Malaria Eradication and Economic Outcomes in Sub-Saharan Africa: Evidence from Uganda

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Abstract

This study evaluates the economic consequences of a 1959-1960 malaria eradication campaign in southwestern Uganda. The effort constitutes a rare attempt to eliminate malaria in sub-Saharan Africa and produced an immediate and large disease reduction. We utilize this quasi-experimental health shock to explore changes in educational and economic outcomes. Our analysis shows that eradication produced improvements in years of schooling, literacy, and primary-school completion with suggestive increases in socioeconomic status. Given that sub-Saharan Africa bears a disproportionate share of the world's current malaria burden, these results provide the best guidance available on the potential long-term economic impact of malaria eradication.

Keywords: malaria, malaria eradication, human capital, economic development, Uganda. I15, I18, I21, O15, O18.

1. Introduction

The damage that malaria inflicts on population health is severe and well established. With an estimated 350 to 500 million cases and over one million deaths per year, malaria represents a major threat to 3.3 billion people in over 100 nations (Christopher JL Murray, et al. 2012). Sub-Saharan Africa

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bears the bulk of the global malaria burden, with 71% of cases and 86% of deaths (WHO, 2009). It is estimated that anywhere from 30% to 50% of outpatient visits and hospital admissions in this region are a result of malaria illness, with severe cases leading to complications including anemia, seizures, coma, and death. Malaria also significantly aggravates the condition of HIV-positive individuals and increases HIV transmission. In this paper. we exploit quasi-experimental variation in childhood malaria exposure by examining the impact of an eradication campaign in southerwestern Uganda on educational attainment and household income.

Malaria exposure in childhood may affect human-capital formation and adult income in multiple ways. First, malaria among pregnant women and during infancy produces anemia and impedes in utero nutrition, which adversely impacts cognitive development (Betsy Lozoff and Michael Georgieff, 2006), delays the development of the central nervous system (John Beard, 2008), and reduces a child's ability to respond to environmental cues (Maggie Burhans, et al., 2006). Malaria exposure during childhood also worsens neurocognitive performance, including attention, memory, visio-spacial skills, and language function, making advancement through school more difficult (Michael Kihara, et al., 2006). Third, children in a household affected by malaria tend to have less educational attainment because of reduced income and greater care-giving demands. Finally, adult mortality attributable to malaria reduces the expected time that individuals can realize gains from human-capital investments, thereby decreasing incentives for schooling. Conversely, child mortality from malaria may raise economic outcomes by differentially affecting individuals that, if they had survived, would have exhibited lower educational attainment and adult income.

In addition to severe health consequences, nations with high malaria incidence also exhibit low levels of economic development (John Luke Gallup and Jeffrey Sachs, 2001; F. Desmond McCarthy, et al., 2000). John Luke Gallup and Jeffrey Sachs (2001), for example, estimate that wiping out malaria in sub-Saharan Africa could increase per capita economic growth by as much as 2.6% per year. In contrast, Daron Acemoglu and Simon Johnson (2007) utilize the reduction in mortality produced by the 1940s international epidemiological transition to identify the effect of increasing life expectancy on economic growth. They find no evidence that large health improvements produced any positive effect on per capita income.[2]

[2]The debate on the sign and magnitude of how health improvements affect income is ongoing. David Bloom, Canning, and Sevilla (2004) review 13 studies that investigate how national differences in health affect income variation at the macro level. They find similar qualitative results overall to their result that a one-year increase in life expectancy raises

Most closely related to this paper, three studies at the individual level investigate how malaria eradication affects income and human-capital formation in the Americas (Hoyt Bleakley, 2010), female educational attainment in Paraguay and Sri Lanka (Adrienne Lucas, 2010), and schooling and economic status in India (David Cutler, et al., 2010). These studies utilize the discovery of dichloro-diphenyl-trichloroethane (DDT) and the WHO-led worldwide malaria eradication campaign that followed as an exogenous health shock to identify the impact of health improvements as malaria was eliminated. Similarly, our analysis employs the sudden and nearly-complete elimination of malaria transmission in southwestern Uganda to identify the impact of disease reduction on educational and economic outcomes. Taken together, these previous studies find mostly positive effects of reduced childhood malaria exposure on adult income and educational attainment, although these results are orders of magnitude smaller than macro estimates. (Further discussion of these studies in the context of our findings is included in section V).[3]

None of these studies, however, estimate the effect of malaria eradication in sub-Saharan Africa, the region which bears the vast majority of the current malaria burden. The location of previous studies is important because the malaria strain most prevalent in sub-Saharan Africa, Plasmodium fal-

output by 4%. David Weil (2007) uses microeconomic estimates to evaluate the effect of better health on economic outcomes through worker productivity and his simulation indicates that eliminating health disparities between nations would reduce the variance of log GDP per capita by 9.9%. Lant Pritchett and Lawrence Summers (1996) instrument for variation in infant and child mortality and conclude that income-per-capita growth produces health improvements, but reject the notion that causality runs from health to income. David Weil (2010) surveys the evidence on the effect of disease control on GDP and finds "at best weak support for the claims that the disease burden in Africa significantly lowers GDP or that improving health would provide a big impetus to economic growth."

[3]Additional literature provides support for a statistically significant and positive effect of health improvements on education and income that is smaller than cross-country estimates. Sharon Maccini & Yang (2009) use variation in early-life rainfall to estimate the impact of health shocks on long-term well-being in Indonesia and find that rainfall 20% above average during early life for girls leads to reduced likelihood of self-reported poor health, 0.57 cm greater height, 0.22 more completed grades, and higher income. Specific to malaria, Sok Chul Hong (2011; 2013) combines Union Army health records from the US Civil War with socioeconomic data from the general population to estimate the impact of malaria exposure on health status and wealth accumulation. Hong (2011) finds that Union Army veterans enlisting from malaria-endemic US counties were up to 0.87 inches shorter than those from malaria-free counties, while Hong (2013) finds an association between malaria exposure and old-age disability. Alan Barreca (2010) found that malaria exposure in the US South during the early twentieth century reduced schooling by 0.26 years, representing 15% of the educational difference between the South and the rest of the US.

ciparum, produces health effects that differ substantially from the malaria prevalent in other regions (Simon Hay, et al. 2009). Therefore, these previous results may not generalize to current eradication efforts. Plasmodium vivax, the malaria strain most prevalent outside of sub-Saharan Africa, is primarily a chronic disease which causes fever and anemia, but rarely death. In contrast, P. falciparum produces morbidity, acute illness, and particularly cerebral malaria (Michael Boivon, et al., 2007; Richard Idro, et al., 2005) and death at a higher rate than P. vivax.

Given recent shifts from malaria control to increased funding for elimination and eradication, knowledge about the potential economic impact is particularly important (Robert Snow and Marsh, 2010). Indeed, aid to low and middle income nations for malaria control has increased from $230 million to 1.86 billion between 2000 and 2010 (IHME, 2012). However, even though this funding expansion has produced the most ambitious malaria control efforts since the original WHO initiative began in 1955, evidence on the potential long-term economic impact in sub-Saharan Africa remains relatively scant (Marie Coll-Seck, 2008; Mark Wilson, et al., 2012).

This paper intends to fill this evidence gap by exploiting a plausibly exogenous malaria eradication campaign in Uganda's southwestern Kigezi region to investigate long-term effects on educational attainment and economic status. During the years of 1959 and 1960, a program of DDT spraying and mass distribution of antimalarial medication rapidly interrupted disease transmission, producing variation in childhood malaria exposure by birth cohort. To identify the impact of malaria eradication, we employ a difference-in-difference methodology to compare changes in outcomes for the intervention district against changes in the rest of Uganda. Our primary results show that malaria eradication produced 0.3 more years of schooling in the intervention area compared to the rest of Uganda. This treatment effect represents a gain in schooling of 10% and 5% for males and females respectively and translates into a 3% to 11% overall income gain, depending on the rate of return to education assumed. We also find statistically significant improvements in primary-school completion and literacy, although the former effect constitutes a 50% increase, while the latter does not represent an economically important change. Importantly, we find that these educational effects are larger in areas with higher pre-treatment malaria incidence, as expected. Finally, we construct an asset-index to proxy for household socioeconomic status and find suggestive increases in this measure of income.

This paper contributes to the existing literature on the long-term economic effects of malaria eradication as the first to produce estimates in

sub-Saharan Africa.[4] Second, given differences in the health effects of P. falciparum compared to P. vivax malaria, this analysis elucidates additional health-to-wealth channels than those evaluated in previous studies. That is, because of P. falciparum's larger effect on mortality, we can test whether the selection effect dominates, such that reduced child mortality from malaria actually lowers economic outcomes in adulthood. Acemoglu and Johnson (2007), in analyzing a health intervention that sharply increased life expectancy, argue that this selection effect in part explains their null results. We find that eradication of P. falciparum malaria in sub-Saharan Africa can produce positive long-term economic outcomes even while significantly reducing child mortality. Third, this analysis contributes to the health-to-wealth debate, by testing the economic impact of malaria eradication in the area where it produces the largest negative health effects. Consistent with previous papers in this literature, we find a positive and economically significant effect from eradication, but results orders of magnitude smaller than previous macro estimates.

This paper continues as follows: section II provides background on malaria and the eradication campaign, section III describes our methodology and main treatment effects, section IV outlines multiple tests of the treatment effect's robustness, section V interprets our results and discusses them in the context of other work, while section VI concludes.

2. Malaria Eradication in southwest Uganda

Launched in 1955, the WHO's global malaria eradication campaign eliminated the disease from Europe, North America, the Caribbean, and parts of Asia and South-Central America. The effort was abandoned in 1969 due to the challenges of eradication in sub-Saharan Africa (SSA), caused in part by increasing mosquito resistance to DDT and heightened parasite resistance to chloroquine treatment. The campaign studied here was intended to test

[4]Other studies answer related questions. Alfredo Burlando (2009) exploits differences in village elevation as an instrument for disease exposure in central Ethiopia and finds that a 10% increase in village malaria is associated with a reduction of 0.25 in years of schooling. Nava Ashraf, et al., (2010) explore the association between recent efforts at malaria reduction in Zambia and individual level health, finding stronger associations between health and bednet provision than for regional spraying operations. A series of randomized trials have also been conducted or are in the field to identify the short-term effect of malaria prevention on educational outcomes. Results so far from these trials in coastal Kenya show that malaria prevention improves school attendance, cognition, and a child's ability to sustain attention in class (Simon Brooker, et al. 2000; Sian Clarke, et al. 2008; Simon Brooker, et al. 2010; Katherine Halliday, et al. 2012).

the feasibility of eradication in SSA after these successes were achieved in peripheral malaria regions. It is, to our knowledge, the only large-scale effort to eliminate malaria in SSA. Previous to this program, the region was excluded from the WHO's campaign due to intense transmission and lack of infrastructure.

The malaria eradication campaign under study took place between 1959 and 1960 in the Kigezi district of southwestern Uganda. This area exhibits significant variation in topography, which in turn determines malaria endemicity. The district can be divided into three zones: the flatlands of the north, the highlands of the center and south, and the high mountains of the extreme south (Zulueta et al., 1964). To track baseline malaria incidence and to monitor operational success, surveys to estimate the rate of enlarged spleen in the population, a measure of long-standing malaria infection, and the rate of parasite infection were carried out monthly before and after spraying, and fever surveys were carried out monthly at visits to dispensaries (Zulueta, et al., 1961). Survey results were used to assign malaria endemicity to areas within Kigezi, in accordance with the classification scheme recommended by the WHO Expert Committee on Malaria (fourth session, 1950). A map of malaria prevalence in Uganda (Figure 1) and Kigezi district (Figure 2) at the time of the experiment illustrates pre-eradication malaria variation. Most of the northern part of Kigezi district was classified as hyperendemic, meaning that a marker of malaria infection, the rate of spleen inflammation among children 2 to 10 years old, was consistently over 50% and permanently high for adults as well. Some villages in this area were measured to have rates of child spleen inflammation greater than 75% throughout the year (Zulueta et al., 1961). The southern areas of the intervention district are situated at a higher altitude, mostly above 3,7000 feet, and classified as mesoendemic, meaning that the measured rate of spleen inflammation among children was found to be between 11 and 50%. Hyperendemic areas around the lakes of the southern area were also observed.

As of the 1991 Census, Although defined administratively as one region during the eradication campaign, by the 1991 Census, the intervention region had been split into two districts that, conveniently for the purposes of our analysis, correspond to the higher versus lower pre-eradication incidence areas. The high pre-eradication malaria incidence district, Rukungiri, was created in the north and the lower incidence district, Kabale, in the south (see Figure 2).[5] In addition to comparing the impact of eradication in Kigezi

[5]Figure 2 shows the variation in pre-eradication incidence and Uganda's district borders as of 2010. For the 1991 Census, Kisoro and Kabale district were combined into one district

versus the rest of Uganda, we also use variation in pre-eradication malaria incidence within the intervention area to verify our findings. Although the eradication did not occur in Uganda's most malarious zones, the nation overall exhibited the highest malaria incidence in the world at 47.8 percent in 2005 (WHO, 2005).

Pre-eradication parasite surveys in the treatment district of Kigezi showed that in hyperendemic areas, 82% of cases were P. falciparum and 17% were P. malariae, while none were vivax. In the mesoendemic areas, the numbers were 91% P. falciparum and 9% P. malariae (Zulueta et al., 1961). This confirms both that the malaria burden in the intervention district area corresponds to the rest of SSA. meaning these results can indeed be generalized across the continent, and differs from the malaria strains analyzed in previous studies on eradication.[6]

The eradication campaign consisted of DDT spraying in human and animal dwellings along with mass distribution of antimalarial medication. 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. Standard treatment under the global malaria campaign only included DDT spraying, but mass distribution of antimalarial medication was carried out as well to completely interrupt malaria transmission. 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% (Zulueta et al., 1961).

In 1959, the Kigezi region of southwestern Uganda had a population of 493,000, according to a census from that year, situated on 1,969 square miles (Zulueta, et al. 1961). Northern Kigezi had a population of 59,000 in 500 square miles pre-eradication, while southern Kigezi supported a higher population density of about 434,000 in an area of 1,500 square miles. Consistent with a substantial decrease in malaria's mortality burden, Zulueta, et al.

called Kabale.

[6]There are four human malaria parasites (P. falciparum, P. vivax, P. malariae, and P. ovale). Cutler et al. (2010) mentions that data on prevalence by malaria type pre-eradication are not available for India, but post-eradication data suggest that about 30% of cases were P. falciparum. Bleakley (2010) explains that P. vivax and P. malariae were prevalent in the Americas pre-eradication, and Lucas (2010) states that both Sri Lanka and Paraguay's malaria came primarily from P. vivax pre-eradication.

(1961) mention a large increase in the population of northern Kigezi after the first DDT spraying campaign. The authors state that "the great increase observed was probably due to the better health conditions brought about by the introduction of DDT" and not migration unrelated to the malaria eradication effort. In addition, as explained below, we use district of birth to identify the impact of eradication instead of current district, thereby mitigating concerns that migration drives our results.

3. Empirical Analysis

3.1. Main Specification

We use data from Uganda's 1991 Census provided by the *Integrated Public Use Micro Sample* (IPUMS, 2007) and our intervention area is therefore defined based on district definitions at the time of the census. As noted above, by 1991 the intervention area had been divided into two separate districts, Rukungiri in the north and Kabale in the south. Pre-intervention birth cohorts are defined as those born prior to 1960, while post-intervention cohorts are born in 1960 and after. The 1991 census is a weighted 10% sample of Uganda's population. The census contains information on years of education, binary indicators for primary-school completion and literacy, and a set of asset questions that we combine into an index of household assets as a proxy for socioeconomic status.

We employ a difference-in-difference (DD) methodology to estimate the impact of malaria eradication on human capital attainment and household assets. This approach compares the differential change in outcomes pre- and post-eradication for individuals born in the intervention area against the change in outcomes for those born in other Ugandan districts. Our main specification is estimated in the following form, for individual i, in birth cohort c, and district d:

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

In this equation, K_d represents a binary variable for birth in Kigezi district, P_c represents an indicator for birth post-intervention, and $K_d * P_c$ represents our treatment variable, the interaction term for being born both in the treatment district and after the eradication occurred. Equation (1) also includes district δ_d and birth-cohort μ_c fixed effects, to control for time-invariant social or environmental characteristics by district and year that may be correlated with both intervention status and outcome. Finally, equation (1) controls for individual-level characteristics in matrix $\mathbf{X}$, such as gender, urban status, religion, marital status, and ethnicity. Our coefficient of interest is β_3 and represents the differential change in outcomes pre- versus

post-eradication for those born in the intervention district compared to the change for those born in other areas of Uganda. In addition to using years of schooling as our primary outcome, we also estimate the effect of eradication on primary school completion and literacy with a probit model.[7]

As with any DD analysis, our essential identification assumption is that the rest of Uganda represents an appropriate control group for the intervention district of Kigezi. That is, we assume these two areas would have had the same outcome trajectory absent the intervention. Table 1 shows dependent and independent variables of interest and compares their averages before and after the eradication program in 1960 for both Kigezi and the rest of Uganda for cohorts born between 1931 and 1971. We observe that the intervention area exhibits lower educational attainment both before and after eradication. We also observe that the increase in years of education pre-versus post-eradication in Kigezi was 1.52 years, while in the rest of Uganda it was 1.56. The same pattern is found for literacy and primary school completion and we therefore proceed using parametric analysis in the following to further explore the treatment effect. 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 educational variables for Kigezi versus the rest of Uganda by birth cohorts from 1941 to 1971. These figures show that although age-heaping (the tendency for lower education individuals to report their ages as ending with a zero or five) drives the variation in education, the rest of Uganda follows a broadly similar trend as the Kigezi and therefore constitutes an appropriate control group for the intervention area.

3.2. Effect on Educational Attainment

Table 2 shows results from estimating equation (1) for dependent variables: years of education, literacy, primary-school completion and an index of household assets used to proxy for socioeconomic status (SES, discussed below). We find that the eradication campaign had a positive and significant effect on educational outcomes for all three education variables using a sample of individuals born between 1951 and 1971. Specifically, we find a treatment effect of 0.29 years in column 1, indicating that the differential increase in years of schooling for those born in the treatment area after the

[7]As clarified by Chunrong Ai and Norton (2003), the interaction term effect in nonlinear models is equal to the cross derivative of the expectation of the outcome and not the commonly calculated marginal effect of the interaction term. Therefore, we employ the CLARIFY software, discussed below, to correctly estimate the magnitude of the interaction effect with a nonlinear version of equation (1).

eradication campaign compared to the rest of Uganda amounts to almost 0.3 school years, after controlling for individual-level, year, and district covariates. Given that total years of schooling in Kigezi for birth cohorts 1951-1971 averaged 3.59, this represents an 8% increase in educational attainment attributable to malaria eradication. 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 (Mincer, 1974), this β_3 estimate implies that eradication produced a 2.9% average increase in yearly earnings. Running equation (1) separately for males and females produces a statistically significant β_3 coefficient of 0.49 for males and statistically insignificant coefficient of 0.12 for females (results not shown). Mean years of schooling for birth-cohorts aged 20 to 40 in Kigezi was 4.75 and 2.54 for males and females, respectively. Therefore, these results indicate that malaria eradication was associated with an increase in years of schooling of over 10% for males and 4.7% for females.

In column 2, being born in Kigezi after the eradication is associated with a β_3 coefficient of 0.158 for primary school completion and 0.065 for literacy, both statistically significant. To interpret the magnitude of these effects in quantities of interest, we estimate the change in probability associated with being born in the intervention area post-eradication using Monte Carlo simulation.[8] Table 3 presents the effect of eradication translated into probability changes for literacy and primary-school completion. Coefficient estimates from equation (1) imply that eradication produced a precisely estimated, but economically insignificant change in literacy of 0.4 percentage points or 0.8%. In contrast, we find that equation (1) implies a 0.063 percentage point or 53% rise in primary-school completion, given that 12% of individuals completed primary school in Kigezi between 1951 and 1971. The discrepancy in the percentage change treatment effect for literacy and primary-school completion can be explained by the fact that the latter is a threshold measure corresponding to five years of education, while the former is a measure of basic educational attainment that in fact may be acquired outside of school. Given that average years of schooling pre-eradication was 3.2 and 2.4 in the rest of Uganda and Kigezi, respectively, the observed 0.29 years-of-schooling increase is consistent with a shift around the modal part of the schooling distribution, pushing a large percentage of individuals above the primary-school threshold.

[8]The change in probability of literacy and primary-school completion associated with the intervention is calculated using the CLARIFY program (Tomz, Wittenberg, & King, 2003; King, Tomz, & Wittenberg, 2000). which draws parameters from the asymptotic distribution of equation (1) while model covariates are set to their mean or modal category.

In addition, we estimate equation (1) for each district within Kigezi separately, using the variation in malaria incidence pre-eradication to validate our results, with the hypothesis that the area with higher malaria prevalence (Rukungiri) should exhibit larger educational gains compared to the lower malaria district (Kabale). Indeed, table 4 shows that individuals born post-eradication in the pre-treatment high malaria area 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 (excluding Rukungiri). 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.

3.3. Birth Cohort Analysis

In addition to the pre- versus post-eradication analysis, we also examine the treatment effect by birth cohort over time. Following Cutler, et al. (2010), we plot cohort-specific relationships between the treatment district Kigezi (and its component districts of Rukungiri and Kabale separately) and years of schooling. This specification intends to test the timing of the treatment effect we identify. If the educational effect we find is indeed produced by malaria eradication, we would expect to observe the differential effect on those born in the intervention district at the time of the eradication project and not before. We estimate these cohort-specific relationships using the following equation:

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

In equation (2), the coefficient β_c represents the differential cohort-specific relationship between being born in the treatment area and years of schooling compared to the rest of Uganda. Other variables are defined as before from equation (1). Subscript c indexes birth cohort and, to minimize age heaping (to be discussed further below), refers not to specific birth years, but 5-year age categories centered on ages ending in zero and five (years ending in 1 and 6).

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 2-6 during treatment also experience positive educational gains, then

we expect this trend break to occur for cohorts in early childhood during eradication. We also run specification (2) separately for each district - Rukungiri and Kabale - to explore which specific district produced the largest increase in educational attainment and whether the timing of this effect coincides with our priors. We would expect a larger increase in educational outcomes for Rukungiri given its higher malaria incidence pre-eradication. Equation (2) is run with years of education as the dependent variable.

Figure 5 plots the β_c coefficients for all 5-year cohorts born between 1929-1933 and 1969-1973 (ages 18-62) using equation (2). These results indicate that the Kigezi region was not 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 the treatment area were losing educational ground compared to the rest of Uganda before eradication. However, after losing ground throughout the 1940s and 1950s, the intervention district begins to reverse fortunes, starting with birth cohort 1959-1963, years that correspond nearly perfectly to the malaria eradication campaign. This strengthens our intuition that the results we see in the main specification are robust to the specific definition of eradication year chosen, since the timing of the improvement in years of schooling follows our expectations. Moreover, we find that the average increase in schooling for birth cohorts born between 1954 and 1973 is larger for the more-malarious district of Rukungiri than for the less-malarious district of Kabale and that the trend break increase in educational outcomes for both districts is also observed when the eradication campaign began.

3.4. Partial Exposure

To investigate the effect of partial exposure to malaria 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. This method follows Bleakley (2010)'s estimate of partial-exposure effects. Cohorts born in 1960 or after have the maximum of five years exposure to malaria eradication. Five years is chosen because evidence suggests that malaria's most important cognitive and health impact occurs in early childhood. We also 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 $K_d * P_c$.

Employing this partial-exposure approach to estimate the impact of malaria reduction, we find that the estimated coefficient on β_3 is significantly reduced when we use partial exposure 5-years before eradication (not shown). 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. 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 impacts in utero and during infancy on cognitive development, instead of reduced malaria exposure in early childhood.

3.5. Effect on Socioeconomic Status

Uganda's 1991 Census does not ask income questions directly. However, it provides 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 is used (following Filmer & Pritchett (2001)). 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 categorical nature of our proxy measure for household income, we employ an ordinal probit model instead of an OLS specification to measure the intervention's effect on SES.

Returning to table 2, column 4 shows the results from running equation (1) with our household asset index as the dependent variable using an ordinal probit model. We find that the intervention is associated with an imprecisely measured increase in SES (p-value 0.18). Although suggestive of an impact on household well-being, this measure of income is not sufficiently accurate to make a determination on how eradication effects income using our main specification equation.

4. Robustness Checks

4.1. Age Heaping

As is common in data from developing nations, Uganda's 1991 Census suffers from severe age heaping. That is, individuals round their age to the nearest number ending in zero or five since they are often unsure of their birth year. This occurs differentially for the poorest and lowest-educated respondents, resulting in artificially low average educational levels for ages ending in zero or five. Given that the identification strategy employed here utilizes variation in malaria exposure by birth-cohort, incorrectly reported years of birth may drive the educational results we find instead of a true effect.[9]

To adjust for age-heaping, we re-run equation (1) using 5-year birth cohort categories instead of 1-year categories. 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. These regressions are run for ages 18 to 42, instead of ages 20 to 40. Table 5 shows results for the four dependent variables as in table 2 and we observe treatment effects for educational outcomes that do not differ substantially from those found in table 2 using 1-year birth cohorts. In addition, in this specification, we observe that the treatment effect estimated for our asset-index proxy for SES increases by more than 50% and becomes statistically significant at the 10% level (p-value of .065). This adds to the suggestive evidence that malaria eradication also raised income in the intervention district.

4.2. Placebo Tests

Marianne Bertrand, Duflo, & Mullainathan (2004) 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 female wages from the Current Population Survey 1979 to 1999 and 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 Bertrand, Duflo, & Mullainathan (2004) and randomly generate placebo

[9]To understand the extent of age misreporting in these data, over 4,000 individuals report their age as 30 while about 1,000 report their age as 29 and 31.

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 the absence of any interventions that affect outcomes, 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 on results from Bertrand, Duflo, & Mullainathan (2004), we would expect the actual rejection of the null hypothesis to be much higher. We interpret the set of interaction terms from the 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 $\beta_3 = 0$.[10] 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.

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 shows 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

[10] See Raj Chetty, Looney, & Kroft (2009) for a more detailed explanation of this method.

performed, all the placebo tests with $\beta_{3,p} > \beta_3$ come from 6 districts, while all the placebo tests with $\beta_{3,p} > \beta_{3,R}$ come from 3 districts. The fact that only a few districts account for all the coefficients in which $\beta_{3,p} > \beta_3$ suggests 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, Uganda gained independence from the British in 1962, close to the time of the eradication program under study. We would expect this change in government to produce public spending effects that would differentially benefit some districts in Uganda compared to others. Although we cannot completely rule out that the results we find in Kigezi were also produced by this differential stimulus, the malaria eradication campaign was, to our knowledge, the largest public-policy investment in the intervention district over this time period.

4.3. Ignoring Time Series Information

Another way that Bertrand, Duflo, & Mullainathan (2004) overcome serial correlation in DD analysis is to ignore the data's time-series structure altogether. Doing so, the authors produce DD estimates in which the null hypothesis of no effect is rejected in approximately 5% of cases. Applying this method here, we first regress educational outcomes on the individual-level covariates from equation (1) . Then, we collect the residuals from this regression and average over birth-cohorts before and after the eradication campaign by district, calling the residuals ν_{cd}, and regress ν_{cd} on the rest of the variables from equation (1). ν_{cd} can be interpreted as district-level variation in educational attainment not explained by differences in individual covariates. The specific equation estimated at the district level is:

$$\nu_{cd} = \beta_0 + \beta_1 K_d + \beta_2 P_c + \beta_3 K_d * P_c + \beta_4 \delta_d + \gamma_{cd} \tag{3}$$

where these variables have the same meanings as in (1). Since there are a total of 35 districts in Uganda, equation (3) 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.

Table 6 displays the results of this procedure. 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 a larger treatment effect in column 1, a 0.4

years-of-schooling increase, compared to our baseline specification in table 2. Although, the treatment effect 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, Duflo, & Mullainathan (2004) identify as a threat to hypothesis testing using a DD methodology.

In addition, we perform the same placebo test as described above with years-of-schooling as the outcome variable, for placebo district-year pairs and equation (3). 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 shocks and that the treatment effect we find is a result of malaria eradication and not random variation.

4.4. Varying the Birth Cohort Window

We perform an additional sensitivity test to determine whether the treatment effects found are robust to variation in the birth-cohort window used. In addition to the baseline specification where ten years pre- and post-eradication are used, we estimate equation (1) using birth cohorts from 1931 to 1971, 1941 to 1971, 1956 to 1966, and 1958 to 1962. It is expected that the estimated β_3 will increase as we narrow the window if eradication produced the positive educational effect observed. That 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 treatment effect downward through attenuation.

Table 7 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 treatment effect. When we narrow the birth-cohort window even more to 1958-1962, we still find a significant β_3, but diminished educational effect. This analysis shows an educational effect when expected (as the birth-cohort window narrows), again suggesting that the human-capital impact we find is indeed due to the eradication campaign under study.

5. Discussion

5.1. Interpretation

To translate our main treatment effect on years of education into income gains, we utilize estimates on rates of return to education (ROREs).[11] Generally, reviews of the ROREs literature suggest that the highest returns to education occur during primary school and in developing nations, especially Africa. In a recent review, Pscharopoulos and Patrinos (2004) calculate that the social and private RORE in Africa during primary school is 25.4% and 37.6%, respectively. These rates represent the highest ROREs of any region.[12] Using this private RORE, our treatment effect of 0.286 years of schooling would imply a 7% and 11% social and private annual earnings return, respectively, while, as noted above, the standard Mincerian estimate of a 10% RORE implies an almost 3% yearly income gain from malaria eradication.

Although we also estimate that malaria eradication positively affects income using a household asset index, this is an approximate measure of underlying economic well-being and the results are significant at the 10% level and only under some specifications. Instead of analyzing these point estimates, we note that they are always positive, and prefer to interpret this as qualitative evidence on the sign of malaria eradication's effect on income, even with a large child mortality effect. That is, even though malaria elimination in SSA averts many more deaths under five years old than previously evaluated malaria eradications, we still find evidence of positive income effects. At the least, this suggests a refutation of findings such as Acemoglu and Johnson (2007), inter alia, which assert that health interventions that raise life expectancy either produce no effect or can reduce per capita economic growth.

A useful way to compare results across papers is to rescale regression coefficients to determine the effect of malaria eradication in terms of malaria incidence, as in Cutler, et al. (2010), and Bleakley (2010). To do this, we follow Bleakley (2010), but normalize our reduced form estimates of malaria eradication's effect on years of schooling, instead of income. To perform this calculation, we compare the treatment effects between the high-malaria intervention area (Rukungiri) against the low-malaria area (Kabale) from table

[11] See George Psacharopoulos (1994) and George Psacharopoulos & Patrinos (2004) for summaries.

[12] Nevertheless, other work cautions that the data in SSA to estimate ROREs are poor quality, unrepresentative (covering only 18 of 46 SSA nations), and suffer from sample selection within nations using only formal sector employees (Bennell, 1996).

4. Instead of comparing the treatment district overall to the rest of Uganda, this analysis exploits variation in pre-eradication incidence within the intervention district. We found in table 4 that the higher pre-eradication malaria incidence area exhibits a 0.262 larger increase in years of schooling compared to the low-malaria district. Given the classification of malariousness from Figure 2, we define these areas as primarily hyperendemic and mesoendemic respectively, which corresponds to approximate malaria incidence rates of 0.625 and 0.3 (Louis Molineaux, 1988). Then, we divide the difference in treatment effects, 0.262 years of schooling, by the difference in approximate malaria incidence between the two areas, 0.325, to obtain an estimate of an educational effect per probability of malaria infection. This yields an estimated increase of 0.81 years of schooling per probability of malaria infection. Using the private RORE for primary education in SSA of 0.376 to translate this educational effect into income gains implies that being infected with malaria during early life increases adult income by approximately 16% per year. This estimate drops to 4% if a 10% RORE is used to translate the educational effect into income changes (we compare this income effect to other papers below).

5.2. Mechanisms

In this section, we detail mechanisms that may be driving the human capital results observed. Theory predicts ambiguous effects on schooling from malaria eradication because while cognitive improvements increase returns to schooling and less illness raises school attendance, being healthier also tends to increase a child's ability to earn wages (the opportunity cost of schooling). Consistent with this opportunity-cost argument, Atheendar Venkataramani (2012) uses the same data as Bleakley (2010) in Mexico and finds that early life exposure to malaria eradication is associated with improved adult cognitive function and earlier school entry and exit, without any change in overall schooling. However, the expected difference in the impact of malaria eradication for P. falciparum versus P. vivax is also theoretically ambiguous. It is uncertain, a priori, whether the larger mortality impact of P. falciparum eradication would tend to increase or decrease incentives to education because this depends both on the extent and magnitude of mortality selection from child and adult mortality. Reduced child mortality would tend to increase the magnitude of mortality selection and reduce years of schooling, in addition to raising population growth and potentially overwhelming school systems, lowering educational outcomes in general equilibrium. However, by extending the time over which returns to human-capital investments can be realized, averted adult mortality would tend to raise incentives for schooling.

Epidemiologically, most estimates suggest that the vast majority of mortality from malaria occurs among children under five years of age. The WHO calculates that close to 90% of current malaria mortality occurs among children under five (WHO, 2012), however some dissent exists on the relative importance of adult versus child mortality from the disease.[13] Given that most of the averted mortality from malaria occurs during childhood, we would expect that the mortality-selection effect to attenuate the benefits of eradication produced through reduced morbidity. Our positive educational results therefore indicate that either selection produces a small effect in practice or that the morbidity averted in SSA is also proportionately larger than for malaria eradications in non-SSA nations. If the larger estimates of malaria's impact on adult mortality are indeed accurate, then increased life expectancy may also raise returns to human capital and mitigate any mortality selection effect. Overall, since we find consistent increases in years of schooling, we can conclude that the cognitive benefits and increased incentives for education from malaria eradication together overwhelm the effects of mortality selection and increased wages from less illness.

The lack of impact on birth-cohorts partially exposed to malaria eradication is also worth comment. This small effect for the cohorts exposed after infancy suggests that the most important channel for improved education comes from improved cognition, instead of a greater ability to attend school and concentrate when in attendance. This small partial-exposure effect, however, could be produced for two reasons. One is that malaria in early childhood does not substantially impede schooling and so its reduction produces little educational improvement. The other, more plausible reason given our clinical knowledge of malaria's effect on childhood illness, is that the mortality selection effect from P. falciparum eradication approximately cancels the educational benefits from less illness during early childhood. Nevertheless, the same forces apply for cohorts exposed to malaria eradication in utero and infancy, indicating that the cognitive benefits from eradication are in fact larger during this period than for early childhood.

[13]The two main estimates of malaria deaths in 2010 come from the WHO and the Institute of Health Metrics and Evaluation (IHME, Murray, et al. 2012). Both estimates indicate that approximately 90% of malaria deaths occur in SSA. However, the IHME estimates are substantially larger overall, over one million compared to 660,000 from the WHO. Both reports also show more under five deaths than over five deaths from malaria. However, the WHO suggests a ratio of under-five to over-five deaths of approximately 10 to 1, whereas the IHME reports a ratio closer to 1.75. That is, nearly all of the difference in overall mortality comes from variation in estimates of adult mortality attributable to malaria.

5.3. Results in Context

This section compares our results to those of Cutler et al. (2010), Bleakley (2010), and Lucas (2010), while table 8 summarizes the qualitative findings of each study. Lucas (2010) focuses exclusively on the educational effects of malaria eradication in Paraguay and Sri Lanka for females. She finds that a 10 percentage point decrease in malaria incidence is associated with a 0.1 year increase in schooling and a 1 to 2 percentage point increase in the probability of literacy. Given that we find a positive, but not statistically significant effect on female educational attainment nor an economically important literacy effect, our results differ somewhat and may be a reflection of the greater impact of mortality selection in our context. However, for years of education, Lucas (2010) reports that if her results were applied to Uganda (chosen because it represents the highest malaria incidence in Africa) they would imply an increase of 0.5 years of schooling. This treatment effect is, in fact, quite close to the treatment effect of 0.48 we find in the high malaria area of the intervention district. Although Lucas (2010) looks specifically at females, while our result apply to males and females together, these results produce effects of a broadly similar magnitude. Translating her results using changes in enlarged spleen rate, Lucas (2010) also finds that a ten percentage point decrease led to an increase of between 0.39 and 0.93 years of schooling. To obtain a similar estimate for our results we use coefficients on the years-of-schooling treatment effect of malaria eradication in the high- and low-malaria areas of the intervention district from table 4 and normalize them by the change in spleen rate reported after the eradication campaign in hyperendemic and mesoendemic areas. We find that for the high- and low-malaria areas, a 10 percentage point decrease in the rate of enlarged spleen produces a 0.089 and 0.126 increase in years of schooling, respectively. Although smaller and, again, for males and females instead of females alone, our results normalized by spleen rate are also of an approximately similar magnitude to those found in Lucas (2010).

For educational outcomes, our findings contrast with those of Cutler, et al. (2010) which finds no statistically significant impact of eradication on educational outcomes (literacy and primary-school completion) for men and mixed evidence for women depending on the specification. Meanwhile, Bleakley (2010) investigates the effect of malaria eradication campaigns on education for males in Colombia, Brazil, and Mexico and finds that eradication is associated with increases in years of schooling in Colombia and Brazil, while most specifications find a decline in Mexico (although none of Mexico's results are significant). For literacy, Bleakley (2010) finds differentially larger increases in literacy rates for all three nations in high compared to low malaria areas pre-eradication.

For income, Cutler et al., (2010) find that the eradication campaign produced modest increases in household per capita income for men aged 20 to 60 and that the effects are larger for men than women. In malaria incidence terms, findings from various models imply a 15% to 68% increase in per-capita consumption per probability point of childhood malaria infection. Bleakley (2010) finds results on income of roughly the same magnitude. In terms of malaria incidence, he calculates that persistent childhood malaria infection reduces adult incomes by approximately 50% across the nations he studies. Bleakley (2010) also decomposes the income improvement attributable to malaria eradication from changes in schooling and finds that approximately 25% and 10% of the increase in income for Brazil and Colombia, respectively, comes from educational improvements. In Mexico, however, exposure to malaria eradication was associated with reduced schooling. By translating our educational results into income effects above, we found increases of 4%-16% in malaria incidence terms, depending on the RORE employed. These results again are of a similar size to the income effects found by Bleakley (2010) that are attributable to educational gains. Nevertheless, we should emphasize that Bleakley (2010) and Cutler, et al. (2010) utilize direct measures of income whereas we are using ROREs and an asset-index proxy of household SES.

5.4. 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 the treatment area of 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 the intervention area differently from other districts in Uganda.

However, threats to attributing causality in this case do exist. The age-heaping or auto-correlation in our data could be causing the effect we observe. Through extensive robustness checks we intend to avoid the possibility that the effect we find is caused either by measurement error or serial correlation. By controlling for age-heaping, performing placebo district-year tests, and ignoring time-series information, 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 expulsion 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, 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 like the discovery of DDT and implementation of the WHO's worldwide malaria eradication program. 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 campaign.

The data in the present study are advantageous relative to similar studies because we use place of birth instead of current residence, thereby reducing concerns that our results are driven primarily by migration.[14] We also have well estimated pre-eradication information on spleen inflammation and malaria parasite rates 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 whereas Lucas (2010) focuses on females alone.

However, this study also has disadvantages 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. Because of the use of one region, we note that the main specification compares the intervention district to the rest of Uganda, which we argue constitutes a reasonable control group to estimate the effects of the eradication campaign. This however represents a difference from the identification strategies of other papers in this literature. We intend to mitigate this concern by comparing educational results between the pre-eradication high and low malaria areas.

6. Conclusion

The preceding analysis evaluates the educational and economic impact of a malaria eradication campaign in southwestern Uganda. The program was

[14]Bleakley (2010) and Lucas (2010) use place of birth whereas Cutler, et al. (2010) employ district of current residence as a proxy for district of birth.

implemented in conjunction with the WHO's global eradication effort and produced quasi-experimental variation in health that we exploit to identify treatment effects. We find that malaria eradication produced an increase in schooling of nearly 0.3 years, corresponding to an 8% increase overall and 10% and 5% increases for males and females, respectively. This educational improvement corresponds to an annual income gain of between between 3% and 11% overall, depending on the rate of return to education used. We find statistically significant increases in literacy and primary-school completion, although the former effect is not of economic importance, while the latter result represents a 50% rise. This educational effect is found to be robust to a host of additional sensitivity tests, including controlling for age-heaping, placebo tests on the timing of the intervention, serial correlation in outcomes by birth cohort, and variation in the birth-cohort window employed. Importantly, we also find larger treatment effects in areas with higher pre-eradication malaria incidence. Comparing educational gains between these high- and low-malaria districts and normalizing by the probability of infection, we find a 4% to 16% income gain in Uganda, depending on the rate of return to education used. The magnitude of our treatment effects generally similar to those found in other studies in this literature. To measure changes in household socioeconomic status directly, we create an index of household assets and find suggestive income improvements, significant at the 10% level under some specifications.

This paper provides the only evidence on the long-term human capital and economic effect of malaria eradication in Africa south of the Sahara. Since the vast majority of the current malaria burden occurs in this region, our findings are the most relevant for predicting the impact of investing in malaria eradication today. Until recently, public health efforts focused on access to anti-malarial medicine and malaria control. However, eradication and vaccine development is again being seriously considered in policy circles (Wilson, et al., 2012).[15] Our results show that, even in sub-Saharan Africa, cheap policies such as the provision of insecticide-treated bednets could actually pay for themselves when long-term educational and income effects are taken into account. Moreover, this analysis indicates that the cost-effectiveness of eradication programs should be reevaluated while including the long-term benefits we find here.

Consistent with previous findings, our results show that fighting malaria

[15]Malaria vaccines have shown promise, with some phase three clinical trials indicating a 50% rate of malaria reduction in field tests among African children (Agnandji, et al., 2011), while near complete protection was observed in a very small patient population when inoculated with a weakened form of P. falciparum (Seder, et al. 2013).

on its own will not pull African nations of out poverty. However, they also indicate that, contrary to theoretical predictions and literature on health interventions that reduce mortality, malaria eradication campaigns in sub-Saharan Africa can indeed induce positive long-term human capital and economic effects. Given the magnitude of the utility benefits generated from increased life expectancy (Gary Becker, et al., 2005) when fighting this disease, our results provide additional support for interventions that reduce the burden of malaria in sub-Saharan Africa.

Disclaimer: The views expressed herein are those of the authors, and may not be attributed to the Economic Research Service or the U.S. Department of Agriculture.

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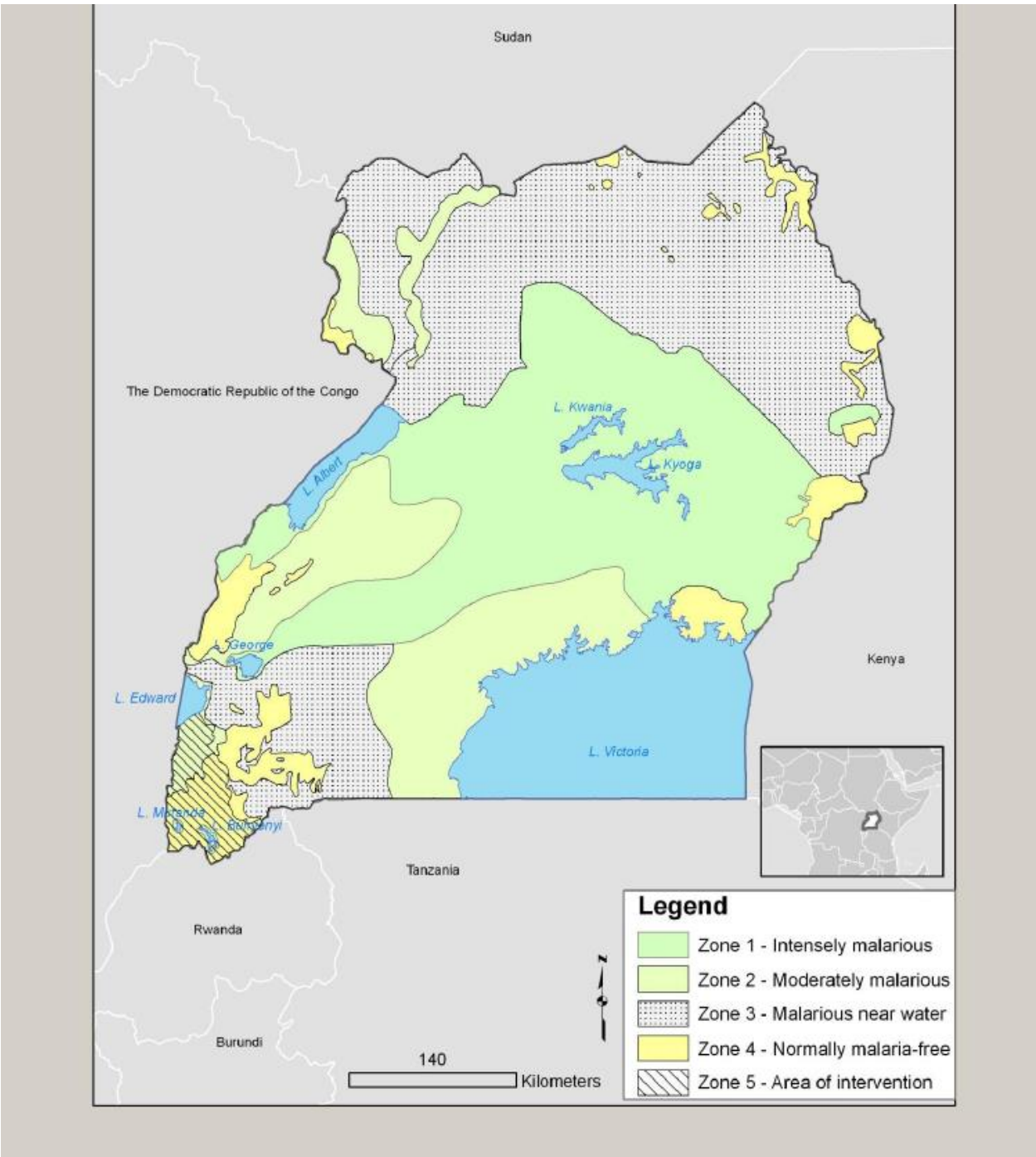

Figure 1: Pre-eradication Malaria Prevalence in Uganda.

Source: Zulueta, et al. (1964)

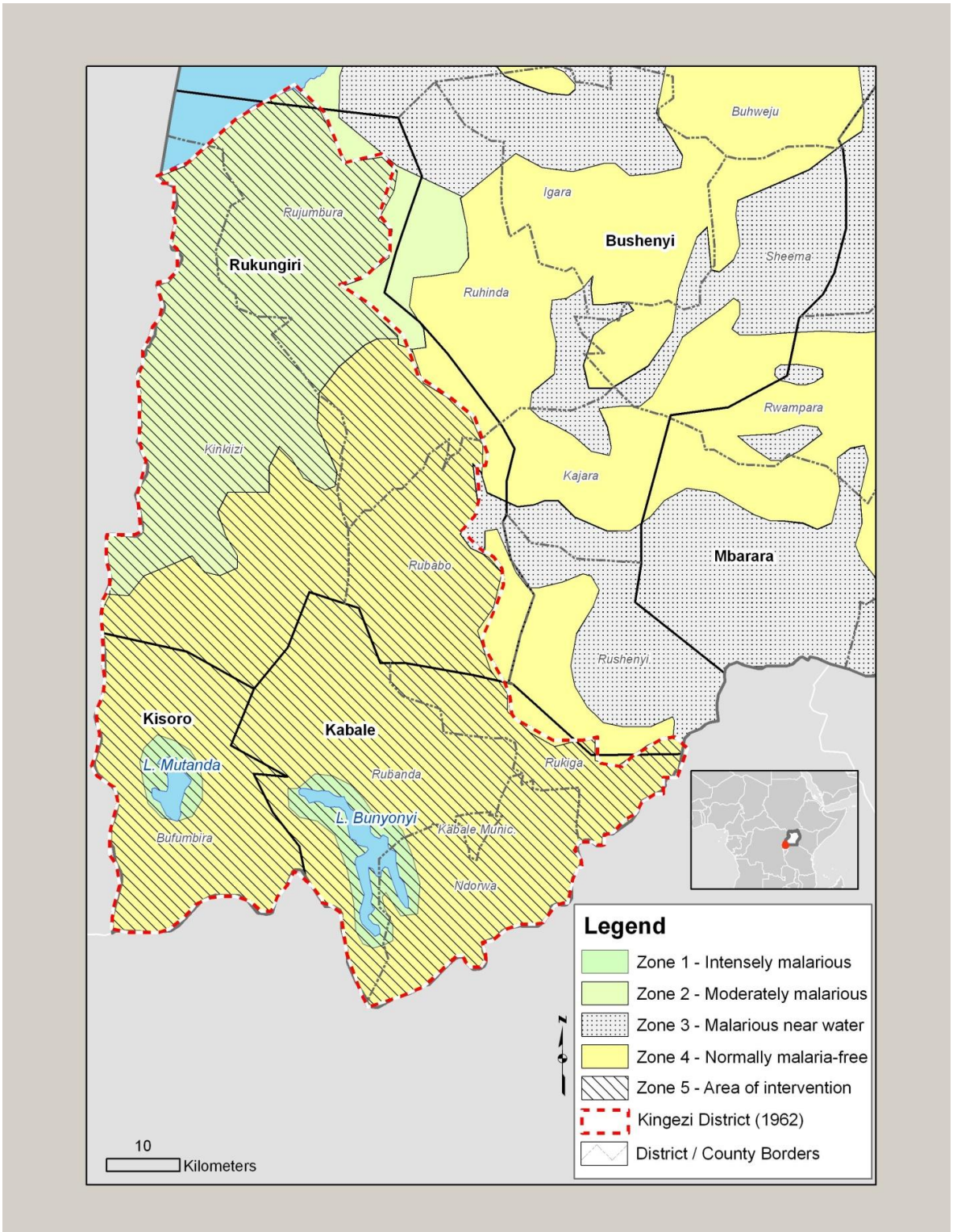

Figure 2: Pre-eradication Malaria Prevalence in the Intervention District (Kigezi) in Uganda.

Note: This map reflects district boundaries as of 2010, in which the intervention district was split into three districts, but as of Uganda's 1991 Census, the data used here, it was split into two districts Rukungiri in the North and Kabale (comprising Kabale and Kisoro districts above).

Source: Zulueta, et al. (1964)

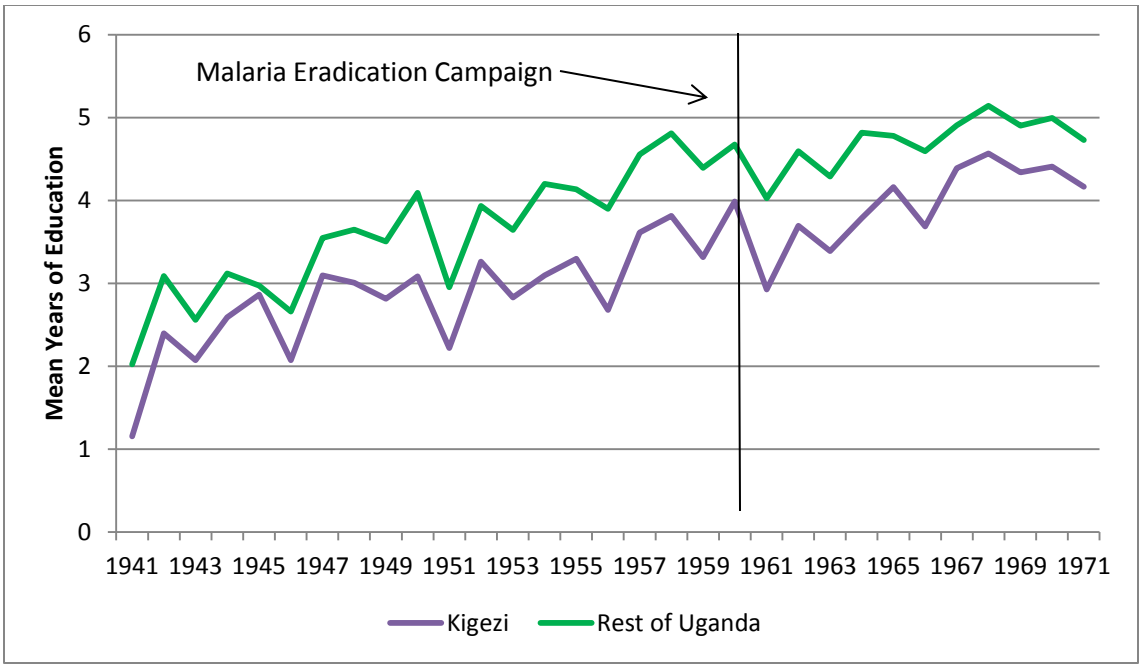

Figure 3: Mean years of schooling by age-cohort for Kigezi and the rest of Uganda, 1941-1971.
Source: Author's calculations from Uganda 1991 10% IPUMS Census sample.

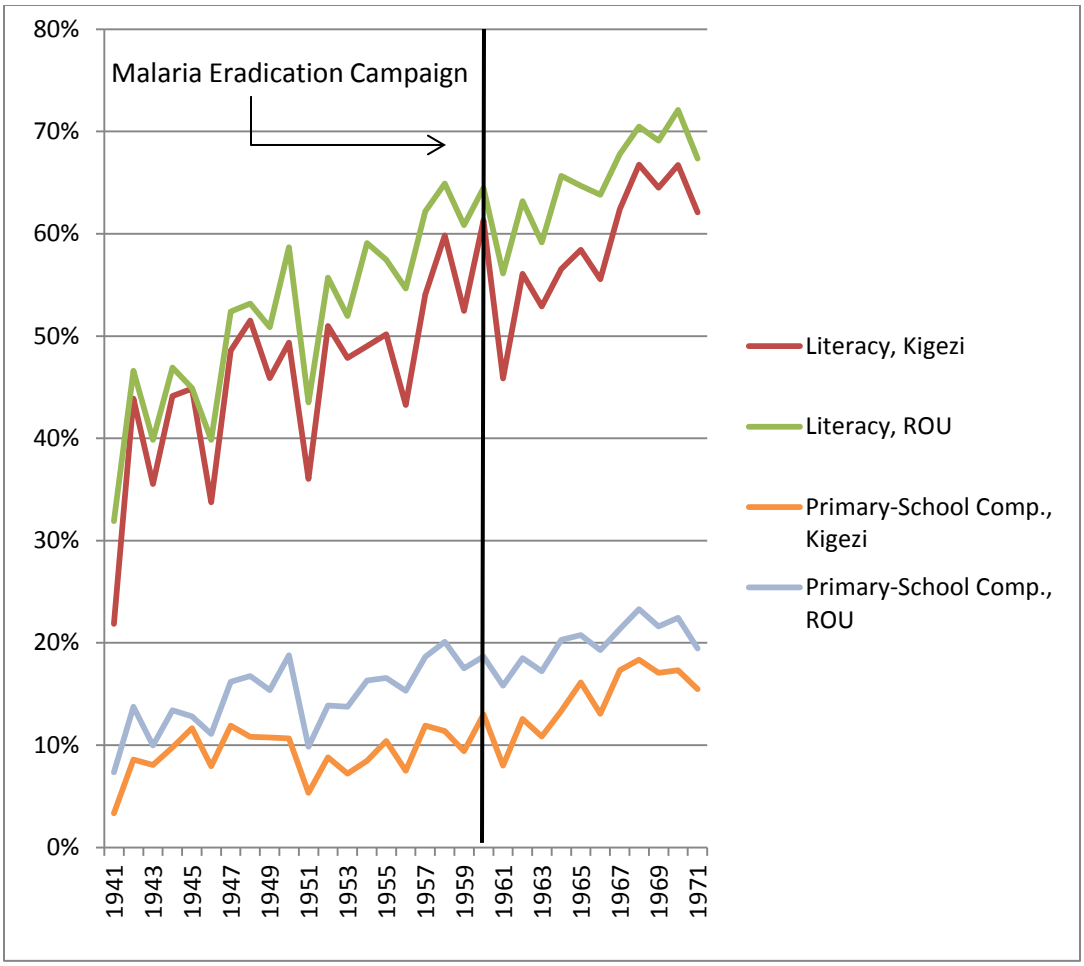

Figure 4: Literacy and Primary School Completion (%) by age cohort in the intervention district (Kigezi) and the rest of Uganda (ROU), 1941-1971.

Source: Author's calculations from Uganda 1991 10% IPUMS Census sample.

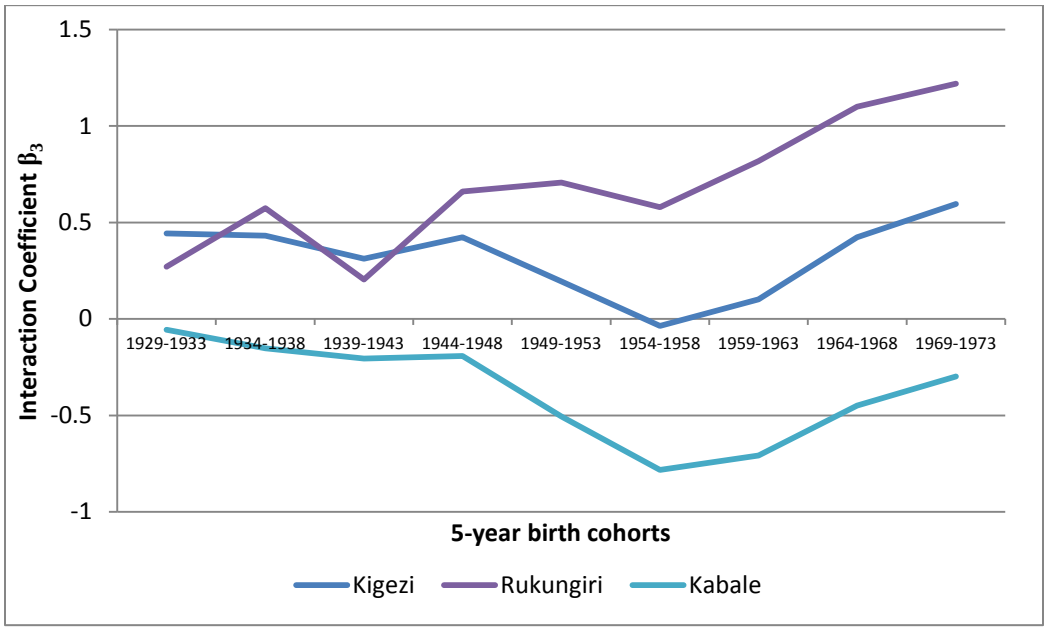

Figure 5: Cohort-specific relationship between years of schooling in the intervention district (Kigezi) and higher-malaria (Rukungiri) and lower-malaria (Kabale) areas pre-eradication, compared to the rest of Uganda.

Note: Figure 5 plots the differential change in years of schooling comparing the intervention district overall (Kigezi) and its higher (Rukungiri) and lower (Kabale) malaria incidence areas pre-eradication to the rest of Uganda. Estimates come from β_c coefficients calculated using equation (2) where c indexes five-year birth cohorts. These five-year birth cohorts are centered on years with individuals aged 0 or 5 to minimize the impact of age heaping on the regression results. Cohorts born between 1929 and 1973 (ages 18 to 62 in 1991) are used. Equation (2) is run with robust standard errors to adjust for heteroskedasticity, is clustered at the district level, and weighted by individual according to the IPUMS 10% sample of the 1991 Uganda Census.

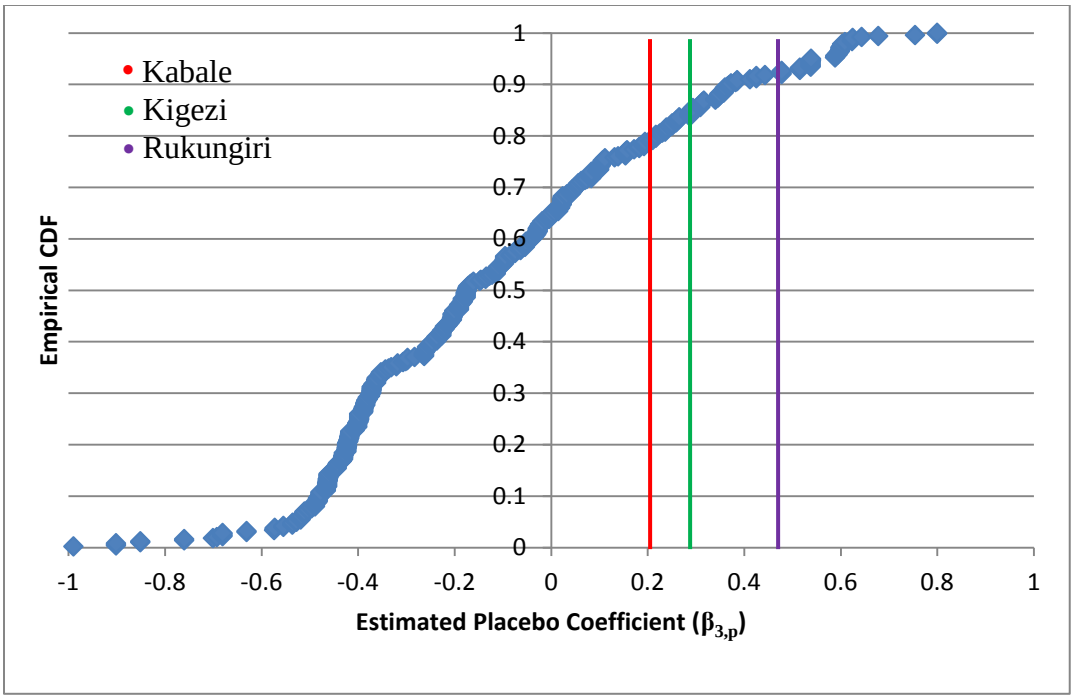

Figure 6: Distribution of placebo estimates ($\beta_{3,p}$).

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 equation (1) where a random district and year pair is randomly drawn from ages 25-35 (excluding age 31 – the year of the eradication campaign) 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 the less malarious area of the intervention district (Kabale, β_3 estimate in column 1 of table 2), the entire intervention district (Kigezi, β_3 estimate in column 1 of table 3), and the more malarious area (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		Intervention District (Kigezi)	
	Pre-	Post-	Pre-	Post-
Percent 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%

Source: Author's calculations from Uganda 1991 10% IPUMS Census sample for birth cohorts 1931 to 1971 where the eradication intervention occurs in the intervention district (Kigezi) in 1960.

Table 2: Malaria eradication's effect on years of schooling, primary school completion, literacy, and household socioeconomic status.

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: Table 2 presents the effect of malaria eradication on years of schooling, primary school completion, literacy, and a proxy for socioeconomic status constructed from an index of assets owned by the household, using equation (1). All regressions include robust standard errors to adjust for heteroskedasticity, are clustered at the district level, and weighted by individual according to the IPUMS 10% sample of the 1991 Uganda Census. Birth cohorts born between 1951 and 1971 are included in this sample. All regressions are run with district- and birth-cohort fixed effects and include the variables shown above as well as binary variables for marital status and 23 ethnicity dummies (not shown).

Table 3: Malaria eradication's effect on an individual's probability of literacy and primary school completion.

	Change in Prob.	95% CI	Mean (%)	Increase (%)	95% CI (%)
Primary-School Comp.	0.063	[0.058 - 0.0674]	12.0	52.5	[48.2 - 56.2]
Literacy	0.0044	[0.004 - 0.005]	54.0	0.81	[0.7 - 0.9]

Note: Changes in the probability of literacy and primary school completion are estimated using the CLARIFY program with Monte Carlo simulation and covariates set to their mean or modal levels for continuous and categorical variables respectively. That is, we estimate the change in an individual's probability of literacy and primary-school completion, for an unmarried, Catholic male, born pre-eradication, living in a rural area from the Baganda tribe compared to that individual's probability of literacy and primary-school completion if they had been born in the intervention district after the eradication campaign.

Table 4: Malaria eradication's effect on years of schooling by pre-eradication malaria incidence within the intervention district using high malaria (Rukungiri) and low malaria (Kabale) areas.

Dep Var: Yrs of School	High Malaria (Rukungiri)		Low Malaria (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: Table 4 presents estimates of the effect of malaria eradication on years of schooling using equation (1), where the high and low malaria incidence districts pre-intervention, Rukungiri and Kabale, are separately defined as the intervention area while the other district is excluded. All regressions include robust standard errors to adjust for heteroskedasticity, are clustered at the district level, and weighted by individual according to the IPUMS 10% sample of the 1991 Uganda Census. Birth cohorts born between 1951 and 1971 are included in this sample. All regressions are run with district- and birth-cohort fixed effects and include the variables shown above as well as binary variables for marital status and 23 ethnicity dummies (not shown).

Table 5: Malaria eradication's effect on outcome variables years of schooling, primary-school completion, literacy, and socioeconomic status using 5-year age categories.

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: Table 5 presents the effect of malaria eradication on years of education, primary school completion, literacy, and a proxy for socioeconomic status constructed from an index of assets owned by the household, using equation (1) with 5-year birth cohorts. All regressions include robust standard errors to adjust for heteroskedasticity, are clustered at the district level, and weighted by individual according to the IPUMS 10% sample of the 1991 Uganda Census. Individuals born between 1949 and 1973 (ages 18 to 42) are included in this sample so that full 5-year birth cohorts can be included. All regressions are also run with district- and 5-year birth-cohort fixed effects and include the variables shown above as well as binary variables for marital status and 23 ethnicity dummies (not shown).

Table 6: Malaria eradication's effect on educational outcomes while ignoring birth-cohort information.

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: Table 6 displays the effect of malaria eradication on educational outcomes, while ignoring potential serial correlation between birth cohorts. That is, table 6 shows estimates of the effect of being born in the intervention district, post-eradication, compared to educational changes in the rest of Uganda using only mean district-level variation in outcomes pre- versus post-eradication. These results are derived by first regressing educational outcomes on the individual-level covariates from equation (1) including gender, urban status, religion, marital status, and 23 ethnicity indicators. This regression is run using person weights and robust standard errors clustered at the district level. Then, the residuals from this procedure are aggregated by district for the pre- and post-eradiation period separately, producing 70 observations from Uganda's 35 districts, with one observation by district for pre- and post-eradication. Finally, these residuals are regressed on district-level variables with robust standard errors to determine the treatment effect using equation (3).

Table 7: Malaria eradication's effect on years of education with varying birth-cohort windows.

Dep Var: Yrs of School	**Ages 20-60**		**Ages 20-50**		**Ages 20-40 (baseline)**		**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: Table 7 displays the educational effect of malaria eradication, while varying the birth-cohorts used to estimate equation (1). The baseline specification uses individuals born between 1951 and 1971 (ages 20 to 40 in the 1991 Census). Ages 20 to 60 correspond to cohorts born between 1931 and 1971, ages 20 to 50 correspond to cohorts born between 1941 and 1971, ages 25 to 35 correspond to cohorts born between 1956 and 1966, and ages 29 to 33 correspond to cohorts born between 1958 and 1962. All regressions include robust standard errors to adjust for heteroskedasticity, are clustered at the district level, and weighted by individual according to the IPUMS 10% sample of the 1991 Uganda Census. Moreover, all regressions are run with district- and birth-cohort fixed effects and include the variables shown above as well as binary variables for marital status and 23 ethnicity dummies (not shown).

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 (2010b) and Lucas (2010).

Authors		**Cutler, et al (2010)**	**Barofsky, et al (2013)**	**Bleakley (2010b)**				**Lucas (2010)**	
DEPENDENT VARIABLES:	**Country:**	**India**	**Uganda**	**US**	**Mex.**	**Col.**	**Brazil**	**Para.**	**Sri Lanka**
Years of Schooling	Males	**X**	**++**	**N/A**	**--**	**++**	**++**	**N/A**	**N/A**
	Females	**X**	**X**	**N/A**	**N/A**	**N/A**	**N/A**	**++**	**++**
Consumption / Income	Males	**++**	**++**[1]	**++**	**++**	**++**	**++**	**N/A**	**N/A**
	Females	**++**	**X**	**N/A**	**N/A**	**N/A**	**N/A**	**N/A**	**N/A**
Literacy	All	**X**	**++**	**N/A**	**++**	**++**	**++**	**++^**	**++^**
Primary School Comp.	All	**X**	**++**	**N/A**	**N/A**	**N/A**	**N/A**	**++*^**	**++*^**
DATA									
Birth Place		**No**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**

Notes:

N/A: Not analyzed,

X: No effect or mixed evidence over multiple specifications,

++: Positive effects from malaria eradication,

--: Negative effects from malaria eradication,

[1] Significant only for some specifications at the 10% level,

* Refers to years of primary school,

^ Regressions only include females.