The impact of malaria control on infant mortality in Kenya

Article (Published Version)

The Impact of Malaria Control on Infant Mortality in Kenya

VIKRAM PATHANIA
University of Sussex

I. Introduction

Malaria is a leading cause of childhood morbidity and mortality worldwide. It is estimated to account for 732,000 deaths among children age 5 or less, or about 8% of all such deaths; the share in Africa is 16% (Black et al. 2010). The childhood burden of malaria also leaves a lasting adverse imprint on lives and economies (Gallup and Sachs 2001; Sachs and Malaney 2002; Bleakley 2010b; Lucas 2010).

There has been a rapid escalation of malaria control efforts in recent years. Funding committed by major global financiers of malaria control has increased more than ninefold from less than US$0.2 billion in 2004 to about US$1.8 billion in 2009 (WHO 2010). The Global Fund to Fight AIDS, Tuberculosis and Malaria (hereafter, Global Fund) has played a major role in this funding surge. Since its inception in 2002, the Global Fund dramatically increased funding levels for HIV/AIDS, TB, and malaria in the developing world. By 2009, the Global Fund had approved proposals worth US $19.2 billion, of which grants for malaria control account for US$5.7 billion (Global Fund 2010).

Kenya adopted a new national malaria strategy in 2001. The strategy emphasized the distribution of insecticide-treated bed nets (ITNs), in line with a large body of evidence that ITNs were effective in reducing malaria. In addition, it recommended selective indoor residual house spraying for epidemic control, a switch to a more effective and prompt treatment of malaria, and prophylactic treatment for pregnant women in endemic areas. Akin to the rest of sub-Saharan Africa, funding for malaria control in Kenya jumped in 2002 and showed major growth by 2005. Figure 1 charts the growth in funding for malaria control in Kenya.[1] The Global Fund, the Presidential Malaria

I would like to thank Chris Dye, Eline Korenromp, Abdisalan Mohamed Noor, and seminar participants at the London School of Economics and Royal Economic Society Meeting, 2013, for their valuable comments. I also gratefully acknowledge the advice of two anonymous referees who have helped improve the article immeasurably. Contact the author at vikram.pathania@gmail.com.

[1] The volatility in fig. 1 reflects the lumpy disbursements by major external donors.

Electronically published February 07, 2014

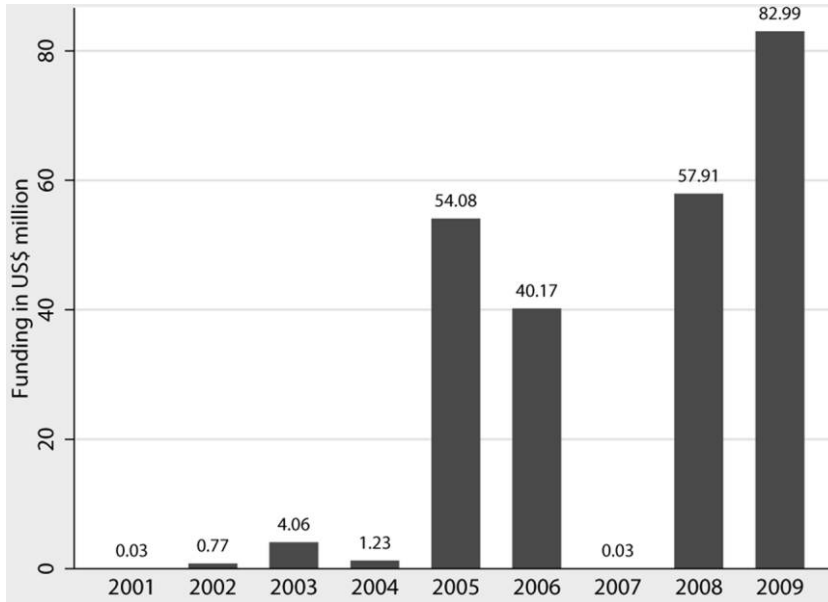

Figure 1. Annual malaria control funding in Kenya, 2001–9. Source: Annex 3, World Malaria Report (WHO 2010).

Initiative, and bilateral donors provided most of the increase in funding. Total funding committed to malaria control in Kenya in 2004–9 amounted to US $236.4 million, 66% of which was provided by the Global Fund (WHO 2010). Cumulative disbursements (actual monies transferred from the Global Fund to the Kenyan government) rose from US$4.6 million in 2004 to US $107 million in 2009.[2] A program review in 2009 found moderate progress in access to effective treatment, prophylaxis for pregnant women, and the use of indoor residual spraying. But it showed a striking increase in the ownership and usage of ITNs by Kenyan households. The proportion of children under 5 who used ITNs rose from 5% in 2003 to 39% in 2007 and 47% by 2008–9. In the malarious regions, ownership had grown to be in excess of 70%, and the proportion of children protected by ITNs was about 60%.

In this article, I investigate the impact of this rapid intensification of malaria control in Kenya. In particular, I use the quasi-experimental variation induced by preexisting geographical variation in malaria in Kenya coupled with a sharp escalation of the campaign to map out the impact of malaria control on infant mortality. I choose to study the impact in Kenya for two main reasons. First, it has geographically distinct regions with widely varying risk of ma-

[2] See http://www.theglobalfund.org/en/commitmentsdisbursements/, accessed on April 28, 2011.

laria. In particular, some regions have very high risk of malaria and would be expected to benefit greatly from enhanced malaria control activities, while other regions have very low risk of malaria and can usefully serve as controls. Second, there are two conveniently timed Demographic and Health Surveys (DHS 2003 and DHS 2008–9; see CBS, MOH, and ORC Macro 2004; KNBS and ICF Macro 2010, respectively) that permit estimation of trends in infant mortality before and after the intensification of malaria control. The latter survey is recent enough to allow detection of early impact.

I define 2004–8 as the postintensification (hereafter, "post") and 1993–2003 as the preintensification (hereafter, "pre") periods. I find large and significant reductions in postneonatal mortality (death in the first 1–11 months of life) in the post period in the malarious regions relative to the nonmalarious ones. Importantly, the pattern of reduction in infant mortality is consistent with a stylized fact about the burden of malaria in infancy: life-threatening malaria during infancy usually occurs in the later part of the first year of life, with very young infants enjoying significant clinical protection that is apparently maternally derived. I do not find evidence for similar preexisting differential trends in infant mortality before the post period; the estimate is robust to the inclusion of regional time trends. I also find no evidence of differential changes in maternal or infant care that could explain the sharper reduction in mortality in the malarious regions. There was a boost in funding for an HIV/AIDS program in the same period, but the findings are robust to controlling for any contemporaneous impact of the HIV/AIDS program on mortality. The preferred specification suggests that the renewed malaria campaign led to a fall of 33% in postneonatal mortality in malarious regions that had a mean postneonatal mortality rate of about 75 per 1,000 in the precampaign period.

This finding is consistent with those of several other studies based on admissions and outcomes data from hospitals and health centers in various African countries including Kenya. These studies have noted a dramatic reduction in malaria case load and deaths in parallel with the intensified campaign against malaria, while nonmalaria cases and deaths have remained stable or declined only slowly (Okiro et al. 2007; O'Meara et al. 2008; Otten et al. 2009; Snow and Marsh 2010; Aregawi et al. 2011).

This article adds to the existing literature on the efficacy of ITNs. While clinical trials have found that ITNs are effective against malaria, such trials are characterized by high coverage and close monitoring. This article suggests that ITNs have large health benefits in the context of routine program implementation with coverage rates of about 60% in malarious regions (for children under 5 who sleep under ITNs). The article also adds to the rapidly

growing set of studies assessing the impact of the recent intensification of malaria control. These studies tend to track changes in outcomes on the basis of patient data from select health facilities. Use of nationally representative survey data such as the DHS allows for a more comprehensive and credible mapping of malaria-related mortality trends as compared to data from health center records and vital statistics registries, which usually have poor coverage in developing country settings.

There has been a recent surge in the literature on the long-run benefits of historical malaria eradication campaigns (Bleakley 2010b; Cutler et al. 2010; Lucas 2010). This article extends this literature to the contemporary context: the renewed campaign against malaria since the early 2000s. It also differs from that literature in two important ways. First, it investigates the impact of contemporary malaria control that is centered on bed nets in contrast to earlier malaria control efforts that were centered on DDT spraying. Second, I focus explicitly on the impact on mortality since the relevant parasite species is *Plasmodium falciparum*, which causes more deadly malaria than *Plasmodium vivax*, which is common in most of the settings in that literature. Credible and timely assessment of the extent of mortality and morbidity reductions is critical for sustaining the recent momentum in malaria financing and program implementation.

II. Malaria in Kenya

Malaria is a leading cause of morbidity and mortality in Kenya. It accounts for about 30%–50% of all outpatient visits and 20% of hospital admissions. Pregnant women and children are particularly vulnerable; malaria is estimated to account for about 20% of deaths among children under 5.[3]

There is wide geographical variation in the risk of malaria in Kenya. Noor et al. (2009) map the malaria risk across Kenya on the basis of *Plasmodium falciparum* parasite rate (PfPR) data assembled through cross-sectional community-based surveys in 2,095 sites across the country from 1975 to 2009. The PfPR is a commonly used measure of malaria risk in the population: it is the proportion of the population that carries asexual blood stage parasites (Smith et al. 2007). The mapping shows that over 86% of the Kenyan population resides in areas with very low malaria risk (PfPR < 5%). Only Nyanza and the Western regions, both around Lake Victoria, have substantial risk of malaria. In Nyanza, over 40%, and in the Western region, about 50%, of the population resides in areas with a high risk of malaria (PfPR > 40%). In con-

[3] Malaria Fact Sheet from the Malaria Control Program of Kenya; see http://www.nmcp.or.ke/section.asp?ID=4.

TABLE 1
MALARIA RISK PFPR IN KENYAN REGIONS

	Distribution of Population by PfPR							PfPR*	HIV Rate†
	<.1%	.1%–1%	1%–5%	5%–10%	10%–20%	20%–40%	>40%	(%)	(%)
Central	99.1	.9	.0	.0	.0	.0	.0	.055	7.6
Coast	.3	28.2	68.0	.0	2.1	1.5	.0	2.96	6.6
Eastern	32.1	66.3	1.6	.0	.0	.0	.0	.429	6.2
Nairobi	100.0	.0	.0	.0	.0	.0	.0	.05	11.9
North Eastern	21.0	75.7	3.3	.0	.0	.0	.0	.526	0
Nyanza	.0	7.9	39.9	.0	.2	11.2	40.8	33.19	18.3
Rift Valley	26.7	51.7	21.2	.0	.2	.1	.1	1.064	6.9
Western	.0	.0	39.9	.0	.7	10.1	49.3	38.84	5.8

Sources. Montana, Neuman, and Mishra (2007) and Noor et al. (2009), table 4.
* Regional PfPR (*Plasmodium falciparum* parasite rate) calculated using the midpoints of the PfPR bands and the share of population in band in columns 1–7. For the >40% band, the midpoint is assumed to be 70%.
† Among women age 15–49, based on Demographic and Health Survey 2003.

trast, four other regions have a very low malaria risk. For instance, the PfPR for the entire population of Nairobi is less than 0.1%. Table 1 shows the estimated distribution of regional populations by malaria risk. On the basis of this distribution, I estimate the regional malaria risk by taking the weighted average of the midpoints of the malaria risk bands—the weights being the population share in each band.[4] Figure 2 maps this regional distribution of malaria risk.

A. Malaria Control in Kenya

By the late 1990s, there was a considerable body of evidence that the use of ITNs reduced childhood mortality in sub-Saharan Africa (see Lengeler [2004] for a review of several randomized controlled trials of ITNs). In 2001 Kenya adopted the National Malaria Strategy, 2001–2010 to combat malaria over the coming decade (Division of Malaria Control 2001); the strategy emphasized the increased use of ITNs. Before 2001, access to nets was limited to sales through the retail sector and localized distribution by some research and nongovernmental initiatives. In 2002, PSI Kenya received support from the UK Department for International Development to market partially subsidized ITNs across Kenya. This was the only major ITN distribution campaign between 2002 and 2004, and it operated through the retail sector. The nets were priced at US$4.70 in urban areas and at US$1.30 in rural areas. Starting in 2004, there were more ambitious campaigns to increase coverage by distributing heavily

[4] Since the >40% band is wide. I have checked the robustness of the findings to different assumptions about the rate in that band. The results can be accessed in the online appendix; the estimates are unchanged if I assume 60% or 80% instead of the 70% midpoint in the band.

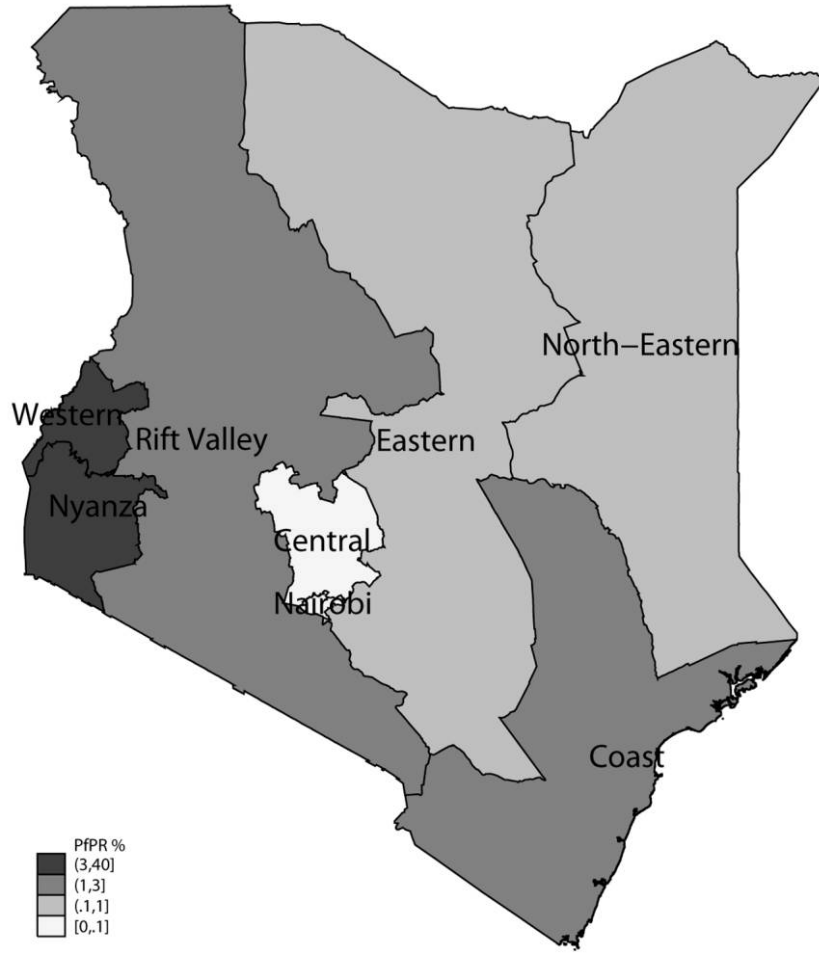

Figure 2. Regional distribution of *Plasmodium falciparum* malaria prevalence in Kenya. Source: Noor et al. (2009), table 4.

subsidized or free ITNs through multiple channels. PSI expanded its program by distributing heavily subsidized ITNs through maternal and child health clinics; these nets came bundled with insecticide retreatment tablets. These ITNs were branded with the Ministry of Health logo and priced at merely US$0.70. In 2004, Kenya also received a Global Fund grant to distribute 5 million long-lasting insecticide nets free of charge to children under 5 years of age. While regular ITNs are usually effective for about a year and a half, the long-lasting versions have a life span of between 3 and 5 years. The distribution took place in two phases in 2006 through health care facilities and mass vaccination campaign points (Noor et al. 2007). From 2002 to 2008, an estimated

16 million bed nets were distributed through various channels (KNBS and ICF Macro 2010).

Other major program components include vector control through indoor spraying of houses, prophylactic treatment for all pregnant women, and more prompt and effective treatment of malaria. The National Malaria Strategy recommended selective use of indoor residual spraying (IRS) in the early detection phase of epidemics in the highlands and arid areas. The strategy also aimed to deliver intermittent presumptive treatment in the second and third trimesters to all pregnant women residing in endemic areas as part of routine antenatal care. For case management, the target was to deliver malaria treatment to 60% of all fever cases within 24 hours of the onset of fever. In response to growing drug resistance, in 2006 Kenya switched its first-line treatment protocol for treating uncomplicated cases of malaria. Instead of being treated with sulfadoxine-pyremethamine, malaria cases were now to treated with the more effective artemisinin-based combination therapy that works well against drug-resistant strains.

Figure 3 illustrates the nationwide progress of the malaria control campaign. It is based on a detailed performance review of the malaria program in 2009 (Division of Malaria Control 2009). There has been only a modest increase in access to effective treatment and in the provision of intermittent

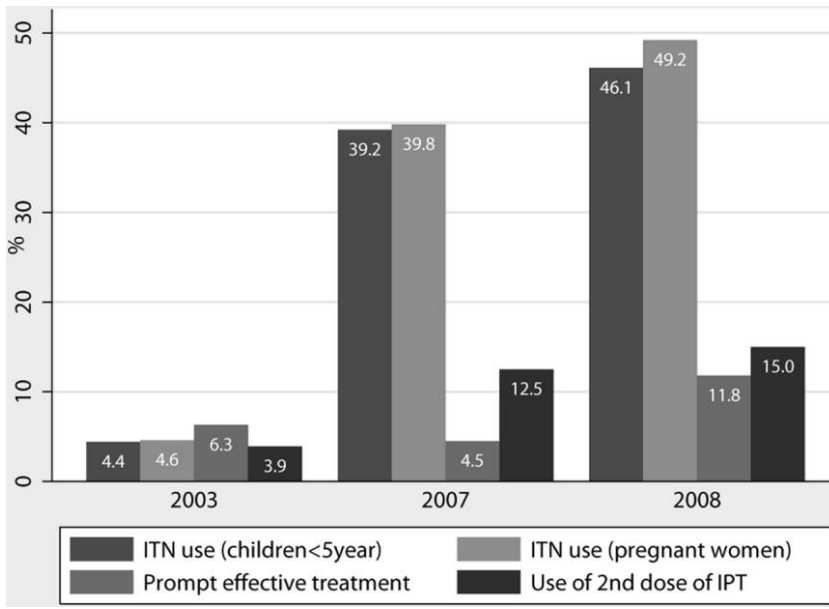

Figure 3. Performance of Kenya Malaria Control Program. Source: Kenya Malaria Program Performance Review (Division of Malaria Control 2009).

presumptive treatment to pregnant women. Access to prompt and effective treatment by artemisinin-based combination therapy has grown slowly due to a number of reasons. There have been frequent drug stock outs due to persistent distribution bottlenecks. Further, much of malaria treatment takes place in the informal sector, and many nonrecommended drugs including substandard antimalarial medicines can be purchased easily over the counter (Chuma et al. 2009). The provision of prophylaxis during pregnancy has been hampered by late presentation of pregnant women for antenatal care, drug stock outs, and a shortage of trained health workers.

The performance review also found that IRS had been implemented annually in epidemic-prone areas since 2005 with support from various donors. IRS campaigns were supported by the British Department for International Development in 2005–9, the Global Fund in 2006–9, and the President's Malaria Initiative in 2007–9. The intensity of IRS campaigns appears to have fluctuated, driven in part by an inconsistent flow of financing. For instance, in 2008, 63% of the targeted houses were sprayed, which afforded protection to about 3.1 million people. But in 2009, only 33% of the target was reached due to delays in donor fund disbursement. Other performance issues are related to lack of proper maps of areas for IRS spraying and poor quality control of pumps and insecticides. In Kenya, the areas west of the Rift Valley and arid and semiarid zones are prone to occasional malaria epidemics. In the current analysis, these areas are included in the nonmalarious regions. To the extent that the IRS campaigns were successful, they likely reduced mortality by mitigating epidemics. Since the nonmalarious regions effectively serve as controls in the analysis, this would create a downward bias in the estimate of the impact on malarious regions.

By far the biggest achievement of the program is the dramatic increase in the use of ITNs. Reliable ownership and usage data are available from the 2003 DHS, the 2007 Kenya Malaria Indicator Survey, and the 2008–9 DHS. Although ITN coverage was emphasized starting in 2001, survey evidence suggests that ITN coverage and usage really took off between 2003 and 2006–7. The proportion of households reporting ownership of at least one bed net of any kind rose from 22% in 2003 to 63% in 2007 before falling slightly to 61% in 2008–9. The proportion of households with at least one ITN rose from a mere 6% in 2003 to 48% in 2007 and 56% by 2008–9. The ITN coverage for children under 5, defined as the proportion of children under 5 who slept under an ITN the night preceding the survey, rose from 5% in 2003 to 39% in 2007 and 46% by 2008–9. Table 2 presents the regional increase in ITN ownership and usage between 2003 and 2008–9. In the highly malarious Nyanza and Western regions, household ownership of ITNs rose from 11.3% and 6.7% in 2003 to 76.5% and 71.4%, respectively, and the proportion of

TABLE 2
REGIONAL CHANGES IN INSECTICIDE-TREATED BED NET (ITN) OWNERSHIP AND USAGE (%)

	ITN Ownership*			ITN Usage (under 5)†		
	2003	2008–9	Δ	2003	2008–9	Δ
Nonmalarious:						
Central	3.4	32.7	29.3	3.9	35	31.1
Coast	10	66.3	56.3	7.5	56.9	49.4
Eastern	4.6	60.4	55.8	3.9	50.6	46.7
Nairobi	6.9	50.8	43.9	8.1	51.9	43.8
North Eastern	2.7	73.3	70.6	1.2	62.7	61.5
Rift Valley	3	41.4	38.4	2.5	29.5	27
Malarious:						
Nyanza	11.3	76.5	65.2	7.4	60.9	53.5
Western	6.7	71.4	64.7	4.8	55.4	50.6

Sources. Kenya Demographic and Health Surveys 2003 and 2008–9.
* Households surveyed who owned an ITN.
† Households with children under 5 years of age who slept under an ITN the night before the survey.

children under 5 who slept under bed nets rose from 7.4% and 4.8% to 60.9% and 55.4%, respectively, by 2008–9 (CBS, MOH, and ORC Macro 2004; KNBS and ICF Macro 2010). In the nonmalarious regions, ITN coverage for children under 5 also rose sharply, although the coverage achieved by 2008–9 was more varied, ranging from a low of 29.5% in the Rift Valley region to a high of 62.7% in the North Eastern region. Accordingly, in the following analysis the post intervention period is defined as starting in 2004 onward.[5]

B. Effect on Mortality in Early versus Late Infancy

When analyzing the impact of improved malaria control on infant mortality, it is useful to analyze separately the impact on early and late infancy because the morbidity and mortality burden of malaria appears to vary substantially between early infancy and the remainder of the first year of life. Snow et al. (1998) conduct a prospective study of hospital admissions of African infants across four sites with widely varying transmission intensity (including three sites in Kenya). They find evidence of significant clinical protection against severe malaria morbidity in the first 3 months of infancy. This protection does not appear to stem from a lower infection rate—neonates appear to get infected at roughly similar rates as older infants (Kitua et al. 1996). Instead protection in the first few weeks of life appears to be derived from Immu-

[5] Given that the malaria strategy was put in place in 2001, and that ITN distribution commenced in 2002, it is conceivable that the campaign began to have an impact before 2004. Hence, as a robustness check, I code 2001–3 as the buildup period, and 2004–8 as the post period, and track the relative changes in mortality in the malarious and nonmalarious regions in both those periods. As an additional check presented in the online appendix, I test and find that the estimates are robust to varying the starting year of the post period.

noglobulin G transferred through the placenta (McGregor 1986; Amaratunga et al. 2011). This is consistent with several older studies that suggest significant clinical protection against malaria in early infancy—congenital malaria is rare, and very young infants reportedly suffer only mild symptoms when infected. Severe, life-threatening malaria in infancy usually occurs during the latter part of the first year of life (Garnham 1949; Macdonald 1950; Colbourne and Edington 1954). In line with standard practice in public health and medical literature, I define neonatal (first month) and postneonatal (1–11 months) mortality indicators and analyze the impact of the campaign on each separately.[6] For the sake of completeness, I also present estimates of the differential impact of the campaign on various phases of infancy, that is, 0 months (neonatal as before), 1–2, 3–5, and 6–11 months.

III. Data and Method

The mortality indicators are calculated from the 1998, 2003, and the 2008–9 Kenya Demographic and Health Surveys. These are nationally representative surveys that collect detailed information on numerous population and health characteristics. The primary respondents are women of reproductive age (15–49). The women's questionnaire covers topics such as socioeconomic characteristics, complete reproductive history, antenatal and delivery care, immunization of infants, infant and child care, and childhood mortality.

Using the birth history and reported age at death in the DHS, I construct indicators of infant mortality (death within the first year of life), neonatal mortality (within the first month), and postneonatal (1–11 months) for each live birth in the sample.[7] The postneonatal and infant mortality indicators are defined only for cohorts that were born at least 1 year before the survey date (these

[6] Strictly speaking, neonatal mortality is defined as death of a live-born infant within the first 0–27 days of life. Postneonatal mortality is defined as the death of a live-born infant during 28–364 days of life. The two types of mortality are usually analyzed separately since most deaths occurring in the neonatal period are driven by events surrounding the prenatal period and the delivery, while most postneonatal deaths are associated with environmental factors that the infant is exposed to after delivery. See Rowley et al. (1994) for a full discussion. In the context of this analysis, it is worth noting that while the literature is not precise about the exact duration of protection, some of the studies above suggest that maternally derived protection against severe malaria extends up to the first 3 months of life. As a check, I have analyzed the impact on mortality in the 0–2- and 3–11-month age brackets, and the point estimate for 0–2-month mortality is essentially zero, while that for 3–11-month mortality is very close to that estimated for postneonatal mortality. Results are available from the author on request.

[7] The DHS contains information on the death of older children. I focus on infant mortality because it accounts for the bulk of child mortality. Also, a birth-cohort-based measure of children-under-5 mortality would restrict me to cohorts born in 2003 and earlier since the most recent survey data come from 2008 to 2009. However, I have analyzed children-under-5 mortality by counting the

indicators are undefined for infants who were alive and younger than 1 year at the time of the survey). Hence, I restrict the sample to births that took place at least 1 year before the survey date. Since the data related to birth history are retrospective, recall bias is a concern, particularly for the more distant births. I further restrict the analysis to recent births, occurring no earlier than 5 years of the date of the interview. I am constrained to using at least 5 years as the cutoff date for the following reason. If I set the cutoff at 4 years, then using the 2008–9 survey, I would be able to include births from 2004 to 2008 but not 2003. I cannot use data from the 2003 survey to construct postneonatal or infant morality for the 2003 birth cohort since that cohort would have been too young (i.e., less than 1 year old at the time of the survey). By setting the cutoff at 5 years, I can use information in the 2003 and the 2008–9 DHS to include births in 2003 and 1998, respectively. Accordingly, using the three surveys, I can construct mortality indicators for infants born between 1993 and 2008. The final sample has 13,897 births.[8] To mitigate any gender-specific recall bias, I include gender-specific birth order as fixed effects in all regressions.

The identification strategy relies on preexisting variation across Kenya in malaria risk. I estimate the following linear probability model for the mortality among infants born between 1993 and 2008:

$$m_{ijy} = \alpha + \gamma_j + \rho_y + \beta \times \text{post}_y \times \text{malaria}_j + \theta X_i + \varepsilon_{ijy}, \qquad (1)$$

where m_{ijy} is a mortality indicator for infant i born in region j in year y, and γ_j and ρ_y are region and year of birth fixed effects respectively. The "malaria" variable is the regional estimate of malaria risk as shown in table 1. The "post" dummy denotes years starting in 2004. This is the intervention period that witnessed a rapid intensification of malaria control. The covariates X_i include gender of the infant, maternal age, maternal education (in completed years), number of household members, and indicators for multiple birth and place of birth (rural or urban). Also included are fixed effects for religion and ethnicity of the head of the household, month of birth (to capture any seasonal variation), birth order, and gender-specific birth order.[9] The coefficient of in-

deaths of children age 0–4 as a proportion of the stock of all children age 0–4 alive in the sample at the start of the year. I find a negative and significant coefficient (the same holds for mortality in the 1–4-years age group).

[8] Even with a 5-year recall period, there remains a potential for bias due to selective migration of mothers. In the online appendix, I test and find that the estimates are robust to trimming the sample to the subset of mothers who were resident in the region for at least 5 years or more.

[9] The online appendix shows that the main findings are robust to the choice of both individual child-level and household-level covariates.

terest is β, which measures the differential change in infant mortality in the post period for a unit rise in malaria risk.[10]

Since the errors may be correlated with a region, I estimate cluster-robust standard errors (Moulton 1986, 1990). However, the cluster-robust standard errors are asymptotically valid only if the number of clusters is large. With a small number of clusters, the cluster-robust standard errors can be biased downward. Since there are only eight clusters (regions), I also calculate the two-tailed p-values using the wild cluster bootstrap-T method developed by Cameron, Gelbach, and Miller (2008).

IV. Results

Before presenting the regression results, it is instructive to examine the sample summary statistics in table 3. The table contains the mean and standard deviation of key variables for births in the pre and post periods by the level of malaria risk. Specifically, the table contrasts the nonmalarious regions (Central, Coastal, Eastern, Nairobi, North Eastern, and Rift Valley) that have low risk with the malarious regions (Nyanza and Western) that have very high risk. The reported p-values are from a test of difference in means between the malarious and the nonmalarious regions. A higher fraction of births in malarious regions occurs in rural areas and to slightly younger but more educated mothers.[11] The biggest difference between the malarious and the nonmalarious regions is in infant mortality, particularly in the pre period.

Table 4 previews the major finding of this article. The table displays the mean infant, neonatal, and postnatal mortality in the pre and post periods for the nonmalarious and the malarious regions. Looking first at the pre period, one finds that infant mortality is much higher in the malarious regions. Al-

[10] It is worth noting that the Nyanza and Western regions both have a similar level of very high malaria risk, while four of the remaining six regions (Central, Eastern, Nairobi, and North Eastern) have close to zero risk. This suggests an alternative and equivalent approach: a textbook difference-in-difference that would use a dummy to tag high malaria risk regions instead of a continuous regional malaria rate variable. The zero-risk regions serve as a control allowing us to account for any secular trends in infant mortality in Kenya. The virtue of this approach would be the ease of interpretation of the relevant coefficient—the prepost change in infant mortality in the high-risk regions relative to the low-risk ones. The downside is that the Coastal and the Rift Valley regions that have small pockets of high malaria risk and bigger pockets of moderate malaria risk would have to be excluded from the analysis. For the sake of completeness, I opt for the estimation strategy in eq. (1). But I have also estimated a standard difference-in-difference model in which I tag Nyanza and the Western regions as malarious and all six other regions as nonmalarious. The results are essentially similar and available in the online appendix.

[11] The religious and ethnic composition of the sample is not reported in table 3 for the sake of brevity. However, the regression analysis controls for differences in religious and ethnic composition across regions.

TABLE 3
SUMMARY STATISTICS FOR BIRTHS REPORTED IN KENYA DEMOGRAPHIC AND HEALTH SURVEYS 1998, 2003, AND 2008–9

	Pre (1993–2003)			Post (2004–8)		
	Nonmalarious*	Malarious†	*p*-Value	Nonmalarious*	Malarious†	*p*-Value
Male	.51	.50	.163	.51	.51	.642
	(.50)	(.50)		(.50)	(.50)	
Multiple	.03	.04	.091	.03	.04	.194
	(.17)	(.19)		(.17)	(.19)	
Rural	.77	.87	.000	.74	.82	.000
	(.42)	(.34)		(.44)	(.39)	
Mother's age‡	26.34	25.97	.014	26.51	25.46	.000
	(6.51)	(6.61)		(6.60)	(6.45)	
Mother's education§	6.26	6.90	.000	5.73	7.57	.000
	(4.18)	(3.28)		(4.69)	(2.87)	
Household size	6.19	5.86	.000	5.83	6.01	.021
	(2.79)	(2.32)		(2.43)	(2.56)	
Birth order	3.47	3.82	.000	3.44	3.49	.459
	(2.46)	(2.67)		(2.30)	(2.37)	
Infant mortality‖	58.66	103.74	.000	46.02	64.84	.007
	(235.01)	(304.98)		(209.55)	(246.32)	
Observations	6,465	2,698		3,238	1,496	

Note. Mean values, with standard errors in parentheses. Only includes births within no earlier than 5 years and no later than 1 year of the survey date. Reported *p*-values are for tests of difference in means between the malarious and the nonmalarious regions. Summary statistics for distribution of births by tribe and religion are available from the author on request.

* Central, Coastal, Eastern, Nairobi, North Eastern, and Rift Valley provinces.

† Nyanza and Western provinces.

‡ In years, at time of infant's birth.

§ In completed years, at time of survey.

‖ Per 1,000 live births.

most all of that excess mortality occurs among postneonates—the neonatal mortality is similar in both types of regions. Next, looking at the post period, one finds that infant mortality falls sharply in the malarious regions but declines only modestly in the nonmalarious regions. Further, the relative fall in infant mortality in the malarious regions is driven largely by a sharp reduction in postneonatal mortality.

I now turn to estimating the regression models to assess the magnitude and significance of these differential changes in mortality. The regressions sample includes live births from all eight regions. The results from estimating equation (1) are presented in table 5. For the sake of brevity, only select controls are reported.[12]

The regression results confirm the basic finding reported in table 4. There is a significant fall in postneonatal mortality in the malarious regions relative to

[12] The full set of results is available from the author on request.

TABLE 4
DIFFERENCE IN MEAN MORTALITY

	Pre (1993–2003)	Post (2004–8)	Δ
Infant mortality:			
Nonmalarious*	58.7	46.0	−12.6
	(235.0)	(209.6)	
Malarious†	103.7	64.8	−38.9
	(305.0)	(246.3)	−26.3
Neonatal mortality:			
Nonmalarious*	33.3	29.6	−3.7
	(179.5)	(169.6)	
Malarious†	27.8	26.7	−1.1
	(164.4)	(161.4)	2.6
Postneonatal mortality:			
Nonmalarious*	25.3	16.4	−8.9
	(157.1)	(126.9)	
Malarious†	76.0	38.1	−37.9
	(265.0)	(191.5)	−28.9

Note. Mean values of mortality per 1,000 live births (standard errors in parentheses) for births within 1–5 years of the survey date.
* Central, Coastal, Eastern, Nairobi, North Eastern, and Rift Valley provinces.
† Nyanza and Western provinces.

the nonmalarious regions in the post period. There is no such change for neonatal mortality; in fact the coefficient on the post × malaria interaction is positive albeit insignificant. The total change in infant mortality in the malarious versus nonmalarious regions (table 5, col. 3) is accordingly smaller than the relative fall in postneonatal mortality. The *p*-values from the wild cluster bootstrap-*T* method are reported in square brackets for the coefficient of interest. The estimated impact on postneonatal mortality remains significant at 5%.

V. Robustness Checks

The excess postneonatal mortality in the high-risk regions preintervention is consistent with malaria being a major source of infant mortality in the malarious regions, particularly among postneonatal infants who have lost at least some of the early infancy protection they originally enjoyed. Below, I further explore the pattern of the relative fall in mortality over the course of infancy. The sharper reduction in postneonatal mortality in the malarious regions postintervention suggests that intensified malaria control is having a substantial impact. However, a skeptic may plausibly contend that the relative fall in postneonatal mortality in the malarious areas is not due to the intensification of malaria control but instead due to a relative improvement in other determinants such as maternal and infant care or HIV/AIDS prevention and care or perhaps

TABLE 5
CHANGE IN INFANT, NEONATAL, AND POSTNEONATAL MORTALITY AND MALARIA PREVALENCE

	0–11 months (1)	0 months (2)	1–11 months (3)
Post × malaria	−.00078	.00015	−.00094**
	(.00046)	(.00019)	(.00034)
	[.332]	[.557]	[.041]
Male	.01450***	.00810**	.00640*
	(.00342)	(.00261)	(.00321)
Multiple birth	.15876***	.13614***	.02262**
	(.04271)	(.03823)	(.00942)
Rural	−.00007	.00145	−.00152
	(.00913)	(.00467)	(.00619)
Household size[a]	−.00773***	−.00328***	−.00444*
	(.00192)	(.00049)	(.00200)
Mother's age[b]	−.00046	−.00004	−.00041
	(.00047)	(.00037)	(.00028)
Mother's education[c]	−.00318***	−.00142**	−.00176***
	(.00053)	(.00043)	(.00049)
Constant	.11882***	.06580***	.05302**
	(.02005)	(.01134)	(.01727)
R^2	.04408	.03522	.03327

Note. Ordinary least squares regression coefficients; other covariates include fixed effects for birth order, gender-specific birth order, religion, ethnicity (tribe), month of birth, year of birth, and region of birth. Robust standard errors clustered on region of birth in parentheses. Sample includes births from 1993 to 2008, with "post" defined as years after 2003. Only includes births that took place within 1–5 years of the survey date. "Malaria" is the regional *Plasmodium falciparum* parasite rate. Square brackets contain two-tailed *p*-values for the post × malaria coefficient based on the wild cluster bootstrap-*T* method. $N = 13{,}897$.

[a] At time of the survey.

[b] In years, at time of birth.

[c] In completed years, at time of survey.

* $p < .1$.

** $p < .05$.

*** $p < .01$.

preexisting differential trends; that is, mortality was already falling faster in the malarious regions. I present results from a series of robustness checks that address these possibilities.

A. Effect on Mortality in Early versus Late Infancy

To track the relative change in mortality over the course of infancy, I construct mortality indicators for the following ages: 0 months (neonatal as before), 1–2, 3–5, and 6–11 months. For each of those indicators, I estimate equation (1). The coefficients on post × malaria are plotted in figure 4. It is evident that the relative postintervention fall in mortality in the malarious regions is entirely concentrated in the postneonatal period (3–5 and 6–11 months). This differential pattern in the change in mortality over the course of the first year of life is strongly suggestive that it is improved malaria control that is driving the reduction in mortality in the malarious regions.

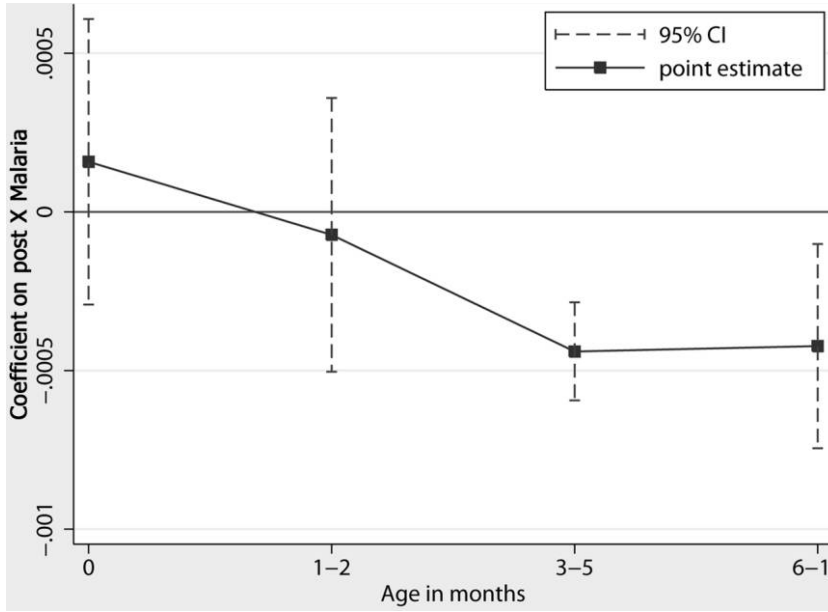

Figure 4. Effect on mortality at different ages in infancy. Sample of births from 1993 to 2008; "post" denotes years starting in 2004.

B. Preexisting Trends in Malarious versus Nonmalarious Regions

Could preexisting trends be driving the observed drop in postneonatal mortality in the malarious regions? To address this possibility, I include region-specific time trends in equation (1) so that the coefficient on post × malaria is now estimated from the variation around regional time trends. The results are displayed in column 2 of table 6. To facilitate comparison, column 1 reproduces the results from table 5. The estimated impact on postneonatal mortality is robust to inclusion of regional trends. Note though that the estimate for neonatal mortality becomes positive and significant, suggesting that neonatal mortality had been dropping in the malarious regions in the pre period but reversed course in the post period (relative to the nonmalarious regions). I also check for possible mean reversion by including the average level of the dependent variable for early birth cohorts in each region, interacted with the post dummy. The results are displayed in table 6 column 3. The estimated impact on postneonatal mortality remains negative and significant, although its magnitude is muted. However, this last result should be interpreted with some caution. If the high level of risk in the malarious regions was keeping postneonatal mortality artificially high in those regions, the post × regional average term could be soaking up some of the malaria effect that would mute the point estimate on post × malaria.

TABLE 6
PREEXISTING REGIONAL TRENDS IN MORTALITY

	Baseline: Table 5 (1)	Regional Trends[a] (2)	Mean Reversion (3)
Dependent variable—infant mortality:			
Post × malaria	−.00078	.00004	−.00008
	(.00046)	(.00021)	(.00025)
	[.332]	[.759]	[.798]
Post × regional average of dependent variable[b]			−.49298**
			(.19461)
R^2	.04408	.04677	.04495
Dependent variable—neonatal mortality:			
Post × malaria	.00015	.00070**	.00021
	(.00019)	(.00021)	(.00020)
	[.557]	[.080]	[.420]
Post × regional average of dependent variable[b]			−.88321
			(.51164)
R^2	.03522	.03727	.03591
Dependent variable—postneonatal mortality:			
Post × malaria	−.00094**	−.00066**	−.00031*
	(.00034)	(.00024)	(.00016)
	[.041]	[.070]	[.212]
Post × regional average of dependent variable[b]			−.46249***
			(.09446)
R^2	.03327	.03424	.03372

Note. Ordinary least squares regression coefficients; covariates include gender, multiple birth indicator, rural residence indicator, mother's age, mother's education, as well as fixed effects for birth order, gender-specific birth order, religion, ethnicity (tribe), month of birth, year of birth, and region of birth. Robust standard errors clustered on region of birth in parentheses. Sample includes births from 1993 to 2008, with "post" defined as years after 2003. Only includes births that took place within 1–5 years of the survey date. "Malaria" is the regional *Plasmodium falciparum* parasite rate. Square brackets contain two-tailed *p*-values for the post × malaria coefficient based on the wild cluster bootstrap-*T* method. $N = 13{,}897$.

[a] Regressions include a region-specific linear time trend.

[b] Mean reversion specification includes an interaction of post with the regional average of the relevant mortality measure for 1993–96.

* $p < .1$.

** $p < .05$.

*** $p < .01$.

Finally, I use a more flexible specification that separately estimates the relative change in infant mortality in the malarious regions during the campaign buildup (2002–3) and the post period (2004–8). The choice of 2004–8 as postcampaign was motivated by the observation that ITN usage appeared to take off after 2003. However, the National Malaria Strategy was adopted in 2001, and ITN distribution (although only modestly subsidized and only through the retail sector) commenced in 2002. The results are displayed in table 7. There appears to have been no change in postneonatal mortality during the buildup, and the estimated impact during the post period remains essentially unchanged.

TABLE 7

CHANGE IN INFANT, NEONATAL, AND POSTNEONATAL MORTALITY DURING CAMPAIGN BUILDUP (2002–3) AND THE POST PERIOD (2004–8)

	0–11 months	0 months	1–11 months
Buildup × malaria[a]	.00049	.00050	−.00000
	(.00105)	(.00054)	(.00055)
Post × malaria[a]	−.00074	.00020	−.00094**
	(.00042)	(.00021)	(.00030)
	[.357]	[.388]	[.036]
Male	.01452***	.00811**	.00640*
	(.00343)	(.00261)	(.00322)
Multiple birth	.15848***	.13585***	.02262**
	(.04225)	(.03804)	(.00913)
Rural	.00004	.00156	−.00152
	(.00928)	(.00473)	(.00627)
Household size[b]	−.00773***	−.00329***	−.00444*
	(.00192)	(.00049)	(.00200)
Mother's age[c]	−.00046	−.00004	−.00041
	(.00047)	(.00037)	(.00028)
Mother's education[d]	−.00318***	−.00142**	−.00176***
	(.00053)	(.00044)	(.00049)
Constant	.11897***	.06595***	.05302**
	(.02007)	(.01138)	(.01728)
R^2	.04412	.03531	.03327

Note. Ordinary least squares regression coefficients; other covariates include fixed effects for birth order, gender-specific birth order, religion, ethnicity (tribe), month of birth, year of birth, and region of birth. Robust standard errors clustered on region of birth in parentheses. Sample includes births from 1993 to 2008. Only includes births that took place within 1–5 years of the survey date. "Malaria" is the regional *Plasmodium falciparum* parasite rate. Square brackets contain two-tailed *p*-values for the post × malaria coefficient based on the wild cluster bootstrap-*T* method. $N = 13{,}897$.

[a] Buildup is defined as 2002–3, while post is defined as 2004–8.

[b] At time of the survey.

[c] In years, at time of birth.

[d] In completed years, at time of survey.

* $p < .1$.

** $p < .05$.

*** $p < .01$.

C. *Contemporaneous Programs*

1. Trends in Maternal and Infant care

It is possible that the differential trends in mortality in the malarious regions are being driven not by intensified malaria control but by relative improvements in maternal and infant care in those regions. The DHS data contain useful information on maternal and infant care indicators for births in the 5 years preceding the survey. Table 8 shows the estimates of differential changes in various measures of antenatal care—one or more antenatal visits to health facilities, receiving iron supplements and tetanus typhoid injections, and delivery at a health facility. There appears to be no relative change in the likelihood of receiving antenatal care between the two types of regions—three of the four coefficients on post × malaria are small and statistically insignificant. If any-

TABLE 8
CONCURRENT CHANGES IN ANTENATAL CARE

	Antenatal Care?[a]	Iron Supplement?	Tetanus Injection?	Delivery at Health Facility?[b]
Post × malaria	−.00069	−.00309	−.00064	−.00135*
	(.00086)	(.00171)	(.00083)	(.00060)
Male	.00539	.00249	.00586	.01820
	(.00588)	(.00397)	(.01053)	(.01230)
Multiple birth	.01231	−.04680	.05166***	.10166**
	(.02786)	(.03854)	(.01287)	(.03015)
Rural	−.03776	−.03115**	−.04471*	−.24491***
	(.02317)	(.01061)	(.02013)	(.02949)
Household size[c]	.00103	−.00388	.00037	−.00073
	(.00239)	(.00274)	(.00265)	(.00295)
Mother's age	.00022	.00116	.00055	.00989***
	(.00105)	(.00092)	(.00153)	(.00213)
Mother's education[c]	.01031***	.01218**	.00796**	.03175***
	(.00085)	(.00351)	(.00283)	(.00243)
Constant	.87294***	.66448***	.80367***	.37476**
	(.07378)	(.08070)	(.10331)	(.14061)
Observations[d]	7,632	5,443	7,737	11,650
R^2	.18572	.12215	.13222	.28108

Note. Ordinary least squares regression coefficients; other covariates include fixed effects for birth order, gender-specific birth order, religion, ethnicity (tribe), month of birth, year of birth, and region of birth. Robust standard errors clustered on region of birth in parentheses. Sample includes births from 1993 to 2008, with "post" defined as years after 2003. Only includes births that took place within 1–5 years of the survey date. "Malaria" is the regional *Plasmodium falciparum* parasite rate.

[a] Indicator for one or more antenatal visits to a health facility.

[b] Public or private health facility.

[c] At the time of the survey.

[d] Limited to births for which the dependent variable is recorded.

* $p < .1$.

** $p < .05$.

*** $p < .01$.

thing, antenatal care in the malarious regions may have worsened relative to the low-risk regions; the coefficient for delivery at a health facility is negative and marginally significant.[13]

Table 9 shows the results of regressions estimating the relative changes in immunization in the malarious versus nonmalarious regions. I investigate the differential changes in the likelihood of receiving each of the several different vaccines that constitute the immunization schedule in the first year. Immunization is a useful indicator of infant care not merely for any protection it confers on the infant in the first year of life but because it proxies for access to and usage of health services. All the coefficients on post × malaria are in-

[13] The reader may notice that the sample size in table 8 varies across the columns and is in some cases markedly less than the sample size in table 5. This is due to missing data for antenatal care indicators. The key findings remain essentially unchanged if the mortality regressions are run only for the subset of women for whom antenatal care data are available. These results are available on request.

TABLE 9
CONCURRENT CHANGES IN IMMUNIZATION RATES

	BCG	Polio 1	Polio 2	Polio 3	DPT 1	DPT 2	DPT 3	Measles
Post × malaria	−.00139	−.00092	−.00048	.00021	−.00092	−.00049	.00015	.00044
	(.00149)	(.00097)	(.00120)	(.00110)	(.00139)	(.00145)	(.00094)	(.00115)
Male	.00389	.00811	.00632	.01097	.00601	.00866	.00943	.01160
	(.00493)	(.00662)	(.00499)	(.00943)	(.00679)	(.00712)	(.00654)	(.00708)
Multiple birth	−.00157	−.00262	−.02249	.01356	−.00223	−.00130	.02031	.03697
	(.01849)	(.01556)	(.02217)	(.02798)	(.01875)	(.02726)	(.03939)	(.03637)
Rural	−.01802*	.00863	.01880	.08015***	−.01161	−.00449	.01555	−.03852*
	(.00780)	(.01094)	(.01340)	(.01525)	(.00702)	(.02238)	(.03290)	(.01878)
Household size[a]	.00164	.00151**	.00296**	.00349***	.00071	.00331**	.00581***	.00462***
	(.00122)	(.00061)	(.00093)	(.00082)	(.00112)	(.00119)	(.00153)	(.00096)
Mother's age	.00081	.00007	.00092	−.00005	.00006	.00045	.00135	.00329**
	(.00088)	(.00077)	(.00094)	(.00137)	(.00071)	(.00065)	(.00124)	(.00127)
Mother's education[a]	.00574***	.00513***	.00857***	.00742***	.00597***	.00973***	.01261***	.01323***
	(.00101)	(.00072)	(.00143)	(.00183)	(.00095)	(.00150)	(.00196)	(.00182)
Constant	.86804***	.89370***	.84673***	.46765***	.88906***	.78032***	.62753***	.82301***
	(.06430)	(.04133)	(.06077)	(.09648)	(.05019)	(.08657)	(.14440)	(.07601)
Observations[b]	10,782	10,798	10,795	10,795	10,782	10,778	10,778	10,770
R^2	.13007	.10031	.11195	.08290	.13654	.14412	.14023	.11232

Note. Ordinary least squares regression coefficients; regressions include fixed effects for birth order, gender-specific birth order, religion, ethnicity (tribe), month of birth, year of birth, and region of birth. Robust standard errors clustered on region of birth in parentheses. Sample includes births from 1993 to 2008, with "post" defined as years after 2003. Only includes births that took place within 1–5 years of the survey date. "Malaria" is the regional *Plasmodium falciparum* parasite rate. Immunization schedule for Kenya: BCG (Bacille Calmette-Guérin) is given at birth; the oral polio vaccine is given at birth, 6 weeks, and 10 weeks, respectively; DPT (diphtheria, pertussis, and tetanus) 1, 2, and 3 are given at 6, 10, and 14 weeks, respectively; and measles is given at 9 months.

[a] At the time of the survey.

[b] Limited to births for which the dependent variable is recorded.

* $p < .1$.

** $p < .05$.

*** $p < .01$.

significant. There is no evidence of any systematic changes in immunization between malarious and nonmalarious regions; in particular the malarious regions are not doing any better in the post period.

2. HIV/AIDS

While malaria has traditionally been a major cause of infant mortality in Africa, HIV has recently emerged as another big contributor (Newell et al. 2004). HIV prevalence in Kenya overlaps to some degree with malaria risk; for example, the Nyanza region, which has high malaria risk, also has the highest HIV prevalence. The Global Fund, which has provided much of the financial support for intensified malaria control in the post period, also funded a major concurrent expansion of HIV prevention and treatment. Early retroviral therapy has been shown to reduce mortality among infected infants by about

TABLE 10
CHANGES IN INFANT, NEONATAL, AND POSTNEONATAL MORTALITY AND MALARIA AND HIV PREVALENCE

	0–11 months	0 months	1–11 months
Post × malaria	−.00054	.00016	−.00070**
	(.00043)	(.00024)	(.00023)
	[.325]	[.618]	[.086]
Post × HIV	−.00156	−.00001	−.00156
	(.00146)	(.00066)	(.00092)
Male	.01437***	.00805**	.00632*
	(.00338)	(.00258)	(.00321)
Multiple	.15897***	.13622***	.02275**
	(.04263)	(.03818)	(.00933)
Rural	−.00030	.00135	−.00166
	(.00917)	(.00466)	(.00624)
Household size[a]	−.00773***	−.00327***	−.00445*
	(.00193)	(.00049)	(.00201)
Mother's age[b]	−.00046	−.00004	−.00042
	(.00047)	(.00037)	(.00028)
Mother's education[a]	−.00316***	−.00143**	−.00173***
	(.00051)	(.00043)	(.00048)
Constant	.12198***	.06623***	.05575**
	(.02129)	(.01209)	(.01801)
R^2	.04431	.03548	.03331

Note. Ordinary least squares regression coefficients; other covariates include fixed effects for birth order, gender-specific birth order, religion, ethnicity (tribe), month of birth, year of birth, and region of birth. Robust standard errors clustered on region of birth in parentheses. Sample includes births from 1993 to 2008, with "post" defined as years after 2003. Only includes births that took place within 1–5 years of the survey date. "Malaria" is the regional *Plasmodium falciparum* parasite rate, while "HIV" is the regional HIV prevalence among women age 15–49. Square brackets contain two-tailed *p*-values for the post × malaria coefficient based on the wild cluster bootstrap-*T* method. $N = 13{,}897$.

[a] At the time of the survey.

[b] In years, at the time of birth.

* $p < .1$.

** $p < .05$.

*** $p < .01$.

75% (Violari et al. 2008). Could some or most of the differential fall in mortality be due to a reduction in HIV burden? Montana, Neuman, and Mishra (2007) use the 2003 Kenya DHS to estimate the regional HIV prevalence rates in Kenya for women age 15–49, which are displayed in the final column of table 1. I reestimate equation (1) but include post × HIV on the right-hand side as an additional control. The coefficient on post × malaria now represents the postintervention change in mortality in the malarious regions after controlling for any concurrent impact of the HIV/AIDS program. The results are presented in table 10. The coefficient on post × HIV is negative but insignificant. There is a modest fall in the magnitude of the coefficient on post × malaria, but it remains highly significant for postneonatal mortality.[14]

[14] Caution should be exercised comparing the magnitudes of the coefficients on post × malaria and post × HIV. The coefficients represent the prepost change in mortality per unit of prevalence of ma-

VI. Discussion

It is useful to interpret the reported estimates in terms of a more conventional mortality metric (i.e., a rate per 1,000 live births). To remain conservative, I choose the coefficient of −.00066 reported in table 6 for the specification that controls for regional time trends. This estimate maps to a fall in postneonatal mortality of about 25 per 1,000 in the malarious Western region.[15] The mean postneonatal mortality rate in the malarious regions in the preperiod was 76 per 1,000 (see the bottom panel in table 4). Thus, the estimate implies that the malaria control campaign led to a fall of about 33% in the mean postneonatal mortality rate in the Western region.

This is likely an underestimate of the true impact of the campaign in the malarious reasons. The reason is that while I use the change in nonmalarious regions to control for any secular trend in infant mortality across Kenya, those "control" regions were also targeted by the campaign. For instance, there had been a large jump in ITN usage in the nonmalarious regions. And, the IRS has been focused on the epidemic-prone parts of those regions. These campaign activities are likely to have reduced malaria-related mortality in the nonmalarious regions as well. Intuitively, using the relative change in mortality in the malarious regions underestimates the true impact of the campaign in those regions because we are netting out the drop in malaria-related mortality in the nonmalarious regions due to the campaign.[16]

Using a back-of-the-envelope calculation, I map this finding to an estimate of infant lives saved in the highly malarious regions. Nyanza and the Western regions have a population of about 5.4 and 4.3 million respectively.[17] The birth rate in Kenya is about 3.84 per 100.[18] The point estimate of −0.00066 (after controlling for regional time trends) maps to relative drops in postneonatal mortality of about 22 and 25 per 1,000, respectively. That implies on average 4,561 and 4,128 infants saved annually in Nyanza and the Western regions, respectively, during 2004–8, or a total of 43,445 infants over the 5-year period.

Measuring the impact purely in terms of deaths averted understates the benefits of malaria control. Malaria is a major cause of morbidity during pregnancy

laria and HIV, respectively. The regional prevalence of malaria is much higher and very different from that of HIV.

[15] The difference in the PfPR rates between the Western region and Nairobi is $38.84 - 0.05 \approx 38$. Multiplying by the estimated effect of −.00066 and scaling to 1,000 births yields about 25.

[16] I thank a perceptive referee for drawing attention to this point.

[17] Table 4 of Noor et al. (2009).

[18] In 2009, the population of Kenya was 39.8 million and the annual number of births was 1.53 million, which yields a birth rate of 3.84 per 100 (http://www.unicef.org/infobycountry/kenya_statistics.html).

and early life, and there is a growing body of evidence that in utero and early life exposure to adverse health shocks leave a lasting imprint. Almond (2006) finds that in utero exposure to the 1918 influenza epidemic lowered adult income and schooling for the affected cohorts. Almond and Mazumder (2011) show that in utero exposure to maternal fasting during Ramadan leads to learning disabilities and adverse effects on schooling and income. Barreca (2010) finds that cohorts born in the warmer and wetter months in the early part of the twentieth century in the southern part of the United States have less education; he attributes this to the higher burden of malaria in those months. Bleakley (2010a) provides a recent review of this literature including several studies showing that childhood morbidity due to infection by tropical parasites can permanently scar human capital and reduce income.

Some of these studies explore the long-run impact of malaria eradication campaigns. Just as in this article, the recent studies exploit the quasi-experimental nature of the campaigns: identification stems from preexisting variation in the burden of malaria due to climatic and geographical differences combined with the large and sudden changes in the disease burden due to the campaigns that led to plausibly exogenous spatial and time variation in malaria exposure across birth cohorts. Cutler et al. (2010) find gains in household consumption for cohorts of adult men born in regions of high malaria prevalence during India's malaria eradication campaign of 1950s. Lucas (2010) looks at the impact of malaria eradication campaigns in Paraguay and Sri Lanka and concludes that reducing malaria incidence by 10 percentage points increases completed years of schooling by 0.1 years and the probability of being literate by 1 percentage point. Bleakley (2010b) finds that malaria eradication campaigns of the United States (ca. 1920) and Brazil, Columbia, and Mexico (ca. 1955) led to higher income and literacy rates among cohorts born in areas with high malaria prevalence (relative to cohorts born in areas with low prevalence). He concludes that being infected with malaria through childhood leads to a reduction in adult income of about 50%. Since the estimates are at the cohort level, any selection and general equilibrium effects are already embedded in that estimate.

VII. Evidence from Other Studies

The findings are consistent with an older and related literature on the efficacy of bed nets. This includes several individual and clustered randomized control trials. Lengeler (2004) provides a comprehensive review. The trials find that, in areas of stable malaria, ITNs reduce the incidence of severe malaria by about 45%. Other studies have found a significant impact on child mortality of past malaria control campaigns. Payne et al. (1976) and Bradley (1991)

found that the insecticide-spraying campaigns in Kenya, Nigeria, and Tanzania in the 1950s and 1970s reduced mortality among infants and children age 1–4 years by as much as 40%–50%. Alonso et al. (1991) report that malaria chemoprophylaxis and ITNs reduced children-under-5 mortality by 42% in Gambia. The magnitude of these findings is in line with the 33% drop calculated above.

Of direct relevance are the newer studies of the impact of the recent intensification of malaria control. These studies are typically before-after comparisons using admissions and outcomes data from health facilities; some have a difference-in-difference flavor in that nonmalaria cases and deaths are used as controls. Otten et al. (2009) have investigated the impact of the recent and rapid intensification of malaria control in Rwanda and Ethiopia by tracking inpatient malaria cases and mortality among children under 5 in select sites before (2001–6) and after (2007) nationwide implementation of long-lasting insecticide nets and artemisinin-based combination therapy. They find that malaria cases and deaths fall by 73% and 62%, respectively, in Ethiopia and by 55% and 67%, respectively, in Rwanda. In contrast, they find that nonmalaria cases and deaths remain stable or increase. Similarly, Aregawi et al. (2011) find that the rapid scale-up of malaria control in Zanzibar has reduced malaria deaths and inpatient and outpatient cases at health facilities in Zanzibar by 90%, 78%, and 99.5%, respectively, by 2008 (as compared to the preintervention period 1999–2003). Studies of malaria admission rates from coastal Kenya report dramatic falls of up to 90% in some hospitals in recent years (Okiro et al. 2007; O'Meara et al. 2008; Snow and Marsh 2010). While such studies are useful in flagging trends, it is difficult to use the findings to construct an accurate national picture of the true impact of malaria control because of poor record keeping and poor coverage of health facilities in developing countries.

Demombynes and Trommlerová (2012) have used DHS data from Kenya to investigate the recent decline in infant mortality. They also find a sharp fall in postneonatal mortality. They use an Oaxaca-Blinder decomposition to conclude that the use of ITNs in malaria endemic areas explains about 39% of the decline in postneonatal mortality. In contrast, I find that the malaria campaign led to a decline of 58% and 65% in postneonatal mortality in the malarious Nyanza and Western regions, respectively.[19]

However, in another recent study, Tarozzi et al. (2011) report an unusual finding. They detect no health improvements from increased ownership and

[19] The relative drops of 22 and 25 per 1,000 in Nyanza and the Western regions as fractions of the overall drop of 38 per 1,000 in postneonatal mortality (see table 4).

usage of ITNs in a large-scale cluster randomized controlled trial in rural Orissa (India). They discuss at length various possible explanations to reconcile their findings with those of several earlier trials that found large reductions in malaria burden after the distribution of ITNs. They conclude that the most plausible explanation is the relatively low coverage of ITNs and no monitoring of usage in their study. Their trial did not achieve more than 50% coverage in any study village, with the typical coverage rate being significantly less than 50%. Hence, the increase in ITN ownership and usage within study villages was insufficient to break the cycle of malaria transmission. In contrast, most clinical trials have high coverage and close monitoring of usage. These findings raise a question about the true efficacy of ITNs in real-world settings where coverage may be low and usage is not monitored.

How does one reconcile the findings of Tarozzi et al. (2011) with the findings in the current study? One possible explanation is the higher ownership and usage of ITNs in Kenya; the ownership in the highly malarious Nyanza region had risen to about 75% by 2008–9, and about 60% of children under 5 years of age were sleeping under ITNs (KNBS and ICF Macro 2010). Studies have documented the existence of substantial nonlinearity in the relationship between coverage and health benefits from ITNs (Gimnig et al. 2003; Hawley et al. 2003). Another reason could be the difference in settings and the species of the parasite—*Plasmodium falciparum* in sub-Saharan Africa versus *Plasmodium vivax* in India—although this remains speculative in the absence of further research. It is instructive to examine the findings of a study from at least one other African setting with comparable ITN coverage (children sleeping under bed nets) of about 60%. D'alessandro et al. (1995) conducted a cluster randomized trial of ITN efficacy in villages in five areas of Gambia. Trained health workers impregnated bed nets with insecticide in treatment villages. The researchers estimated that about 60% of children slept under ITNs in the treated villages. Treated and control villages had similar postneonatal mortality before the intervention. After the intervention, postneonatal mortality was 43 per 1,000 in the control villages but only 31 per 1,000 in the treated villages—a drop of about 28%. They also found a drop of 25% in all-cause mortality of older children age 1–9 years, a finding in line with the estimate of the current analysis.

VIII. Conclusion

Kenya has witnessed a major campaign against malaria since the adoption of the National Malaria Strategy in 2001. Funding for the malaria program has risen several fold. The strategy emphasized the use of ITNs, and its biggest achievement had been a large increase in ITN coverage since 2003. Data from

health facilities point to a major drop in the burden of malaria. However, it is challenging to construct a representative national picture on the basis of such data due to poor formal health system coverage. I use large preexisting differences in the regional risk of malaria coupled with the sharp timing of the renewed campaign to study the impact on infant mortality. I find that before the intervention, infant mortality in the malarious regions was substantially higher than that in the nonmalarious regions. Much of that difference was due to higher postneonatal mortality in the malarious regions, consistent with a key stylized fact from malaria epidemiology: malaria is most deleterious in later infancy with very young infants enjoying maternally conferred protection. Postintervention, I find a significant fall in postneonatal mortality in the malarious regions vis-à-vis the nonmalarious regions. There is no such relative fall in neonatal mortality. Robustness checks show that the sharp reduction in postneonatal mortality in the malarious regions cannot be explained by preexisting trends. Neither can it be explained by differential changes in maternal and infant care across the two types of regions. Further, the findings are robust to any potential impact on infant mortality of the contemporaneous expansion of HIV/AIDS prevention and treatment.

The preferred specification suggests that the renewed malaria campaign led to a fall in postneonatal mortality of 22–25 per 1,000 live births in the malarious regions. This amounts to a drop of 33% in postneonatal mortality, which maps to an estimated 43,445 infant lives saved in those regions during 2004–8. While clinical trials have already demonstrated the efficacy of ITNs, the findings here suggest large health benefits from the use of ITNs during routine program implementation where coverage is lower and usage is not closely monitored. The findings in this article support the case for sustaining the momentum of the current campaign against malaria.

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