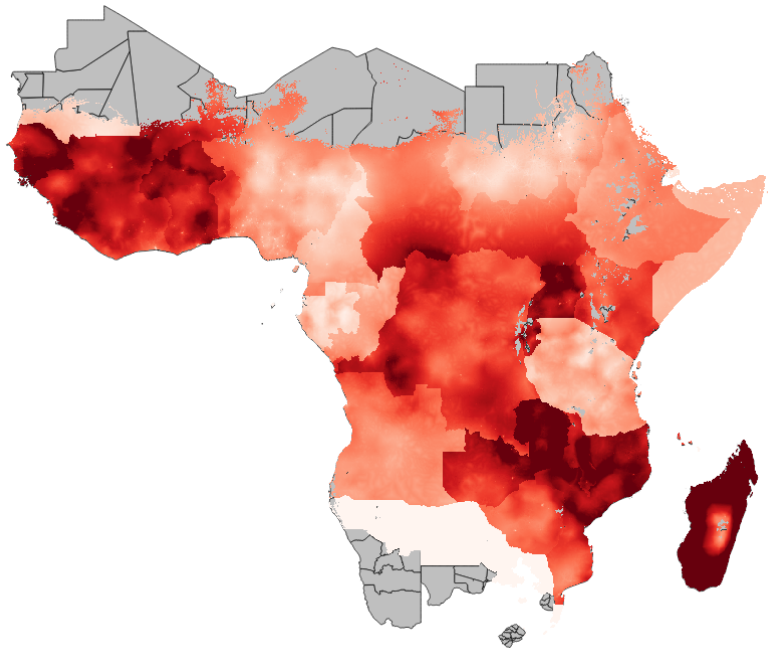
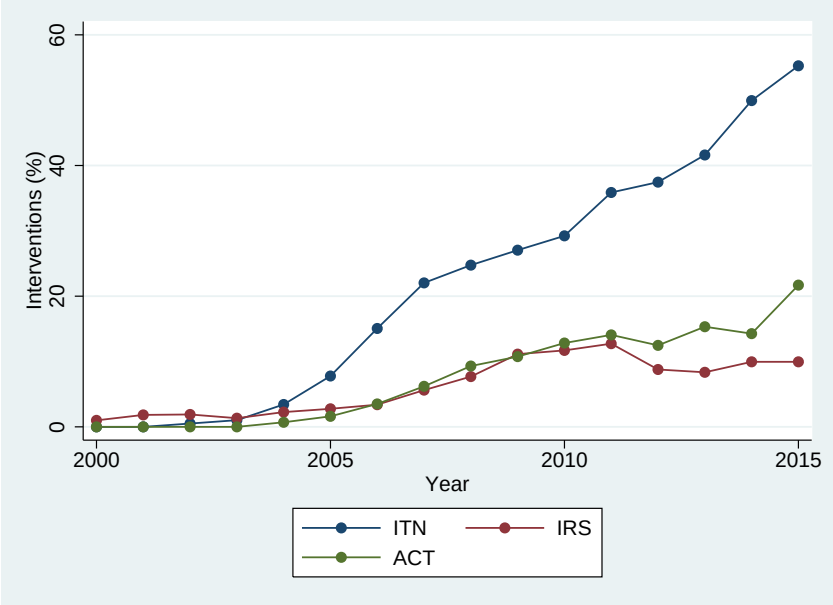
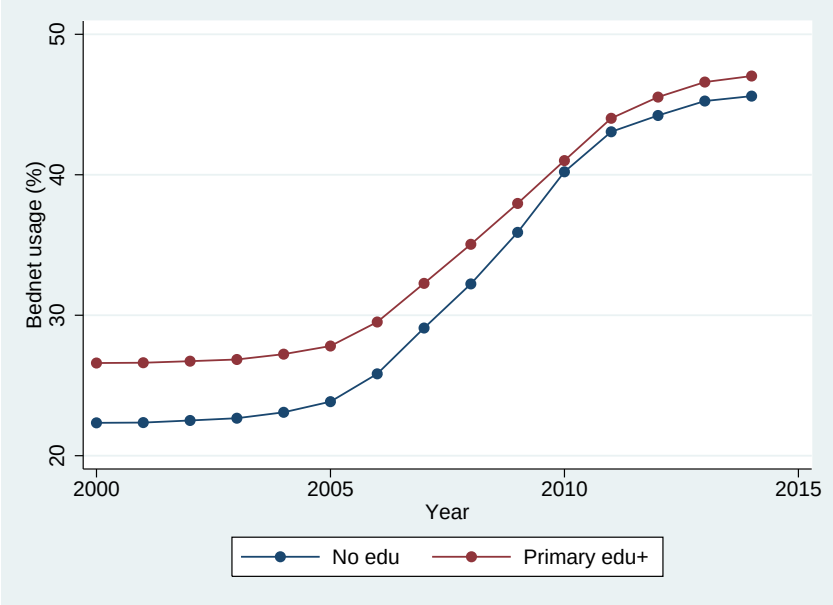


Figure 3: Evolution of regional malaria prevalence, ITN usage, IRS and ACT uptake since 2000



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: Bed Net Usage by Education Level



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 children 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.

Additionally, the above theoretical model assumes that women actively choose their fertility levels to maximize their utility. While this is the dominant view within economics as a field, many scholars – especially those in sociology and anthropology – propose that women don't always have perfect control over fertility decisions due to social norms (such as the influence of a husbands, parents, or other members of society in fertility decisions), low levels of female empowerment, and lack of contraception or information on preventing pregnancy. In this context, another channel – the fecundity channel – may also lead to an increase in fertility when malaria falls. If a woman is exposed to becoming pregnant at the same rate, and cannot prevent pregnancy (at least not perfectly), then an increase in fecundity due to a better disease environment could lead to more births.

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.[23] 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 budget constraint is

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

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

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

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

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

Since the mean of the binomial is nq,

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

[23] 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.

From log utility, the partial derivatives are:

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

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

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

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

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

which can also be rewritten as

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

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

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

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

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

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:

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

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

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). So 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.

Bed Nets and Fertility over the Life Cycle

Not only does our model incorporate different channels by which reductions in malaria would affect fertility, but it also suggests how these channels

might change fertility differentially during the life cycle. On the one hand, if bed nets campaigns increase fertility by reducing the cost of child-bearing, we expect fertility to rise faster for women in ages where child-bearing is relatively more costly. For very young women, child-bearing is relatively more costly than for women in their prime child-bearing years both biologically and because of competing investments in education. For older women, child-bearing is also relatively more costly mainly for biological reasons. As a result, if we find that reducing malaria increases fertility, we would expect these increases to be concentrated among very young women and older women.

On the other hand, if introducing bed nets reduces fertility, the expected change in life-cycle fertility looks much different. In our model, if the introduction of bed nets decreases fertility, this should be caused by reductions in precautionary child-bearing and replacement children. In the case of precautionary child-bearing, we expect bed net campaigns to mainly decrease the fertility of very young and older women. Indeed, bed nets campaigns will reduce the overall number of children needed to achieve the desired number of surviving adult offspring. A woman could react the nets campaigns in two ways: by increasing spacing but continuing having children over the same span of years, or by keeping spacing constant but reducing the number of years bearing children. If a woman decides to reduce the number of years she has children, she is likely to reduce it in ages where child-bearing is more costly, i.e. when she is very young and older.

In the case of replacement children, we expect to see larger reductions in fertility for older women. Consider a women who loses a child early in her child-bearing years. She has two choices – intensify her child-bearing by decreasing spacing, or keep her spacing constant but continue to bear children later in life. In this case, it is unclear at which ages we should see an increase in fertility. However, now consider a woman who loses a child later in life. She has no option but to have a child in her older ages. Since the population consists of women who lose children at younger and older ages, the only clear prediction is that fertility should decline relatively more at older ages as bed nets are introduced.

Putting it all together, our model predicts that overall increases or decreases in fertility should be driven by women who are very young or older. If fertility rises due to a reduction in the cost of child-bearing, those increases should

be concentrated in women who are very young and older, since child bearing is most costly for them. If fertility falls due to a decline in precautionary child-bearing and replacement children, we expect these declines to be relatively larger for very young and older women.