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Malaria eradication and economic outcomes in sub-Saharan Africa: Evidence from Uganda☆

Jeremy Barofsky[a,*], Tobenna D. Anekwe[b], Claire Chase[c]

[a] Harvard University, School of Public Health, United States
[b] USDA Economic Research Service, United States
[c] World Bank, United States

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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, large-scale, and well-documented attempt to eliminate malaria in sub-Saharan Africa and produced an immediate disease reduction. We use this quasi-experimental health shock to identify long-term changes in educational and economic outcomes. Comparing the treatment district to a similar synthetic control, we find malaria eradication raised educational attainment by about a half year for both males and females, increased primary school completion among females and generated an almost 40% rise in the likelihood of male wage employment.

1. Introduction

The damage that malaria inflicts on population health is severe and well established. With an estimated 120–300 million cases and close to 600,000 deaths per year, the disease represents a major threat to 3.2 billion people in over 100 nations (World Health Organization, 2014; Murray et al., 2012). Sub-Saharan Africa (SSA) and children under five bear the bulk of the global malaria burden, with 70% of cases and 90% of deaths (World Health Organization, 2012) occurring in SSA and almost 90% of deaths occurring before age five. However, since 2000, substantial reduction in malaria cases and mortality has been achieved, increasing the salience of understanding the long-term impact of elimination.

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 (Lozoff and Georgieff, 2006), delays the development of the central nervous system (Beard, 2008), and reduces a child's ability to respond to environmental cues (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 (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, to the extent that adult mortality attributable to malaria reduces the expected time that individuals can realize gains from human capital investments, the disease decreases incentives for schooling.

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* Corresponding author. Current address: The Brookings Institution, United States. Tel.: +1 2027976248.
E-mail addresses: jbarofsky@brookings.edu, jtb856@mail.harvard.edu (J. Barofsky), tanekwe@ers.usda.gov (T.D. Anekwe), cchase@worldbank.org (C. Chase).

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Conversely, child mortality from malaria may improve economic outcomes by differentially affecting individuals that, if they had survived, would have exhibited lower educational attainment and adult income.

In this paper, we investigate the relationship between reduced exposure to childhood malaria and later life outcomes using an eradication campaign in southwestern Uganda as a quasi-exogenous case study. Most closely related to this paper, three studies investigate how malaria eradication affects income and human capital formation in the Americas (Bleakley, 2010), female educational attainment in Paraguay and Sri Lanka (Lucas, 2010), and schooling and economic status in India (Cutler et al., 2010). These studies use the discovery of dichlorodiphenyltrichloroethane (DDT) and the WHO led Global Malaria Eradication Program (GMEP) 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. The macro evidence is mixed with some studies showing that nations with high malaria incidence also exhibit low levels of economic development (Gallup and Sachs, 2001; McCarthy et al., 2000). In contrast, Acemoglu and Johnson (2007) use the reduction in mortality produced by the 1940s international epidemiological transition to identify the effect of increasing life expectancy on economic growth and find no evidence that large health improvements produced any positive effect on per capita income.[1]

Our 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 impact 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 sharp variation in childhood malaria exposure by birth cohort. The intervention occurred in one district within Uganda and consequently we identify its impact by employing the synthetic control method (Abadie and Gardeazabal, 2003; Abadie et al., 2010) to construct a valid counterfactual district against which to compare outcome changes. Using individual-level data from the 1991 Uganda Census and district-level data from the 1959 Census, this procedure weights other districts in Uganda such that the control area closely resembles the treatment district in pre-eradication characteristics. Given that the synthetic control model represents a generalization of the differences-in-differences framework with varying district weights, we also employ a differences-in-differences model to estimate eradication's impact by subdistrict when the synthetic control is not feasible.

First, we use summary birth history data by mother's age and district from the 1969 Uganda Census and investigate how under five mortality changes over time and observe an almost 16% decline in child mortality for the treatment district around the malaria eradication campaign. Comparing the treatment district to its synthetic counterfactual, our primary results show that malaria eradication increased years of schooling by 0.52 (11%) and 0.53 (21%) for males and females, respectively, which translates into income gains of approximately 5–20% annually, depending on the rate of return to education assumed. We also find that primary school completion among females increases by 2.4 percentage points (34%) compared to the synthetic control, but no significant increases for males are observed. In addition, we find that the five year birth cohort completely exposed to eradication (born 1964–1968), experienced a 5.6 percentage point (37%) rise in likelihood of wage work. We employ a differences-in-differences (DD) model using the synthetic control district weights and find statistically significant changes in outcomes similar to those identified previously. Importantly, using the DD model, we also find that educational effects are uniformly larger in areas with higher pre-treatment malaria incidence, as expected if these effects are driven by a reduction in early-life malaria exposure. We also test the timing of our results using the DD model with years of schooling for males and females separately and find educational improvements that coincide with eradication. A placebo analysis is run, investigating the treatment effect for all districts, and we verify that the treatment district consistently exhibits one of the largest improvements in education and wage work after eradication.

This paper contributes to the existing literature on the economic effects of malaria eradication as the first to produce estimates in SSA on long-term effects. The location of previous studies is important because the malaria strain most prevalent in SSA, *Plasmodium falciparum*, produces health effects that differ significantly from the malaria prevalent in other regions (Hay et al., 2009). Therefore, these previous results may not generalize to current eradication efforts, while studies on the impact of recent malaria control efforts[2] cannot yet answer questions on long-term effects. *Plasmodium vivax*, the malaria strain most prevalent outside of SSA, 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 (Boivin et al., 2007; Idro et al., 2005) and death at a substantially higher rate than *P. vivax*. 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 morbidity benefits, such that reduced child mortality from malaria worsens economic outcomes in adulthood. Acemoglu and Johnson (2007), in analyzing the epidemiological transition post-World War II argue that this selection effect in part explains their null results. We find that eradication of *P. falciparum* malaria in SSA 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 that is similar to or larger than *P. vivax*, but results orders of magnitude smaller than macro estimates even in the long term.

This paper continues as follows: Section 2 provides background on malaria and the eradication campaign, Section 3 describes our methodology and main results, Section 4 interprets our results and discusses them in the context of other work, while Section 5 concludes.

2. Malaria eradication in southwest Uganda

Launched in 1955, the WHO's GMEP 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 SSA, caused in part by increasing mosquito resistance to DDT and heightened parasite resistance to chloroquine treatment. By 1950, the WHO had already recommended "to governments responsible for the administration of African territories that malaria should be controlled by modern

[1] Further description of this literature is included in the discussion and Appendix A.

[2] For example, Kuecken et al. (2014).

methods as soon as feasible, whatever the degree of endemicity, and without awaiting the outcome of further experiments," (World Health Organization, 1950). Snow et al. (2012) highlights 25 malaria control and eradication initiatives in SSA between 1945 and 1979. Of these, indoor residual spraying and mass drug administration was implemented in six, including the Kigezi eradication analyzed here. In general, this effort at malaria elimination in middle Africa failed (Snow et al., 2012). The reasons include the high cost of insecticide that newly independent nations could not bear, fears of rapid escalation of vector resistance to insecticide, and mixed results from the various pilot elimination projects in the region. Given that most SSA nations did not have the resources nor the capacity to expand malaria control and elimination programs, by the 1970s, malaria was seen as a problem best solved through health system strengthening (see Appendix C for additional background on GMEP). The malaria eradication campaign we analyze occurred in the Kigezi district of southwestern Uganda and constitutes, to our knowledge, the only large-scale, effective, and well-documented intervention from this period.

The malaria eradication campaign in Kigezi took place between 1959 and 1960. The 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 were carried out monthly before and after spraying, and fever surveys were carried out monthly at visits to dispensaries to estimate the rate of enlarged spleen in the population (a measure of chronic malaria infection) and the rate of parasite infection (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).

Figs. 1 and B1 illustrate pre-eradication malaria variation in the intervention district of Kigezi and Uganda, respectively. Most of the northern part of Kigezi district was classified as hyperendemic, meaning that the rate of spleen inflammation among children 2–10 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 3700 ft., and classified as mesoendemic, meaning that the measured rate of spleen inflammation among children was found to be between 11 and 50%. Small hyperendemic areas around the lakes of the southern area were also observed.

Although Kigezi was defined administratively as one district during the eradication campaign, by the 1991 Census the area had been split into two districts that, conveniently for the purposes of our analysis, correspond to 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 Fig. 1).[3] In addition to comparing the impact of eradication in Kigezi versus the rest of Uganda, we also use variation in pre-eradication malaria incidence within the intervention area to verify our findings. As of 2005, Uganda exhibited the highest malaria incidence in the world at 47.8 percent (World Health Organization, 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 *Plasmodium 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 corresponds to the rest of SSA and differs from the malaria strains analyzed in previous studies on eradication.[4]

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, in March, April, May, September, and October of 1960. DDT spraying and mass distribution of antimalarial drugs ceased thereafter in Kigezi. Standard treatment under GMEP only included DDT spraying, but mass distribution of antimalarial medication was carried out as well to completely interrupt 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 summary, the campaign "produced the almost complete disappearance of malaria within the first year of operations," (Zulueta et al., 1964).[5]

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 1500 square miles. Consistent with a substantial decrease in malaria's mortality burden, Zulueta et al. (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. Mortality change

To investigate the effect of malaria eradication, we first explore the change in under five mortality in the treatment district using summary birth histories (SBHs) from the 1969 Uganda Census. That is, the 1969 Census provides information on the aggregate number of children born and the number of children surviving for all women by five year age group and district. As noted by those that first translated SBHs into indirect mortality estimates (Brass and Coale, 1968), the proportion surviving of children ever born by female age group

[3] Fig. 1 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 called Kabale.

[4] 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 only *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.

[5] Unfortunately, data is limited on how malaria prevalence in Kigezi changed after the initial follow-up approximately one year after eradication. Presumably, this is because Uganda gained independence soon after, touching off political turmoil that did not subside until the mid 1980s. There is suggestive evidence that reduced malaria prevalence persisted. Yeka et al. (2012) shows that under-five malaria prevalence in southwestern Uganda remains substantially lower than the rest of the country in 2009.

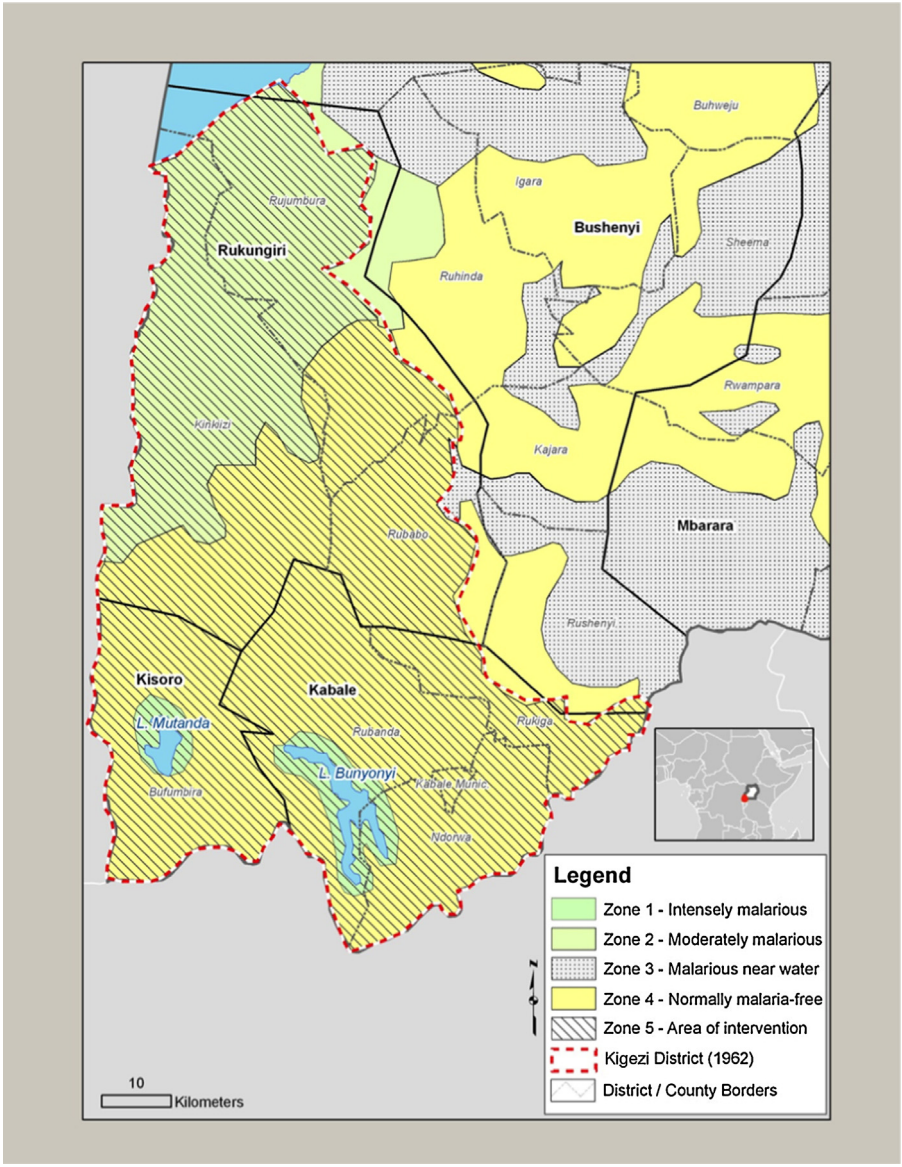

Fig. 1. Pre-eradication malaria prevalence in intervention district (Kigezi) of southwestern Uganda. This figure shows pre-eradication malaria prevalence measured in conjunction with Kigezi's eradication campaign. The intervention district of Kigezi is outlined in red. The boundaries in black reflect district boundaries as of 2010, in which the intervention district was split into three districts. As of Uganda's 1991 Census, (the data used here for educational and economic outcomes), Kigezi was split into two districts Rukungiri in the North and Kabale (comprising Kabale and Kisoro districts above) in the South. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Source: Harvard Pusey Library; Zulueta et al. (1964).

is an indicator for child mortality. Mother's age proxies for a child's exposure to the risk of dying in a given year, with older mothers, all else being equal, representing longer exposure to death risk. To attribute an approximate date and mortality level to each mother's age group, we follow methods first developed by Feeney (1976, 1980). However, given that children are born to mothers at different ages and are exposed to changing mortality rates over time, the calendar year time reference should be treated as the center of a distribution around which child mortality occurred. Particularly when mortality is changing rapidly, these methods are best suited to measuring general mortality time trends, instead of the yearly mortality fluctuations that can be derived from more detailed data (see Appendix B for additional details on mortality calculations). Therefore, we interpret results from these methods cautiously, but note that they permit additional insight into the mortality pattern of the treatment district around the malaria eradication period.

With this caveat, Fig. 2 displays the time path of under five mortality in Kigezi compared to the weighed average in the rest of Uganda. First, we observe that the treatment district exhibits lower child mortality than the rest of Uganda. This is driven by both the higher altitude of the district, but also undoubtedly reflects the smoothed impact of malaria eradication. Nevertheless, we observe a 16% decline in child mortality between the end of 1958 and the middle of 1960 for Kigezi, which closely corresponds to the malaria eradication period.[6] Given that malaria mortality during this time was estimated to represent between 10% and 15% of under five child mortality in tropical Africa (Bruce-Chwatt, 1952), this effect is

[6] We also calculate that child mortality in the rest of Uganda declines by 11% over the analogous time period. Although larger in the treatment district, we cannot reject the hypothesis that the decline in child mortality in Kigezi is equal to the decline in the rest of Uganda. See Appendix B for hypothesis test specifics.

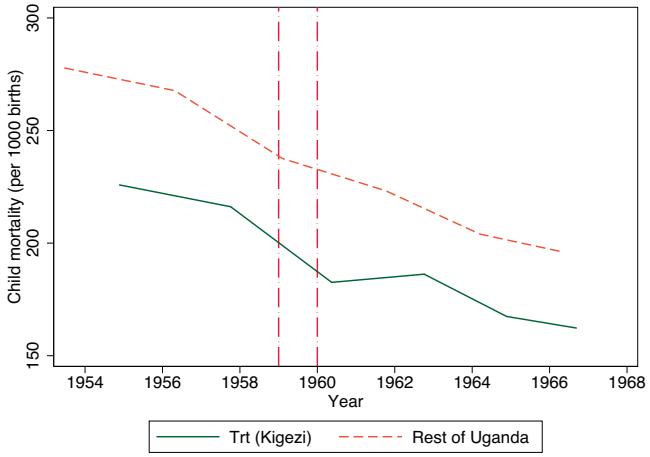

Fig. 2. Child mortality per thousand in the malaria eradication district (Kigezi) compared to the rest of Uganda. This figure compares under five mortality in the district that received malaria eradication (Kigezi) compared to the population weighted average in the rest of Uganda, 1953–1967. The red dotted lines show the extent of the eradication campaign from 1959 to 1960. Mortality rates are calculated using summary birth histories by mother's age and district from the Uganda Census 1969. Since mortality rates use mother's age as a proxy for year of death, the timing of deaths represent an approximation to the actual time profile of mortality. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

broadly consistent with a close to complete elimination of malaria-related mortality. This first stage of our analysis, therefore, provides suggestive evidence that the changes we explore to education and economic outcomes indeed reflect the impact of reduced malaria burden.

3.2. Creating a synthetic control district

Given that the malaria eradication program occurred at the aggregate level in one district of southwestern Uganda, we could compare changes in outcome variables to the rest of the country using a differences-in-differences (DD) model. However, pre-eradication, the treatment district differed from the rest of Uganda in education, economic level, and other characteristics associated with the growth of the former two quantities. We want to avoid a comparison between Kigezi and the rest of Uganda that is biased by differences in pre-eradication determinants of educational and economic trajectories. Therefore, we employ synthetic control methods (Abadie and Gardeazabal, 2003; Abadie et al., 2010) to construct a weighted combination of other districts in Uganda to serve as a valid counterfactual that is similar to the treatment area in pre-treatment characteristics likely to affect future educational and economic outcomes. The method therefore weakens the DD assumption of parallel trends and allows the effect of unobservables on the outcome to change over time.

We use pre-eradication data from the 1959 Uganda Census at the district level to create a synthetic control district for each dependent variable analyzed. The educational and economic outcome data come from Uganda's 1991 Census provided by the *Integrated Public Use Micro Sample* (IPUMS, 2007), which is a weighted 10% sample of Uganda's population. The census contains information on years of education, a binary indicator for primary school completion, and a binary indicator for wage work. Wage work is defined using the Uganda 1991 Census question on worker class, with categories self-employed, wage/salary worker, and unpaid family worker. Worker class is recoded into a binary variable for wage work and used because data on income or consumption is not provided. As noted above, by 1991, the intervention area had been divided into two separate districts, Rukungiri in the north and Kabale in the south. Given the use of Census 1959 data by district, we cannot make use of this variation when employing the synthetic control method. However, DD methods are also used to investigate how the effect of malaria eradication varies by initial malaria prevalence in these two districts.

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 and large artificial variability in education by one year birth cohort. Given that the identification strategy employed here uses variation in malaria exposure by birth cohort, incorrectly reported years of birth may drive the educational results we find instead of a true effect.[7] To adjust for age heaping, we employ five year categories centered on ages where age heaping is most severe and implement the synthetic control method for ages 18–62 (born 1929 to 1973). The eradication cohort is defined as those born between 1959 and 1963, which includes cohorts that are partially exposed during eradication,[8] and post-eradication birth cohorts are defined as the two groups born 1964–1968 and 1969–1973.

To understand the treatment effect more formally, let Y_{it}^N represent the outcome variable that we would observe in district i and time t in the absence of the intervention and Y_{it}^I represents the outcome variable in district i and time t if the intervention had occurred. We assume that the Kigezi malaria eradication intervention has no effect before time T_0 so that for the pre-intervention period (1929–1958) $t \in (1, \ldots, T_0)$, $Y_{it}^N = Y_{it}^I$. The weighted average of Uganda's control districts are then proxies for Y_{it}^N, what would have happened in Kigezi in the absence of eradication. The treatment effect for unit i at time t then is defined as $\alpha_{it} = Y_{it}^I - Y_{it}^N$. Because we observe Y_{it}^I for the intervention district of Kigezi, Y_{it}^N must be estimated to obtain α_{it}. This is done by finding a set of weights for the other districts in Uganda such that the resultant combination of districts (the synthetic control) approximates the treatment district's pre-intervention outcome variable trajectory and set of observed covariates. Let $\mathbf{W}$ represent a $(J \times 1)$ vector of weights, where J is the number of control units (in this case the 16 non-treatment districts in Uganda as of 1959), such that $0 \leq W_j \leq 1$ and all W_j sum to 1. Each value of $\mathbf{W}$ constitutes a possible synthetic control, that is, a weighted average of existing control units. $\mathbf{W}$ is chosen to minimize the difference between the treatment district and the composite control in the trajectory of the dependent variable and chosen pre-intervention covariates.[9] For each dependent variable, we implement this synthetic control method, creating an estimate of the trajectory of Y_{it}^N for a given vector $\mathbf{W}$, thereby identifying the treatment effect α_{it}.[10]

The primary threat to identification in this study is that we investigate an eradication effort that occurred in one region of Uganda. We address this by constructing a counterfactual control district with similar characteristics to the treatment pre-intervention. Other threats include potential economic changes occurring simultaneously with eradication and generalizability given that malaria control in the studied intervention differs from

[7] To understand the extent of age misreporting in these data, over 4000 individuals report their age as 30 while about 1000 report their age as 29 and 31.

[8] This group is partially exposed because, given that eradication occurred from early 1959 to late 1960 moving north to south in Kigezi, a significant proportion of this cohort was in utero and infants before eradication.

[9] For further explanation see Abadie and Gardeazabal (2003) and Abadie et al. (2010).

[10] Table G.9 displays the district weights used to create the synthetic control region for each dependent variable.

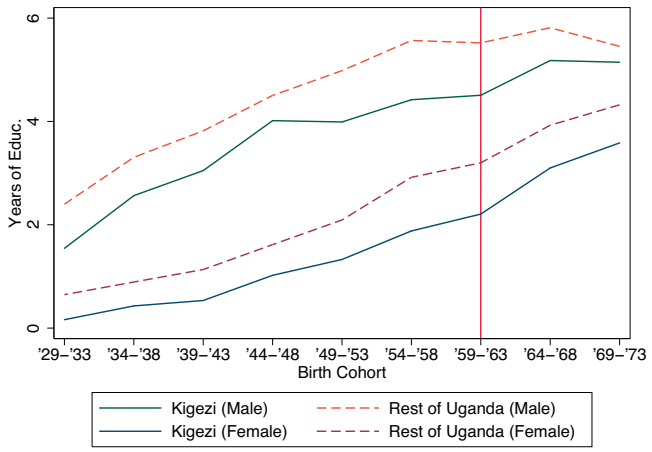

Fig. 3. Mean years of education by five year age group in treatment district (Kigezi) compared to the rest of Uganda. Data from Uganda's 1991 Census using five year age groups between treatment district Kigezi and the rest of Uganda for males and females separately. Census reports district of birth, not current district of residence.

methods applicable today. We examine each of these issues in the discussion section.

3.3. Effects on educational attainment

Fig. 3 plots the trajectory of mean years of education for males and females in the treatment district of Kigezi compared to the rest of Uganda for five year cohorts born 1929 to 1973. These figures show that all of Uganda experienced substantial increases in educational attainment over this period. In addition, we observe that the treatment district of Kigezi exhibited educational attainment consistently below the rest of Uganda for all birth cohorts. For cohorts born 1954 to 1958, immediately before eradication, mean years of schooling for males and females combined was 31% lower in Kigezi compared to the rest of Uganda. Although educational attainment trends are approximately similar between the treatment district and the rest of Uganda, this non-parametric investigation does not reveal any systematic pre-eradication catch-up in schooling for those born in the treatment area. Nevertheless, pre-eradication characteristics associated with more rapid educational and economic growth may have differed between Kigezi and the rest of Uganda, making the latter poised for more rapid growth and confound our findings.

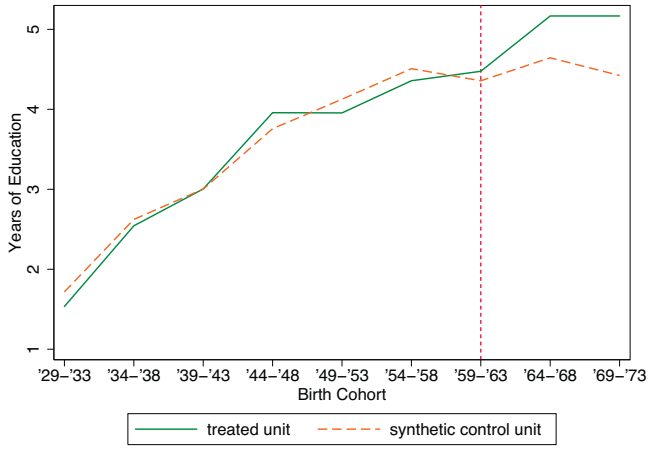

Fig. 4. Treatment effect for male years of schooling. Using data from Uganda Census 1959 and Census 1991 to create a synthetic control district similar to the treatment district (Kigezi) in the time path of years of education and pre-intervention characteristics. Synthetic control method employed for each district in Uganda 1959 with 5 year birth cohorts.

Table 1
Years of schooling predictor means (males).

Variables	Treatment (Kigezi)		Rest of Uganda	Std. dev.
	Real	Synthetic		
Yrs of Schl Born 34–38	2.543	2.623	3.6	0.95
Yrs of Schl Born 44–48	3.957	3.757	4.932	1.20
Yrs of Schl Born 54–58	4.357	4.508	5.785	1.21
General Fert. Rate 1959	231	220.877	187.686	35.60
Ever in Schl, M., 1959	34.3	33.168	50.529	14.96
7–10 yrs Schl, M. 1959	11.7	16.176	17.226	7.94

Predictor variables include mean years of education pre-intervention for birth cohorts 1934–1938, 1944–1948, 1954–1958, general fertility rate (Census 1959), percent of district males that had ever attended school among ages 6–15 (Census 1959), and percent of district males with 7–10 years of education (1959 Census).

Our main analysis therefore applies the synthetic control method described above to equalize both the trajectory of dependent variables pre-intervention as well as characteristics associated with schooling and employment growth. This is achieved through a weighted average of districts in Uganda, chosen to reproduce pre-eradication predictors of the educational or economic dependent variable. For years of education, Tables 1 and 2 compare the pre-intervention characteristics of Kigezi with synthetic Kigezi and population weighted averages in the rest of Uganda for males and females, respectively. Those predictors include mean years of education for three pre-eradication birth cohorts, the general fertility rate, the percent of males (females) ever in school, and the percent of males (females) with 7–10 years of education. Using Census 1959 data, these tables show that prior to malaria eradication the rest of Uganda exhibited higher levels of education than Kigezi and lower fertility. In contrast, synthetic Kigezi (the convex combination of other Uganda districts) is similar to real Kigezi in pre-intervention education, fertility, percent of males (females) ever in school as of 1959, and percent of males (females) in 1959 with high levels of education. Characteristics that differ between real and synthetic Kigezi (for example, percent with high education in some models), do so because those factors provide limited predictive power for the dependent variable.

Figs. 4 and 5 show the treatment effect α_{it} by five year birth cohort comparing the district Kigezi and its composite synthetic region for males and females, respectively. These figures indicate that the synthetic district's years of education trajectory closely follows that of Kigezi, pre-intervention. However, that trend diverges after eradication, revealing a significant positive effect on years of schooling attributable to reduced malaria exposure in early life. We find that the treatment effect α_{it} is 0.52 (11.3%) and 0.53 (21.2%) greater years of schooling for males and females, respectively, among those born 1964 to 1968, the first five year cohort born entirely after eradication.

Table 2
Years of schooling predictor means (females).

Variables	Treatment (Kigezi)		Rest of Uganda	Std. dev.
	Real	Synthetic		
Yrs of Schl Born 34–38	.434	.439	1.19	.94
Yrs of Schl Born 44–48	1.01	.997	1.999	1.22
Yrs of Schl Born 54–58	1.841	1.845	3.308	1.44
General Fert. Rate 1959	231	218.99	187.686	35.60
Ever in Schl, F. 1959	13.6	16.959	31.632	14.99
7–10 yrs Schl, F. 1959	11	7.684	7.833	4.45

Predictor variables include mean years of education pre-intervention for birth cohorts 1934–1938, 1944–1948, 1954–1958, general fertility rate (Census 1959), percent of district females that had ever attended school among ages 6–15 (Census 1959), and percent of district females with 7–10 years of education (1959 Census).

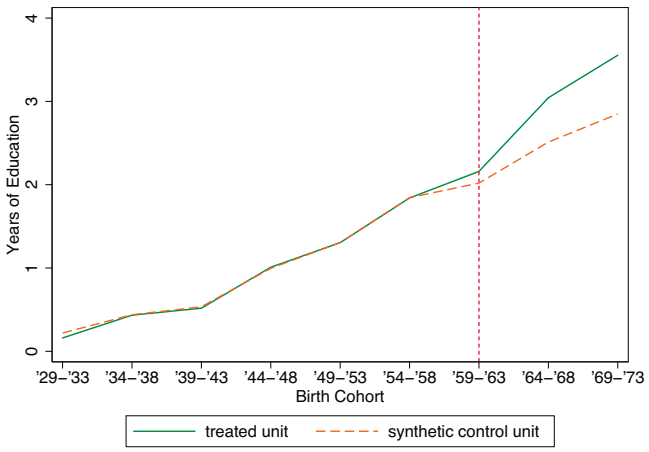

Fig. 5. Treatment effect for female years of schooling. Using data from Uganda Census 1959 and Census 1991 to create a synthetic control district similar to the treatment district (Kigezi) in the time path of years of education and pre-intervention characteristics. Synthetic control method employed for each district in Uganda 1959 with 5 year birth cohorts.

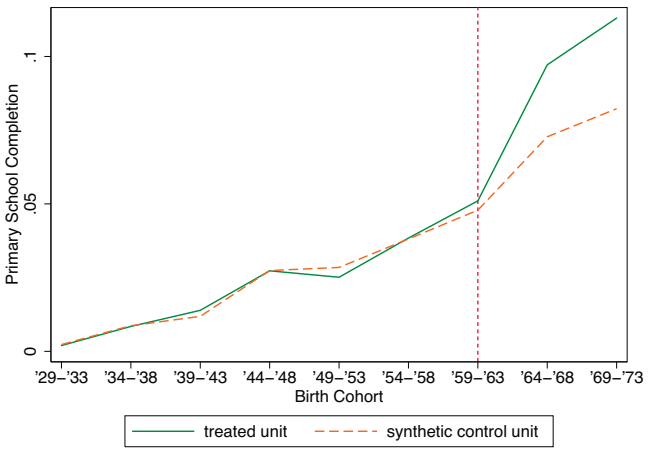

Fig. 7. Treatment effect for female primary school completion. Using data from Uganda Census 1959 and Census 1991 to create a synthetic control district similar to the treatment district (Kigezi) in the time path of percent reporting primary school completion and pre-intervention characteristics. Synthetic control method employed for each district in Uganda 1959 with 5 year birth cohorts.

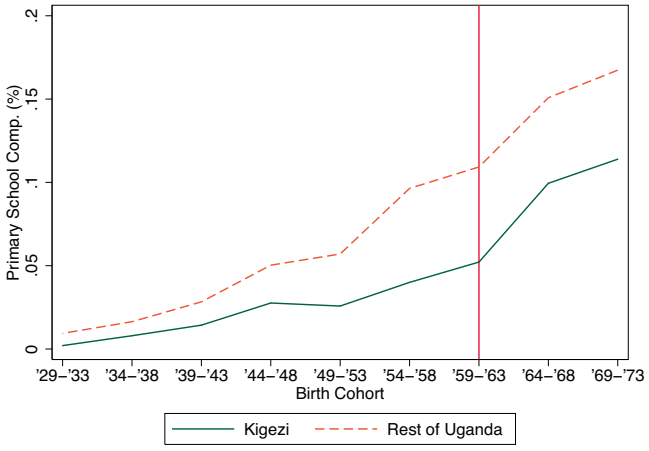

Fig. 6. Primary school completion (%) for females by five year age group in treatment district (Kigezi) compared to the rest of Uganda. Data from Uganda's 1991 Census using five year age groups between treatment district Kigezi and the rest of Uganda. Census reports district of birth, not current district of residence.

Fig. 6 compares the trajectory of primary school completion for females by cohort between those born in the eradication district against those born in the rest of Uganda. Again, trends look generally similar although Kigezi exhibits lower primary school completion pre-eradication and does not exhibit any differential catch-up before treatment. Table 3 shows that female primary school completion (in %) was less than half of the rate in Kigezi compared to the rest of Uganda immediately preceding eradication. Creating synthetic Kigezi produces a comparison district much more similar to Kigezi in lagged primary school completion, as well as general fertility rate and percent of females ever in school as of 1959. Fig. 7 shows the treatment effect for primary school completion comparing the district Kigezi and its composite synthetic region for females. This figure indicates that synthetic Kigezi reproduces the path of primary school completion of the treatment district closely pre-intervention and then diverges such that the treatment district grows more rapidly post eradication. For females born 1964 to 1968, primary school completion is 34% (2.4 percentage points) greater in Kigezi compared to its control. Also in Appendix, this analysis was performed for primary school completion among males, although no beneficial effect is observed (see Appendix Table G.10 and Fig. G.17).

3.4. Effects on wage work

Fig. 8 displays the percent of males with wage work for the treatment district of Kigezi compared to the rest of Uganda for five year birth cohorts aged 18–62. Given that these data are cross-sectional, we find that work exhibits the expected age pattern, peaking in prime ages and dropping off both for older and younger cohorts. We also observe that, as with education, cohorts born pre-eradication in Kigezi are less likely to be engaged in wage work than cohorts born in the rest of Uganda; approximately 10% less likely for the cohort born immediately before malaria eradication. The same predictors used to create synthetic Kigezi are employed to create a synthetic control district for Kigezi to investigate wage work. Table 4 shows these predictors for males in real and synthetic Kigezi, as well as the rest of Uganda. This table confirms that applying the synthetic control method to wage work for males produces

Table 3
Primary school completion predictor means (females).

Variables	Treatment (Kigezi)		Rest of Uganda	Std. dev.
	Real	Synthetic		
Primary Schl Comp. Born 34–38	.008	.009	.023	.02
Primary Schl Comp. Born 44–48	.027	.027	.064	.04
Primary Schl Comp. Born 54–58	.038	.038	.117	.08
General Fert. Rate 1959	231	224.637	187.686	35.60
Ever in Schl, F. 1959	13.6	14.979	31.632	14.99
7–10 yrs Schl, F. 1959	11	7.307	7.833	4.45

Predictor variables include mean years of education pre-intervention for birth cohorts 1934–1938, 1944–1948, 1954–1958, general fertility rate (Census 1959), percent of district females that had ever attended school among ages 6–15 (Census 1959), and percent of district females with 7–10 years of education (1959 Census).

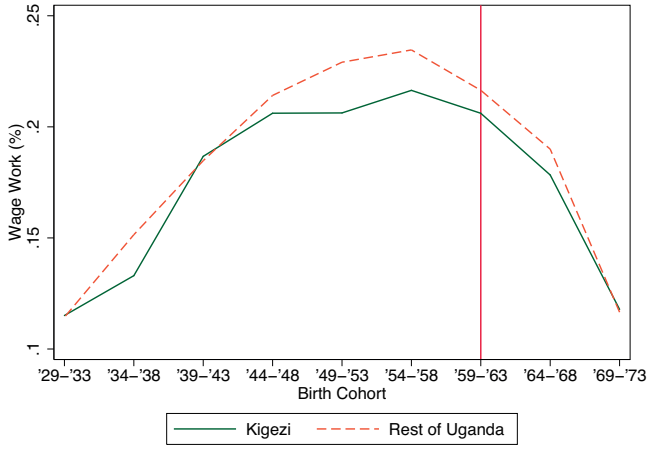

Fig. 8. Wage work (%) for males by five year age group in treatment district (Kigezi) compared to the rest of Uganda. Data on percent with wage work from Uganda's 1991 Census using five year age groups between treatment district Kigezi and the rest of Uganda. Census reports district of birth, not current district of residence.

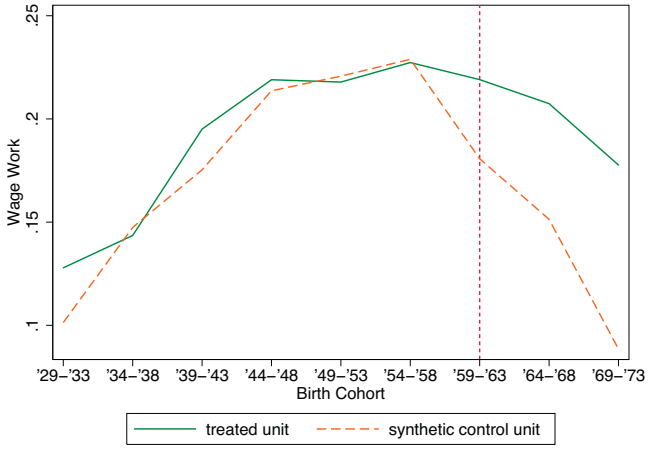

Fig. 9. Treatment effect for male wage work. Using data from Uganda Census 1959 and Census 1991 to create a synthetic control district similar to the treatment district (Kigezi) in the time path of percent reporting wage work and pre-intervention characteristics. Synthetic control method employed for each district in Uganda 1959 with 5 year birth cohorts.

a synthetic control district that resembles Kigezi more closely than the rest of Uganda both in percent with wage work pre-eradication, as well as fertility and educational attainment. Fig. 9 shows our treatment effect α_{it}, the gap between real and synthetic Kigezi wage work by birth cohort. Likelihood of wage work is quite similar between the treatment and composite counterfactual district pre-eradication and then diverges substantially for the cohort born 1959 to 1963, which is coincident with malaria eradication. For the cohort born 1959 to 1963, wage work is 3.8 percentage points higher in real Kigezi compared to synthetic and 5.6 percentage points higher for the cohort born 1964 to 1968. These magnitudes imply that individuals born in Kigezi were, respectively, 21% and 37% more likely to be engaged in wage work than those born in the synthetic control. In contrast to our educational results, we also observe immediate positive effects on wage work for the partially exposed cohort born 1959 to 1963. Appendix Fig. G.19 shows that being born in the eradication district is not associated with a higher likelihood of wage work for females.

3.5. Differences-in-differences model

We also employ a DD model to estimate the impact of malaria eradication on human capital attainment and wage work when questions cannot be addressed using the synthetic control. These include an investigation of effects by baseline malaria incidence and by birth cohort for robustness. The DD model 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 five year birth cohort c, and district d:

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

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. Eq. (1) also includes district, δ_d, and birth cohort fixed effects, μ_c, to control for time invariant social or environmental characteristics by district and year that may be correlated with both intervention status and outcome. Finally, Eq. (1) controls for individual-level characteristics in $\mathbf{X}_{icd}$, such as gender, urban status, religion, and ethnicity, while standard errors are clustered at the district level. 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. We again use years of schooling, primary school completion, and wage work as our outcomes. In this DD specification, cohorts born between 1949 and 1973 are used. This differs from the synthetic control analysis because we are interested in changes on the dependent variable directly around the intervention. Since similarity between the trajectory of dependent variables pre-intervention is assumed, the longer lag is unnecessary. When estimating Eq. (1) for the entire treatment district alone, the district weights from the synthetic analysis are applied.

Tables 5 and 6 display results from estimating Eq. (1) for males and females, respectively. We observe that for males, cohorts born after eradication in Kigezi experience differentially greater increases in years of schooling and wage work with statistical significance. For females, we find that the treatment effect is positive and statistically significant for years of schooling and positive for primary school completion without significance (p-value 0.17). These tables test the statistical significance of the synthetic control treatment effects and generally find that the null of no effect can be rejected.

Table 4
Wage work (%) predictor means (males).

Variables	Treatment (Kigezi)		Rest of Uganda	Std. dev.
	Real	Synthetic		
Wage Work Born 34–38	.144	.147	.147	.03
Wage Work Born 44–48	.219	.214	.224	.04
Wage Work Born 54–58	.227	.229	.247	.04
General Fert. Rate 1959	231	217.015	187.686	35.60
Ever in Schl, M., 1959	34.3	35.634	50.529	14.96
7–10 yrs Schl, M. 1959	11.7	12.961	17.226	7.94

Predictor variables include mean years of education pre-intervention for birth cohorts 1934–1938, 1944–1948, 1954–1958, general fertility rate (Census 1959), percent of district males that had ever attended school among ages 6–15 (Census 1959), and percent of district males with 7–10 years of education (1959 Census).

3.6. Variation in baseline malaria

In addition, we use Eq. (1) to investigate how the impact of eradication differs by areas with high versus low pre-intervention malaria incidence, corresponding to districts Rukungiri and Kabale,

Table 5
Malaria eradication's effect on years of schooling, primary-school completion, and wage work for males using DD model with synthetic control district weights.

	Yrs School (1)	Prim. Schl Comp. (2)	Wage Work (3)
Born Post-Erad.	.518	.068	−.081
	(.191)***	(.022)***	(.022)***
Born Trt.	−.480	.046	−.004
	(.328)	(.039)	(.036)
Trt. * Post-Erad.	.549	.011	.042
	(.158)***	(.021)	(.019)**
Intercept	5.776	.165	.261
	(.285)***	(.031)***	(.021)***
Obs.	70,109	54,055	60,290
R^2	.664	.241	.308

This table displays the differential change in dependent variables years of schooling, primary school completion, and wage work for those cohorts born in Kigezi post-eradication compared to a synthetic control district. Eq. (1) is used for males only and using 5 year birth cohorts with control districts weighted to reflect the treatment pre-eradication. All regressions are run with district- and birth-cohort fixed effects and include the variables shown above as well as binary variables for living in an urban area, religious affiliation, and 23 ethnicity dummies. Standard errors are clustered at the district level.
* $p < 10\%$.
** $p < 5\%$.
*** $p < 1\%$.

Table 6
Malaria eradication's effect on years of schooling, primary-school completion, and wage work for females using DD model and 5 year birth cohorts with synthetic control weights.

	Yrs School (1)	Prim. Schl Comp. (2)	Wage Work (3)
Born Post-Erad.	1.613	.050	.004
	(.183)***	(.013)***	(.005)
Born Trt.	1.976	.051	.010
	(.348)***	(.020)**	(.006)*
Trt. * Post-Erad.	.425	.021	−.0006
	(.216)**	(.014)	(.006)
Intercept	1.539	.127	.024
	(.683)**	(.049)***	(.037)
Obs.	85288	83105	58534
R^2	.491	.176	.189

This table displays the differential change in dependent variables years of schooling, primary school completion, and wage work for those cohorts born in Kigezi post-eradication compared to a synthetic control district. Eq. (1) is used for females only and using 5 year birth cohorts with control districts weighted to reflect the treatment pre-eradication. All regressions are run with district- and birth-cohort fixed effects and include the variables shown above as well as binary variables for living in an urban area, religious affiliation, and 23 ethnicity dummies. Standard errors are clustered at the district level.
* $p < 10\%$.
** $p < 5\%$.
*** $p < 1\%$.

respectively. As noted, these districts comprised the treatment district as of the 1959–1960 intervention, but had been administratively split by the 1991 Census. Therefore, we rerun (1) with five year birth cohorts, assuming variously that either only those born in high versus low pre-eradication malaria incidence areas were treated, dropping the other district that comprised Kigezi in 1959, and compare educational and economic outcomes to the rest of Uganda. Tables 7 and 8 for males and females, respectively, display the results for dependent variables years of schooling, primary school completion, and wage work. As expected, we find that coefficients on our treatment effect β_3 for all dependent variables are larger in the district with high malaria pre-eradication. The difference is particularly large for males in primary school completion and wage work. For females, the increase in years of schooling observed in Table 6 is driven by increases for those born in the high malaria district of northern Kigezi. Increases in primary school completion and wage work for females are also observed in the high malaria district. That we find larger positive effects in the area with higher pre-eradication malaria incidence supports the conclusion that these gains were indeed generated by the reduction in malaria exposure during childhood.

3.7. Birth cohort analysis

In addition to the synthetic control and DD results, we also examine the treatment effect by birth cohort over time using Eq. (1). 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. Given the importance of malaria exposure's impact in utero and during infancy described above, we expect to find effects beginning for cohorts born 1959

Table 7
Malaria eradication's effect by pre-eradication malaria incidence for years of schooling, primary-school completion, and wage work for males using DD model and 5 year birth cohorts.

	Yrs School		Prim. Schl Comp.		Wage Work	
	High (1)	Low (2)	High (3)	Low (4)	High (5)	Low (6)
Born Post-Erad.	.358	.371	.007	.009	−.067	−.064
	(.118)***	(.117)***	(.012)	(.012)	(.010)***	(.011)***
Born Trt.	.451	−.467	.002	−.025	.045	−.001
	(.206)**	(.258)*	(.016)	(.018)	(.013)***	(.014)
Trt. * Post-Erad.	.535	.452	.070	.033	.030	.013
	(.076)***	(.075)***	(.008)***	(.008)***	(.006)***	(.006)**
Intercept	6.371	6.272	.281	.276	.185	.179
	(.279)***	(.279)***	(.020)***	(.019)***	(.017)***	(.019)***
Obs.	229,381	239,057	229,381	239,057	185,803	194,773
R^2	.74	.733	.325	.322	.319	.317

This table displays the differential change in dependent variables years of schooling, primary school completion, and wage work for those birth cohorts born in high (Rukungiri) versus low (Kabale) pre-eradication malaria incidence areas compared to the rest of Uganda. Eq. (1) is used for males only, separately assuming the treatment area is either Rukungiri or Kabale district, respectively, and using 5 year birth cohorts. All regressions are run with district- and birth-cohort fixed effects and include the variables shown above as well as binary variables for living in an urban area, religious affiliation, and 23 ethnicity dummies.
* $p < 10\%$.
** $p < 5\%$.
*** $p < 1\%$.

Table 8
Malaria eradication's effect by pre-eradication malaria incidence for years of schooling, primary-school completion, and wage work for females using DD model and 5 year birth cohorts.

	Yrs School		Prim. Schl Comp.		Wage Work	
	High (1)	Low (2)	High (3)	Low (4)	High (5)	Low (6)
Born Post-Erad.	1.924	1.904	.087	.085	.007	−.006
	(.096)***	(.096)***	(.007)***	(.007)***	(.005)	(.003)*
Born Trt.	.673	−.197	.015	.005	.002	−.012
	(.176)***	(.243)	(.008)*	(.011)	(.005)	(.007)*
Trt.* Post-Erad.	.491	−.013	.023	−.012	.006	−.007
	(.065)***	(.060)	(.005)***	(.004)***	(.002)**	(.002)***
Intercept	4.078	3.974	.149	.146	.058	.068
	(.207)***	(.205)***	(.010)***	(.011)***	(.005)***	(.006)***
Obs.	256,344	267,513	256,344	267,513	161,341	171,004
R^2	.635	.627	.263	.261	.232	.23

This table displays the differential change in dependent variables years of schooling, primary school completion, and wage work for those birth cohorts born in high (Rukungiri) versus low (Kabale) pre-eradication malaria incidence areas compared to the rest of Uganda. Eq. (1) is used for males only, separately assuming the treatment area is either Rukungiri or Kabale district, respectively, and using 5 year birth cohorts. All regressions are run with district- and birth-cohort fixed effects and include the variables shown above as well as binary variables for living in an urban area, religious affiliation, and 23 ethnicity dummies.

* $p < 10\%$.
** $p < 5\%$.
*** $p < 1\%$.

to 1963. We estimate these cohort-specific relationships using the following equation:

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

In Eq. (2), the coefficient β_c represents the differential five year birth 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 Eq. (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. We run specification (2) separately for each district and by gender to explore whether the timing of this effect coincides with our priors by gender and for each subdistrict as well.

Figs. 10 and 11 plot the β_c coefficients for all five year cohorts born between 1929–1933 and 1969–1973 (ages 18–62) using Eq. (2). These results indicate that the Kigezi region was not poised for human capital takeoff pre-eradication. Indeed, if anything, the differential trend in years of schooling for pre-eradication Kigezi (green, middle line in both figures) was declining, suggesting 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. The pre-eradication high- and low-malaria subdistricts of Rukungiri and Kabale, respectively, also generally experience a trend break at the time of eradication. We observe a sharper trend break at the time of eradication for males compared to females, but the same pattern. This pattern varies slightly for females in the high-malaria area, which experienced slowly improving educational outcomes relative to the rest of Uganda throughout the study period and then a sharper improvement

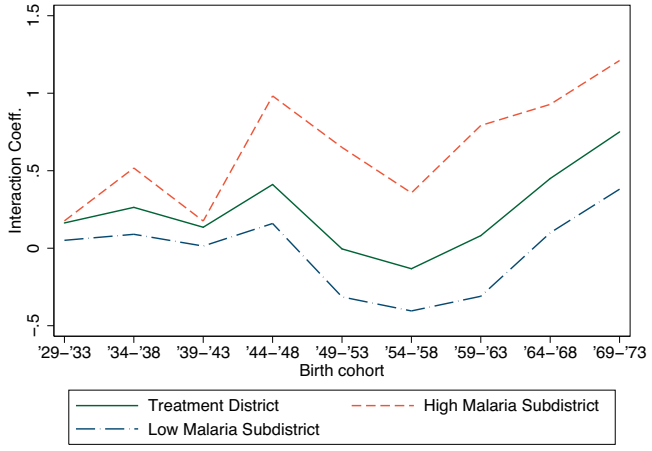

Fig. 10. Differential change in years of schooling by birth cohort for males. This figure plots cohort-specific relationships between the differential change in years of schooling for males in the intervention district (Kigezi) and higher (Rukungiri) and lower (Kabale) pre-eradication malaria areas, compared to the rest of Uganda. Estimates come from β_c coefficients calculated using Eq. (2) where c indexes five year birth cohorts centered on years with individuals aged 0 or 5 to minimize the impact of age heaping. Cohorts born between 1929 and 1973 (ages 18–62 in 1991) are used. Eq. (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.

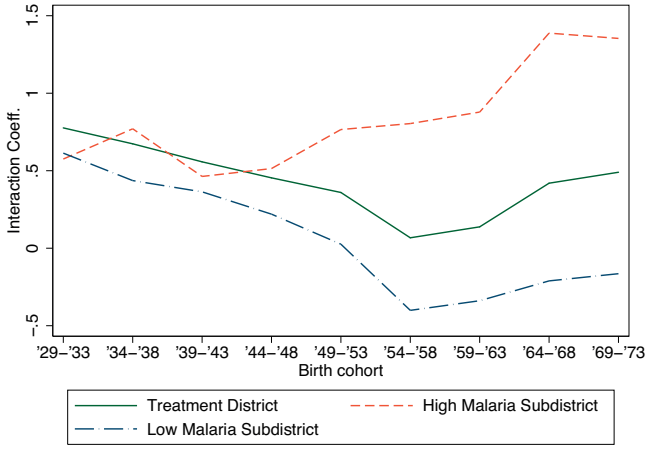

Fig. 11. Differential change in years of schooling by birth cohort for females. This figure plots cohort-specific relationships between the differential change in years of schooling for females in the intervention district (Kigezi) and higher (Rukungiri) and lower (Kabale) pre-eradication malaria areas, compared to the rest of Uganda. Estimates come from β_c coefficients calculated using Eq. (2) where c indexes five year birth cohorts centered on years with individuals aged 0 or 5 to minimize the impact of age heaping. Cohorts born between 1929 and 1973 (ages 18–62 in 1991) are used. Eq. (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.

compared to the lower malaria district for cohorts born 1964 to 1968. These findings strengthen our intuition that the results we see in the main specification are driven by the eradication since the timing of the improvement in years of schooling follows our expectations.

4. Discussion

4.1. Interpretation

To translate our main treatment effect on years of education into income gains, we use estimates on rates of return to education (ROREs).[11] In a review, Psacharopoulos and Patrinos (2004) calculate that the social and private RORE in Africa during primary school is 25.4% and 37.6%, respectively, the highest ROREs of any region. Using the private RORE, our treatment effect for males of 0.52 and females of 0.53 years of schooling would imply an approximately 20% annual earnings return, while applying the standard Mincerian estimate of 10% earnings gain per year of additional education (Mincer, 1974) implies income increases of more than 5% from malaria eradication. Even though elimination of *P. falciparum* malaria in SSA averts many more child deaths than *P. vivax*, we find positive effects in years of schooling for males and females and wage work for males. This provides evidence to support the possibility that health interventions which improve life expectancy need not also reduce per capita income 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 Tables 7 and 8 (treatment effect in column 1 minus 2) averaged between males and females. This is equal to 0.294 more years of schooling in the high versus low malaria district, with the difference driven by greater increases among females.

Given the classification of malariousness from Fig. 1, we define these areas as primarily hyperendemic and mesoendemic respectively, which corresponds to approximate malaria incidence rates of 0.625 and 0.3 (Molineaux, 1988). Then, we divide the difference in treatment effects, 0.294 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 0.9 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 decreases adult income by approximately 17.7% per year. This estimate drops to 4.7% if a 10% RORE is used to translate the educational effect into income changes, which is similar to our result using the synthetic control (we compare this income effect to other papers below).

4.2. Mechanisms

In this section, we detail mechanisms that may be driving the human capital and wage work 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, 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. The fact that years of schooling is found to increase, in contrast to Mexico, may be due to lower employment opportunities for young adults as exhibited by the low incidence of wage work for this group. 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. Less child mortality would tend to weaken the impact of mortality selection, thereby reducing average years of schooling, in addition to raising population growth and potentially overwhelming school systems. However, by extending the time over which returns to human capital investments can be realized, averted adult mortality would 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 (World Health Organization, 2012), however some dissent exists on the relative importance of adult versus child mortality from the disease.[12] 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 eradication 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 any attenuation through mortality selection and higher wages.

4.3. Results in context

This section compares our results to those of Cutler et al. (2010), Bleakley (2010), and Lucas (2010). Lucas (2010) focuses exclusively on the educational effects of malaria eradication in Paraguay and Sri Lanka for females and finds that a 10 percentage point decrease in malaria incidence is associated with a 0.1 year increase in schooling (close to the 0.09 year rise identified above). In addition, 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 approximately 0.5 for males and females already noted, highlighting the similarity

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

[12] 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 about 600,000 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 in SSA attributable to malaria.

in impact found even with greater averted mortality in the Uganda context.

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. 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 (non-significant) declines in Mexico.

For income, Cutler et al. (2010) 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. 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. By translating our educational results into income effects above, we found increases of 4.7–17.7% in malaria incidence terms, depending on the RORE employed (or a 5–20% income increase translating directly from the change in schooling). These results again are of a similar size to the income effects found by Bleakley (2010) attributable to educational gains. Nevertheless, we should emphasize that Bleakley (2010) and Cutler et al. (2010) use direct measures of income whereas we are using RORE and wage work as proxies for income change and are not able to make calculations using the intensive margin of economic impact.

It should also be noted that 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.[13] 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 wage work changes for both males and females separately whereas Lucas (2010) focuses on females alone.

4.4. Threats to identification

The primary disadvantage of this study is that, unlike the other papers in this literature, we are investigating the impact of an eradication campaign for one region of the country (roughly 10% of the population), not its entirety. We address this by creating a weighted average of control districts that most closely resemble the treatment area in pre-intervention trajectory of outcome variables and their predictors. In addition, for the analyses using the DD model, our identification strategy rests on the parallel trends assumption. This is supported by the fact that results using the synthetic control method and DD models are broadly consistent. In addition, we have provided evidence that, like the other interventions described in this literature, the Kigezi eradication campaign was initiated exogenously as a result of the discovery of DDT and the WHO's expansion of the GMEP to SSA. By controlling for age heaping with five year birth cohorts, performing placebo district tests, and exploring the differential effect of eradication by malaria incidence and timing of effect we intend to avoid the possibility that the results we find are caused by factors other than disease reduction. Mortality selection also represents a potential threat to our results. Mortality is correlated with worse educational and economic outcomes, while we only observe individuals that survive to the 1991 Census. It is possible that differential mortality in the treatment district compared to control obscures null or negative effects of eradication if outcomes were observed for full birth cohorts. However, Fig. C.12 shows that mortality in the treatment district is in fact comparatively low compared to other districts such that mortality selection, if accounted for, would differentially improve average outcomes in Kigezi compared to the rest of Uganda. This suggests that mortality selection biases our results downward.

In addition, during the time frame of our study, 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 and the expulsion of 80,000 Asian Ugandans in September 1972. In addition, the HIV epidemic hit Uganda in the mid-1980s. Although, we cannot control for all of these explicitly, we note that by constraining our sample to largely 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 nor Amin, both from different districts in northern Uganda, provided patronage to the Kigezi region. This reduces the likelihood that the results we observe were driven by political factors present in the Kigezi region before or after eradication or endogenous to the decision to implement this malaria reduction campaign.[14] We also observe increasing outcome gains for cohorts born in 1969–73 compared to 1964–68. This could reflect delayed improvements in education and labor market outcomes because of schooling supply constraints, but we cannot completely rule out that the expanding benefits found are driven by omitted variables.

When it comes to external validity, although this analysis provides unique evidence on the long-term human capital and economic effects of malaria eradication, there are differences between the intervention and current practice that may reduce the generalizability of these results. First, the eradication was implemented by a colonial government, not a current African government nor an NGO and each vary in their effectiveness and scale. Moreover, current methods of malaria control necessarily differ from the combination of DDT spraying and anti-malarial medication investigated here. Nevertheless, we argue that this quasi-experimental change in malaria exposure constitutes the best available evidence to guide decision-making on probable impacts.

5. Conclusion

The preceding analysis evaluates the educational and economic impact of a malaria eradication campaign in southwestern Uganda. The program was implemented in conjunction with the WHO's Global Malaria Eradication Program and produced quasi-experimental variation in health by birth cohort that we exploit to identify treatment effects. First, we translate summary birth histories into child mortality estimates and observe a decline of almost 16% in under five deaths in the treatment area coincident with eradication. This decline is larger than, but cannot be statistically differentiated from, the decrease found in the rest of Uganda. For the main analysis, we find that malaria eradication produced an increase in schooling of over 0.5 years for males and females, with improvements in primary school completion for females, and an almost 40% rise in the likelihood of wage work for males. This

[13] 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.

[14] See Appendix D for additional background on the political and historical context in Uganda during the time of Kigezi's eradication campaign.

educational improvement corresponds to an annual income gain of between 5% and 20%, depending on the rate of return to education used. These effects are found to be robust to a host of additional tests using both the synthetic control method and a differences-in-differences model. Importantly, we also find larger treatment effects in areas with higher pre-eradication malaria incidence and that the timing of the rise in schooling coincides with the eradication campaign. Comparing educational gains between these high- and low-malaria districts and normalizing by the probability of infection, we find a 4.7–17.7% annual income gain in Uganda, depending on the rate of return to education used. Even though the type of malaria prevalent in this intervention district and throughout Africa may reduce the long-term benefits of eradication, the magnitude of our treatment effects are similar or larger than those found in other studies in this literature.

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, elimination, eradication, and vaccine development are again being seriously considered in policy circles.[15] Our results suggest that policies such as the comprehensive provision of insecticide-treated bednets or anti-malarial medication can improve long-term economic outcomes. Moreover, this analysis indicates that cost-effectiveness studies of malaria control should include these economic benefits.

Consistent with previous findings, we observe that fighting malaria on its own will not pull African nations out of poverty. However, these results also show that, contrary to theoretical predictions and literature on mortality-reducing interventions, malaria eradication in sub-Saharan Africa can indeed induce positive long-term human capital and economic effects. Consequently, expenditure to fight this terrible disease should be considered both a humanitarian endeavor as well as an investment in future economic well-being.

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.

Appendix A. Additional literature review on health's effect on economic outcomes

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. Maccini and 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, 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 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 in. shorter than those from malaria free counties, while Hong (2013) finds a positive association between malaria exposure and old-age disability. 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.

This paper is one of the first to identify the long-term impact of malaria eradication in SSA. Other studies however answer related questions. 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. Ashraf et al. (2010) explore the association between 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 from these trials in coastal Kenya show that malaria prevention improves school attendance, cognition, and a child's ability to sustain attention in class (Brooker et al., 2000, 2010; Clarke et al., 2008; Halliday et al., 2012).

The debate in the macroeconomic literature on the sign and magnitude of how health improvements affect income is ongoing. For malaria, Gallup and Sachs (2001) estimate that wiping out the disease in SSA could increase per capita economic growth by as much as 2.6% per year. For health overall, Bloom et al. (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 output by 4%. 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%. Pritchett and 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. 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."

Appendix B. Pre-eradication malaria prevalence in Uganda

See Fig. B.1.

Appendix C. Calculating child mortality using indirect methods

Using aggregated summary birth history (SBH) data from the 1969 census, we employ indirect estimation methods, pioneered by Brass and Coale (1968), to investigate infant and child mortality in Uganda around the Kigezi malaria eradication period. Although SBH data (the proportion surviving of children ever born by women of given age groups) is an indicator of child mortality, additional assumptions on the age pattern of childbearing and child survivorship probabilities are required to translate this information into mortality estimates. Following Hill (2013), SBHs are translated into mortality estimates by (1) calculating proportions of dead children ever born (CEB) by mother's five year age group, (2) calculating the average number of children ever born to women in each five year age group, (3) applying the Princeton 'North' family of model life tables to convert the proportions dead of children ever born

[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).

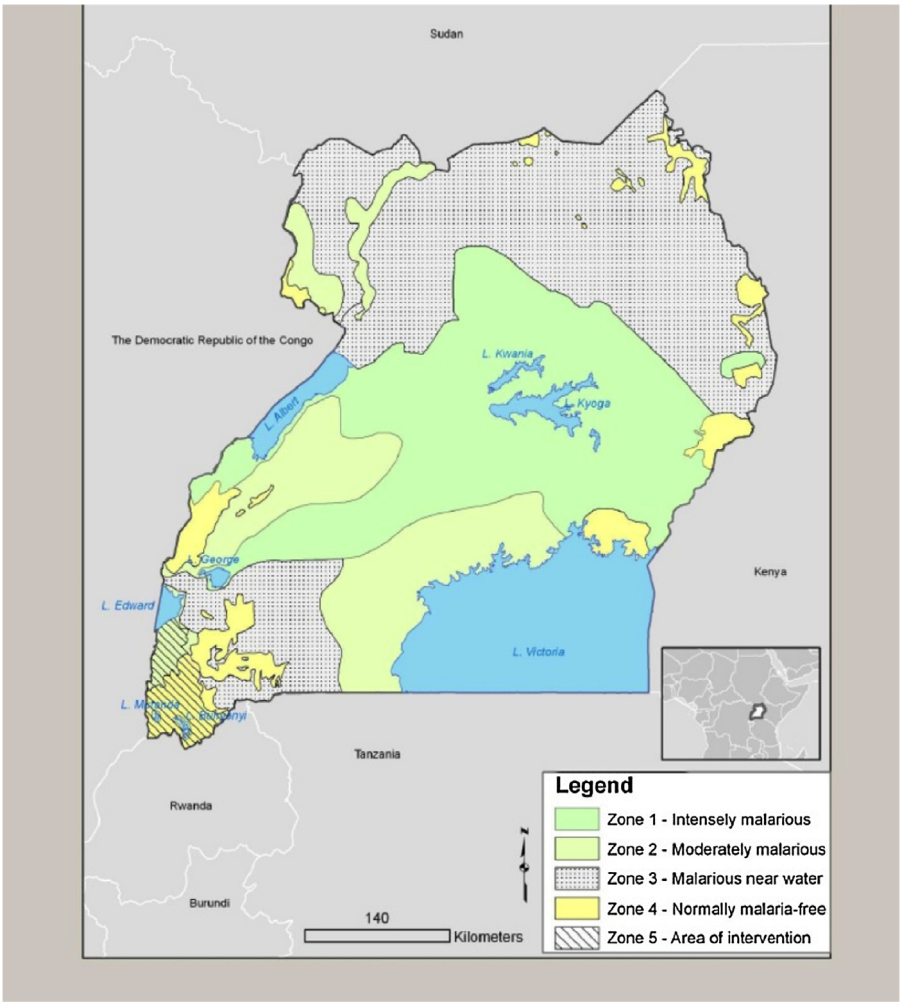

Fig. B.1. Source: Harvard Pusey Library; Zulueta et al. (1964).

by five year age group into probabilities of death between age 0 and n. This is done by applying coefficients that relate proportion dead and probability of death for the Princeton 'North' model life table to the proportion dead of CEB by mother's five year age group. (4) Then, the time reference (the center of the distribution of child mortality for a five year age group of mothers) is calculated, which entails applying regression coefficients obtained from the Princeton 'North' model life table, relating empirical parity ratios from the SBH to time of birth with full birth histories from a model population using the relationship given in the model life table. Given the paucity of direct estimates of child mortality for this period in Uganda, the North model life table is chosen because it exhibits the highest ratio of infant to child mortality at each mortality level of all model life tables commonly employed.

This method assumes that fertility and childhood mortality are accurately reflected in the choice of model life table, child mortality does not vary within the five year age group of mothers, there is no correlation between mortality risk and mother's survival, and that changes in child mortality were gradual. If, for example, fertility has been changing, then the ratio of average parities by age group obtained from a cross-sectional survey may not accurately reflect the distribution in time of births to the women in each age group. Also of note, estimates of child mortality using the birth history of women aged 15–19 are biased by the fact that younger mothers are more likely to be poor, less educated, experience higher risk pregnancies, and therefore have higher child mortality rates than what is representative of the overall population at that time. As is standard practice, we therefore exclude the youngest age group of mothers 15–19 from this analysis. When it comes to estimation of standard errors around the mortality estimates, these data represent the total population of children ever born and children died in Uganda for women aged 20–49 in 1969, and therefore are not a sample. However, uncertainty exists when attributing mortality levels to a specific date because these data reflect accumulated fertility and child mortality experienced around each five year age group. Therefore, these calculations represent smoothed mortality estimates instead of cohort specific mortality at a given year. Fig. C.12 displays the results of our mortality analysis by district, showing average child and infant mortality per thousand births by district from 1953 to 1967.

We also perform a statistical test of whether the decline in child mortality in Kigezi before and after eradication differs statistically from the decline observed in the rest of Uganda. This is accomplished by running a fully saturated model to identify mean child mortality for each birth cohort in the mortality data and then a separate interaction term for mortality in Kigezi. The specification run is the following:

$$Y_{cd} = \alpha + \beta_1 * K_d + \sum_c \beta_c \mu_c + \sum_c \beta_c (\mu_c * K_d) + \epsilon_{cd} \quad \text{(C.1)}$$

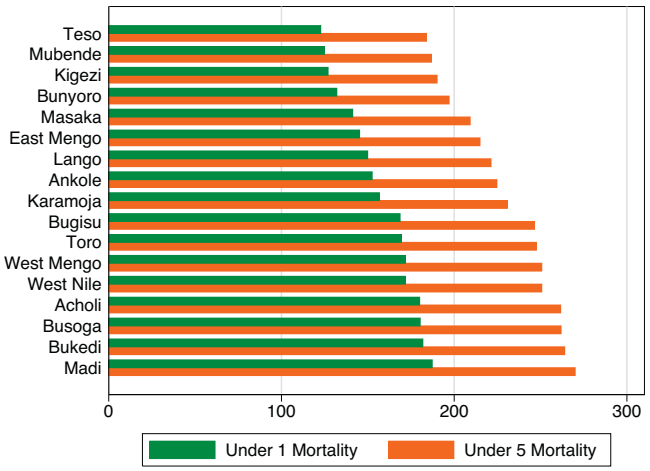

Fig. C.12. Average under 5 and under 1 mortality per thousand births by district in Uganda. This figure shows average child and infant mortality per thousand births by district from 1953 to 1967, calculated using summary birth histories from the Uganda Census 1969.

where α represents the intercept, K_d a dummy variable for child mortality in the treatment area of Kigezi, μ_c a set of birth cohort dummy variables with the pre-eradication time period being the omitted category, and $\mu_c * K_d$ representing the interaction between each birth cohort and being born in Kigezi. Because the omitted birth-cohort category immediately precedes pre-eradication, testing $H_0 : \mu_3 * K_d = 0$ is a test of whether the change in child mortality in the treatment district before and after eradication differs statistically from the change in child mortality in the rest of Uganda. Although we observe a 16% decline in child mortality in the treatment district compared to an 11% decrease in the rest of Uganda, this difference is not statistically different from zero (p-value of 0.75).

Appendix D. WHO's Global Malaria Eradication Campaign in middle Africa

In 1950, the WHO recommended to governments responsible for the administration of African territories that "malaria should be controlled by modern methods as soon as feasible, whatever the degree of endemicity, and without awaiting the outcome of further experiments" (World Health Organization, 1950). The WHO strengthened its call to action in 1955 by launching the Global Malaria Eradication Program (Nájera et al., 2011). Uganda's malaria eradication campaign in Kigezi district was part of the new WHO agenda, and it was initiated jointly by the WHO and the government of Uganda. Initially, the campaign was limited to northern Kigezi, but after a successful first year that produced an almost complete disappearance of malaria, project officials decided to extend the anti-malaria operations to all parts of Kigezi that had evidence of transmission (Zulueta et al., 1964). The campaign was started in northern Kigezi because malaria endemicity there had been a significant obstacle for a government resettlement program that was attempting to relocate people of the Bachiga ethnic group from southern Kigezi.

The presence of such a resettlement program might raise concerns that it could bias our study results. For example, if the government installed better health and educational programs in northern Kigezi for the Bakiga people compared to what was present before resettlement, this could upwardly bias the average level of educational outcomes in Kigezi compared to non-Kigezi regions of Uganda. However, reports describing this malaria eradication campaign, resettlement program, and the social history of post-independence Uganda make no mention of any such health or educational interventions apart from the malaria eradication campaign itself (Zulueta et al., 1961, 1964; Mutibwa, 1992). And indeed, the birth cohort analysis we undertake shows that, if anything, the educational trajectory of the treatment area was declining relative to the rest of Uganda immediately preceding the intervention.

Snow et al. (2012) identifies 25 malaria control or elimination projects in middle Africa between 1945 and 1979. Of the 25, fourteen included only indoor residual spraying (IRS), five included only drug-based malaria control without administration of IRS, and six included both drug-based malaria control and IRS. The Kigezi intervention, as noted above, involved both IRS and mass drug administration and was, of the six similar programs, the largest in scope and most successful. There was one other malaria control project in Uganda that was much more limited in scope than the Kigezi campaign and occurred around two sugar plantations near Kampala between 1964 and 65. The WHO discontinued the Global Malaria Eradication Program in 1969 upon recognizing that eradication was not achievable with available means in many areas, although eradication remained the ultimate objective (Nájera et al., 2011). Major barriers to malaria elimination in SSA were the high cost of insecticides, fears of rapid escalation of vector resistance to insecticides, and mixed results from various malaria elimination pilot projects in the region. In addition, much of Africa did not have the strong and effective health systems necessary for successful elimination programs (Snow et al., 2012).

Appendix E. Background on Uganda's political history

As with so many nations in post-colonial Africa, the decisions made by the colonizers had an important effect on future political and social development. To implement their administration, the British favored one ethnicity over all others, the Baganda, which meant that this group dominated the upper echelons of education, politics, and business immediately preceding and following independence in 1962. Although favored in all other realms, the British did not want the Baganda to become too powerful and therefore recruited military and security forces from the North and East (Mutibwa, 1992). This is of interest for the purposes of this investigation because the Baganda comprised less than 1% of Kigezi's population in 1991. Instead, the districts that made up Kigezi in 1959 (Rukungiri and Kabale) were 75% Bakiga and 10% Bafumbira, according to the 1991 Census. Educational attainment is a particularly important determinant of economic prospects in Uganda. Indeed, in Uganda, "the most powerful determinant of occupational attainment was said to be the level of an individual's past academic performance; it was more powerful than either sex or social status. This was attributable to two things: the high degree to which educational qualifications have been used as formal determinants of labor market entry; and the degree to which educational attainment itself is determined by academic performance rather than by quotas or by political recommendations," (Heyneman, 1983, p. 407). After the elections that followed Uganda's independence in 1962, the Democratic Party (DP) formed the single largest party in Parliament. The DP favored national unity and was popular in the non-Baganda South. However, in an unlikely alliance, the two smaller parties – the Uganda People's Congress (UPC), heavily supported in the North, and Kabaka Yekka (KY), a pro-Baganda independence party – combined to form an alliance headed by the northerner Milton Obote of the UPC. Over time, this alliance frayed as Obote became increasingly dictatorial until he was deposed in 1971 by another northerner from West Nile district, the commander of Uganda's army, Idi Amin. This history is important to understand the nature

of potential political patronage during and after Kigezi's eradication campaign and how these political changes did not involve politicians from the relatively under-developed southwest. The following certainly cannot completely rule out the possibility that socioeconomic characteristics differed in the treatment district of Kigezi resulting from differential political patronage. However, this lack of tribal connection generally supports the assertion that there was no link between political or ethnic favoritism and the effects found.

Appendix F. Placebo analysis of main effects

Following Abadie et al. (2010), we test the robustness of our synthetic control results by re-running the same model for each district in Uganda that did not experience malaria eradication. If we find that the effect produced in Kigezi is large in relation to the impact found in other districts, then we can conclude the observed changes are unlikely to be driven by chance, but Kigezi's malaria eradication campaign instead. Of course, this judgment must be tempered by the fact that large unknown changes in education and wage work may have obtained in other districts of Uganda during this time as well. When estimating the effect on placebo districts, mean squared prediction error (MSPE, the average of the squared difference in pre-eradication dependent variables between the real and synthetic district) is used to determine how effectively the synthetic control is implemented. For two districts, their MSPE is consistently greater than fives times the average and are therefore excluded from the placebo analysis, resulting in a comparison of the remaining 15 Ugandan districts in 1959.

Figs. F.13 and F.14 show placebo analysis implemented with years of education for males and females, respectively. Both figures indicate that the difference between years of education in real versus synthetic Kigezi in cohorts born completely after eradication is either the largest or among the largest increase in years of education observed among all other districts. For male cohorts born 1964–1968 and 1969–1973, the increase in years of schooling in Kigezi is the largest of 15 districts. The probability of estimating such a gap as that observed in Kigezi for these two birth cohorts is 1/15 = 6.67%, given that there are 14 other control districts. For females, we find that the years of education gap in Kigezi is the second or third largest of all placebo districts, representing a probability of observing that gap, assuming a random distribution, of

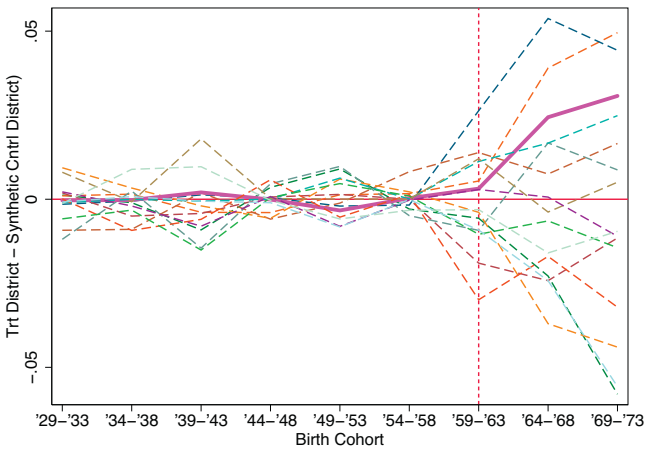

Fig. F.15. Placebo analysis for female primary school completion. Placebo analysis assuming that each district in Uganda experienced malaria eradication. Two districts with model error greater than five times the average are excluded. The thick line represents the actual treatment district of Kigezi and the dotted lines represent all other 1959 Uganda districts.

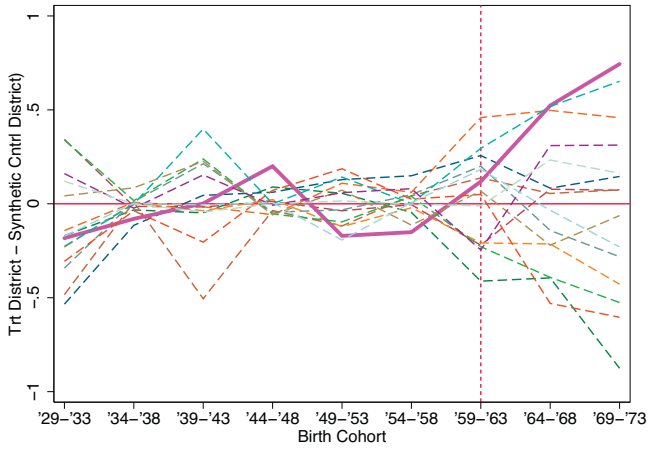

Fig. F.13. Placebo analysis for male educational attainment. Placebo analysis assuming that each district in Uganda experienced malaria eradication. Two districts with model error greater than five times the average are excluded. The thick line represents the actual treatment district of Kigezi and the dotted lines represent all other 1959 Uganda districts.

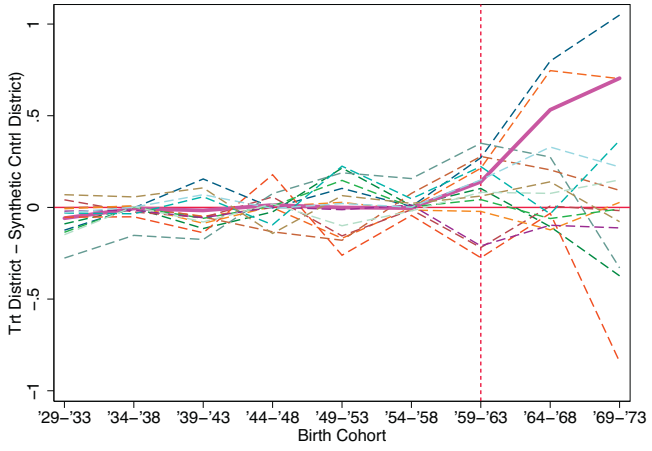

Fig. F.14. Placebo analysis for female educational attainment. Placebo analysis assuming that each district in Uganda experienced malaria eradication. Two districts with model error greater than five times the average are excluded. The thick line represents the actual treatment district of Kigezi and the dotted lines represent all other 1959 Uganda districts.

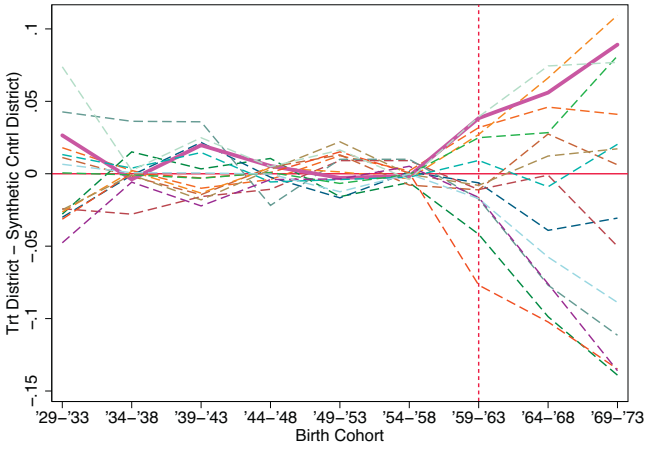

Fig. F.16. Placebo analysis for male wage work. Placebo analysis assuming that each district in Uganda experienced malaria eradication. Two districts with model error greater than five times the average are excluded. The thick line represents the actual treatment district of Kigezi and the dotted lines represent all other 1959 Uganda districts.

Table G.9
District weights used to create the synthetic control district by gender and dependent variable.

District	Male			Female		
	Years Educ.	Prim. Schl	Wage Work	Years Educ.	Prim. Schl	Wage Work
Acholi	0.197	0.113	0.086	0.323	0.347	0
Ankole	0	0	0	0	0	0
Bugisu	0.13	0	0	0	0.064	0
Bukedi	0	0	0.151	0.09	0	0.12
Bunyoro	0	0	0	0	0	0
Busoga	0	0	0	0	0	0
East Mengo	0	0	0	0	0	0
Karamoja	0.301	0.248	0.247	0.21	0.391	0.3
Lango	0	0	0	0	0	0.258
Madi	0.215	0	0.438	0.144	0.025	0.098
Masaka	0	0	0	0	0	0
Mubende	0	0	0	0	0	0
Teso	0	0	0	0	0	0
Toro	0.158	0.639	0	0.233	0.173	0.223
West Mengo	0	0	0.078	0	0	0
West Nile	0	0	0	0	0	0

13% and 20%. Similar results are found when we implement the placebo district analysis for females using primary school completion in Fig. F.15 again resulting in a 20% probability. We also observe that the same two districts are the ones that are larger than Kigezi for female schooling and primary school completion, suggesting that educational expansion may have also been occurring in these areas as well.

Fig. F.16 displays placebo analysis for wage work among males. We observe that the treatment effect is either the second or third largest in Kigezi compared to all other districts in Uganda for cohorts born 1959–1963 and 1969–1973 (second largest), 1964–1969 (third largest). The probability of observing these magnitudes is 13.3% and 20%, respectively. Taken together, these placebo tests indicate that over all dependent variables for which we find treatment effects, we observe changes for Kigezi that are in the upper tail of the distribution found among all districts in Uganda.

Appendix G. More synthetic control results

See Table G.9.

G.1. Primary school completion

See Table G.10, Figs. G.17 and G.18.

G.2. Wage work

Wage work is measured by recoding the census variable worker class with categories: not in universe, self-employed, wage/salary worker, unpaid family worker, and unknown into a binary variable

Table G.10
Primary school completion (%) predictor means (males).

Variables	Treatment (Kigezi)		Rest of Uganda
	Real	Synthetic	
Primary Schl Comp. Born 34–38	.082	.086	.138
Primary Schl Comp. Born 44–48	.159	.154	.253
Primary Schl Comp. Born 54–58	.143	.154	.264
General Fert. Rate 1959	231	208.236	187.686
Ever in Schl, M. 1959	34.3	32.249	50.529
7–10 yrs Schl, M. 1959	11.7	12.062	17.226

Predictor variables include mean years of education pre-intervention for birth cohorts 1934–1938, 1944–1948, 1954–1958, general fertility rate (Census 1959), percent of district males that had ever attended school among ages 6–15 (Census 1959), and percent of district males with 7–10 years of education (1959 Census).

Fig. G.17. Treatment effect for male primary school completion. Using data from Uganda Census 1959 and Census 1991 to create a synthetic control district similar to the treatment district (Kigezi) in the time path of percent reporting primary school completion and pre-intervention characteristics. Synthetic control method employed for each district in Uganda 1959 with 5 year birth cohorts.

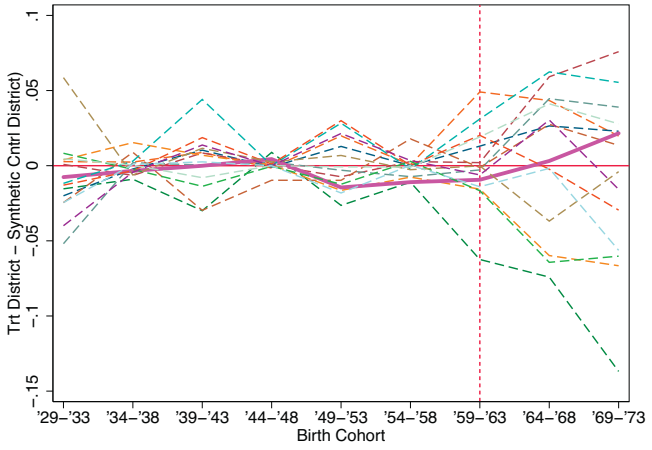

Fig. G.18. Placebo analysis for male primary school completion. Placebo analysis assuming that each district in Uganda experienced the malaria eradication treatment. Two highest MSPE excluded. Thick line represents actual treatment district – Kigezi. Dotted lines represent all other 1959 Uganda districts.

Table G.11
Wage work (%) predictor means (females).

Variables	Treatment (Kigezi)		Rest of Uganda
	Real	Synthetic	
Wage Work Born 34–38	.013	.014	.021
Wage Work Born 44–48	.025	.024	.047
Wage Work Born 54–58	.04	.04	.083
General Fert. Rate 1959	231	214.362	187.686
Ever in Schl, F. 1959	13.6	14.824	31.632
7–10 yrs Schl, F. 1959	11	5.33	7.833

Predictor variables include mean years of education pre-intervention for birth cohorts 1934–1938, 1944–1948, 1954–1958, general fertility rate (Census 1959), percent of district females that had ever attended school among ages 6–15 (Census 1959), and percent of district females with 7–10 years of education (1959 Census).

for wage/salary work and making the unknown and not in universe categories missing. Of all non-missing workers 18–42, 15% are categorized as wage workers, 22% of males and 7% of females. This variable is used because it represents a proxy for higher and more stable income than agricultural work, while this data does not include information on income or consumption.

See Table G.11, Figs. G.19 and G.20.

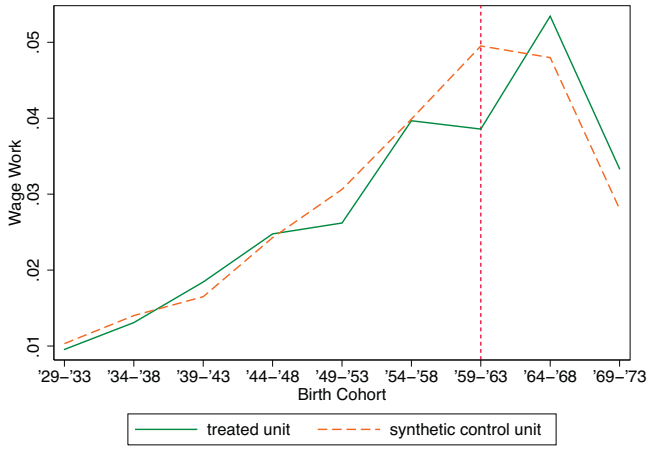

Fig. G.19. Treatment effect for female wage work. Using data from Uganda Census 1959 and Census 1991 to create a synthetic control district similar to the treatment district (Kigezi) in the time path of percent reporting non-agricultural work and pre-intervention characteristics. Synthetic control method employed for each district in Uganda 1959 with 5 year birth cohorts.

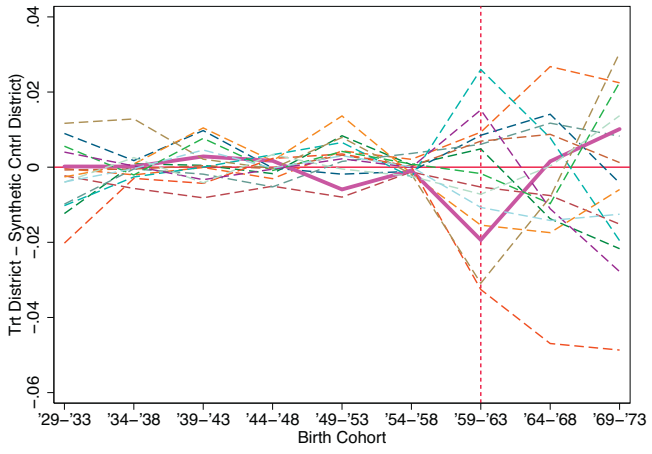

Fig. G.20. Placebo analysis for female wage work. Placebo analysis assuming that each district in Uganda experienced the malaria eradication treatment. Two highest MSPE excluded. Thick line represents actual treatment district – Kigezi. Dotted lines represent all other 1959 Uganda districts.

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