Malaria, Child Mortality, and Fertility: The Effect of Mosquito Net Distribution in Sub-Saharan Africa

Joshua Wilde* Bénédicte Apouey† Stacey Gelsheimer‡

Gabriel Picone§

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Abstract

Over the last decade, there has been a large international effort to distribute insecticide-treated bed nets to reduce the incidence of malaria in sub-Saharan Africa. At the same time, child mortality and female fertility have substantially declined. In this paper, we examine whether these declines in mortality and fertility can be attributed to mosquito net usage. Taking advantage of the rapid increase in the distribution of nets in the mid-2000s, we employ a difference-in-differences estimation strategy to identify the causal effect of bed nets on mortality and fertility across regions with diverse malaria ecological conditions. Using data on net usage and ownership, mortality, and fertility, combined with information on regional malaria ecology, for 22 sub-Saharan countries between 1999 and 2013, we show that nets have a greater impact on mortality in regions with ecological conditions more conducive of malaria, for children born two to three years before the date of the interview. In addition, bed nets reduce fertility of women ages 15-24 and 35-44.

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

JEL codes: I15, J13, O10.

*Corresponding Author. University of South Florida. E-mail: jkwilde@usf.edu

†Paris School of Economics - CNRS. E-mail: benedicte.apouey@psemail.eu

‡University of South Florida. E-mail: sgelshei@usf.edu

§University of South Florida. E-mail: gpicone@usf.edu

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1 Introduction

Over the last decade, under-5 child mortality has substantially declined in sub-Saharan Africa. According to the UNICEF, it fell from 187 for every 1,000 live births in 1990 to 160 in 2006, which represents a reduction of 14.4%.[1] Malaria, which is an infectious disease that is transmitted through the bites of infected mosquitoes, is a leading cause of child mortality in that part of the world. As a matter of fact, there were 219 million episodes of malaria leading to 660,000 deaths in 2010, and about 90% of these deaths occurred in Africa. In addition, 16% of all deaths among children under 5 in Africa are due to malaria (WHO, 2012). The disease is particularly dangerous for these young children because they have not yet developed partial immunity against the disease. Using available technologies, malaria can be prevented, diagnosed, and cured, which makes the large burden of the disease particularly tragic.

In the light of the preventable nature of malaria, there has been renewed emphasis to coordinate the global efforts against the disease within the past 25 years. The Roll Back Malaria Partnership (RBM) was created in 1998 to scale up preventive interventions against the infection. Examples of such preventive measures include the use of insecticide-treated mosquito nets (ITNs), indoor residual spraying, and the uptake of intermittent preventive treatments during pregnancies (IPTps). One of the goals of RBM is to achieve full coverage of people at risk of malaria in areas targeted for malaria prevention. This includes universal usage of ITNs and IPTps for children under 5 and pregnant women. To achieve these goals, large amounts of resources have been spent: for example, international disbursements for malaria control rose from less than US$100 million in 2000 to US$1.71 billion in 2010 (WHO, 2012). A good fraction of these resources has been used to disseminate ITNs, as they are considered to be an effective and cheap way to prevent the disease. Consequently, the percentage of households owning at least one ITN in sub-Saharan Africa rose from 3% in 2000 to 53% in 2011 (WHO, 2012). According to the WHO, approximately 90% of persons with access to an ITN in their household actually use it (WHO, 2012).

[1] See http://www.worldwatch.org/node/5875.

Given the emphasis placed on ITNs within the campaigns to reduce malaria, a natural question is that of the extent to which the reduction in infant mortality in sub-Saharan Africa can be attributed to mosquito net usage. Estimating this effect is not trivial, because the recent decline in mortality cannot be fully attributed to the increase in bed nets coverage. Specifically, improvements in sanitation and nutrition also contributed to the observed decline in mortality.

In addition to the effect of bed nets on mortality, there are several theoretical reasons to believe that nets could also have an effect on fertility. First, a decrease of mortality could also cause a decline in fertility by reducing precautionary child-bearing. Second, a decline in mortality could cause a decline in fertility by eliminating the need for replacement children. Third, the introduction of bed nets is likely to increase fertility if bed nets reduce the cost of child-bearing (by reducing the probability of death of the mother from malaria during pregnancy). Fourth, bed nets could decrease fertility if there is a trade-off between child quantity and quality. As a matter of fact, if bed nets reduce the in-utero exposure to malaria, leading to improved child heath and potentially higher educational attainment, child quality will increase. In the presence of a trade-off between child quantity and quality, this mechanism will induce a decline in the number of children per woman. Taken together, these points suggest that the total effect of bed nets on fertility could be either positive or negative.

In this paper, we take advantage of the rapid increase in ITN usage in the mid 2000s in sub-Saharan African to identify the effect of mosquito net usage on mortality on the one hand and fertility on the other hand. We employ a difference-in-differences approach, which exploits the fact that malaria ecological conditions are not constant across regions in sub-Saharan Africa, and that the regional roll out of bed nets was largely independent of these malaria ecological conditions. As a result, we can estimate the effect of bed nets on mortality (or fertility) by testing whether the change in mortality (or fertility) in regions in which ecological conditions are suitable to malaria is greater than in regions in which ecological conditions are less suitable for malaria. Note that our identification strategy is similar to that of Acemoglu and Johnson (2007), Bleakley (2007), Fortson

(2011), and Lucas (2010), among others.

To estimate our models, we construct a unique dataset that merges information on bed net ownership and usage, child mortality outcomes, fertility outcomes, and malaria ecology, for 22 sub-Saharan countries between 1999 and 2013. Specifically, data on mosquito nets and birth histories come from the Demographic and Health Surveys, Malaria Indicator Surveys, and Multiple Indicator Cluster Surveys. The measure of malaria ecology, which is region-year specific, is created using malaria prevalence information from the Malaria Atlas Project, and precipitation and temperature data from the Climatic Research Unit at the University of East Anglia. Overall our sample contains information on approximately 1,050,000 births.

Our results indicate that an increase in net ownership and usage reduces more child mortality in regions where ecological conditions are conducive of malaria, for children who were born two to three years before the survey. This gives weight to the arguments that nets distribution campaigns are effective and that international donations should continue supporting them. The impact of nets on mortality is independent of malaria ecology for children who were born less than two years, or more than three years, before the survey. In addition, we show that nets ownership and usage have a negative impact on fertility for women ages 15-24 and 35-44, but a positive impact for women ages 25-34.

The rest of the paper is organized as follows. Section 2 contains a conceptual framework on the relationships between bed net usage, mortality and fertility. Section 3 presents background information on the recent increase in nets ownership in Africa. Section 4 outlines our methodology while Section 5 describes the data. Section 6 presents our results and Section 7 contains some robustness tests. Finally Section 8 presents some concluding remarks.

2 Conceptual Framework

That an increase in bed net usage should reduce child mortality is rather straightforward and does not require detailed explanations. In contrast, the channels through which net usage could affect fertility are less intuitive. This section is devoted to that point.

Theoretical Model on Bed Nets and Fertility

Our model is a static model which is heavily based on the work of Kalemi-Ozcan (2003). In this model, the prevalence of malaria affects a woman's fertility choice through three channels. The first two 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. The third 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.

Consider a woman who derives utility from consumption and children in the following manner:

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

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.[2] As a result, the woman's budget constraint is

$$C = w\left[1 - v(m)n\right] \tag{2}$$

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

[2]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. 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.

probability that N out of n children will live to adulthood is

$$f\left(N;\ n,\ q\right) = \left(\begin{array}{c} n \\ N \end{array} \right) q(m)^N \left[1 - q(m)\right]^{n-N} \tag{3}$$

for each integer N between 0 and n. Combining (1) and (2) and introducing this uncertainty into the model, the woman maximizes her expected utility

$$E\left(U\right) = \left\{\gamma \ln\left(w\left[1 - v(m)n\right]\right) + \left(1 - \gamma\right) \ln\left(wN\right)\right\} f\left(N;\ n,\ q(m)\right) \tag{4}$$

We simplify this utility function by using a third-order Taylor expansion around the mean of N to get:[3]

$$E\left(U\right) = \gamma \ln\left(w\left(1 - v(m)n\right)\right) + \left(1 - \gamma\right) \ln\left[wnq(m)\right] - \frac{\left(1 - \gamma\right)\left[1 - q(m)\right]}{2nq(m)} \tag{5}$$

Taking the first order condition of (5) with respect to n and multiplying by n^2 for simplicity yields

$$G\left[n, m\right] = \frac{-\gamma v\left(m\right) n^2}{1 - v(m)n} + \left(1 - \gamma\right) n + \frac{\left(1 - \gamma\right)\left[1 - q(m)\right]}{2q(m)} = 0 \tag{6}$$

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

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

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

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

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

[3]See Appendix A of Kalemli-Ozcan (2003) or Appendix A of this paper.

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 Sri Lanka 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.

3 Background on Malaria and ITN Roll out

Malaria has historically been a major cause of mortality worldwide. Malaria, or diseases very similar to malaria, have been recorded across the globe since 3000 B.C. In the late 19th century, the malaria parasite was discovered, and in the early 20th century, programs to control malaria began to be developed. During the 1930s and early 1940s, great strides were made in malaria control, including the invention of anti-malarial drugs such as chloroquine, and the discovery of vector-controlling chemicals such as DDT. Immediately following WWII, several national malaria eradication programs began and had great success, especially in the United States, India, and Sri Lanka. In the United States, the National Malaria Eradication Program was launched in July of 1947, and had succeeded in completely eradicating malaria in the American South by 1951.

In 1955, the World Health Organization (WHO) launched the Global Malaria Eradication Campaign. Mostly through the use of insecticide spraying, malaria was eradicated from 37 countries by 1973 when the campaign was terminated. However, by the mid-1980s, the prevalence of malaria began to rise again. This led to a renewed interest in malaria and the creation of the Roll Back Malaria Partnership in 1998. Malaria control then became an important component of the Millennium Development Goals of the United Nations.

In Africa, most cases of malaria are caused by the bite of a female anopheline mosquito that is infected with protozoan parasites. Although there are several species of the parasite, the Plasmodium falciparum strain is the most common (responsible for 98% of infections) and the deadliest in Africa (RBM, 2012). In this study, we refer to "malaria episodes" and "malaria prevalence" as those caused by the P. falciparum parasite.

The P. falciparum parasite needs a human host to complete its cycle and only 40 out of the 430 known species of anopheline mosquito can carry the parasite (Crawley and Nahlen, 2004). Following the bite by an infected mosquito, the parasites leave the skin and migrate to the liver. After release, the parasites penetrate red blood cells where they multiply, causing an infection. An infected individual with no previous immunity is almost certain to develop severe flu-like symptoms that may lead to death depending on

the age and general health of the individual.

Since children between 6 months and 3 years approximately have not yet developed immunity, they are the most vulnerable population (Crawley and Nahlen, 2004). By age 10, most infected individuals suffer at worst mild complications (febrile episodes), and by 15, most individuals have asymptomatic infections and the risk of developing even mild complications is very low.

Malaria infections can be controlled using several preventive interventions: sleeping under an insecticide-treated mosquito net (ITN), indoor residual spraying (IRS), intermittent preventive treatment during pregnancy (IPTp), use of mosquito repellents, cleaning drains, and treatment of standing water with larvicidal chemicals. These interventions work by reducing the number of mosquitoes and / or by preventing bites.

Sleeping under an ITN is considered the most cost-effective intervention to prevent malaria (Lengeler, 2004). Indeed, Anophelene mosquitoes tend to bite at night and then rest inside a house (RBM, 2012). They immediately die when they come into contact with ITNs, which does not only prevents infections but also reduces the mosquito population.

Before the creation of the Roll Back Malaria Partnership in 1998, the use of mosquito nets including ITNs was very limited. Even in the early stages of the campaign, the distribution of nets was slow and not uniform across countries. The earliest scale up in the distribution of nets did not start until 2006 (RBM, 2012). The Global Malaria Action Plan (GMAP), endorsed in the 2008 Millennium Development Goals Malaria Summit of the United Nations, set a target of achieving universal coverage with ITNs for all endemic areas in Africa by 2010 (RBM, 2012).

In order to meet this goal, RBM helps individual governments in Africa to secure funding to scale-up their anti-malaria programs. The actual distribution of nets is most often done by the individual countries' National Malaria Control Programs. household received a voucher for every two members which could be redeemed for a netlater. [4]

[4]In a personal correspondence with Dr. Mea Antoine Tanoh, the director of Côte d'Ivoire's National Malaria Control Program, we established that, in general, once a national malaria control program decided that the whole country should have universal coverage with ITNs, they did not emphasize particular regions based on malaria incidence.

4 Empirical Specification

We estimate the causal effect of the malaria campaigns on fertility and child mortality by using a difference-in-differences model that exploits exogenous variation in the timing and intensity of the campaigns in different regions, along with exogenous variation in pre-campaign malaria intensity. This estimation strategy is rather common in the literature on the effect of campaigns to reduce malaria, and is broadly similar to work from Bleakley (2010), Barofsky et. al. (2011), Lucas (2010), Culter et al. (2010), Venkataramani (2012), and Kuecken et. al. (2014). Specifically, in our most basic specification we use the fraction of individuals sleeping under a bed net at the time of the interview as a proxy for campaign intensity to estimate the following mortality equation:

$$\mathrm{M}_{i,r,t} = \alpha + \gamma_1^m \mathrm{Net}_{rt} + \gamma_2^m (\mathrm{ME}_r - \overline{\mathrm{ME}}) \cdot \mathrm{Net}_{rt} + \Pi^m \mathrm{X}_{irt} + \alpha_r^m + \psi_r^m t + \epsilon_{irt}^m \qquad (7)$$

where $M_{i,r,t}$ is a dummy variable for whether the child i in region r died in the 12 months preceding the interview at time t. In all our specifications, we divide our sample into five yearly age groups: children who were born less than 12 months before the interview, between 13-24 months before the interview, etc. until our oldest group born between 48 and 60 months before the interview.[5] $\mathrm{Net}_{r,t}$ represents the fraction of individuals sleeping under a net in the region at the time of the interview, which we call bed net usage. In some specifications, we use other measures of bed nets besides usage, such as bed net ownership or ITN usage. ME_r is our pre-campaign measure of malaria prevalence, which we demean from the average malaria prevalence measure $\overline{\mathrm{ME}}$ in our sample in order to interpret the coefficient γ_1^m as the average partial effect of $\mathrm{Net}_{r,t}$ evaluated at $\overline{\mathrm{ME}}$ instead of zero. The control variables $\mathrm{X}_{i,r,t}$ include the mother's age at birth, birth interval, and child gender. We also include a control for the number of months the child was exposed to malaria which we call $\mathrm{Risk}_{i,r,t}$. For children less than 12 months at the time of the

[5] Because very few people are born exactly 12 months or 24 / 36 / 48 / 60 months before a survey, our dependent variables will not give the exact mortality probabilities for each age group. However, they allow us to study whether the effects of bed nets on children mortality is different for different age groups in a consistent manner across surveys.

interview, $\text{Risk}_{i,r,t}$ controls for the age of the child in months at t.[6] For all other age groups, exposure is always 12 months, meaning the effect of exposure on mortality is captured by the intercept. We also include region and time fixed effects in our regression, captured by α_r^m and θ_r^m.

While similar to the rest of the literature on the effect of malaria campaigns on outcomes, our empirical strategy differs in several important ways. In general, this literature uses an interaction variable between the level of malaria prevalence and some indicator variable measuring exposure to a malaria eradication campaign, usually based on the cohort's year of birth. Our study differs in that our exposure variable is not an indicator variable for being before or after the campaign, but rather a continuous one which incorporates the timing as well as the intensity of the of campaigns by region. Therefore, by using $\text{Net}_{r,t}$ as our exposure variable, we make a methodological contribution by incorporating an third source of variation (campaign intensity) compared with the rest of the literature which only uses two (timing and pre-campaign malaria prevalence).

Another advantage to our study is setting. While the current literature looks at the effect of interventions to eradicate malaria over 50 years ago, this study focuses on a recent intervention which is still ongoing. As such, our paper has more current policy relevance. In addition, our paper focuses on sub-Saharan Africa where the more deadly and agressive *P. falciparum* strain of malaria is prevalent, in contrast to the existing literature set in countries with the *P. vivax* strain, which is relatively less harmful. Another advantage is that we do not limit our study to one or two countries, but rather analyze a large population in 29 countries over 89 surveys and 327 sub-national regions, which has the advantage that our estimates can plausibly extrapolate to large populations and do not suffer from the external validity or scale concerns of a randomized trial methodology.

However, our methodology is not without drawbacks. The majority of this literature relies on the assumption that malaria rates go to zero after the campaigns, implying that the pre-campaign measure of malaria prevalence is a measure of the change in malaria. This assumption allows the other studies to claim that their estimated effects are the

[6]For children who are dead at the time of the interview, we control for their age had they survived.

causal effect of malaria on the outcome of choice. In our case, malaria is not eradicated, meaning we cannot interpret our results as such. In addition, a reliable panel measure of malaria prevalence over time does not exist, meaning that we cannot use the campaigns as an instrument for malaria prevalence in a first stage to estimate the effect of malaria itself on our outcomes. As a result, our estimates must be interpreted as the effect of the bed net campaigns on outcomes, not the effect of malaria itself. Since we believe 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.[7] However, from a policy perspective, finding the effect of the campaigns is the correct thing to do, since policy makers wishing to conduct a cost-benefit analysis of campaigns are more concerned with the aggregate effects of campaigns rather than isolating specific channels.

Another potential concern is the endogeneity of the main interaction term, malaria prevalence times the fraction of individuals sleeping under a bet net. Notice that it is not necessary for our identification strategy that the intensity of the campaigns themselves be exogenous to general mortality in the region or even pre-existing levels of malaria prevalence. In fact, it is highly unlikely that decisions about the number and location of bed net distribution were made without taking into consideration the underlying level of malaria mortality or prevalence in a region. In addition, places which have higher levels of malaria may get more of other types of aid in addition to bed nets than areas which do not. Since our regression specification includes region fixed effects, we are implicitly controlling for the fact that regions with higher malaria prevalence attract more aid in general, and specifically more bed nets once the campaigns began.[8]

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

[8]To see this, consider the case where before the beginning of the campaigns, the fraction of individuals sleeping under a bed net varies only at the region level. However, after the beginning of the distribution the intensity of the bed nets distribution is a linear function of the level of malaria prevalence in a region and a random component uncorrelated with malaria prevalence μ_{rt}:

$$\text{Net}_{rt} = \eta_r + [\sigma(\text{ME}_r - \overline{\text{ME}}) + \mu_{rt}] \cdot \text{Post}_{rt} \tag{8}$$

Plugging this into our main regression specification yields:

$$\text{M}_{irt} = \zeta_r^m + \psi_r^m t + \gamma_1^m \mu_{rt} \cdot \text{Post}_{rt} + \gamma_2^m (\text{ME}_r - \overline{\text{ME}}) \cdot \mu_{rt} \cdot \text{Post}_{rt} + \Pi^m \text{X}_{irt} + \theta_{irt}^m \tag{9}$$

A more realistic problem is if the policy makers systematically assigned more nets to those places where mortality was already falling faster – violating the parallel trends assumption. This is more difficult to test directly since we do not have an annual panel of mortality rates by region. Instead, we attempt to control for this problem in two ways. First, we include region-specific time trends in all our specifications. Second, we run placebo tests for other diseases to test whether areas which received more bed nets saw differentially larger reductions in mortality based on the incidence of other diseases besides malaria which should not be affected by bed nets. Specifically, we replace malaria prevalence with the prevalence of these other diseases in our regression model, and test whether the interaction term is significant. If we find a significant effect of bed nets on diseases which should not have been affected by bed nets, this implies that the distribution of bed nets was likely correlated with pre-existing trends in mortality.

Another concern may be that the bed nets were distributed in conjunction with some other program which reduced mortality. This is especially important since bed nets were many times distributed to women going to antenatal care visits, and who also likely had an opportunity to get vaccinations for their children at the same time. If these antenatal care visits and vaccinations were the main reason for the trip to the clinic, then the campaigns may not have increased the overall amount of these public health interventions. However, if people went to the clinics for bed nets, and then decided to also get antenatal care and vaccinations, then the mortality and fertility effects of these additional public health measures may be driving our results. 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 difference in difference 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 disproportionately more in

where $\zeta_r^m = \alpha_r^m + \gamma_1^m \eta_r + \gamma_2^m \eta_r (\text{ME}_r - \overline{\text{ME}})$, $\theta_{irt}^m = \omega_r \text{Post}_{rt} + \epsilon_{irt}^m$, and $\omega_r = \sigma[\gamma_1^m (\text{ME}_r - \overline{\text{ME}}) + \gamma_2^m (\text{ME}_r - \overline{\text{ME}})^2]$. Therefore, the parameters of interest, γ_1^m and γ_2^m, identified with only the random component of the intensity of bed net distributions, and *not* with the component correlated with malaria prevalence. Also of note is the fact that, with the inclusion of region fixed effects in our model, θ_{irt} will be orthogonal to $\mu_{rt} \cdot \text{Post}_{rt}$ since the effect of Post_{rt} varies at the region level (captured by ω_r in the error term). As a result, our main specification will correctly identify the parameters of interest even when the intensity of bed net distribution is correlated with underlying malaria prevalence in a region.

areas with higher malaria prevalence.[9]

Obtaining good pre-campaign measures of malaria prevalence is difficult. The best malaria prevalence data currently available is from the Malaria Atlas Project (MAP), but their data is for current prevalence, not pre-campaign prevalence. Using current malaria prevalence as a proxy for pre-campaign malaria prevalence is problematic since they campaigns themselves likely affected malaria prevalence – in fact, this is precisely what we are hoping happens. Instead, we use two different measures of malaria ecology to proxy for the pre-campaign malaria prevalence. First, we use the Kizewski index, a measure of malaria ecology based on the life cycle of the mosquito, which should be exogenous to the campaigns, and which will be described in detail in section XXXX. Second, using climate data on the precipitation and temperature patterns at the region level, we regress malaria prevalence in the region (in 2010) on precipitation and temperature variables, using a flexible functional form. We then compute the predicted malaria prevalence and use it as our malaria ecology index. Since precipitation and temperature are good predictors of malaria prevalence, but are exogenous to the timing of the roll out of bed nets within a country, this malaria ecology index is likely to be exogenous in our model.

We use a similar equation to estimate the effect of the malaria campaigns on fertility:

$$\mathrm{F}_{i,r,t} = \alpha_r^f + \psi_r^f t + \gamma_1^f \mathrm{Net}_{rt} + \gamma_f^m (\mathrm{ME}_r - \overline{\mathrm{ME}}) \cdot \mathrm{Net}_{rt} + \Pi^f \mathrm{X}_{irt} + \epsilon_{irt}^f \tag{10}$$

In this specification, the unit of observation is the woman, not the child. We estimate this model separately for women aged 15-19, 20-24, 25-29, 30-34, 35-39, and 40-44 at the time of the interview to test whether the effect of the malaria campaigns are heterogenous by age. The dependent variable $\mathrm{F}_{i,r,t}$ equals 1 if the woman had a child during the year prior to t, and 0 otherwise. In contrast with the mortality equation, the fertility equation does not include a control for risk exposure since the woman could have become pregnant at any time during the previous year. *XXX OTHER CONTROLS? XXX*

[9] 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.

The parameter of interest in these specifications is γ_2^m and γ_2^f. If bed nets are effective in reducing malaria mortality among children, then we expect γ_2^m¡0. The sign on γ_2^f is ambiguous, since the theoretical effect of bed net distribution on fertility is ambiguous as explained in section (2).

In the case of mortality, distributing bed nets is likely to have an immediate effect. However, in the case of fertility there may be time lags. For example, treated children in utero may be born up to nine months later, and mothers who are treated are more likely to get pregnant in the future. To solve this, we follow Bleakley (2010) by using a long differences approach. Formally, we estimate the following regression equation:

$$\Delta Y_{i,r,t} = \alpha + \gamma(\mathrm{ME}_r - \overline{\mathrm{ME}}) \cdot \mathrm{Net}_{rt} + \Gamma \mathrm{X}_{irt} + \epsilon_{irt} \tag{11}$$

XXX Discussion here XXX

5 Data Construction

5.1 Data Sources

The individual- and household-level data on net usage and ownership, child mortality, fertility, and socio-demographic characteristics, come from the MEASURE-Demographic Health Surveys (MEASURE-DHS) program and the Malaria Indicator Cluster Surveys (MICS) program.

Within the MEASURE-DHS program, we use data from the standard Demographic Health Surveys (DHS), the Interim Demographic Health Survey (DHS(I)), the Malaria Indicator Surveys (MIS) and the AIDS Indicator Surveys (AIS). The standard DHS contains pieces of information on health preventive behaviors and sociodemographic characteristics of children, women, and men. The DHS women questionnaire includes a detailed birth history module which provides birth histories for all children born to women in the sample. It includes the date of birth and the date of death for deceased children. DHS(I), MIS, and AIS use the same basic questionnaire as the standard DHS, but do not include all the modules of the standard DHS. They usually include a shorter birth history module that

may contain the date of birth for the last three births and data on whether the children are alive at the interview date. MIS contain more detailed questions about malaria than DHS and DHS(I).

The MICS program is funded by UNICEF and it is used to monitor the situation of children and women. MICS also includes a birth history module that can be used to calculate mortality and fertility. As with the DHS program, the MICS modules can be either long or short. Most MICS surveys use the short birth history module.

All these surveys are nationally representative of the population. Starting in 1999-2000, DHS / DHS(I) / MIS / AIS and MICS began collecting information on the use and ownership of mosquito nets and whether the nets are ITNs. Because malaria eradication was not a health policy priority before the creation of the Roll Back Malaria Partnership, DHS / DHS(I) / MIS / AIS and MICS do not include questions related to malaria prevention before 1999-2000.

Using the region of residence of individuals, we are able to merge the DHS / DHS(I) / MIS / AIS and MICS datasets with our malaria ecology index. The latter is constructed using data on regional malaria prevalence in 2010, from the Malaria Atlas Project (MAP), and regional levels of precipitation and temperature between 2000 and 2010, from the Climatic Research Unit at the University of East Anglia (see details below).

We restrict our data to countries for which we have at least two usable DHS / DHS(I) / MIS / AIS or MICS surveys between 1999 and 2013. Table 1 shows the countries and years which appear in our sample. Overall, the dataset includes 89 surveys from 28 countries and 312 regions.

[Insert Table 1 here]

5.2 Region and Time Definitions

The region r is the geographical region where the household is located. For some countries, the boundaries of some regions have changed between surveys conducted in different years. In these instances, we combine regions using maps provided in the public report of each survey to get consistent regions over time. Region identifiers are used to merge the DHS

/ DHS(I) / MIS / AIS and MICS with the malaria ecology index, and to compute the average levels of net usage and ownership for each region-year.

Time t represent the survey year. In our mortality and fertility models, we control for country-specific time trends. To get these trends variables, we normalize year 1999 to be time "1" ($t = 1$). If a survey was conducted between two years, we assign the middle value: for instance, a DHS survey was conducted 2010-2011 in Senegal (DHS 10-11), and we assume that the corresponding time is 11.5.

5.3 Nets Variables

Using data from the DHS / DHS(I) / MIS / AIS and MICS surveys, we construct three measures of net usage or ownership for each region-year. Our first variable, called "net usage," captures the percentage of children under five who slept under any type of bed net in the region of interest the night preceding the interview. Our second variable, "ITN usage," reflects the percentage of children under five who slept under an ITN in the region the night preceding the interview. Note that in many surveys, there is no information on the type of bed net the child used the night before the interview, so we do not know whether this was an ITN or not. For this reason, we have fewer observations for "ITN usage" than for "net usage." Our third variable is "net ownership" and it reflects the percentage of households who own at least one net in the region-year.

In our models, we use the within-region variation in these variables to identify the effects of net usage and ownership on mortality and fertility. Ideally, we would like to have information on net usage and ownership for each region-year, to draw a precise picture of the scale-up of net usage and ownership over time, and to take full advantage of the variations in nets variables for our identification. However, the DHS / DHS(I) / MIS / AIS and MICS are not conducted every year for all countries. But these data are precise enough to enable us to identify the timing of the scale-up in each country. Moreover, they exhibit sufficient variations in the timing and in the intensity of the scale-up to get accurate estimates of the impact of nets.

In Table 2, we compute the average net usage and ownership, using our data. In

early years, between 1999 and 2005, net usage and ownership rates are low. In particular, in 2000, the average usage rate is less than 10% in Burundi, Côte d'Ivoire, Malawi, and Swaziland. In 2006-2013, these averages are larger. Specifically, in the most recent survey, more than 50% of children use a net in Benin, Burkina Faso, Burundi, Congo-Brazaville, Kenya, Madagascar, Malawi, Rwanda, Tanzania, and Uganda.

For most countries, our data enable us to identify the precise years of the scale-up of bed nets. In Cameroon, net usage starts at 13.5% in 2000 and stays low at 11.4% in 2004, but then jumps to 27.9% in 2006. At the same time, between 2004 and 2006, nets ownership increases from 19.5% to 32.2%. We can infer that there is a large scale-up of the distribution of nets in Cameroon sometime between late 2004 and early 2006.

The timing of the scale-up is different for different countries – some are rapid, while others are slow and gradual. For instance, the scale-up in Senegal is rapid: net usage remains low around 17.5% until 2006, then it jumps to 30.4% in 2006, rises to 36.5% in 2008, and reaches 46.3% in 2010-11. Some countries experience an early scale-up, while others still have very low levels of net usage and ownership (see Swaziland). Such variations in the timing and intensity of nets campaigns are ideal for our identification strategy.

[Insert Table 2 here]

5.4 Mortality and Fertility

To construct the mortality and fertility variables, we use three different dates in CMC (century-month-code) format: the net-date, woman-date, and birth-date. The net-date is the CMC date of the survey that we use to calculate net usage, the woman-date is the CMC date of the survey that have the information on the woman, and the birth-date is the CMC date of birth of the child. The net-date and woman-date are not always the same. Also, for every child and woman, there are multiple net-dates, but only one birth-date and woman-date.

The data for the child mortality model are created as follows. We first calculate the difference in months between the net-dates and the birth-date, for each net-date. For

the surveys that use the detailed birth module, we then create five different samples: (1) all children who were born 12 months before a net-date, (2) all children who were born between 13 to 24 months before a net-date and were still alive 12 months before the net-date, (3) all children who were born between 25 to 36 months before a net-date and were still alive 12 months before the net-date, (4) all children who were born between 37 to 48 months before a net-date and were still alive 12 months before the net-date, and (5) all children who were born between 49 to 60 months before a net-date and were still alive 12 months before the net-date. For the surveys that use the short birth history data module, we only have the children who were born in the 12 months preceding the interview. For each sample, we create a dummy for whether the child dies within the 12 months preceding the net-date.

The data for the fertility equation are prepared as follows. For every woman, using her woman-date and each of her net-dates, we calculate her age at each of her net-dates. We then construct six different samples: (1) all women between ages 15 and 19 at the net-date, (2) all women between ages 20 and 24 at the net-date, (3) all women between ages 25 and 29 at the net-date, (4) all women between ages 30 and 34 at the net-date, (5) all women between ages 35 and 39 at the net-date, and (6) all women between ages 40 and 44 at the net-date. For each of these samples, our fertility variable indicates whether the woman had a birth in the 12 months preceding the net-date.

5.5 Malaria Ecology

Ideally, we would like to use the pre-intervention levels of malaria in each region as our measure of malaria ecology in equations (7) and (8). However, such measures at the region level are difficult, if not impossible, to find. As a result, we need to create our own measure of malaria ecology, that needs to be exogenous to the subsequent provision of bed nets. We create our measure of malaria ecology using information on malaria prevalence, precipitation, and temperature.

Malaria Prevalence

Our measure of malaria prevalence is the percentage of individuals infected with the

Plasmodium falciparum parasite in the region. This Plasmodium falciparum parasite rate is commonly called PfPR. Information on PfPR for each region is obtained from the Malaria Atlas Project (MAP) available at www.map.ox.ac.uk. The best malaria prevalence data currently available at MAP is PfPR_{2-10} for 2010, which captures the percentage of children between the ages of 2 to 10 who have detectable levels of the Plasmodium falciparum parasite using peripheral blood (Picone et al., 2014). Hay et al. (2009) and Gething et al. (2011) provide details on the construction of PfPR_{2-10}.

Precipitation and Temperature

We use information on precipitation and temperature for each region and month, from 2000 to 2010, from the Climatic Research Unit at the University of East Anglia.

For each region, we first compute the average temperature over all months between 2000 and 2010. We then find the average maximum temperature – we find the month with the highest average temperature in the region for each year, and then average over all the years. We then find the average minimum temperature using the same method as we did to find the average maximum.

We then repeat the exact same process to find the average, minimum, and maximum levels of precipitation for each region.

Malaria Ecology

To get our malaria ecology index, we regress malaria prevalence in a region on the precipitation and temperature variables, using a non-linear, flexible form. Specifically, our explanatory variables are the average temperature, maximum temperature, minimum temperature, precipitation, maximum precipitation, minimum precipitation, as well as their squares, cubes, and quartics. In total, we use 24 explanatory climate variables, which are meant to capture the non-linearities and seasonalities of the effect of climatic conditions on malaria prevalence.

The fit of the model is very good, with an R^2 of 0.68 (see Appendix B). Our malaria ecology index is then the prediction from this regression (Apouey and Picone, 2014).

5.6 Descriptive Statistics

Figure 1 shows the evolution of bed net usage and mortality between 1999 and 2013 in our sample. The share of children under 5 sleeping under a net has increased over time, especially starting in 2004. At the same time, mortality has steady declined.

[Insert Figures 1]

Table 3 provides summary statistics for some variables. Average malaria ecology in the regions of our sample is 34.6%. The average mother's age is around 26 years old. 50% of births are boys.

[Insert Table 3 here]

6 Results

6.1 The Effect of Nets on Mortality

Table 4 reports our estimates of the effect of net usage and ownership on child mortality in the 12 months preceding the interview. 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 survey. Panels A, B, and C present the estimates when the main explanatory variable is either use any net usage, or ITN usage, or any net ownership. In all specifications, the coefficient of interest is on the interaction between malaria ecology and the nets variable.

In Panel A, the coefficient on net usage gives the average partial effect of net usage on mortality. As expected, this effect is generally negative. Specifically, the effect is negative and significant in columns (2)-(5), and insignificant in column (1). In column (2), our estimate implies that when net usage in the region increases by 10 percentage points, the probability that a child dies decreases by 0.18 percentage points. The coefficient on the interaction between malaria ecology and nets usage is systematically negative, but only significant for children born 25-36 months before the interview, while it is insignificant for children born 0-24 and 37-60 months before the survey. To quantify the magnitude

of this interaction effect for children born 25-36 months before the survey, we use the case of Nigeria as an example. For this country, our predicted malaria ecology measure is 50.7%, making its climate one of the most conducive of malaria in our sample. Nigeria had a decently large increase in net usage over time in our sample, from 4.3% in 2007 to 31.8% in 2010, with most of the scale up occurring in the time period 2008-2010. Using these figures, we estimate that this increase in net usage should have led to a decrease of 0.87 percentage points $(0.507 * (0.318 - 0.043) * (-0.063) = -0.0087)$ in the probability of dying for children born 13-24 months before the survey.

That the coefficient on the interaction term between malaria ecology and net usage is significant for children born 25-36 months before the interview, but insignificant for children born less than 24 months or more than 37 months before the survey, is somewhat unsurprising. On the one hand, many children born less than 24 months before the survey are likely to still be breastfed during the 12 month preceding the interview, which increases their resistance to malaria. On the other hand, children born more than 37 months before the survey may have started to acquire partial immunity against the disease. These explain why bed nets do not have a greater impact on mortality in regions with bad malaria ecology for these two groups of children.

In Panels B and C, we re-estimate our mortality model using ITN usage and any net ownership as our explanatory variables. The results are very similar to those obtained in Panel A. Indeed, we find a negative correlation between ITN usage and net ownership on the one hand and death on the other hand, for all age groups, but this correlation is only significant for children who were born more than 13 months before the date of the interview. In addition, the coefficient on the interaction term between malaria ecology and the net variable is significant for children born 25-36 months before the interview, and insignificant for children born less than 24 months and more than 37 months before the interview.

For all specifications, the estimates on our control variables are as expected. In particular, we find that risk exposure, having a birth interval less than 24 months, and being male are all positively associated with mortality.

[Insert Table 4 here]

6.2 The Effect of Nets on Fertility

Table 5 reports our estimates of the effect of bed nets on fertility. In the columns, we report the results for the different age groups of women, while in the panels, we use the three alternative explanatory variables (any net usage, ITN usage, and any net ownership). Just as with our results for mortality, our coefficient of interest is on the interaction term between malaria ecology and the bed net variables. This coefficient is generally insignificant, with four exceptions. To interpret this coefficient, we take the example of the significant and positive estimate in Panel A, column (1), for women ages 15-19. Using the data for Nigeria between 2003 and 2010 presented above, our estimate suggests that the increase in bed net usage from 4.3% in 2007 to 31.8% in 2010 should have led to an decrease of 1.88 percentage points $(0.507 * (0.318 - 0.043) * (-0.135) = -0.0188)$ in the probability of a woman having a baby.

We observe a life-cycle pattern in the impact of the interaction term on fertility. Specifically, the coefficient on the interaction term is generally positive for younger and older women ages 15-19, 20-24, 35-39 and 40-44, whereas it is positive for middle-aged women ages 25-29 and 30-34. This provides clear evidence for a life cycle pattern in the impact of nets (malaria) on fertility. Thus, for younger and older women, the negative impact of malaria on fertility is mainly due to the fact that malaria decreases precautionary childbearing and the need for replacement children; in contrast, for middle-aged women, the positive impact of malaria on fertility is mainly due to the fact that malaria decreases the cost of having children.

[Insert Table 5 here]

6.3 The Relationship Between Mortality and Fertility

One important implication of our research is that we can compare the declines in mortality and fertility due to the introduction of bed nets and see which is falling faster. This relationship between mortality and fertility is a topic which has been studied for a very

long time with little consensus. Some scholars have argued that reductions in mortality rates are a necessary condition for reductions in fertility rates, citing the need for less births in order to achieve the same desired number of surviving offspring. But others have argued that economic factors drive both mortality and fertility rates, so observing that they move together is not sufficient evidence to suggest that mortality is the driving factor.

In addition, the fertility effects of interventions which reduce mortality are interesting from a development policy perspective. Improving health as been suggested as a major driver for economic development due to improvements in labor productivity and returns to education, among others. However, in a Malthusian framework, even if health interventions in fact do have these positive effects of productivity, they still may not lead to higher incomes due to population pressure in the presence of a fixed factor of production. For example, Ashraf et al. (2009) show that curing malaria may actually hurt economic growth in the short run since the rapid increase in population size would reduce land per capita, offsetting (and perhaps exceeding) the positive productivity effects. Acemoglu and Johnson (2007) find no effect of health interventions on economic growth during the epidemiological transition, and similarly hypothesize that any positive labor productivity gains were mitigated by increases in population size. A central assumption in these studies is that in the short run, there is little effect of these health interventions on fertility. For example, the model of Ashraf et al. (2009) assumes that fertility adjusts slowly over a period of 50 years.

Since we estimate both the mortality and fertility effects of bed net usage, we can test for the existence of an immediate fertility affect. We can also test whether these interventions should be population increasing as assumed in Acemoglu and Johnson (2007) and Ashraf et al. (2009), population neutral, or even population reducing. To do this, we use data from the UN Population Projections 2012 (UN, 2013) and apply our estimates to the age structure of the population to calculate the ratio of the number of children prevented from being born via reductions in fertility against the number of children saved via reductions in mortality. If this ratio is greater than one, then introducing bed nets is

population reducing, and if it is less than one, it is population increasing.

We begin by finding the number of children ages 0-4 in 2010 for the countries in our sample. We then take this number and divide it into annual age groups consistent with the mortality rates given in Table 4.[10] We then calculate the new age-specific mortality rates as implied by our estimates in Tables 4 and 5, under the assumption that bed net usage / ownership increases by 25 percentage points, evaluated at the mean level of malaria ecology. Comparing these two mortality rate schedules, we estimate the number of children "saved" in our sample countries as a result of this hypothesized intervention according to our estimates.

We do a similar analysis for fertility. We calculate the number of children born in 2010 by multiplying the vector of age-specific fertility rates in 2010 from our sample with the vector of the number of women in each age group. We then compare that with the number of which would have been born under the new fertility rates implied by our estimates. We then take the ratio of the number of children prevented from being born to the number of children saved from dying.

Using our estimates for "any net usage" (Panel A) in Tables 4 and 5, we find that for each child who did not die as a result of the introduction of bed nets, 0.305 less children were born. This implies that introducing bed nets is strongly population increasing as assumed by the previous literature. Stated differently, for each child who was not born as a result of the policy, there are approximately three children saved from death. This ratio becomes even smaller if we consider the estimates from Panels B (ITN usage) and C (any net ownership) – we find that for each child not born, there were approximately 5 and 7 children spared from death respectively (ratios of 0.192 and 0.136).

However, one criticism of this analysis is that we only estimate the causal effect of the interaction term in our regression equation, but not the level of bed net usage. Since the partial effect of bed nets on fertility / mortality is $\gamma_1 + \gamma_2 \cdot ME$, an unidentified γ_1 could lead to a poor estimate of this ratio. As a robustness check, we re-estimate the ratios for

[10]Note that since the mortality rates calculated in Table 4 are an average across all children in our sample, we observe the average child at age 6 months. As a result, the correct mortality rate we use in this calculation is twice as large as our reported rate.

all three panels in Tables 4 and 5, but using only the interaction terms in our calculations. We find ratios of 0.365, 0.511, and -0.043 for Panels A, B, and C, respectively.

7 Robustness Checks

To check the robustness of our findings on the impact of nets on mortality and fertility, we implement a falsification test, using as our dependent variables several health behaviors which are not directly affected by bed net usage. Specifically, we use the number of antenatal care visits of pregnant women, a dummy for whether the child has been vaccinated against polio, and a dummy for whether the child has been given full vaccination (more precisely: 1 BCG, 1 measles, 3 polio, and 3 DPT).

If the interaction term (between malaria ecology and the net variable) is positively and significantly correlated with health behaviors, then we will conclude that our main model in Tables 4 and 5 gives an upper bound of the effect of bed nets on mortality and fertility. If the interaction term is negatively and significantly correlated with health behaviors, then our models provide a lower bound of the effect of bed nets on mortality and fertility.

The results of the falsification test are given in Table 6. We find a positive and significant association between any net usage and any net ownership on the one hand and antenatal visit, child polio vaccination, and child full vaccination on the other hand. This likely captures reverse causation, since women are likely to get nets for free when they go to health care facilities for their antenatal visits or for child vaccination. Moreover, the coefficient on the interaction terms are systematically negative and significant. Consequently, if anything, our main models underestimate the effect of nets on mortality and fertility.

[Insert Table 6 here]

8 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 female fertility. We find that bed nets have been effective in their goal of reducing child mortality, for children ages 2 to 3 – for instance, in our example of Nigeria, the increase of bed net usage have led to a decrease of the probability of a child dying of 0.87 percentage points.

We also find that the introduction of bed nets has a negative impact on fertility for younger and older women, whereas it has a positive effect for middle-aged women. The negative effect is mainly due to a decreased need for replacement children and precautionary child-bearing.

Although our paper explores the reduced-form effect of bed net on mortality, and of bed net on 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.

The fertility effects we find, at least partially, are driven by the change in infant mortality we uncover. However, we cannot disentangle the pure effect of mortality on fertility from the effect of the cost of child-bearing which changes with the introduction of

bed nets. Neither can we disentangle the effect of movements along the quality-quantity frontier which come from improved child health and educational outcomes on fertility. Given the important theoretical relationship between mortality and fertility in theories of fertility transitions, we hope that this paper may spur further interest in research on this question.

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.

Our findings support the contention that erosion of international funding for malaria control, specifically of ITNs, could not only cause an increase in child mortality due to malaria, but also lead to higher fertility for younger and older women. Inasmuch as higher fertility is associated with lower education achievement, higher maternal mortality, and lower income per capita, this reduction in funding could be especially detrimental – not only for the children who die, but also for the families which are left behind.

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Table 1. List of Surveys

Country	# of surveys	# of regions	Survey sources
Angola	3	18	MICS01, MIS06-07, MIS11
Benin	3	6	DHS01, DHS06-07, DHS11-12
Burkina Faso	3	13	DHS03, MICS06, DHS10
Burundi	4	17	MICS00, MICS05, DHS10, MIS12
Cameroon	4	10	MICS00, DHS04, MICS06, DHS11
Congo(Brazza)	2	11	DHS05, DHS11-12
Congo(DRC)	3	9	MICS01, DHS07, MICS10
Côte d'Ivoire	3	9	MICS00, AIS05, DHS11-12
Ghana	3	10	DHS03, MICS06, DHS08
Guinea	2	5	DHS05, DHS12
Kenya	3	8	MICS00, DHS03, DHS08
Lesotho	2	10	DHS04, DHS09
Liberia	3	15	DHS06-07, MIS08-09, MIS11
Madagascar	4	6	DHS03-04, DHS08-09, MIS11, MIS13
Malawi	5	26	DHS00, DHS04, MICS06, DHS10, MIS12
Mali	2	9	DHS01, DHS06
Mozambique	3	10	DHS03-04, MICS08, DHS11
Namibia	2	13	DHS00, DHS06-07
Niger	2	8	MICS00, DHS06
Nigeria	4	37	DHS03, MICS07, DHS08, MIS10
Rwanda	5	10	MICS00, DHS00, DHS05, DHS(I)07-08, DHS10
Senegal	5	11	MICS00, DHS05, MIS06, MIS08-09, DHS10-11
Sierra Leone	3	4	MICS00, MICS05, DHS08
Swaziland	3	4	MICS00, DHS06-07, MICS10
Tanzania	4	25	DHS04-05, AIS07-08, DHS09-10, AIS11-12
Uganda	3	4	DHS06, MIS09-10, DHS11
Zambia	3	9	MICS99, DHS01-02, DHS07
Zimbabwe	3	10	DHS05-06, MICS06, DHS10-11

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. Evolution of Bed Net Usage and Ownership by Country and Survey Year

Country	Net	Evolution in percentage	
		1999-2005	2006-2013
Angola	Usage	10.2(01)	22.5(06-07), 26.6(11)
	Ownership		35.1(06-07), 36.1(11)
Benin	Usage	35.2(01)	45.8(06), 73.5(11-12)
	Ownership	40.2(01)	56.4(06), 85.6(11-12)
Burkina Faso	Usage	17.8(03)	16.3(06), 54.7(10)
	Ownership	38.4(03)	49.4(06), 67.6(10)
Burundi	Usage	2.7(00), 17.8(05)	48.1(10), 54.8(12)
	Ownership	17.6(05)	54.9(10), 64.0(12)
Cameroon	Usage	13.5(00), 11.4(04)	27.9(06), 29.1(11)
	Ownership	19.5(04)	32.2(06), 51.1(11)
Congo(Brazza)	Usage	66.2(05)	77.3(11-12)
	Ownership	77.1(05)	81.2(11-12)
Congo(DRC)	Usage	13.7(01)	22.0(07), 38.7(10)
	Ownership		30.9(07), 52.8(10)
Côte d'Ivoire	Usage	9.3(00)	17.5(06), 39.9(11-12)
	Ownership		28.4(06), 72.4(11-12)
Ghana	Usage	15.8(03)	36.6(06), 42.7(08)
	Ownership	19.9(03)	33.5(06), 48.0(08)
Kenya	Usage	15.7(00), 17.6(03)	54.0(08-09)
	Ownership	24.9(03)	64.4(08-09)
Liberia	Usage		30.7(08-09), 38.4(11)
	Ownership		55.5(08-09), 53.5(11)
Madagascar	Usage	39.9(03-04)	50.8(08-09), 77.2(11), 56.6(13)
	Ownership	41.6(03-04)	64.9(08-09), 81.5(11), 65.8(13)
Malawi	Usage	3.6(00)	30.6(06),46.6(10),59.5(12)
	Ownership	17.8(00)	51.4(06), 67.6.5(10), 60.2(12)
Mali	Usage	42.5(01)	40.3(06)
	Ownership	55.2(01)	66.6(06)
Mozambique	Usage		41.3(08), 37.4(11)
	Ownership		57.2(11)
Niger	Usage	21.6(00)	19.1(06)
	Ownership		72.4(06)
Nigeria	Usage	5.5(03)	4.3(07), 12.3(08), 31.8(10)
	Ownership	10.6(03)	4.8(07), 18.3(08), 45.3(10)
Rwanda	Usage	16.4(05)	60.8(07-08), 69.7(10)
	Ownership	9.2(00), 19.8(05)	60.0(07-08), 82.6(10)
Senegal	Usage	17.2(02), 17.6(05)	30.4(06), 36.5(08-09), 46.3(10-11)
	Ownership	45.8(05)	62.6(06), 76.0(08-09), 82.0(10-11)
Sierra Leone	Usage	14.0(00), 20.5(05)	28.9(08)
	Ownership	20.2(05)	40.6(08)
Swaziland	Usage	0.1(00)	0.9(06-07), 1.8(10)
	Ownership		6.7(06-07), 11.1(10)
Tanzania	Usage	30.5(04-05)	41.8(07-08), 68.9(09-10), 74.1(11-12)
	Ownership	46.2(04-05)	61.8(07-08), 76.7(09-10), 93.4(11-12)
Uganda	Usage		20.3(06), 40.7(09-10), 52.3(11)
	Ownership		33.6(06), 59.2(09-10), 74.4(11)
Zambia	Usage	7.8(99), 15.7(01-02)	34.4(07)
	Ownership	27.2(01-02)	66.6(07)
Zimbabwe	Usage		6.3(05-06), 22.2(09), 13.6(10-11)
	Ownership		20.5(05-06), 40.8(10-11)

Table 3. Descriptive Statistics

Variable	Mean	S.d.	n
Net usage	0.261	0,211	190 531
Any net ownership	0.429	0.269	171 457
PfPR 2010	0.3388	0.1937	190 531
Malaria ecology	0.3462	0.1654	190 531
Risk exposure	6.035	3.698	190 531
Mom age at birth/10	2.631	0.686	190 531
Birth interval under 24 months	0.190	0.392	190 531
Male	0.507	0.499	190 531
Trend	7.679	3.493	190 531

Table 4. Nets and Mortality (Individual-Level Model)

Sample	(1)	(2)	(3)	(4)	(5)
	Number of months that the child was born before the survey				
	0-12	13-24	25-36	37-48	49-60
Mean death	0.0539	0.0301	0.0164	0.0086	0.0059
	(0.2259)	(0.1710)	(0.1272)	(0.0922)	(0.0765)
Panel A. Any net usage					
Net usage	0.0086	-0.0189***	-0.0213***	-0.0073***	-0.0050***
	(0.0057)	(0.0048)	(0.0031)	(0.0021)	(0.0017)
Malaria ecology ×	-0.045	-0.0366	-0.063***	-0.0090	-0.0029
Net usage	(0.0311)	(0.0255)	(0.0169)	(0.0117)	(0.0093)
Observations	234,570	198,544	212,889	209,045	204,959
R-squared	0.015	0.012	0.012	0.006	0.004
Panel B. ITN usage					
ITN usage	-0.0063	-0.0320***	-0.0381***	-0.0165***	-0.0126***
	(0.0074)	(0.0059)	(0.0031)	(0.0024)	(0.0020)
Malaria ecology ×	-0.023	-0.0060	-0.0460***	-0.0033	-0.0049
ITN usage	(0.0312)	(0.0251)	(0.0163)	(0.0114)	(0.0094)
Observations	173,442	147,627	159,846	157,541	155,488
R-squared	0.014	0.014	0.013	0.007	0.005
Panel C. Any net ownership					
Net ownership	-0.0031	-0.0205***	-0.0214***	-0.0100***	-0.0051***
	(0.0059)	(0.0047)	(0.0029)	(0.0020)	(0.0016)
Malaria ecology ×	-0.0068	-0.0018	-0.0391***	0.0017	0.0006
Net ownership	(0.0300)	(0.0245)	(0.0159)	(0.0112)	(0.0088)
Observations	205,801	176,100	191,707	189,715	185,914
R-squared	0.015	0.013	0.011	0.006	0.004

Notes. Net usage, ITN usage, and net ownership and malaria ecology are measured at the region level. We also control for risk exposure, mothers age at birth, birth interval, gender, region fixed effects, and country time trends.
Robust standard errors in parentheses.
*** p<0.01, ** p<0.05, * p<0.1.

Table 5. Nets and Fertility (Individual-Level Model)

	Sample					
	Woman's age at the date of the survey					
	15-19	20-24	25-29	30-34	35-39	40-44
Mean birth	0.1036	0.2490	0.2622	0.2318	0.1765	0.0896
	(0.3406)	(0.4299)	(0.4398)	(0.4220)	(0.3804)	(0.2856)
Panel A. Any net usage						
Net usage	-0.0046	-0.0017	-0.0300	-0.0053	0.0002	0.0028
	(0.0120)	(0.0179)	(0.0193)	(0.0207)	(0.0204)	(0.0172)
Malaria ecology ×	-0.135**	-0.135	0.0382	0.111	-0.242**	-0.0682
Net usage	(0.0570)	(0.0869)	(0.0945)	(0.101)	(0.0999)	(0.0813)
Observations	134,738	119,667	108,857	85,457	71,760	55,457
R-squared	0.029	0.024	0.017	0.020	0.022	0.019
Panel B. ITN usage						
ITN usage	-0.0043	-0.0210	-0.0317	0.0225	-0.0120	-0.00714
	(0.0127)	(0.0192)	(0.0206)	(0.0221)	(0.0217)	(0.0184)
Malaria ecology ×	-0.0713	-0.133*	0.0879	0.117	-0.242***	-0.101
ITN usage	(0.0521)	(0.0798)	(0.0869)	(0.0923)	(0.0911)	(0.0741)
Observations	128,046	113,794	103,339	81,252	68,091	52,674
R-squared	0.029	0.023	0.017	0.020	0.022	0.019
Panel C. Any net ownership						
Net ownership	-0.0011	-0.0209	-0.0224	-0.0176	0.0004	-0.00487
	(0.0110)	(0.0163)	(0.0174)	(0.0188)	(0.0184)	(0.0157)
Malaria ecology ×	-0.0720	-0.0229	0.119	0.157*	-0.139	-0.0393
Net ownership	(0.0517)	(0.0787)	(0.0849)	(0.0917)	(0.0899)	(0.0747)
Observations	135,370	120,476	109,558	86,096	72,348	55,853
R-squared	0.028	0.023	0.017	0.020	0.022	0.019

Notes. Net usage, ITN usage, and net ownership and malaria ecology are measured at the region level. We also control for region fixed effects and country specific time trends. Robust standard errors in parentheses.
*** p<0.01, ** p<0.05, * p<0.1.

Table 6. Falsification Tests

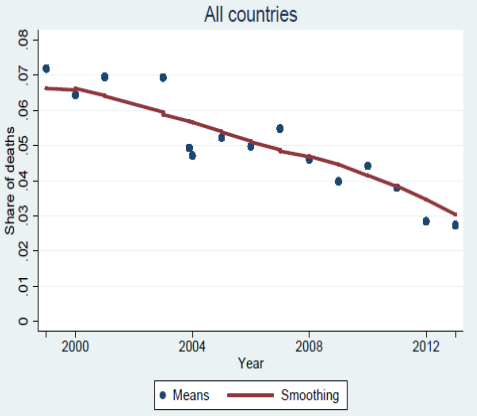
	Antenatal visits and vaccines		
	(1)	(2)	(3)
	Visits	Polio at birth	Full Vaccination
Mean	3.601	0.587	0.241
	(2.847)	(0.492)	(0.428)
Panel A. Any net usage			
Net usage	0.333***	0.0438**	0.132***
	(0.120)	(0.0192)	(0.0173)
Malaria ecology ×	-1.126**	-0.220**	-2.037***
Net usage	(0.543)	(0.0926)	(0.0834)
Observations	104,248	111,636	113,953
R-squared	0.298	0.221	0.140
Panel B. ITN usage			
ITN usage	0.228	0.0872***	0.186***
	(0.153)	(0.0279)	(0.0237)
Malaria ecology ×	-1.527***	-0.0544	-1.068***
ITN usage	(0.518)	(0.0960)	(0.0817)
Observations	88,614	89,137	90,917
R-squared	0.269	0.207	0.107
Panel C. Any net ownership			
Net ownership	0.622***	0.126***	0.237***
	(0.117)	(0.0200)	(0.0176)
Malaria ecology ×	-1.733***	-0.595***	-1.753***
Net ownership	(0.545)	(0.0958)	(0.0840)
Observations	100,562	106,082	107,483
R-squared	0.301	0.214	0.125

Notes. Visits is the number of antenatal care visits for that birth. Polio0 is whether the child received a polio vaccine at birth or within 18 days. Fully vaccinated is whether the child had all vaccines recommended by WHO (1 BCG, 1 measles, 3 polio, and 3 DPT). Net usage, ITN usage, and net ownership and malaria ecology are measured at the region level. We also control for mother's age, gender, region fixed effects and country specific time trends.
Standard errors in parentheses.
*** p<0.01, ** p<0.05, * p<0.1.

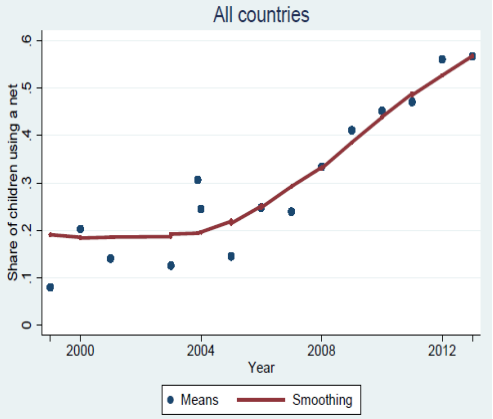
Figure 1. Trends in Net Usage, Mortality, and Fertility

Evolution of Mortality and Bed Net Usage in sub-Saharan Africa (1999-2013) (from DHS)

Infant Mortality Rate

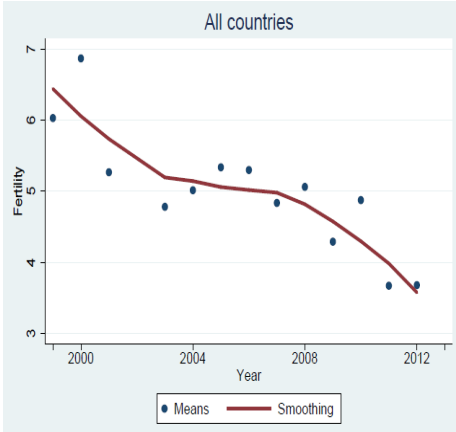


Bed Net Usage

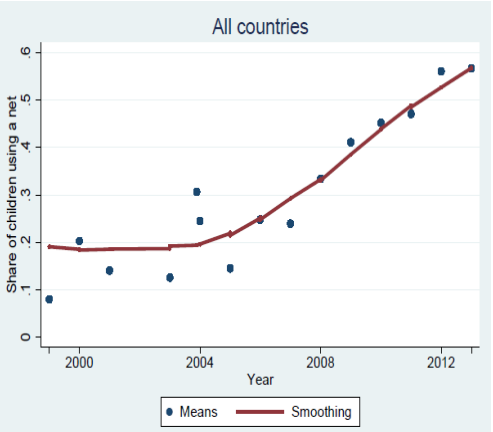


Evolution of Fertility and Bed Net Usage in sub-Saharan Africa (1999-2013) (from DHS)

Fertility Rate



Bed Net Usage



Appendix A. Details on the Theoretical Model

Since the mean of the binomial is nq,

$$U(N) = U\left[nq\left(m\right)\right] + \left[N - nq\left(m\right)\right]U_N\left[nq\left(m\right)\right] + \frac{\left[N - nq\left(m\right)\right]^2}{2!}U_{NN}\left[nq\left(m\right)\right] + \frac{\left[N - nq\left(m\right)\right]^3}{3!}U_{NNN}\left[nq\left(m\right)\right] \quad (12)$$

From log utility, the partial derivatives are:

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

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

$$E\left(U\right) = U\left[nq\left(m\right)\right] + E\left\{\left[N - nq\left(m\right)\right]\frac{(1-\gamma)}{nq(m)}\right\} - E\left\{\frac{\left[N - nq\left(m\right)\right]^2}{2!}\frac{(1-\gamma)}{\left[nq(m)\right]^2}\right\} + E\left\{\frac{\left[N - nq\left(m\right)\right]^3}{3!}\frac{2\left(1-\gamma\right)}{\left[nq(m)\right]^3}\right\} \quad (13)$$

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 $E\left[N - nq(m)\right]^2 = nq(m)\left[1 - q(m)\right]$. Therefore, (6) can be rewritten as

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

which can also be rewritten as

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

Appendix B. The Effect of Climate on Malaria Prevalence

		PfPR
Avg. rain		0.024
		(2.73)**
Avg. rain	squared	-0.000
		(1.72)
Avg. rain	cubed	0.000
		(1.35)
Avg. rain	quartic	-0.000
		(1.23)
Avg. max. rain		-0.006
		(1.64)
Avg. max. rain	squared	0.000
		(1.37)
Avg. max. rain	cubed	-0.000
		(1.16)
Avg. max. rain	quartic	0.000
		(0.98)
Avg. min. rain		-0.014
		(2.48)*
Avg. min. rain	squared	0.001
		(1.20)
Avg. min. rain	cubed	-0.000
		(0.70)
Avg. min. rain	quartic	0.000
		(0.46)
Avg. temp		-33.693
		(3.62)**
Avg. temp	squared	2.081
		(3.53)**
Avg. temp	cubed	-0.057
		(3.43)**
Avg. temp	quartic	0.001
		(3.33)**
Avg. max. temp		21.673
		(3.74)**
Avg. max. temp	squared	-1.186
		(3.68)**
Avg. max. temp	cubed	0.029
		(3.61)**
Avg. max. temp	quartic	-0.000
		(3.53)**
Avg. min. temp		0.140
		(0.05)
Avg. min. temp	squared	0.016
		(0.07)
Avg. min. temp	cubed	-0.001
		(0.19)
Avg. min. temp	quartic	0.000
		(0.28)
Constant		52.635
		(1.76)
R^2		0.68
N		269

Notes. Average rain and temperature are measure in millimeters and Celsius, respectively. Averages are calculated by finding the average temperature for each month from 2000-2009, and then averaging over all the months. Average maximum (minimum) rain and temperature are calculated by first finding the month with maximum (minimum) of each year 2000-2009, then averaging these averages. PMP refers to our predicted malaria prevalence measure.

* $p<0.05$; ** $p<0.01$.