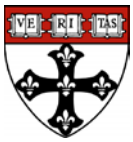

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The economic effects of malaria eradication: evidence from an intervention in Uganda

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Abstract

This study evaluates the economic consequences of a malaria eradication campaign in the southwestern Ugandan district of Kigezi. The project was a joint venture between the WHO and Uganda's Ministry of Health, designed to test for the first time the feasibility of malaria eradication in a sub-Saharan African country. During the years of 1959 and 1960, eradication efforts employing DDT spraying and mass distribution of anti-malarials were implemented, beginning in northern Kigezi. Follow-up studies reported a drop in overall parasite rates from 22.7 to 0.5% in hyperendemic areas and from 12.5 to 0% in mesoendemic areas. We use this campaign as a plausibly exogenous health shock to explore changes in human-capital formation and income. We employ a difference-in-difference methodology to show that eradication produced differential improvements in Kigezi compare to the rest of Uganda in years of schooling, literacy, and primary school completion. In addition, we find suggestive evidence that eradication increased income levels. Keywords: human capital, malaria, economic development and health

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1 Introduction

The damage that malaria inflicts on population health is severe and well established. With an estimated 350 to 500 million cases per year and nearly 900,000 deaths, malaria represents a major threat for 3.3 billion people in 109 nations. Sub-Saharan Africa (SSA) carries the bulk of the global malaria burden, with 71% of cases and 86% of global deaths [26]. It is estimated that anywhere from 30 to 50% of outpatient visits and hospital admissions in much of SSA are a result of malaria illness, with severe cases leading to complications including anemia, seizures, delirium, coma, and death. Malaria also significantly aggravates the condition of HIV-positive individuals and increases HIV transmission.

In addition to severe health consequences, nations with high malaria incidence also exhibit low levels of economic development. The present study intends to explore whether the association between malaria and poverty is causal. We exploit a plausibly exogenous eradication campaign in Uganda's southwestern Kigezi region to investigate the impact of malaria reduction on educational attainment and economic status. The eradication campaign began in 1959 with collaboration between the World Health Organization (WHO) and Uganda's Ministry of Health to bring the WHO's Global Malaria Eradication Campaign, launched in 1955, to SSA. That campaign eventually eliminated malaria from Europe, North America, the Caribbean, and parts of Asia and South-Central America. However, the effort was abandoned in 1969 due to the challenges of eradication in SSA, caused in part by increasing mosquito resistance to dichloro-diphenyl-trichloroethane (DDT) and increasing parasite resistance to chloroquine treatment.

Understanding the impact of malaria on development in SSA represents an increasingly important question, given renewed calls to fund eradication efforts; particularly since a 2007 Bill and Melinda Gates Foundation conference at which the Gateses boldly called for global malaria eradication. Their statement was seconded by Margaret Chan, the Director General of the WHO). This clarion call has been credited with reinvigorating the malaria scientific community and shifting attention from malaria control towards the long-term goal of eradication. No similar effort to shift from control to eradication has occurred since the original WHO campaign in 1955.

Malaria affects human capital and income in multiple ways. First, infection during pregnancy causes anemia and reduced in utero nutrition, leading to reduced neurocognitive function and decreasing a child's likelihood of attending or advancing through school. Second, infection during childhood causes cognitive impairment as well and can reduce educational outcomes or labor productivity. Third, malaria in the community or household can

reduce overall investment and income available for schooling. The present research shows that malaria eradication efforts focused on SSA can indeed raise human capital and income. In addition, these effects override the population pressure that some argue has reduced the economic impact of public health interventions in other contexts [1]. This paper continues as follows: section 1 describes the literature on malaria in more detail, section 2 provides background on the eradication region and campaign, section 3 describes our methodology, section 4 outlines our results, section 5 provides additional discussion of these results compared to other work, and section 6 concludes.

malaria that followed, the use of nonmalarious areas for comparison, and the differential incidence of eradication benefits across cohorts) combine to form the research design of the present study. p. 9 or so

to conduct the analysis, which is therefore an intention-to-treat design." p. 11

1.1 Effect of malaria on population health and developmental outcomes

Malaria reduction or elimination obviously increases a child's ability to attend school and learn once there. In addition to the immediate effects of malaria on health, there is evidence that malaria exposure during infancy and childhood can have detrimental effects on long-term cognitive development. A 2006 systematic review of 18 studies concluded that Plasmodium falciparum malaria in childhood affects short- and long-term neurocognitive performance, including attention, memory, visio-spacial skills, and language functioning [21]. These effects are present and most pronounced for cerebral malaria [9],[20] the most severe form, but also obtain for less-severe infections. Another possible route from malaria to cognitive development is anemia, since repeated malaria exposure during childhood can lead to childhood rates of underweight and anemia [35]. There is strong evidence that iron-deficiency anemia during gestation and early lactation can adversely impact cognitive development and delay the development of the central nervous system [4]. These adverse outcomes are are often irreversible, depending on the child's developmental stage during iron deficiency. Moreover, evidence indicates that anemia reduces a child's ability to respond to environmental cues [12], while anemic infants test lower in cognitive, motor, and social-emotional development compared to comparison group infants [23]. Moreover, randomized evaluations have previously shown the impact that malaria has in hte short-term on school-attendance and cognitive ability [15], [11].

1.2 Effect of malaria on income and human capital attainment

Aside from the population health impact of malaria reduction, there is a large body of work that looks at the effects of malaria reduction on income and human capital attainment. In addition, three recent studies have examined this issue in quasi-experimental settings using the WHO's eradication efforts during 1950s [16], [7], [24] and this study uses a similar identification strategy. Cutler et al., 2010 examines a nationwide malaria eradication program that occurred in India in the 1950s. The authors exploit pre-intervention geographic variation in malaria prevalence to estimate the impact of India's eradication program on educational outcomes. They find that the eradication campaign produced modest increases in household per capita income for men aged 20-60 and that the effects are larger for men than women. They use a sample of ages 15-75 and find no statistically significant effect of malaria eradication in India on human capital outcomes (literacy and primary school completion) [16]. Another study, Bleakley, 2010, combines malaria mortality rates with an ecology index to estimate malaria prevalence during the 1920s in the United States, and the 1950s in Brazil, Colombia, and Mexico [7]. It finds that, relative to non-malarious areas, cohorts born after the eradication programs had higher income and literacy rates than those born prior to eradication.

Bleakley 2010 also notes that the effect of malaria eradication in nations with widespread child labor is ambiguous because health improvements will increase returns to both work and schooling. In addition, since malaria reduces mortality and morbidity, the impact on schooling can be ambiguous because although morbidity will tend to raise schooling / reduce school absences, decreased mortality may move these variables in the opposite direction as reduced mortality differentially benefits children with weaker health and cognitive abilities. Finally, Lucas, 2010 explores female educational attainment following the WHO Global Malaria Eradication Campaign in Paraguay and Sri Lanka. These findings suggest that a ten percentage point decrease in enlarged spleen rate (a measure of long-standing malaria) led to an increase of between 0.39 and 0.93 years of schooling, with a corresponding increase in a female's probability of literacy of between 2.5 to 7.8 percentage points [24].

A related recent paper uses exogenous variation in early-life rainfall to estimate the impact of health shocks on long-term health, education, and socioeconomic status for Indonesian birth cohorts 1953-1974 [25]. For girls, they find early-life positive health shocks in the form of rainfall 20% above average leads to reduced likelihood of self-reported poor health, 0.57 cm

greater height, 0.22 more completed grades, and higher income.

Where malaria exposure data are not readily available, researchers have reconstructed estimates of exposure and linked these to health and productivity outcomes. For instance, one study combines Union Army health records with socioeconomic data from the general population to estimate the impact of malaria exposure on health status and wealth accumulation [19]. It finds significant impacts on both health and physical capital accumulation; for example, Union Army veterans enlisting from malaria-endemic US counties were up to 0.87 inches shorter than those from malaria-free counties. Moreover, exposure to malaria in the US South during the early part of the twentieth century reduced mean years of schooling by 0.26 years, which represented 15% of the difference in years of schooling between the South and the rest of the US at the time [3].

1.3 Effect of health on economic growth

Much evidence already suggests that long-term exposure to malaria can lower educational attainment, depress literacy rates, and damage long-term health. However, do these impacts collectively translate into worse economic growth and development? There are several pathways through which health, as a broad category, may affect growth. For example, healthy workers work more hours and more productive hours than their sick counterparts. Health improvements may also increase returns to human capital and encourage savings for retirement, although recent models have suggested that improvements in health may actually deter savings. In combination, a more productive workforce and higher savings rates can attract investment, which further enhances economic growth.

But empirically, there is some debate as to whether population health leads to economic growth, and the size of this effect. For example, research uses a set of exogenous growth determinant instruments to explain variation in infant and child mortality [32]. While they conclude that income-per-capita growth produces health improvements, they reject the notion that causality runs from health to income. More recently, Acemoglu and Johnson, 2007 have argued that large declines in disease-specific mortality associated with the epidemiological transition after 1940 raised life expectancy with very little impact on growth in income per capita [1].[1]

In response, Bleakley, 2006 has argued that Acemoglu and Johnson fail to account for the economic impacts of morbidity because their analysis does

[1]See Bloom, Canning, and Fink for a dissenting view on the impact of health improvements on growth [8]

not distinguish the effect of mortality and morbidity improvements on population health. Bleakley, for example, points to the impact of malaria and hookworm eradication (Kremer and other Bleakley, which affected morbidity more than mortality, and subsequently improved human capital attainment. Still other studies have suggested that population health contributes to economic growth and development. Using country-level regressions, Gallup and Sachs, 2001 show that nations with endemic malaria exhibit over 1% lower growth per year compared to non-endemic nations for the years 1965 to 1990 [18]. They also note that nations with successful malaria eradication programs enjoyed substantially higher growth rates in the five years following malaria eradication. McCarthy, Wolf, and Wu look specifically at SSA and estimate that, given the prevalence of malaria in the region, the disease reduces GDP growth per annum by 0.55% [27].

The question of malaria eradication addresses two major themes in the economic development literature today: First, what are the costs of malaria illness on health, education, and labor productivity, and how would these costs translate into benefits if the malaria vector were eliminated? Second, how will improvements in population health, achieved through eradication, affect economic growth and development? Answers to these questions are essential to drive the allocative efficiency argument for malaria eradication. That is, what are the relative benefits to eradication versus long-term control?

This study uses a natural experiment to identify and quantify the long-run impact of a short-term malaria eradication program on human capital attainment. It is the first study of its kind to examine the long-term economic effects of malaria eradication in an African context, and contributes to an area where there is growing demand for evidence on the economic benefits of malaria control versus eradication. In the context of today's struggle against malaria, almost exclusively taking place in sub-Saharan Africa, these findings are of even greater importance. Because we focus on a region where the deadliest malaria parasite, Plasmodium falciparum, predominates, the findings contribute to our understanding of whether an intervention that averts many deaths can still improve human capital attainment and lead to economic growth.

2 Background: The malaria eradication experiment in Kigezi District, Uganda

2.1 The setting

During the eradication period, the Kigezi region of southwestern Uganda had a population of 493,000 according to the 1959 census [37] in 1,969 square miles. The state was bound by two game parks, one in the north and one in the south. Altitude increases toward the south. The initial malaria spraying operations occurred in northern Kigezi with a population of 59,000 in 500 square miles. The southern Kigezi region therefore had about 434,000 people in an area of 1,500 square miles, representing a density of almost 290 persons per square mile. This high density helps explain the population pressure to move north and why the Ugandan government was interested in malaria eradication to ease this population movement. Zuleta, 1961 notes a large increase in the population of northern Kigezi after the first DDT spraying campaign: in May-June 1959 the population was 40,562 but by second spraying in December 1959-March 1960 it had increased to 59,605. However, the authors claim that "the great increase observed was probably due to the better health conditions brought about by the introduction of DDT" and not migration. In addition, pre-operational parasite surveys from April-May 1960 showed that in hyperendemic areas (those below 3,700 feet), 82% of cases were falciparum and 17% were malariae, while none were vivax. In the mesoendemic areas (between 3,700 and 4,500 feet) the numbers were 91% falciparum and 9% malariae. This confirms that the malaria found in Uganda, as in most of SSA, is much more deadly than the malaria found in south Asia and Latin America where the other studies done on the impact of malaria eradication were located. Malaria with the P. vivax strain is mostly a chronic disease, while P. falciparum is acute and causes morbidity and mortality. For this reason we would expect the malaria eradication campaign to move educational attainment in two directions. The decrease in morbidity from malaria would tend to improve human capital attainment, while the reduced mortality would tend to, ceteris paribus, reduce human capital, since those surviving are those who are least likely to survive - a variable correlated with low overall educational attainment.

2.2 The malaria eradication project in Kigezi District, Uganda

The malaria eradication experiment took place between 1959 and 1961 in Kigezi District of southwestern Uganda. After a Ugandan government attempt to resettle the Bakiga (Bachiga) people in northern Kigezi (then Rukungiri District) failed due to high malaria prevalence, the Ugandan Ministry of Health teamed up with the WHO to pilot an eradication experiment in Kigezi. The experiment aimed not only to lower malaria prevalence in the area, but also to simultaneously test the feasibility of malaria eradication in SSA, which had previously been excluded from the 1955 Global Malaria Eradication Campaign due to intense transmission and lack of infrastructure. The eradication campaign consisted of DDT spraying in human and animal dwellings twice yearly in mesoendemic areas and three times a year in hyperendemic areas. In contrast to the Global Malaria Eradication Campaign, mass distribution of anti-malarials was carried out alongside the DDT spraying in all areas of Kigezi District in order to completely interrupt transmission and eliminate the disease-causing parasite from the area. (Records indicate malaria eradication efforts were extended to Masaka District in 1961, however this district is not considered in the present analysis due to insufficient data regarding the specifics of that eradication effort.) Records indicate that the project extended from January 1959 until November 1961. DDT spraying and drug administration occurred in northern Kigezi four times throughout the life of the project; in May, September, and December 1959, and May 1960. Southern and central Kigezi received five rounds of spraying and drug administration, namely in March, April, May, September, and October of 1960.

In order to track baseline malaria prevalence and to monitor success of the operation, spleen and parasite surveys were carried out monthly before and after spraying, and fever surveys were carried out monthly at visits to dispensaries. Survey results were used to assign malaria endemicity to Kigezi, in accordance with the classification scheme recommended by the WHO Expert Committee on Malaria (4th session 1950). Available data suggest that hyperendemic conditions existed from the shores of Lake Edward, at an altitude of 2,995 feet, up to 3,700 feet. Above this altitude, conditions were classified as mesoendemic. A map of malaria prevalence in Uganda (Figure 1) and Kigezi District (Figure 2) at the time of the experiment illustrates pre-eradication malaria prevalence.

Results after the first year of the experiment reported a drop in overall parasite rates from 22.7 to 0.5% in hyperendemic areas and from 12.5 to 0% in mesoendemic areas. In areas of hyperendemicity, rates of enlarged

spleen decreased from 68.5% of the population surveyed to 14.4%, while in mesoendemic areas they went from 20.7 to 3.6%. The drastic drop in malaria prevalence seen in Kigezi is a positive health shock which we exploit as part of the identification strategy in the present analysis. Indeed, we argue that although the campaign occurred because of a desire to increase migration to the area, it was directed by the WHO and associated primarily with the fact that the WHO had just implemented campaigns in peripheral malaria regions and wanted to pilot a program to determine whether DDT-spraying could work in SSA. Therefore, we claim that the campaign was exogenous to other factors that would have increased educational attainment and can use the eradication campaign to identify the impact of this health shock on human capital and income.

3 Methodology

3.1 Main Specification

We use Ugandan Census Data from 1991 [29] and our intervention area is therefore defined based on district definitions at the time of the census. By 1991, Kigezi District had been divided into two separate districts, Rukungiri in the north and Kabale in the south. These two districts make up the intervention area in the present study. Furthermore, we define "pre-intervention" in this study as the years prior to 1960, and "post-intervention" as the years 1960 and after. The 1991 census is a weighted 10% sample of the Ugandan population.

We employ a difference-in-difference (DD) methodology to compare human capital attainment before and after the eradication program for Kigezi compared to the rest of Uganda. This approach compares the difference in outcome changes over time for individuals in the treatment group (Kigezi) to those in the control group (the rest of Uganda), while at the same time controlling for time-invariant social or environmental characteristics that may be correlated with both treatment status and outcome. In other words, we estimate the differential change in educational outcomes in Kigezi before and after the eradication treatment compared to the change in outcomes before and after treatment in the rest of Uganda.

Our main specification is estimated in the following form, for individual i, in birth cohort c, and district d:

$$Y_{icd} = \beta_0 + \beta_1 K_d + \beta_2 P_c + \beta_3 K_d * P_c + \beta_4 \vec{\mathbf{X}} + \delta_d + \mu_c + \epsilon_{icd} \qquad (1)$$

In this equation, K_d represents a binary variable for birth in Kigezi dis-

trict, P_c represents a binary variable for birth post-eradication (born in 1960 or after), and $K_d * P_c$ represents the interaction term of being born both in Kigezi and after the eradication campaign. Equation (1) also includes district-level fixed effects δ_d and birth-cohort fixed effects μ_c, to control for linear trends by district and year. Finally, (1) controls for individual-level characteristics in matrix $\mathbf{X}$, such as gender, urbanity, religion, marital status, and ethnicity. Unlike other similar studies (eg: Cutler, et al, 2010) the Uganda Census 1991 contains information on current district and district of birth. We employ the latter throughout this analysis, thereby eliminating concerns that migration drives our results.

Our coefficient of interest is β_3 and interpreted as the mean change in educational outcomes for being born in Kigezi post-eradication versus pre-eradication compared to the change in educational outcomes for being born in the rest of Uganda post- versus pre-eradication, controlling for individual covariates. In addition to using years of schooling as our baseline outcome variable, we also estimate (1) using binary variables primary school completion and literacy with probit regression to check the robustness of our human capital results. We interpret these results carefully, accounting for the interaction coefficients impact on schooling given that we are using non-linear models [2], [31]. Baseline regressions are run with standard errors adjusted for heteroskedasticity using the Huber-White method, clustered at the district level, and weighted by individual using the IPUMS 10% sample of the 1991 Uganda Census.

As with any DD analysis, our essential identification assumption is that the rest of Uganda represents an appropriate control group for the Kigezi malaria campaign and that these two areas would have had the same educational trajectory absent the treatment in Kigezi. If so, β_3 represents the true impact of the eradication campaign on human capital attainment. Table 1 shows dependent and independent variables of interest and compares their average before and after the eradication program in 1960 for both Kigezi and rest of Uganda over the years 1931 to 1971. We see that among birth cohorts born before 1960, those born in Kigezi have 0.75 fewer years of education than the rest of Uganda and for birth cohorts post-eradication, those born in Kigezi have a 0.8 fewer years of schooling than the rest of Uganda. We also observe the same pattern for literacy and primary school completion - either no change or a larger gap in these dependent variables after the eradication than before, demonstrating that we cannot find a positive program effect using this non-parametric comparison. For the other independent variables, we see that Kigezi is less Muslim, more Anglican, and has a higher percentage of married individuals than the rest of Uganda. Figures 3 and 4 show changes in our dependent variables for Kigezi versus the rest of Uganda by birth cohorts

from 1941 to 1971. These figures show that although educational changes are dominated by variation in educational outcomes caused by age-heaping (to be discussed below), Kigezi and the rest of Uganda follow broadly similar trends and therefore the rest of Uganda can be called an appropriate control group for Kigezi in this DD estimation.

3.2 Age Heaping

As is common in data from developing nations, the Uganda Census 1991 suffers from severe age heaping. Individuals round their age to the nearest number ending in 0 or 5 since they are often unsure of their birth year. This occurs differentially for the poorest and lowest educated individuals, resulting in artificially large numbers of individuals reporting ages ending in 0 and 5 and artificially low average educational outcomes for these ages. For example, the 1991 census contains 4,164 individuals reporting their age as 30 while 1,078 and 1,296 reporting their age as 31 and 29 respectively. We graphically illustrate this age heaping clearly in figure 3 for years of education and in figure 4 for literacy and primary school completion for birth-cohorts born between 1941 and 1971. Figures 3 and 4 show that educational attainment is trending upward over time, but that a discontinuity is not immediately visible around the intervention period, given the large variations due to age heaping by birth-cohort.

Instead of visual inspection, age heaping means we must perform a parametric test to visually represent the differential impact in educational outcomes for the Kigezi region after the eradication campaign compared to the rest of Uganda. To solve the age-heaping problem, we follow Cutler, et al, 2010 and plot cohort-specific relationships between Kigezi (and the treatment districts of Rukungiri and Kabale separately) and our educational outcomes. We estimate these cohort-specific relationships using the following equation:

$$Y_{icd} = \beta_0 + \sum_c \beta_c(\mu_c * K_d) + \beta_4 \mathbf{X} + \mu_c + \epsilon_{icd} \tag{2}$$

In equation (2), the coefficient β_c provides us with the cohort-specific relationship between being born in the treatment area and years of schooling. Because we also include birth-cohort fixed effects (μ_c), we can interpret β_c as the differential impact of being born in Kigezi on educational outcomes compared to the rest of Uganda, controlling for $\mathbf{X}$ - the same individual covariates as (2). However, to minimize age heaping, subscript c indexes birth cohort and now refers not to specific birth years, but 5-year age categories centered on ages ending in 0 and 5 (years ending in 1 and 6). Equation (2) is

run with heteroskedasticity-consistent Huber-White standard errors, person weights, and household clustering as with (1).

If the malaria eradication campaign in Kigezi discontinuously increased educational outcomes, we should observe its impact in a break from the previous trend seen for coefficients β_c. This approach also sheds light on the impact of eradication for partially exposed cohorts 1954-1958. If children aged 2-5 during treatment also experience positive educational gains, then we expect this trend break to occur for this birth cohort. We also run specification (2) separately for each district - Rukungiri and Kabale - to explore which specific district produced the largest increase in educational attainment. We would expect a larger increase in educational outcomes for Rukungiri given its higher level of pre-eradication malaria prevalence. Equation (2) is run with years of education as the dependent variable.

3.3 Placebo district-year robustness tests

Bertrand et al. 2004 [6] argue that since many applications of DD estimation use panel data and rely on serially correlated outcomes, they often suffer from inconsistent standard errors. In particular, they use a sample of women's wages from the Current Population Survey 1979 to 1999 and (in the spirit of [10]) designate a random year and set of states as those affected by a new law. They estimate DD regressions using these 'placebo laws' without correcting for correlated standard errors and find that the null of no effect is rejected over 50% of the time.

To test the level of type I error in Uganda's 1991 census, we follow [6] and randomly generate placebo district-year pairs and then re-estimate equation (1). Since our main specification of (1) includes the years 1951-1971 (ages 20 to 40), we pull a year randomly from birth cohorts 1956-1966 (ages 25-35) with equal probability to ensure that we retain at least 5 years of data on either side of each placebo intervention (but excluding 1960 the eradication campaign year). We then randomly select one of 33 Ugandan districts with equal probability (including foreign born individuals as a separate district and excluding the two intervention districts). Define the set of interaction terms from these placebo tests as $\beta_{3,p}$. In theory, our null hypothesis states that this set of placebo interactions $\beta_{3,p}$, from equation (1) should produce normally distributed t-statistics, an expected value of zero, and, by chance, reject the null hypothesis of no positive educational effect in approximately 5% of the placebo regressions. However, this null hypothesis corresponds to data that is independently-distributed without serial correlation. Based the resutls in [6], we would expect the actual rejection of the null hypothesis to be much higher. Instead of using this null hypothesis, we follow [14] and interpret the

set of interaction terms from our placebo regressions $\beta_{3,p}$'s as the empirical distribution of interaction terms from our placebo tests. If we define $G(\beta_{3,p})$ to be the empirical CDF of these placebo regressions, then the test statistic $1 - G(\beta_3)$ provides us with a p-value for the null hypothesis that $\beta_3 = 0$. Intuitively, we would expect that if the eradication experiment had a large impact on educational outcomes, the actual β_3 estimate we find in Kigezi should be in the upper-tail of the $\beta_{3,p}$ distribution. This nonparametric test that $\beta_3 = 0$ allows us to refrain from making additional assumptions about error structure in the distribution of $G(\beta_{3,p})$ and therefore, does not suffer from the t-test over-rejection problem noted above.

3.4 Ignoring time-series information robustness tests

Another way that [6] overcome serial correlation is to completely ignore the data's serially correlated time-series structure. To implement this method, we first regress the individual-level covariates from (1) on educational outcome. Then, we collect the residuals from this regression and average the data before and after the Kigezi, 1960 malaria eradication campaign and re-estimate the rest of the variables in(1). Using this method, [6] produce DD estimates in which the null hypothesis of no effect is rejected in approximately 5% of cases. The specific equation estimated at the individual level is:

$$Y_{icd} = \beta_0 + \beta\vec{\mathbf{X}} + \epsilon_{icd} \tag{3}$$

where $\vec{\mathbf{X}}$ includes gender, urban status, religion, marital status, and ethnicity. The residuals from (3) are then averaged for each district in Uganda both pre- and post-eradication campaign to produce Y_{cd}, the district-level variation in educational outcomes not explained by differences in individual co-variates. Finally, we regress the rest of the dependent variables from (1) on Y_{cd} using the following specification:

$$Y_{cd} = \beta_0 + \beta_1 K_d + \beta_2 P_c + \beta_3 K_d * P_c + +\beta_4 \delta_d \epsilon_{cd} \tag{4}$$

where these variables have the same meanings as in (1). Equation (3) is run with person weights, robust standard errors, and clustered at the district level, while equation (4) is run with robust standard errors. Since there are a total of 35 districts in Uganda, equation (4) contains 70 observations and since the data is collapsed to the district and pre- versus post-eradication level, we exclude birth-cohort fixed effects, but retain district fixed effects. This procedure is run for years of schooling, primary-school completion, and literacy as dependent variables and the deviance residuals for non-linear models are used to average errors in (3) for the latter two equations.

3.5 Additional analyses: Partial exposure and birth-cohort windows

To investigate the educational impact of partial exposure to eradication on educational outcomes, we adjust our exposure variable, changing it from a binary variable for birth-cohorts born in 1960 or after. Instead, we employ a parameterization with the number of childhood years exposed to the eradication campaign, called EXP_{icd} that is zero for cohorts born in 1955 or before and that increases linearly for those born in the five years previous to 1960 (following [7]). Cohorts born in 1960 or after have the maximum of five years exposure. Five years is chosen because evidence suggests that malaria's most important cognitive and health impact occurs in early childhood. Alternatively, we define EXP_{icd} using a 10-year exposure window with a value of one given to cohorts born in 1951 and linear increases in EXP_{icd} for each birth-cohort until 1960, where those born in 1960 and after are given a value of ten in our exposure variable EXP_{icd}. Equation (1) is then re-estimated except with binary variable EXP_{icd} and $K_d * EXP_{icd}$ as the interaction term of interest instead of P_c and $K_d * P_c$.

We perform an additional sensitivity analysis by varying the birth-cohort window used to estimate our effect to see whether the effect is robust to changes in the window away from 1951 to 1971. To vary the birth-cohort window, we re-estimate equation (1) and vary the birth-cohort windows used to 1931-1971, 1941-1971, 1951-1971, and 1956-1966. It is expected that the estimated β_3 will increase as we narrow the window if eradication produced the positive educational effect observed. This is, if we are identifying an effect that occurred because of the eradication, the effect should increase as we narrow the window, while adding additional data to the regression should bias the β_3 coefficient downward through attenuation.

3.6 Creating a socio-economic status (SES) variable

Uganda's 1991 census does not ask income questions directly. However, it does provide comprehensive information on household assets such as type of cooking fuel, water supply, and toilet used, in addition to electricity, kitchen, and dwelling ownership. Principal components analysis is employed to produce a measure of household income using these variables and loading on the first component (following [17]). Because of clustering among the types of assets owned, our SES variable can be split into three categories with 36% of the total sample in the lowest asset category, 13% in the middle category, and 51% in the highest category. We also use SES as a dependent variable for our main specification, equation (1), and report the results below. However,

because of the lack of precision with which we are measuring income, we employ an ordinal probit model instead of an OLS specification to measure the differential impact of post-eradication Kigezi on SES. The model still adjusts for heteroskedasticity with robust (Huber-White) standard errors, clusters at the household level, and uses person-weights.

4 Results

4.1 Effect of Eradication Experiment on Educational Attainment and SES

Table 2 shows our main specification, estimating equation (1), for dependent variables: years of education, literacy, primary school completion, and socio-economic status. We find that the eradication campaign had a positive and significant effect on educational outcomes for all three dependent variables and a positive, but not significant at the 95% level effect on socio-economic status. Specifically, we find a β_3 coefficient in the first column of 0.286, indicating that the differential increase in years of education for being born in Kigezi after the eradication campaign compared to the rest of Uganda amounts to more than one-fourth of a school year. Given that total years of schooling in Kigezi for birth cohorts 1951-1971 averages 3.59, this represents an 8% increase in educational gains that we argue can be attributed to this rapid malaria reduction campaign. Moreover, using the standard Mincerian result that one additional year of education corresponds to a 10% increase in yearly income over the course of one's life [28], this β_3 estimate implies that eradication produced a 2.9% average increase in yearly earnings for those born in Kigezi.

In column 2, we see that being born in Kigezi after the eradication is associated with a statistically significant β_3 coefficient of 0.158 with primary school completion as the dependent variable and, in column 3, a statistically significant β_3 coefficient of 0.065 with literacy as the dependent variable. In column 4, we find a non-significant β_3 coefficient of 0.031 using an ordinal probit model and socio-economic status as the dependent variable (p-value of 0.18). To interpret the magnitude of these effects in quantities of interest, we first acknowledge the difficulty in evaluating interaction terms in nonlinear models [2], [31] and implement the CLARIFY program to understand these β_3's as probability changes using Monte Carlo simulations [36], [22]. We use the program to simulate parameters from each estimation of equation (1) to explore the change in probability of literacy and primary-school completion (setting covariates to their reference categories) for a single, rural, Catholic

male in birth-cohort 1959, from the Baganda tribe if that individual were born in Kigezi after 1960. As table 7 shows, our β_3 coefficient estimate for literacy from equation (1) implies an increase in the probability an individual is literate of 0.0044 [0.0038, 0.0049], all other covariates held constant at their reference level. Given overall literacy rates of 54% in Kigezi for birth cohorts 1951-1971, this implies being born in Kigezi post-eradication is associated with a 0.81% [0.7%, 0.9%] increase in literacy. The gains in literacy are, therefore, statistically significant and positive, but not economically important. In contrast, we find that equation (1) implies an increase in the probability of an individual completing primary school of 0.063 [0.0578, 0.0674], ceteris paribus. Given that only 12% of individuals completed primary school in Kigezi for birth cohorts 1951-1971, this represents an increase of 52.5% [48.2% , 56.2%] in rates of primary school completion that can be differentially attributed to birth cohorts born in Kigezi after the eradication campaign. The CLARIFY simulations are run using the same variables as equation (1), but standard errors are not clustered at the district level, nor are they adjusted for heteroskedasticity using the Huber-White method, meaning that the confidence intervals on these effects are smaller than they would be with these additional corrections made in equation (1). The large difference in probability changes between literacy and primary-school completion can partly be explained by the fact that the latter is a threshold measure corresponding to 5 years of education. Since years of schooling move from an average of 2.44 to 3.9 in Kigezi pre- to post-eradication campaign, a relatively larger percentage of the educational distribution completes primary schooling in Kigezi than the rest of the Uganda. Nevertheless, the increase in percentage of primary-school completion implied by our β_3 estimate represents a large change.

In addition, we estimate (1) for each district within Kigezi separately, excluding the other Kigezi district, with the prior that the area with higher malaria prevalence (Rukungiri) should produce the larger educational gains compared to the lower malaria district (Kabale). Indeed, table 3 shows that individuals born post-eradication in Rukunigiri attained 0.48 years of schooling more than individuals born post-eradication in the rest of Uganda (excluding Kabale), while individuals born post-eradication in Kabale received 0.22 more years of schooling than their counterparts in the rest of Uganda. Both of these results are statistically significant at the 0.1% level and coincide with our priors concerning where we should find the largest educational impact if that educational change were due to malaria reduction.

When we run equation (1) separately for males and females, we find statistically significant β_3 coefficient of 0.49 for males and statistically insignificant coefficient of 0.12 for females (results not shown). This is not surprising

given that returns to education in this environment for males would surely be larger for males than females. Nevertheless, it does differ from other results on the impact of malaria reduction on female educational outcomes [24]. For context, mean years of schooling for birth-cohorts aged 20 to 40 in Kigezi is 4.75 and 2.54 for males and females respectively. Therefore, these results indicate that the being born in Kigezi post-eradication was associated with an increase in years of schooling of over 10% for males and 4.7% for females, although not significant.

4.2 Age Heaping Results

Figure 5 plots the β_c coefficients for all 5-year birth cohorts from 1929-1933 to 1969-1973 (ages 18-62) using equation (2). Since these birth cohorts are centered on ages where age heaping is most severe, this analysis intends to reduce the likelihood that our initial positive estimates were driven by chance inclusion of a given birth-cohort on one side of the eradication window.

Figure 5 does not indicate that the Kigezi region was poised for human capital take-off pre-eradication. Indeed, if anything, the differential trend in years of schooling for pre-eradication Kigezi (dark blue, middle line) was declining, indicating that birth-cohorts in Kigezi were losing educational ground compared to the rest of Uganda before eradication. However, starting with birth cohort 1959-1963 we see an increase in Kigezi's educational outcomes compared to the rest of Uganda during the exact time we would expect, corresponding to the malaria reduction. This strengthens our intuition that the results we see in the main specification are robust to age-heaping and the specific definition of eradication year. We see some evidence for the effect of partial exposure as the birth-cohorts 1959-1963 show increased educational outcomes compared to the previous cohort 1954-1958, but we do not see evidence of a partial exposure schooling impact for children age 2 to 6 during the eradication. Moreover, we find that the average increase in β_c from 1954-1973 for Rukungiri is 0.16 years of schooling and 0.12 for Kabale. Again, this coincides with our expectations given that Rukunigiri contained more malarious areas pre-eradication.

We also run the main specification equation (1) except use 5-year birth cohort categories instead of 1-year categories to reduce age-heaping. These regressions are run for ages 18 to 42, instead of ages 20 to 40. Table 4 shows results for the four dependent variables as in table 2. The interaction coefficients of interest β_{c3}, for columns 1-3 decline only slightly. This suggests that partial exposure, at the least, does not produce a large decline in our observed main human capital effect. Column 4 shows that the interaction coefficient on SES increases by more than 50% in table 3 and becomes

significant at the 10% level (p-value of .068). It is possible that since our asset-index is measured less precisely than the educational variables, that 5-year birth-cohort categories reduce attenuation bias more for SES than the educational variables. However, this table provides additional evidence that the eradication increases educational outcomes and suggestive evidence on an increase in our asset-based proxy for socio-economic status.

4.3 Placebo Robustness Tests

Figure 6 illustrates the results of the placebo test by plotting the empirical distribution of $G(\beta_{3,p})$ with years of schooling as the dependent variable in all regressions. The vertical lines in figure 6 represent the treatment effect β_3's reported in column 1 of table 2 for Kigezi overall and columns 1 and 2 in table 3 for Rukunigiri and Kabale respectively. We see that the equivalent p-value from this empirical distribution for the actual β_3 is $1-G(\beta_3) = 0.156$ for Kigezi overall. In addition, using the β_3 results from equation (1) run by district in table 3, we find that the $\beta_{3,R}$ for Rukungiri of 0.477 corresponds to a p-value in this empirical CDF of $1-G(\beta_{3,R}) = 0.08$ and the $\beta_{3.K}$ for Kabale of 0.215 corresponds to a p-value of $1 - G(\beta_{3,K}) = 0.21$. This test does show that our actual $\beta_3 = 0.286$ and especially $\beta_{3,R} = 0.477$ lie in the upper tail of the distribution of placebo tests. Moreover, out of the 500 placebo tests performed, all the placebo tests with $\beta_{3,p} > \beta_3$ come from 6 districts, while all the placebo tests with $\beta_{3,p} > \beta_{3,K}$ come from 3 districts.

The fact that only a few districts account for all the coefficients in which $\beta_{3,p} > \beta_3$) indicates that other, unknown interventions likely produced these large effects. If, in contrast, the $\beta_{3,p}$'s $> \beta_3$) were randomly distributed within Ugandan districts, then we would worry more that our estimates of β_3 and $\beta_{3,R}$ were produced by chance. For example, British parliamentary papers from the years 1957 up through Ugandan independence in 1962 report on a large education and school building program which took place in Uganda throughout the 1950s. Although, this program was nationwide and its effects should therefore be filtered out through the use of district and cohort fixed effects, it may have produced differential educational gains on other districts. Since, as figure 5 shows, Kigezi if anything experienced a differential decline in educational attainment over the 1950s, we cannot claim that this educational intervention produced the results we find around 1960.

The mean of the 500 placebo tests in the $G(\beta_{3,p})$ empirical distribution is -0.11 such that we reject the null hypothesis that the distribution of $\beta_{3,p}$ has a mean value of 0. We also reject the null that $\beta_{3,p}$ is normally distributed using a Shapiro-Wilks test for normality. Although our district and birth-cohort fixed effects do not control for all short-term differential trends

in educational attainment throughout Uganda, this analysis shows that the effect we attribute to eradication falls within the upper tail of human-capital responses in Uganda around this time period.

4.4 Ignoring time-series information robustness tests

A simple and extreme method to control for serially correlated data with DD analysis is to assume full correlation over birth-cohorts and completely ignore the time-series information, treating the data as having only pre- and post-eradication cohorts. Table 5 shows results from equation (4) for dependent variables: years of schooling, primary school completion, and literacy. Columns 1 and 2 indicate that the differential increase in educational attainment for Kigezi post-eradication is robust to eliminating time-series information as the β_3 coefficients on years of schooling and primary-school completion are significant at the 0.1% level. Indeed, we find an increase in mean years of schooling in column 1 of 0.404, significantly larger than our result in table 2 even though n = 70 and k, the number of independent variables, estimated is 34 for equation (4). Although, β_3 on literacy is not significant at the 5% level, we find a t-statistic of 1.9 and a p-value of 0.66 (table 5, column 3). These results again strengthen our intuition that the positive educational effect found in table 2 is not an artifact of the serial correlation that Bertrand, et al 2004 identify as a serious threat to hypothesis testing with DD methodology.

In addition, we perform the same placebo test as above using the placebo district-year pairs and equations (3) and (4). Implementing 500 placebo tests, we find a p-value for our β_3 with no times series information of $1 - G(\beta_3) = 0.126$ (empirical CDF not shown). Again, of the 500 random placebo tests, those where $\beta_{3,p} > \beta_3$ can be attributed to three districts, suggesting that these large coefficients are picking up other positive educational health shocks, not randomness.

4.5 Partial exposure and birth-cohort windows results

Employing Bleakley's approach to estimate the impact of partial exposure to malaria reduction, we find that the estimated coefficient on β_3 is significantly reduced when we use partial exposure 5-years before eradication. Our β_3 estimate declines to a (still significant) 0.05 years-of-schooling increase for those born in Kigezi post- (or partially exposed to) eradication. When we extend the partial exposure period to ten years prior to the eradication campaign, we find our β_3 estimate is still significant at the 10% level (p-value of .06) but halves to 0.025 years of education (results not shown). Using this

test, we find little evidence that partial exposure to eradication differentially improves educational outcomes, suggesting that the channel through which malaria reduction improves educational attainment comes from in utero and infant effects on cognitive development.

Table 6 shows that as the birth-cohort window narrows for specification (1), we do not find significant β_3 terms for larger birth cohort windows over years 1931-1971 nor 1941-1971. In addition, when we narrow the birth cohort window to five years around the malaria reduction campaign, estimating equation (1) over years 1956-1966, we obtain virtually the same β_3 coefficient. When we narrow the birth-cohort window even more to 1958-1962, we still find a significant β_3, but diminished educational effect. We find an educational effect during the expected time periods (as the birth-cohort window narrows) and not when we expect not to find one, again suggesting that the human-capital impact we find is indeed due to the eradication campaign.

5 Discussion

5.1 Summary

The preceding analysis evaluates the educational and economic impact of a malaria eradication campaign in southwestern Uganda. We find that individuals born in Kigezi post-eradication experienced an increase in schooling of nearly 0.3 years compared to the rest of Uganda and controlling for a variety of individual-level characteristics along with birth-cohort and district fixed effects. This corresponds to an increase of 8% [2.7%, 13.1%]. Previous research indicates that this increase in educational attainment should translate into an approximately 3% increase in income per year for these individuals. This increase in educational outcomes is consistent for two other binary measures of education: literacy and primary education. We find a much larger effect on increases in the probability of completing primary school compared to literacy. The bulk of these educational increases occur for males instead of females and in the more malarious district of Rukunigiri. In addition, we employ an asset-based household wealth index as a proxy for income and find suggestive results that income also increased differentially in Kigezi for post-eradication birth cohorts. When we control for age-heaping the impact of eradication on SES doubles and becomes statistically significant at the 10% level. These estimated effects are robust to a host of additional analyses. We control for age heaping by running our main specification with 5-year birth cohorts and find evidence that Kigezi's educational trajectory was downward sloping pre-eradication and upward sloping post-eradication. We also per-

form placebo DD analyses and find that a 0.286 differential increase in years of schooling falls roughly in the top 15% of the empirical placebo-effect distribution. Moreover, we ignore time-series information in the data to find an even larger positive educational affect for Kigezi post-eradication. The coefficient estimated from this specification falls in the top 10% of the empirical placebo-effect distribution.

5.2 Results in Context

To our knowledge, this paper provides the only results on the long-term human capital and economic impact of malaria eradication in Africa south of the Sahara.[2] Eradication differs in this region not only because climatic factors make vector control more difficult, but because the type of malaria present produces severe mortality along with morbidity. Indeed, as mentioned above, the P. falciparum malaria strain dominates in SSA, whereas P. vivax reigned in southern Europe, Latin America and south Asia pre-eradication. This means that over 80% of global malaria deaths occur in SSA and it is the region where all control and eradication efforts are now focused. Although three recent studies (Cutler, 2010, Lucas, 2010, Bleakley 2010) employ comparable methodologies to that described here, none directly estimate the impact on educational and income outcomes of malaria eradication in the region where the vast majority of malaria-control efforts remain to be done. Their results are primarily driven by reduced morbidity allowing improved educational and income outcomes. However, the preceding analysis shows that even when malaria causes a large mortality burden, its eradication still produces positive outcomes. This is important because we would expect those lives that are saved by eradication to represent either more sickly individuals or those in lower socio-economic circumstances - both of which are less likely to attain high levels of schooling. For this reason, although the mortality effect could attenuate the measured educational impact of eradication, it does not completely swamp the overall positive effects observed.

The data in the present study is advantageous relative to other similar studies because we use place of birth instead of current residence (Bleakley, 2010 and Lucas, 2010 use place of birth). We also have well-estimated pre-eradication information on spleen inflammation and malaria infection incidence for use as proxies of endemicity instead of malaria indices used in Bleakley, 2010. Moreover, we estimate educational and income changes overall, and males and females separately. However, this study also has disad-

[2]Ashraf, N.,et al 2010 [30] and Burlando, A. 2009 explore similar questions [13].

vantages relative to the others, namely that we are investigating the impact of an eradication campaign for one region of the country (roughly 10% of the population), not its entirety. In addition, this study along with Lucas, 2010 and Cutler, 2010 employ only one cross-section to measure eradication impact, while Bleakley, 2010 uses pooled cross sections from multiple census years. A Uganda census from 2002 is available, however, and future work could analyze this dataset as well. Finally, another disadvantage of this study is that the consumption and income information in the other studies allows more precise estimates of the economic impact of malaria control.

Our results contrast with those of Cutler, et al who finds no statistically significant impact of eradication on educational outcomes for men and mixed evidence for women. Our results using a household-level asset index as a proxy for income are not directly comparable to Cutler, et al's results on increases in household per capita consumption for prime age men. However, both papers find positive income improvements post-eradication. To compare these results with the others, we noted above that parasite rates dropped from 22.7 to 0.5% in hyperendemic areas and that this was associated with a 54 percentage point decrease in spleen rate. This means a 10% drop in malaria incidence is associated with a 24% drop in spleen rate. For mesoendemic areas, the eradication campaign reduced malaria incidence from 12.5 to 0%, which produced a decline in spleen rate of 17.1 percentage points. This means a 10% drop in malaria incidence produced a decline in spleen rate of 13.7%. The former estimate is similar to Cutler's estimate of 28.8, but the latter is not. Using the more conservative estimate of a reduction of 22.2 percentage points in malaria incidence, we divide our main coefficient estimate on years of schooling by 2.22 and find that a 10% decrease in malaria incidence is associated with an increase in educational attainment of 0.14 years or 3.6% in years of education for ages 20 to 40 in Kigezi. These estimates change to increases of 4.5% and 2.1% for males and females respectively when we run the main specification regression separately by gender. These results compare favorably to those found by the other three papers. One possible reason for finding these large and robust results that swamp mortality attenuation is that the rate of enlarged spleen was larger in Kigezi (a low malaria area in Uganda) than nations in the other studies. For example, we find a pre-eradication spleen rate of 54% while the most malarious area of Sri Lanka had a spleen rate of less than 40% immediately preceding eradication. (See table 8 for a side-by-side qualitative comparison of results).

A vast literature exists to summarize estimates on rates of returns to education (ROREs) throughout the world [33], [34]. Generally, these reviews suggest that the highest returns to education occur during primary school and in developing nations, especially Africa. In the most recent review [34],

the social RORE in Africa during primary school is found to be 25.4% and the private RORE in Africa during primary school 37.6%. These rates represent the highest ROREs of any region. If true, our estimate of 0.286 years of educational increase for malaria reduction would translate into a 7% and 10% social and private annual earnings returns respectively. Nevertheless, other work argues that the data in SSA to estimate ROREs is poor quality, unrepresentative (covering only 18 of 46 SSA nations), and suffers from sample selection within nations using only formal sector employees [5]. Even taking the standard Mincerian estimate of a 10% RORE as an approximate average to adjudicate this debate, we find that our results imply an almost 3% increase in yearly income for birth cohorts born in Kigezi post-eradication. Since we find consistent increases in education, this indicates that ROREs were larger than child wages during the post-eradication period studied (such that individuals stayed in school longer as opposed to entered the labor market earlier with their improved productivity from malaria reduction) and that malaria eradication represents a high cost-benefit investment for nations in SSA.

Moreover, this paper also takes seriously the potential serial auto-correlation in this panel data series and provides multiple attempts to solve this problem. We find that our results are mostly robust to many specifications designed to address this correlation problem.

5.3 Threats to Identification

In line with other natural experiments that use a DD methodology to infer an intervention's impact, our identification strategy rests on the assumption that the trajectory of educational attainment in Kigezi, in comparison with the rest of Uganda, would have followed a similar long-term trend, in the absence of the eradication project. Given that these are observational data, this assumption cannot be fully verified. However, attempts to identify other social programs that could have caused the increase in educational attainment seen in Kigezi provide no convincing evidence that a social program, other than the malaria eradication experiment, affected Kigezi differently from other districts in Uganda (British House of Commons Reports, 1957-1962).

However, threats to attributing to causality in this case do exist. The age-heaping or auto-correlation in our data could be causing the effect we see. Through extensive robustness checks we also intend to avoid the possibility that the effect we find is caused either by chance or serial correlation in the data. By controlling for age-heaping, performing placebo district-year tests both for the main specification and the averaged data, and ignoring time-

series information (equations (1)-(4) respectively), we show that our effect remains consistent throughout.

In addition, from 1951 to 1971 Uganda faced significant historical changes that may have produced differential educational and economic trends by region. For example, independence from the British government occurred in 1962, setting off a decade of low-level turmoil with the rule of Milton Obote that climaxed with the rise of Idi Amin in 1971, the explusion of 80,000 Asian Ugandans in September 1972, and an almost total collapse of the economy. In addition, the HIV epidemic hit Uganda in the mid-1980s. Although, we cannot control for all of these changes explicitly, other than using district fixed effects, we note that by constraining our sample to before the rule of Amin, we reduce the impact of these changes. In addition, patronage by the ruling party represents an important potential confound to the results described above. Nevertheless, neither Obote, from northern Uganda, nor Amin, born in central Uganda provided patronage to the Kigezi region. Finally, we note that the malaria intervention itself was implemented based on exogenous technological factors with the widespread use of DDT and the WHO's worldwide malaria eradication efforts. This reduces the likelihood that the results we observe were driven by factors present in the Kigezi region before eradication or endogenous to the decision to implement this malaria reduction program.

6 Conclusion

Using an eradication experiment that began in Kigezi District, Uganda in 1959, this analysis demonstrates the long-term effects on human capital and income that malaria eradication can produce in SSA. The experiment utilized DDT spraying and mass distribution of anti-malarials to reduce malaria transmission in a malaria-endemic area. The program successfully eradicated malaria from the region within one year. Overall, Kigezi District experienced significant gains in educational attainment compared with other districts of Uganda that were not exposed to the eradication efforts. Moreover, areas of primarily hyperendemic malaria prior to the intervention experienced greater increases in educational attainment, which is consistent with eradication producing the estimated schooling effect.

If these results are not due to chance, they provide strong evidence that malaria eradication significantly benefits human development and human capital in the long term. While most funding commitments in recent years have focused on a strategy of access to medicine and malaria control, there are perhaps larger gains to be made through malaria reduction and eradica-

tion efforts. The 8% overall increase in schooling indicates that eradication represents a cost-effective option in the fight to reduce deaths from malaria and promote economic growth in SSA. In general, previous papers on the economic and educational impact of malaria eradication indicate positive effects, but an impact not large enough to drive economic growth. Although we do not purport to disentangle partial and general equilibrium effects, we find a somewhat more optimistic story. We hypothesize this effect is due to higher malaria prevalence and heavier burden of malaria in our eradication area. These results show that fighting malaria on its own will not pull African nations of out poverty. However, they also indicate that malaria eradication campaigns are far from hopeless in sub-Saharan Africa and, indeed, that we should continue to implement them given the positive long-term impact found in Uganda.

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Figure 1: Pre-eradication Malaria Prevalence in Uganda

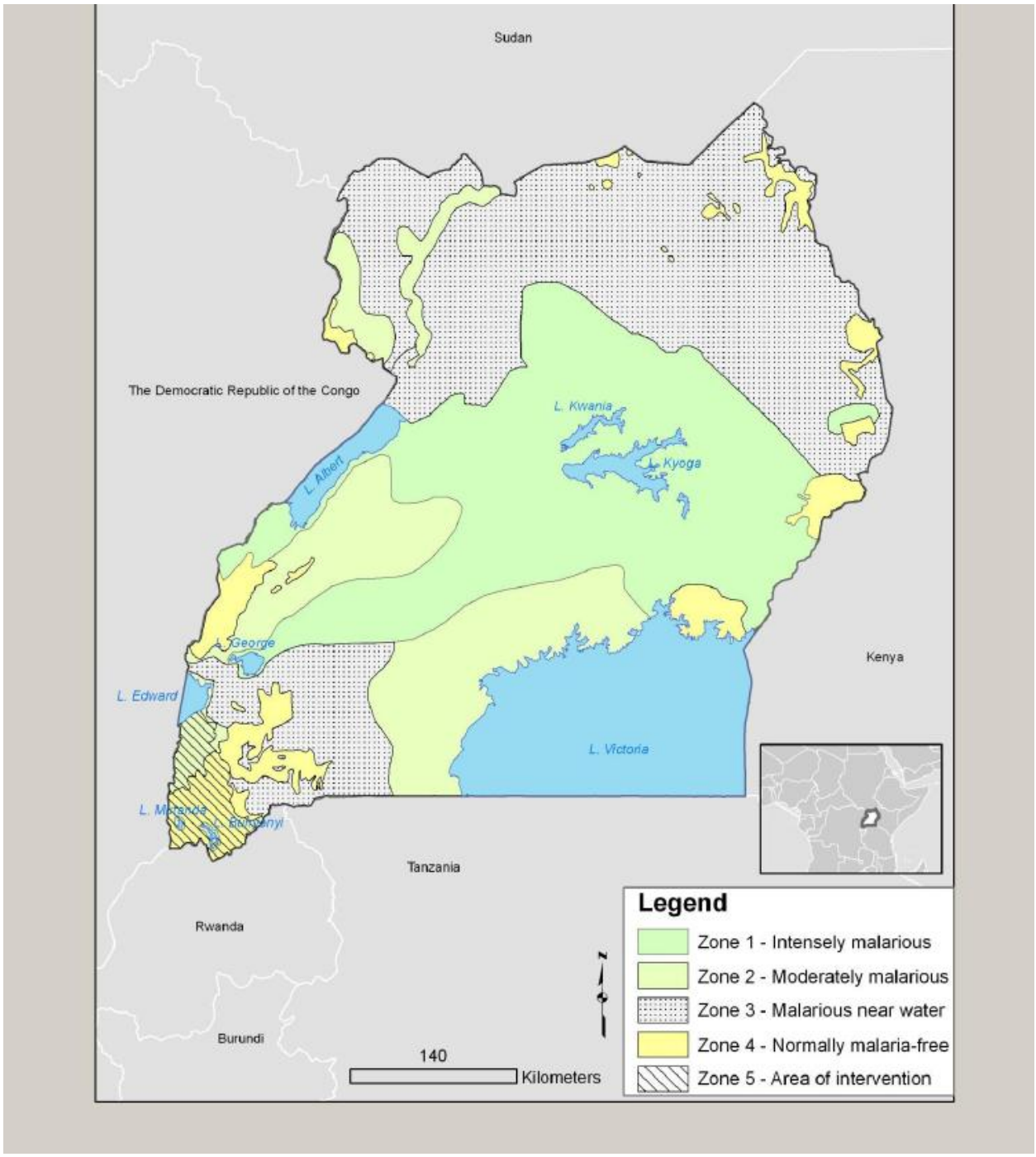


Figure 2: Pre-eradication Malaria Prevalence in Kigezi District, Southwestern Uganda.

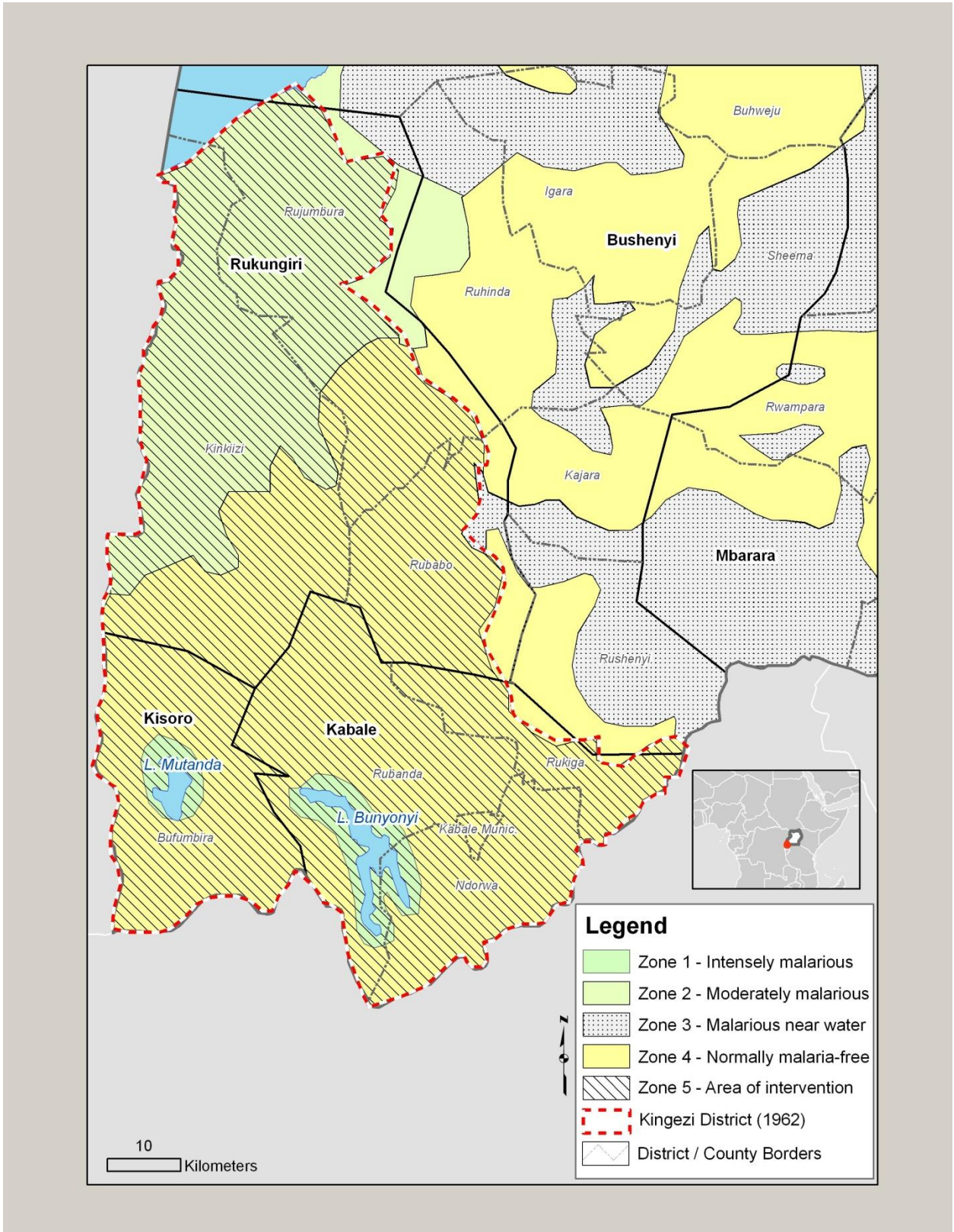


Figure 3: Mean years of schooling by age-cohort for Kigezi and the rest of Uganda, 1941-1971

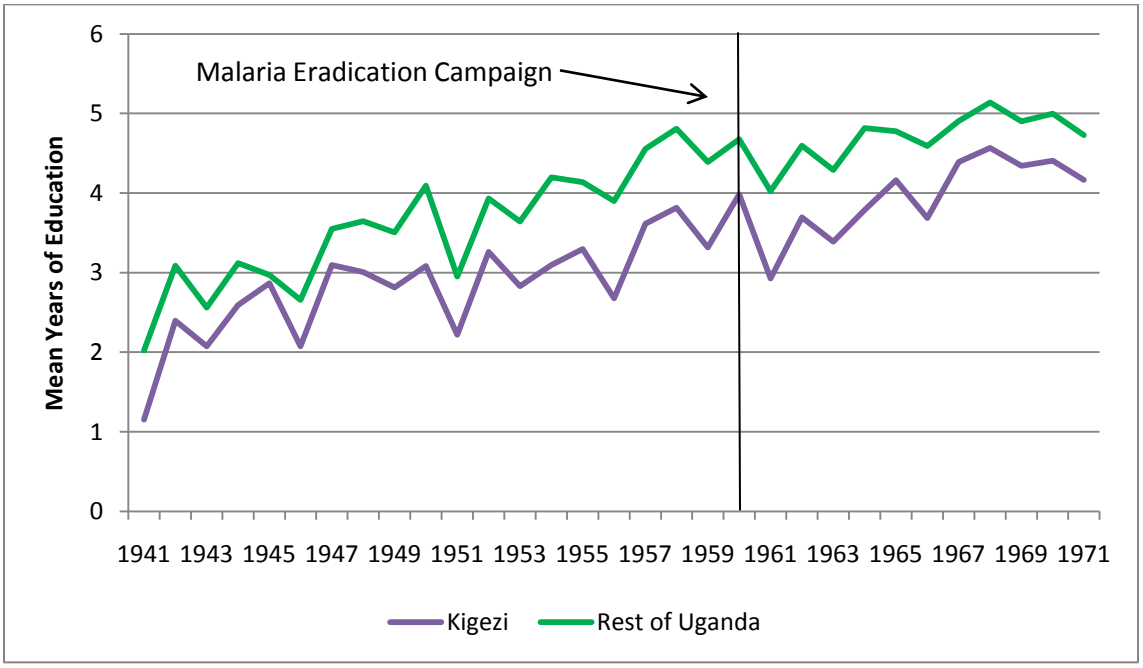


Figure 4: Literacy and Primary School Completion (%) by age cohort in Kigezi and the rest of Uganda (ROU), 1941-1971

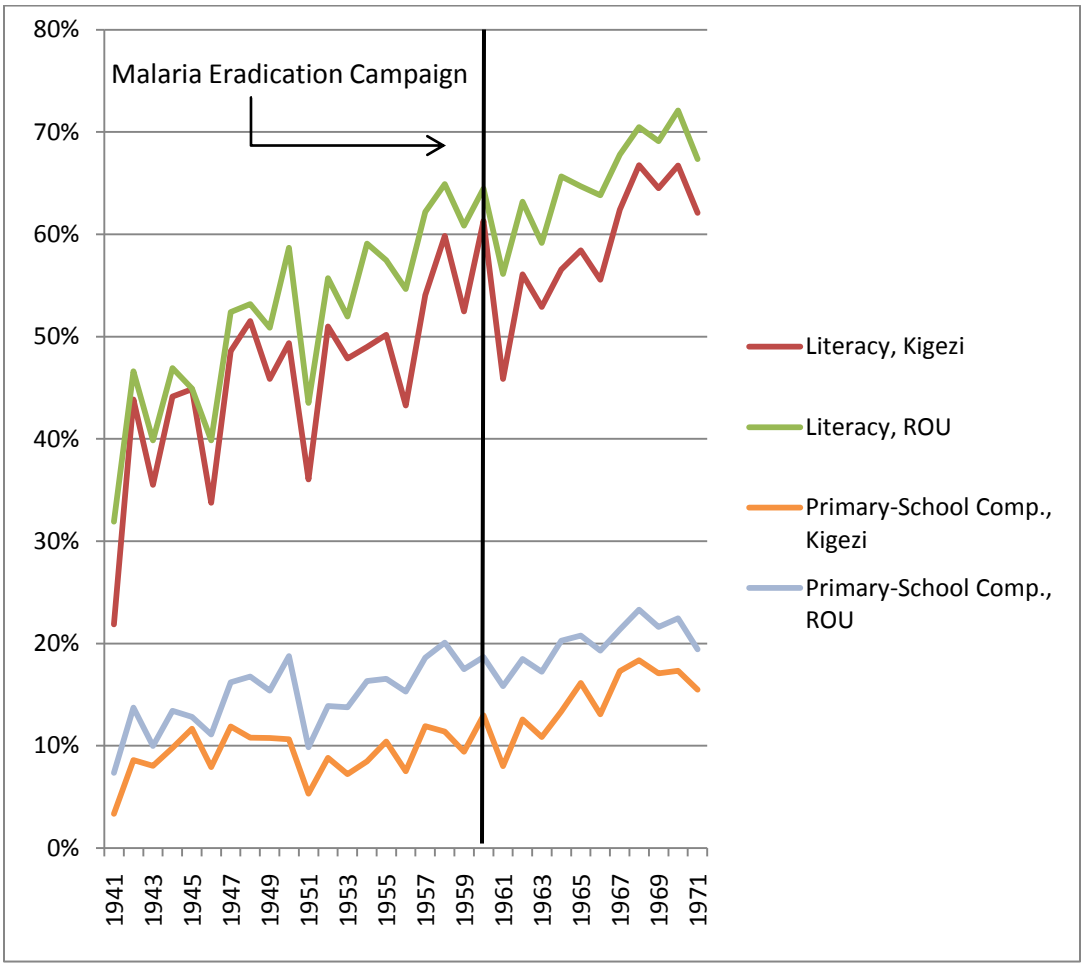
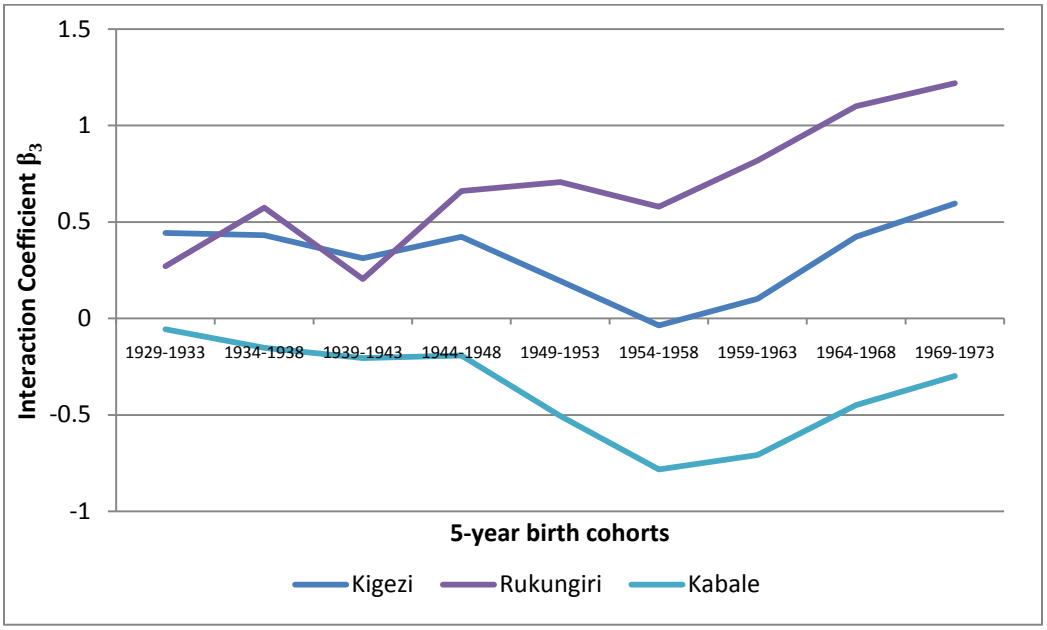
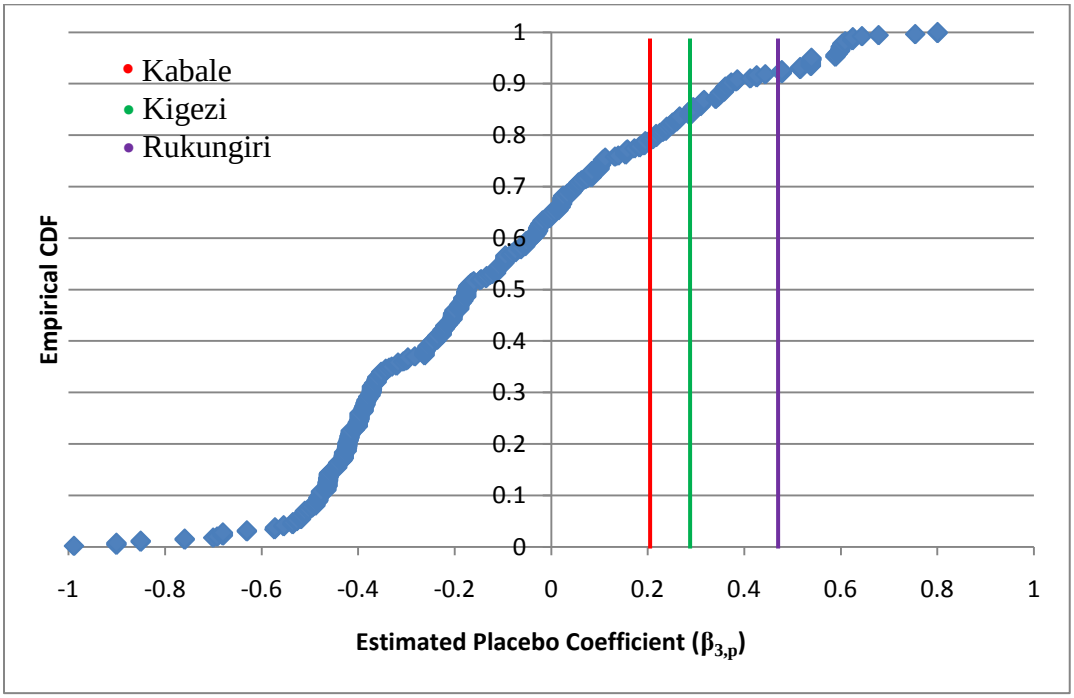


Figure 5: β_3 Interaction Coefficient for Kigezi, Rukungiri, and Kabale by 5-year birth cohort, 1929-1973 (ages 18-62).



Note: Figure 5 plots the β_3 coefficients by 5-year birth cohort using estimated from equation (2) for Kigezi, Rukunigiri, and Kabale. These coefficients are centered on years with individuals aged 0 or 5 to minimize the impact of age-heaping on the regression results.

Figure 6: Distribution of placebo estimates ($\beta_{3,p}$)



Note: Figure 6 plots the empirical distribution of placebo coefficients, $G(\beta_{3,p})$, with years of schooling as the dependent variable. This distribution represents the results from 500 placebo estimations of specification (1) where a random district and year pair is randomly drawn from ages 25-35 (excluding age 31) and all districts in Uganda (excluding the treatment districts) and assumed to represent a placebo treatment . The vertical lines from right to left represent the treatment effect estimate for Kabale (β_3 estimate in column 1 of table 2), Kigezi (β_3 estimate in column 1 of table 3), and Rukungiri (β_3 estimate in column 2 of table 3). The empirical p-values (produced using 1 - $G(\beta_3)$, 1- $G(\beta_{3,R})$, and 1- $G(\beta_{3,,K})$) for Kigezi, Kabale, and Rukungiri are 0.156, 0.201, and 0.082 respectively.

Table 1: Comparison of Kigezi intervention area and the rest of Uganda

	Rest of Uganda		Kigezi	
	Pre-	Post-	Pre-	Post-
% of data	42.07%	48.14%	4.89%	4.91%
Years of Education	3.19	4.71	2.44	3.90
Primary School Completed	12.40%	19.92%	7.52%	14.03%
Literacy	46.68%	65.08%	40.01%	58.06%
Age	43.58	25.00	43.07	25.22
Catholic	49.13%	46.51%	43.86%	41.68%
Anglican	36.26%	37.63%	51.36%	53.95%
Muslim	8.46%	10.99%	2.05%	2.24%
Married	74.03%	68.44%	80.59%	69.38%

Table 2: Estimates of equation (1) for dependent variables: years of education, primary school completion, literacy, and asset-based income category.

Dep Var:	**Yrs of Schooling**		**Primary School**		**Literacy**		**SES**	
	b/se		b/se		b/se		b/se	
Post-Treat.	0.13	*	0.004		0.057	*	-0.005	
	0.051		0.024		0.023		0.019	
Kigezi	-0.364		0.054		0.276	***	-0.09	
	0.229		0.064		0.07		0.047	
Post * Kigezi	**0.286**	**	**0.158**	***	**0.065**	***	**0.031**	
	0.091		0.032		0.019		0.023	
Female	-1.864	***	-0.447	***	-0.73	***	0.115	***
	0.151		0.039		0.056		0.011	
Urban	2.654	***	0.902	***	0.709	***	-1.574	***
	0.162		0.043		0.031		0.051	
Anglican	0.461	***	0.203	***	0.106	**	0.041	*
	0.091		0.019		0.036		0.02	
Muslim	-0.433	***	-0.15	***	-0.092	*	0.071	**
	0.097		0.03		0.039		0.022	
Other relig.	-0.361	*	0.021		-0.221	***	0.006	
	0.144		0.048		0.061		0.038	
R^2	0.27							
Pseudo R^2			0.171		0.175		0.143	
N	4,526,849		4,526,849		4,554,446		4,554,446	

*p<0.05, ** p<0.01, ***p<0.001

Note: All regressions use adjusted standard errors clustered at the district level and employ Huber/White/sandwich variance estimates to adjust for heteroskedasticity and intra-district correlation, and are weighted by individual according to the IPUMS 10% sample of the 1991 Uganda Census. All regressions are run with district and birth-cohort fixed effects, along with binary variables for marital status and 23 ethnicity dummies.

Table 3: Estimates of equation (1) for years of education as the dependent variable by district within Kigezi using Rukungiri (high malaria) and Kabale (low malaria).

Dep Var: Yrs of School	**Rukungiri**		**Kabale**	
	b/se		b/se	
Post-Treat.	0.132	*	0.122	*
	0.052		0.051	
Kigezi	0.614	**	-0.278	
	0.186		0.242	
Post* Kigezi	**0.477**	***	**0.215**	***
	0.063		0.055	
Female	-1.854	***	-1.872	***
	0.162		0.155	
Urban	2.596	***	2.64	***
	0.156		0.165	
Anglican	0.504	***	0.471	***
	0.09		0.093	
Muslim	-0.41	***	-0.427	***
	0.096		0.097	
Other relig.	-0.314	*	-0.352	*
	0.145		0.147	
R^2	0.27		0.27	
N	4,210,838		4,403,579	

*p<0.05, ** p<0.01, ***p<0.001

Note: All regressions use adjusted standard errors clustered at the district level and employ Huber/White/sandwich variance estimates to adjust for heteroskedasticity and intra-district correlation, and are weighted by individual according to the IPUMS 10% sample of the 1991 Uganda Census. All regressions are run with district and birth-cohort fixed effects, along with binary variables for marital status and 23 ethnicity dummies.

Table 4: Estimates of equation (1) with 5-year age categories for dependent variables: years of education, primary school completion, literacy, and asset-based income category.

Dep Var:	**Yrs of Schooling**		**Primary School**		**Literacy**		**SES**	
	b/se		b/se		b/se		b/se	
Post-Treat.	0.829	***	-0.115	***	0.298	***	-0.01	
	0.082		0.03		0.031		0.017	
Kigezi	0.693	**	0.086		-0.122		0.014	
	0.199		0.066		0.076		0.055	
Post* Kigezi	**0.267**	**	**0.15**	***	**0.051**	**	**0.048**	
	0.093		0.036		0.019		0.026	
Female	-1.749	***	-0.397	***	-0.7	***	0.106	***
	0.149		0.038		0.056		0.011	
Urban	2.559	***	0.868	***	0.683	***	-1.57	***
	0.157		0.043		0.03		0.051	
Anglican	0.44	***	0.191	***	0.101	**	0.041	*
	0.09		0.019		0.036		0.019	
Muslim	-0.439	***	-0.15	***	-0.097	*	0.067	**
	0.098		0.03		0.04		0.022	
Other relig.	-0.394	**	0.011		-0.236	***	0.006	
	0.142		0.048		0.061		0.037	
R^2	0.258							
Psuedo R^2			0.158		0.167		0.143	
N	5,397,320		5,397,320		5,425,965		5,425,965	

*p<0.05, ** p<0.01, ***p<0.001

Note: All regressions use adjusted standard errors clustered at the district level, employ Huber-White sandwich estimator standard errors, and are weighted by individual according to the IPUMS 10% sample of the 1991 Uganda Census. All regressions are run for ages 18 to 42 with district and 5-year birth-cohort fixed effects (centered on ages ending in 0 and 5), along with binary variables for marital status and 23 ethnicity dummies.

Table 5: Estimates of equation (4) run at the district-level pre- and post-eradication for dependent variables: years of education, primary school completion, literacy, and asset-based income category.

Dep. Var:	**Yrs of Schooling**		**Primary School**		**Literacy**	
	b/se		b/se		b/se	
Post-Treat.	0.32	***	-1.525	***	-1.65	***
	0.062		0.362		0.333	
Kigezi	-0.319		-2.061		-1.315	*
	0.226		2.29		0.59	
Post* Kigezi	**0.404**	***	**3.03**	***	**0.854**	
	0.103		0.419		0.449	
R^2	0.89		0.942		0.941	
N	70		70		70	

*p<0.05, ** p<0.01, ***p<0.001

Note: These results are produced by first estimating equation (3) using the individual-level covariates from equation (1) including gender, urban status, religion, marital status, and 23 ethnicity indicators. Equation (3) uses adjusted standard errors clustered at the district level, employs Huber-White sandwich estimator standard errors, and is weighted by individual according to the IPUMS 10% sample of the 1991 Uganda Census. The residuals from equation (3) (interpreted as variation in the dependent variables unexplained by individual covariates) are then averaged by district and pre- and post-eradication to create educational outcome dependent variables, controlling for individual characteristics. The remaining independent variables from equation (1) are regressed on these mean residuals by district both pre- and post-eradication, following equation (4) with Huber-White sandwich estimator standard errors. Seventy observations come from 35 districts, with one observation each for pre- and post-malaria intervention.

Table 6: Estimates of equation (1) using years of education as the dependent variable over varying birth-cohort windows.

Dep Var: Yrs of School	**Ages 20-60**		**Ages 20-50**		**Ages 20-40**		**Ages 25-35**		**Ages 29-33**	
	b/se		**b/se**		**b/se**		**b/se**		**b/se**	
Post-Treat.	1.46	***	1.051	***	0.13	*	0.125	*	0.137	*
	0.058		0.048		0.051		0.05		0.053	
Kigezi	-0.22		0.701	**	-0.364		0.663	**	0.808	***
	0.222		0.204		0.229		0.209		0.196	
Post* Kigezi	**0.12**		**0.189**		**0.286**	**	**0.27**	*	**0.174**	*
	0.156		0.135		0.091		0.103		0.073	
Female	-2.045	***	-2.035	***	-1.864	***	-2.082	***	-2.189	***
	0.136		0.144		0.151		0.158		0.149	
Urban	2.657	***	2.663	***	2.654	***	2.831	***	2.859	***
	0.148		0.156		0.162		0.182		0.19	
Anglican	0.428	***	0.454	***	0.461	***	0.489	***	0.49	***
	0.084		0.088		0.091		0.096		0.1	
Muslim	-0.533	***	-0.5	***	-0.433	***	-0.465	***	-0.478	***
	0.085		0.09		0.097		0.098		0.097	
Other relig.	-0.481	**	-0.44	**	-0.361	*	-0.328	*	-0.285	
	0.144		0.145		0.144		0.157		0.166	
R^2	0.3		0.285		0.27		0.28		0.288	
N	6,116,704		5,469,774		4,526,849		2,371,661		969,341	

*p<0.05, ** p<0.01, ***p<0.001

Note: All regressions use adjusted standard errors clustered at the district level and employ Huber/White/sandwich variance estimates to adjust for heteroskedasticity and intra-district correlation, and are weighted by individual according to the IPUMS 10% sample of the 1991 Uganda Census. All regressions are run with district and birth-cohort fixed effects, along with binary variables for marital status and 23 ethnicity dummies. Ages 20-60 correspond to birth-cohorts 1931-1971, ages 20-50 correspond to 1941-1971, ages 20-40 correspond to 1951-1971, ages 25-35 correspond to 1956-1966, ages 29-33 correspond to 1958-1962.

Table 7: Change in probability of literacy and primary-school completion and percentage change in literacy and primary-school completion with covariates set to their reference levels using equation (1).

	Change in Prob.	95% CI		Mean (%)	Increase (%)	95% CI (%)	
Primary-School Comp.	0.063	0.0578	0.0674	12.00%	52.50%	48.17%	56.17%
Literacy	0.0044	0.0038	0.0049	54.00%	0.81%	0.70%	0.91%

Note: Probability changes estimated using the CLARIFY program with Monte Carlo simulations and covariates are set to their reference levels. That is, we estimate the change in an individual's probability of literacy and primary-school completion, investigating the probability change for a single, Catholic male, born in 1959, living in a rural area from the Baganda tribe to that individual's probability of literacy and primary-school completion if they had been born in Kigezi, post-eradication campaign.

Table 8: Summary of previous evidence on the impact of malaria eradication on educational and income outcomes compared to results from the present study (Cutler, et al, 2010, Bleakely, 2010, and Lucas, 2010).

Authors		Cutler, et al	Barofsky, et al	Bleakely				Lucas[t]	
DEPENDENT VARIABLES	**Country:**	**India**	**Uganda**	**US**	**Mex.**	**Col.**	**Brazil**	**Para.**	**Sri Lanka**
Years of Schooling	Males	X	++	N/A	X	X	X	N/A	N/A
	Females	X	X	N/A	N/A	N/A	N/A	++	++
Consumption / Income	Males	++	X	++	++	++	++	N/A	N/A
	Females	++	X	N/A	N/A	N/A	N/A	N/A	N/A
Literacy	All	X	++	N/A	++	++	++	++^	++^
Prim Comp	All	X	++	N/A	N/A	N/A	N/A	++*^	++*^
DATA									
Birth Place		No	Yes	Yes	Yes	Yes	Yes	Yes	Yes

KEY: N/A = Not applicable, X = No effect or mixed evidence over multiple specifications, ++ = Positive educational or income effects from malaria eradication, t Refers to decreases educational attainment for being in either in pre-epidemic or epidemic cohorts before malaria eradication in Sri Lanka and Paraguay, *Refers to years of primary schooling, ^ Regressions only include females.