MALARIA IN THE AMERICAS:
A RETROSPECTIVE ANALYSIS OF CHILDHOOD EXPOSURE*

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Abstract

This study considers the malaria-eradication campaigns in the United States (circa 1920), and in Brazil, Colombia and Mexico (circa 1955) in order to measure how much childhood exposure to malaria depresses labor productivity. The campaigns began because of advances in health technology, which mitigates concerns about reverse causality. Malarious areas saw large drops in the disease thereafter. Relative to non-malarious areas, cohorts born after eradication had higher income as adults than the preceding generation. These changes coincided with childhood exposure to the campaigns rather than with pre-existing trends.

Keywords: Malaria, returns to health, eradication campaigns.

JEL Classification: I12, J24, 010, H43.

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MALARIA EN LAS AMÉRICAS:
UN ANÁLISIS RETROSPECTIVO DE LA EXPOSICIÓN INFANTIL

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Resumen

Este estudio considera las campañas de erradicación contra la malaria en los Estados Unidos (circa 1920), Brasil, Colombia y México (circa 1955) con el objetivo de medir como la exposición infantil a la malaria suprime la productividad laboral. Las campañas empezaron por causa de avances en conocimiento médico, lo cual amortigua el problema de endogeneidad. Áreas maláricas experimentaron reducciones grandes en la enfermedad después del comienzo de las campañas. En términos absolutos y relativos a las áreas no-maláricas, cohortes nacidos después de las campañas recibieron mayores ingresos que los cohortes de la generación anterior. Los cambios en ingreso por cohorte coincidieron con la exposición infantil a las campañas, en vez de una tendencia pre-existente.

Palabras clave: Malaria, retornos de salud, campañas de erradicación.

Clasificación JEL: I12, J24, 010, H43.
1 Introduction

The disease known as malaria, a scourge of mankind through history, persists in tropical countries up to the present day. These same tropical areas have, generally speaking, a much lower level of economic development than that enjoyed in the temperate climates. These facts lead us to a natural question: does malaria hold back economic progress?

The simple correlation between tropical disease and productivity cannot answer this question. Malaria might depress productivity, but the failure to eradicate malaria might equally well be a symptom of underdevelopment. Indeed, tropical countries also tend to have debilitating institutions, such as the poor protection of property rights and weak rule of law, the latter of which makes it difficult to marshal resources in support of public health. This important international question has an interesting parallel among regions within countries. For example, southern Mexico, the southern United States, the tierra caliente of Colombia, and the north of Brazil have borne a disproportionate burden of malaria infection in those countries, but these regions were also disproportionately host to colonial, extractive institutions for several centuries. Both factors plausibly have a role in the failure to eradicate malaria. How can we cut through this Gordian knot of circular causality? The standard econometric answer is to consider plausibly exogenous variation in malaria. A possible source of such variation comes from targeted interventions in public health.

The present study considers two major attempts to eradicate malaria in the Americas during the Twentieth century. The first episode analyzed took place in the southern U.S., largely in the 1920s. In the decades before, the cause and transmission mechanism of the disease were first understood by European physicians, and this knowledge allowed the U.S. Army to attempt scientifically based campaigns against malaria in areas it had recently occupied: Havana and the Panama Canal Zone. Subsequently, this wealth of new knowledge and experience related to disease was applied to the malaria problem in the South. The second episode is the worldwide malaria eradication campaign, and in particular as it was implemented in Brazil, Colombia, and Mexico (principally in the 1950s). The efforts to eradicate malaria worldwide were spurred on by the discovery of DDT, a powerful pesticide. After World War II, the World Health Organization (WHO) helped many afflicted countries put together programs of spraying to combat malaria transmission.

The campaigns in these regions partially interrupted the malaria transmission cycle and brought about marked drops in infection in a relatively short period of time. (Further background on the disease and the eradication efforts is found in Section 2.) Additionally, sufficient time has passed that we can evaluate the long-term consequences of eradication.

The relatively rapid impact of the treatment campaigns combine with cross-area heterogeneity to form the research design of the present study. These four countries are geographically variegated, such that, within each country, some regions have climates that support malaria transmissions, while other regions do not. Areas with high malaria infection rates had more to gain from eradication,
but the non-malarious areas serve as a comparison group, filtering out common trends in national policy, for example. Moreover, the reductions in disease burden occur in the space of a few years, and resulted from critical innovations to knowledge and spending, and these innovations came largely from outside the studied areas. This latter fact mitigates the usual concern about policy endogeneity.

The particular goal of the present study is to identify the role that childhood exposure to malaria has in subsequent labor productivity as an adult. While direct effects of malaria on adults can be partially measured with lost wages from work absences, little is known about effects that persist from infection in early life. Children are more susceptible to malaria than adults, most likely because prolonged exposure to the disease brings some degree of resistance. Although partial immunity is conferred by age, the damage from childhood exposure to malaria may be hard to undo: Most of a person's human-capital and physiological development happens in childhood. On the physiological side, a malaria-free childhood might mean that the individual is more robust as an adult, with concomitant increases in labor supply. On the human-capital side, fewer school absences and less anemia translates into more learning. This would be manifested in the data as greater literacy, higher adult earnings, and, for a fixed time in school, higher returns to schooling. This also affects the schooling decision, but, because malaria also affects the childhood wage (the opportunity cost of schooling), this latter effect is ambiguously signed by theory. Malaria's possible effect on contemporaneous wages implies that an additional channel is via parental income.

To identify the effects of early-life malaria infection, I begin by noting that the timing of the eradication campaign induces variation in childhood malaria that has a marked pattern across year-of-birth cohorts. Cohorts that were already adults before the campaigns were too old to have any early-life exposure to the eradication efforts. In contrast, later cohorts experienced reduced malaria infection during their childhood. I therefore compare cohorts based on (i) the pre-eradication malaria burden in their area of birth and (ii) their year of birth relative to the malaria-eradication campaigns. Being born later and in an area with high pre-campaign malaria burden implies more exposure to the benefits of the eradication programs. To test this hypothesis, census microdata samples from all four countries are used to construct data on a panel of cohorts by birth year and place. Pre-campaign malaria intensity across areas is estimated from a variety of sources. (The identification strategy is further described in Section 2.4, while the data construction is detailed in Section 3 and the appendices.)

Cohorts more exposed to the eradication efforts went on to earn higher incomes and have higher literacy rates as adults. Mixed results are found for years of schooling, consistent with the economic theory of schooling. In Section 4, I present estimates of regressions of adult outcomes on pre-campaign malaria for each year of birth in the sample. Graphs of the resulting coefficients reveal a shift in the malaria-income relationship that coincides with childhood exposure to the
eradication efforts. Furthermore, statistical tests tend to favor this childhood-exposure hypothesis over pre-existing polynomial trends.

These results are not sensitive to accounting for a variety of alternative hypotheses. In Section 5, I show that this shift was systematic across areas of these countries, and not due to a few outliers. Moreover, I obtain essentially similar estimates of malaria coefficients even when controlling for different indicators of health and economic development. In Section 6.2, I present evidence that these results are not due to mortality selection, the failure to account for general-equilibrium spillovers across agents, or the decline of other vector-borne diseases.

The estimates below indicate that childhood malaria has a large, depressing effect on adult productivity. Reduced-form effects, when comparing the least malarious to the most malarious areas within a country, are on the range of 10 to 40 percent. To get a sense of the magnitude of the effect per probability of childhood infection, I normalize the reduced-form numbers with estimates of the pre-campaign infection rates in Section 6.1. Although it is impossible to completely rule out that the intervention had effects through channels besides measured malaria infection, the results suggest persistent childhood malaria infection reduces adult income by 40 to 60 percent. In Section 6.3, I show that these estimates explain a modest fraction of the gap between the areas under study and more developed economies.

2 Malaria and the Eradication Campaigns

2.1 The Disease

Malaria is a parasitic disease that afflicts humans. Acute symptoms of infection include fever and shivering. The main chronic symptom is anemia. Malaria results in death on occasion, but the strains prevalent in the Americas (vivax and to a lesser extent malariae) have low case-fatality rates compared with the predominantly African variety (falciparum).

The parasite has a complicated life-cycle that is partly spent in a mosquito vector and partly in the human host. The disease is transmitted when a mosquito takes a blood meal from an infected person and, some time later, bites another person. Because of the crucial role played by mosquitoes in the transmission cycle, warm and wetter climates are more likely to sustain endemic malaria.

2.2 Efforts against Malaria in the Southern U.S., circa 1920

The turn of the 20th century saw considerable advances in the scientific understanding of the disease. Doctor Charles Louis Alphonse Laveran, of the French army, showed in the early 1890s through microscopic studies that malaria is caused by a single-celled organism. Dr. (later Sir) Ronald Ross, of the British Indian Medical Service, discovered in the late 1890s that malaria is
transmitted via mosquitoes. These discoveries proved invaluable to taking on the malaria problem in a scientific and systematic way, and both men later won Nobel Prizes for Medicine.

The U.S. government’s interest in vector-borne diseases arose in the 20th century not because of a new-found interest in the Southern region, but because of the acquisition of Cuba and of the Panama Canal Zone. Early in the occupation of Cuba, the U.S. Army dispatched a team of physicians, among them Dr. Walter Reed, to Havana to combat yellow fever and malaria. Armed with the new knowledge about these diseases (from Laveran, Ross, and others), the Army was able to bring these diseases under control in that city. Another team of American physicians, this time led by Dr. William Gorgas, was able to bring these diseases under control in the Canal Zone, which was a considerable challenge given that much of the area was a humid, tropical jungle.¹

The progress made by U.S. Army doctors against malaria in Cuba and Panama inspired work back home in the South in the latter half of the 1910s. Several physicians in the United States Public Health Service (PHS) began collecting information on the distribution of malaria throughout the South and the prevalence of the various species of parasites and mosquitoes.² The PHS began actual treatment campaigns in a limited way, first by controlling malaria in a handful of mill villages (to which the Service had been invited by the mill owners). The Rockefeller Foundation, having mounted a successful campaign against hookworm in the early 1910s, also funded anti-malarial work later in the decade through its International Health Board (IHB). These two groups sponsored demonstration projects in a number of small, rural towns across the South. They employed a variety of new methods (spraying, water management, screening, and mass administration of quinine) and most of these demonstrations were highly successful, resulting in 70% declines in morbidity.

The federal government’s large-scale efforts against malaria in the South began with World War I (WWI). In previous wars, a significant portion of the troops were made unfit for service because of disease contracted on or around encampments. The PHS, working now with both a strong knowledge base on malaria control and greatly increased funding, undertook drainage and larviciding operations in Southern military camps as well as in surrounding areas. After the War, the IHB and PHS expanded the demonstration work further. By the mid-1920s, the boards of

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¹It is doubtful that the construction of the Canal would have been economically feasible were it not for these sizable innovations to knowledge. The following anecdote is illustrative of the primitive state of medical knowledge about malaria just a few years earlier:

And all the while, in the lovely gardens surrounding the hospital, thousands of ring-shaped pottery dishes filled with water to protect plants and flowers from ants provided perfect breeding grounds for mosquitoes. Even in the sick wards themselves the legs of the beds were placed in shallow water, again to keep the ants away, and there were no screen in any of the windows or doors. Patients, furthermore, were placed in the wards according to nationality, rather than by disease, with the results that every ward had its malaria and yellow-fever cases. As Dr. Gorgas was to write, had the French been consciously trying to propagate malaria and yellow fever, they could not have provided conditions better suited for the purpose. (McCullough, 1977)

History records that the French effort to build a canal across the isthmus did indeed fail, in part because of malaria.

²Williams (1951) presents a thorough history of the U.S. Public Health Service.
health of each state, following the IHB/PHS model, had taken up the mantle of the malaria control in all but the most peripheral areas of the region (Williams, 1951).

During this period, the South experienced a substantial decline in malaria. Malaria mortality per capita is seen in Panel A of Figure 1. Apart from a hiccup in the first years of the Depression, the region saw a drop of around 50 percent in the 15 years after WWI.

2.3 The Worldwide Campaign to Eradicate Malaria, circa 1950

While some of the innovations in malaria control diffused to less-developed regions, the tropical countries of the Americas would wait for further technological advance before launching serious campaigns against malaria. These campaigns had a peculiar starting point: In 1941, a Swiss chemist seeking to build a better mothball re-discovered a chemical known today as DDT (short for dichloro-diphenyl-trichloro-ethane). Early tests showed this new chemical to be of extraordinary value as a pesticide: it rapidly killed a variety of insects and had no immediately apparent effects on mammals. DDT proved enormously valuable to the Allied war and occupation efforts in combating typhus (transmitted by lice) and later malaria. The United Nations Reconstruction and Relief Agency used DDT in the late 1940s to essentially eradicate malaria from Sardinia in the lapse of a few years.

The World Health Organization (WHO) proposed a worldwide campaign to eradicate malaria in the late 1940s and early 1950s. While the WHO mostly provided technical assistance and moral suasion, substantial funding came from the USAID and UNICEF. The nations of Latin America took up this task in the 1950s. While individual nations had formal control of the design and implementation of the programs, their activities were comparatively homogeneous as per the dictates of their international funders. The central component of these programs was the spraying of DDT, principally in the walls and eaves of houses. Its purpose was not to kill every mosquito in the land, but rather to interrupt the transmission of malaria for long enough that the existing stock of parasites would die out. After that, the campaigns would go into a maintenance phase in which imported cases of malaria were to be managed medically.

The Latin American countries analyzed in the present study (Mexico, Colombia, and Brazil) all mounted malaria eradication campaigns, and all saw large declines in malaria prevalence. Panel B of Figure 1 shows malaria cases per capita in Colombia. A decline of approximately 80 percent is evident in the graph. Throughout Latin America, the campaign ultimately proved inadequate to the task, and, in many areas, malaria partially resurfaced two decades later. But in almost all parts of the hemisphere, malaria never returned to its levels from before the application of DDT.

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3 The historical narrative on the worldwide campaign is drawn from Harrison (1978). For a review of health programs in Latin America prior to the 1950s, see Cueto (1994, 2004) and Quevedo (2005).

4 DDT had residual action as a pesticide, meaning that its effect was sufficiently persistent after spraying that only 1-3 applications per year were mandated.
2.4 Research Design

The first factor in the research design is that the commencement of eradication was substantially due to factors external to the affected regions. The eradication campaign relied heavily upon critical innovations to knowledge from outside the affected areas. Such innovations were not related to or somehow in anticipation of the future growth prospects of the affected areas, and therefore should not be thought of as endogenous in this context. This contrasts with explanations that might have potentially troublesome endogeneity problems, such as, for example, positive income shocks in the endemic regions.

Second, the anti-malaria campaigns achieved considerable progress against the disease in less than a decade. This is a sudden change on historical time scales, especially when compared with trend changes in mortality throughout recent history, or relative to the gradual recession of malaria in the midwestern U.S. or Northern Europe. Moreover, I examine outcomes over a time span of 60 to 150 years of birth, which is unquestionably long relative to the malaria eradication campaigns. This is a further advantage of examining these anti-malaria campaigns: enough time has passed since their inception that we can assess their long-term consequences.

An additional element in the identification strategy is that different areas within each country had distinct incidences of malaria. In general terms, this meant that the residents of the U.S. South, southern Mexico, northern Brazil, and lowland Colombia were relatively vulnerable to infection. Populations in areas with high (pre-existing) infection rates were in a position to benefit from the new treatments, whereas areas with low endemicity were not. This cross-regional difference permits a treatment/control strategy.

The advent of the eradication effort combines with the cross-area differences in pre-treatment malaria rates to form the research design. The variable of interest is the pre-eradication malaria intensity. By comparing the cross-cohort evolution of outcomes (e.g., adult income) across areas with distinct infection rates, one can assess the contribution of the eradication campaigns to the observed changes. (Specific estimating equations are presented below.)

How realistic is the assumption that areas with high infection rates benefited more from the eradication campaign? Mortality and morbidity data indicate drops of 50 to 80 percent in the decade after the advent of the eradication efforts. (See Figure 1.) Such a dramatic drop in the region’s average infection rate, barring a drastic reversal in the pattern of malaria incidence across the region, would have had the supposed effect of reducing infection rates more in highly infected areas than in areas with moderate infection rates. The decline in malaria incidence as a function of intensity prior to the eradication campaign is found in Figure 2. The basic assumption of the

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5 Humid areas with slow-moving water were the preferred nursery for mosquitoes, the vector that transmits malaria.

6 This figure embodies the first-stage relationship. Consider the aggregate first-stage equation:

\[ M_{jt} = \gamma M_{jpre} \times \text{Post}_t + \delta_j + \delta_t + \eta_{jt} \]
present study — that areas where malaria was highly endemic saw a greater drop in infection than areas with low infection rates — is borne out across areas in the countries where data are available.

Finally, the timing of the eradication campaign should induce variation in childhood malaria infection that has a marked pattern across year-of-birth cohorts. The present study considers the effects of childhood malaria infection on later-life outcomes, so it is useful to characterize childhood exposure to an eradication campaign. This is shown in Figure 3. Consider a campaign that starts in year zero and takes effect instantaneously. Cohorts born after this date will be exposed to the campaign for their entire childhood. On the other hand, those cohorts who were already adults in year zero will have no childhood exposure to the campaign, while the ‘in between’ cohorts will be partially exposed during childhood, as shown in the figure. In reality, the campaigns took effect over the better part of a decade. This implementation delay would tend to elongate the line in Figure 3 (if measured as effective childhood exposure to the campaign), inasmuch as being born at the time of the commencement of the campaign does not actually bring the full benefits of the eradication efforts. Note, however, that the left-hand side of the curve (representing those already in adulthood before the campaign started) would remain unchanged. In any case, the effects of partial childhood exposure might not take on the straight-line form suggested in the graph, depending on the relative importance of malaria infection at various points in childhood. For example, if the most sensitive years are in adolescence (when the child is near the margin of staying in school or entering the workforce), the line in Figure 3 would need to be adjusted to rise more sharply for those cohorts in adolescence at the time of the campaign. If, in contrast, the most deleterious point in the life cycle to have malaria is as an infant, the impact-adjusted line in the figure would rise most sharply for those born just before the campaign. For the moment, I maintain the uniform (agnostic) weighting of childhood exposure, but I discuss in Section 6.2 what we learn on this point from the empirical results.

2.5 Related Literature

Over a century has passed since the birth of the interdisciplinary field of malariology, and the associated literature has grown to Brobdingnagian proportions since that time. A thorough reference is Wernsdorfer and McGregor (1988), who edited an encyclopedic tome on medical and social aspects of the disease. There is also an excellent survey by Nájera, Liese, and Hammer (1992).

An important first step in quantifying the output costs of malaria has been to survey the contemporaneous effects of malaria fever. Numerous studies address the question: Following a

\[
\Delta M_{j}^{post} = \gamma M_{j}^{pre} + \text{constant} + \nu_{jt},
\]

For area \(j\) in year \(t\). This equation can be written in first-differenced form and evaluated in the post-campaign period:

\[
\Delta M_{j}^{post} = \gamma M_{j}^{pre} + \text{constant} + \nu_{jt},
\]

an equation that relates the observable variables graphed in Figure 2.
measured case, how much time is lost at work and/or in leisure/home production? For example, Conly (1975) presents such an analysis in Paraguay, while Bonilla Castro, Kuratomi, Rodríguez, and Rodríguez (1991) perform this exercise in Colombia. These studies also consider spillovers within the household (e.g., parents’ caring for sick children). Furthermore, Conly links the time-allocation data to contemporaneous measures of farm output.

There are important reasons to believe that these estimates provide a limited picture of malaria’s economic impact, however. (See Malaney, 2003, for a thorough discussion.) First, the fever is simply the most acute realization of morbidity from malaria. For a variety of reasons, malaria infection can cause anemia that persists for a considerable time after infection, and Thomas et al. (2003) show that anemia has depressing effects on contemporaneous adult productivity. Second, while this methodology considers costs that are evaluated at current prices and constraints, a much larger cost might be that investment in physical capital and land improvement are suppressed by the threat of malaria. Utzinger, Tozan, Doumani, and Singer (2001) argue, for example, that the control of malaria transmission was a key factor in the development of Zambian copper mining. Furthermore, as mentioned above, the control of malaria in the Panamanian isthmus was crucial for the successful completion of the canal. Third, and most relevant for the present study, a full accounting of how malaria in childhood might affect adult outcomes is absent from the literature.7

Several studies also consider the role of broadly defined health in Latin America. In the interest of space, I mention only a few examples here. Lopez Alonso and Porras Condey (2003) and Meisel (2004) use data on stature to analyze changes in health across birth cohorts in Mexico and Colombia, respectively. Sanchez and Nuñez (2000) and Mendoza and Rosas (2004) consider the role of geographic and health factors in explaining cross-municipio income differences within Colombia at a point in time. Additionally, using local resources in childhood as an instrument for adult height, Ribero and Nuñez (2000) analyze the effect of health endowments on income in Colombia.

The present study is most closely related to recent work by three authors. Bleakley (2006) finds that hookworm eradication in the U.S. South was followed by an increase in school attendance and literacy. Furthermore, using a retrospective/cohort design similar to the present study, he finds evidence that childhood exposure to the hookworm-eradication campaign increased adult income. Using a database of Union Army veterans, Hong (2007) considers the effect of early-life exposure to malaria on later-life health outcomes in 19th century America. Lucas (2005) shows that women born after malaria eradication in Sri Lanka completed more years of schooling, suggesting that returns to education rose faster than child wages in that episode. None consider the direct impact of childhood malaria on income.

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7This point is made by Barlow (1967) and Wernsdorfer and Wernsdorfer (1988), but neither evaluates the empirical magnitude of this channel.
3 Data Sources and Definitions

The micro-level data employed in the present study come from the Integrated Public Use Micro Sample (IPUMS), a project to harmonize the coding of census microdata from the U.S. and several other countries (Ruggles and Sobek (1997); Sobek et al. (2002)). I analyze the census data from the U.S., Brazil, Colombia, and Mexico.

The geographic units employed in this analysis are place of birth rather than current residence. Matching individuals with malaria rates of the area where they end up as adults would be difficult to interpret because of selective migration. Instead, I use the information on malaria intensity in an individual’s area of birth to conduct the analysis, which is therefore an intention-to-treat design. For the U.S., Mexico, and Brazil, this means the state of birth. The Colombian census also contains information on birthplace by municipio, a second-order administrative unit similar to U.S. counties.

For the United States, the base sample consists of native-born white males in the Integrated Public Use Micro Sample or IPUMS (Ruggles and Sobek, 1997) and North Atlantic Population Project (NAPP, 2004) datasets between the ages of 25 and 55, inclusive, for the census years 1880-1990, which includes cohorts with years of birth ranging from 1825 to 1965. I use two proxies for labor productivity that are available for a large number of censuses. The occupational income score and Duncan socioeconomic index are both average indicators by disaggregated occupational categories that were calibrated using data from the 1950 Census. The former variable is the average by occupation of all reported labor earnings. The measure due to Duncan (1961) is instead a weighted average of earnings and education among males within each occupation. Both variables can therefore measure shifts in income that take place between occupations. The Duncan measure has the added benefit of picking up between-occupation shifts in skill requirements for jobs. Occupation has been measured by the Census for more than a century, and so these income proxies are available for a substantial stretch of cohorts.

The data on native-born males from the Brazilian and Mexican IPUMS-coded censuses from 1960 to 2000 are similarly pooled, resulting in birth cohorts from 1905 to 1975. These censuses contain questions on literacy, years of education, and income (both total and earned).

For Colombia, I use the IPUMS microdata on native males from the censuses of 1973 and 1993 (those for which municipio of birth was available). This yields birth cohorts from 1918 to 1968. I use the census-defined variables for literacy and years of schooling. I also construct an income score benchmarked from the Mexican and Brazilian data.

I combine microdata from various censuses to construct panels of average outcomes by cohort. Cohorts are defined by both when they were born and where they were born. To construct these panels, I pool the micro-level census data. The individual-level outcomes in the microdata are projected onto dummies for year-of-birth $\times$ census year $\times$ country. I then take the average residual
from this procedure for each cell defined by period of birth and state (or municipio in the case of Colombia) of birth. (This procedure is described in detail in Appendix A.) In Section 4, I consider how cross-area outcomes change by year of birth, so the panels are constructed with year of birth × area of birth as the units of observation. For Section 5, I compare two groups—cohorts born well before or just after the campaign—so the period of birth is defined accordingly.

Malaria data are drawn from a variety of sources. U.S. data are reported from by the Census (1894), Maxcy (1923), and later in the Vital Statistics (Census, 1933). Mexican data are drawn from Pesqueira (1957) and from the Mexican Anuario Estadístico (Dirección General de Estadística, 1960). SEM (1957) and the Colombian Anuario de Salubridad (DANE, 1970) are the sources for the Colombian data. Data on malaria ecology are derived from Gallup, Mellinger, and Sachs (1999a) and Poveda, Graham, and Epstein (2000). The ecology data were matched with states and municipios using a geographic information system (GIS). Appendix B contains further details.

4 Cohort-specific Results

The shift in the malaria-income relationship coincides with childhood exposure to the eradication efforts. This can be seen graphically in this section. I also provide statistical tests that indicate the break is indeed coincident with exposure to eradication rather than with some pre-existing linear trend. I compare changes in socioeconomic outcomes by cohort across areas with distinct malaria intensities in order to assess the contribution of the eradication campaign to the observed changes. For each year of birth, OLS regression coefficients are estimated on the resulting cross section of states/municipios of birth. Consider a simple regression model of an average outcome, \( Y_{jk} \), for a cohort with state of birth \( j \) and year of birth \( k \):

\[
Y_{jk} = \beta_k M_j + \delta_k + X_j \Gamma_k + \nu_{jk}
\]

in which \( M_j \) is the pre-campaign malaria intensity in area of birth \( j \), \( \beta_k \) is year-of-birth-specific coefficient on malaria, \( X_j \) is a vector of other state-of-birth controls, and \( \delta_k \) and \( \Gamma_k \) are cohort-specific intercept and slope coefficients. I estimate this equation using OLS for each year of birth \( k \). This specification allows one to examine how the relationship between income and pre-eradication malaria (\( \hat{\beta}_k \)) differs across cohorts. (Note that the coefficients on the control variables are similarly flexible by year of birth.)

I start with a simple graphical analysis using this flexible specification for cross-cohort comparison. Figures 4, 5, 6, and 7 display plots of the estimated \( \beta_k \), for the various outcomes and countries under study. The \( x \) axis is the cohort’s year of birth. The \( y \) axis for each graphic plots

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8These additional controls are used in constructing the ultimate panels of Tables 2, 3, and 4.
the estimated cohort-specific coefficients on the area-of-birth measure of malaria. Each cohort’s point estimate is marked with a dot.

4.1 Graphical Results for the United States

Results for the U.S. are shown in Figure 4, which displays the coefficient on state-of-birth 1890 malaria mortality for each year of birth. These estimates are produced controlling for both region of birth and unskilled wages in 1899. Additionally, the summarized regressions include controls for health conditions and educational resources. The broad shape of these curves is not sensitive to excluding various subsets of these controls from the estimation procedure.

To consider the effects of childhood exposure to malaria, observe that U.S. cohorts that were already adults in 1920 were too old to have benefited from the eradication efforts during childhood. On the other hand, later cohorts experienced reduced malaria infection during their childhood. This benefit increased with younger cohorts who were exposed to the anti-malaria efforts for a greater fraction of their childhood. The dashed lines therefore measure the number of years of potential childhood exposure to the malaria-eradication campaign. (The line is rescaled such that pre-1890 and post-1940 levels match those of the $\hat{\beta}_k$. The exposure line is not rescaled in the x dimension.) Cohorts born late enough to have been exposed to eradication during childhood generally have higher income than earlier cohorts, and this shift correlates with higher potential exposure to the eradication campaign.

4.2 Graphical Results for Brazil, Colombia, and Mexico

Results for Brazilian states are shown in Figure 5. The range of birth years is smaller in the Latin American samples because only the more recent censuses are available in microdata form, but the design is otherwise similar to that above. The graphs display, for each year of birth, regression coefficients of the outcome variables on malaria intensity. Because data on pre-eradication malaria prevalence is limited, I use an index of malaria ecology (based on Gallup, Mellinger, and Sachs, 9

The latter variable, from Lebergott (1964) serves as a correction for possible mean reversion in income. If the oldest cohorts had high malaria infection and low productivity because of some mean-reverting shock, we might expect income gains for the subsequent cohorts even in the absence of a direct effect of malaria on productivity.

The health controls include fertility, infant mortality, and the proportion of deaths from various childhood diseases in 1890; late-1910s hookworm infection; state public-health spending and the number of doctors per capita in 1898; and the fraction of recruits rejected from service for health reasons by WWI-era Army physicians. The education-related controls are as follows: the 1910 adult literacy rate, and the logarithmic change (circa 1902–32) of teacher salaries, pupil/teacher ratios and total school expenditures. In addition, the regressions include the male unemployment rates from 1930, the 1910 fraction black, and the 1910 fraction living in urban areas. Appendix C has details on these variables. Section 5 below considers the sensitivity of these results to the choice of control sets.

Specifically, the formula is $\text{Exp}_k = \max(\min(18, k – (1920 – 18)), 0)$, which treats 1920 as an approximate start date for exposure. Because the campaigns had their effect over a decade or more, the childhood-exposure measure represents an optimistically fast guess.
1999a). The log of electrical capacity circa 1950 is used as a control for the pre-program level of development. (Neither gross state product nor income were available this early for Brazil.) Infant mortality in 1950 is used to control for possible catch-up in general health, while a number of other variables are included to account for sectoral and regional effects. The outcome variables are literacy, years of schooling, the logarithm of total income, and the logarithm of earned income. The range of years of birth for earned income is further limited by data availability. (See the appendices for details on the sample and variables.)

In Brazil, the malaria-related change in outcomes across cohorts coincides roughly with childhood exposure to the campaign. Childhood exposure is measured as above, except that the starting point of the campaign is taken to be 1955, the year of widespread implementation of DDT spraying. When comparing cohorts with zero versus full exposure to the campaign, the estimated coefficients for each outcome tend to be lower for those who were already adults before the campaign began. Furthermore, the shift in each estimated series occurs as childhood exposure to the campaign increases. Literacy and both measures of income rise with childhood exposure to the campaign, consistent with the central hypothesis of the present study. Education increases as well, which suggests that reducing malaria raised returns to schooling more than it improved childhood wages in this episode.

Similar results are found for Colombia, as shown in Figure 6. The unit for area of birth for this analysis is the municipio, and an ecological index is used for malaria intensity. The estimated malaria coefficients for literacy, education, and income tend to be higher for those fully exposed to the eradication campaign. (Note that the income is measured using a sectoral proxy based on one-digit industry and class of worker.) Moreover, the series of coefficients shift across cohorts with increasing childhood exposure to the eradication efforts, although this result is weaker for literacy because of the imprecise estimates for earlier cohorts.

Results for Mexican states are shown in Figure 7. The outcome and control variables are similar to those above for Brazil, except that only earned income is available for the appropriate sets of cohorts. Malaria mortality is available by state for circa 1950, and this variable is used to measure the malaria prevalence. Estimates for literacy and income tend to be higher for later cohorts. On the other hand, the malaria coefficients for education actually decline initially with childhood exposure to the campaign, suggesting that the reduction in malaria raised the return to child labor more than the return to schooling. Nevertheless, in the microdata, sample sizes for the earlier cohorts are quite small, and the imprecision with which the pre-1940 coefficients are estimated complicates visual analysis for the Mexican case. I therefore turn now to time-series regressions of the cohort-specific malaria coefficients.
4.3 Analysis of the Time Series of Coefficients

Formal statistical tests indicate that the shift in the income/malaria ecology relationship coincided with exposure to malaria eradication, rather than with some polynomial trend. This can be seen by treating the estimated $\beta_k$ as a time series and estimating the following regression equation:

$$\hat{\beta}_k = \alpha \text{Exp}_k + \sum_{i=1}^{n} \gamma_n k^n + \text{constant} + \epsilon_k^t$$

(2)

in which $\text{Exp}_k$ is exposure to the malaria-eradication campaign (defined above), and the $k^n$ terms are $n$th-order trends. To account for the changing precision with which the generated observations are estimated, observations are weighted by the inverse of the standard error for $\hat{\beta}_k$. Table 1 reports estimates of equation 2 under a variety of order assumptions about the polynomial trend. The dependent variables are the cohort-specific regression estimates of outcomes on malaria that are shown in the figure above.\(^{12}\)

Panel A contains estimates for the United States. For the occupational income score, the estimates on the exposure term are similar across specifications, and there is no statistically significant evidence of trends in these $\beta_k$. When the Duncan SEI is used instead, there is evidence of a downward trend, but estimates of the exposure coefficient are stable once this is accounted for.

These point estimates imply reasonable reduced-form magnitudes for the effect of childhood exposure to malaria. I start by considering the distribution of 1890 malaria mortality, and use it to convert the coefficients into more easily interpretable units. The 5th-percentile states were essentially malaria free, while the 95th-percentile states had 7 percent of their deaths attributed to malaria. Multiplying the point estimates in Panel A by 0.07 yields approximately 2.4 and 4 for the occupational income and Duncan indices, respectively. To further convert this into a proportional effect, I note that white males born in the South between 1875 and 1895 had an average of 21 for the former index and 26 for the latter. Therefore, these point estimates suggest an reduced-form effect on income of ten to fifteen percent when comparing the 95th and 5th percentile states. (These re-normalized terms are reported in curly brackets in Table 1.)

The pattern of coefficients for Brazil is broadly consistent with a model of childhood exposure to the campaign, rather than a pre-existing trend. These results are found in Panel B. For education and log total income, estimates of the exposure effect are statistically significant even when controlling for up to a third-degree polynomial trend, while, for literacy and log earned income, exposure is only significantly different from zero when compared with polynomials up to second degree. It

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\(^{12}\)An alternative approach would be to take the panel data and estimate the interaction of pre-campaign malaria and potential childhood exposure in one step. I adopt this strategy in Appendix D. The point estimates are similar to those reported here. The standard errors tend to be larger, but never large enough to affect the inferences in this section.
bears mentioning the span of years in the Latin American data is much shorter than the range for the U.S., and horse-racing the exposure variable with a third-degree polynomial is a difficult test to pass. Comparing states at the 95th and 5th percentile of malaria ecology, these estimates imply that childhood exposure to the eradication campaign brought about relative increases of ten percentage points in literacy, one year of schooling, 40-50% of total income, and 25-40% of earned income in the malarious areas.

The results for Colombia and Mexico are consistent with the childhood-exposure hypothesis, but the evidence is weaker than for the other countries. Estimates are found in Panel C for Colombia and Panel D for Mexico. For Colombia, the childhood-exposure coefficient is significantly different from zero when controlling for up to a quadratic in year of birth. The 95/5-percentile comparison shows malaria-related increases in literacy of three percentage points, in schooling of 0.275 years, and 5% of income (measuring only those changes that occur across one-digit industry and class of worker). For literacy in Mexico, it is difficult to distinguish the effect of exposure versus a trend, while results for schooling and income are more robust to the inclusion of polynomials in year of birth. The 95/5 contrast shows malaria-induced increases of 0.3–0.6 years of school and perhaps 20% of income. However, for both of these countries estimates on exposure are insignificant when controlling for a third-degree polynomial trend. This may be because the Colombian data cover even fewer years than the Brazilian sample, and the Mexican data have the problem that the samples are quite thin for the early cohorts. To ameliorate these problems, I consider estimates in the next section based on pooling the data across broader ranges of birth years.

5 Pre/Post Comparisons

I obtain similar results using a simple pre/post comparison. Once again, I compare outcomes across cohorts while separating along two lines: (i) by year of birth relative to the campaign and (ii) by the degree of pre-campaign malaria intensity in the area of birth. In contrast with the methodology of Section 4, however, I aggregate the different year-of-birth cohorts into two groups: those born well before the campaigns and those who were already adults when the campaign began. (The partially exposed cohorts are therefore not treated in this section.) Therefore, for each area of birth, the outcome variables employed in this section are cross-cohort differences (i.e., ‘born after’ minus ‘born well before’) in the socioeconomic measures. This permits the use of simple scatter plots, the analysis of which indicates that the results do not arise from the undue influence of a few observations. I also show that the results are generally robust to the inclusion of alternative controls and/or the use of instruments to correct for measurement error. A further advantage of the long
differencing is to reduce the bias in inference stemming from higher-frequency serial correlation.\footnote{While this methodology does not account for pre-existing trends, these were considered above and shown in most cases to have only modest effects on the estimates of childhood exposure to the eradication campaigns.}

The basic equation to be estimated is

$$\Delta Y_{j,t} = \beta M_{j,t-1} + X_{j,t-1}\Gamma + \alpha + \varepsilon_{j,t}$$

in which $Y$ is some socioeconomic outcome for state or municipio $j$. The time subscript $t$ refers to a year of birth following the malaria-eradication campaign, while $t - 1$ indicates being born (and having become an adult) prior to advent of the campaign. The pre-program malaria incidence is $M_{j,t-1}$, the $X$ variables are a series of controls, and $\alpha$ is a constant term. The parameter of interest is $\beta$. This parameter can be thought of as coming from a reduced-form equation, in the sense of two-stage least squares.\footnote{The model is derived as follows. Consider an individual $i$, born in area $j$, with year-of-birth $t$, we start with an individual-level model with individual infection data and linear effects of malaria:

$$Y_{ijt} = \alpha M_{ijt} + \delta_j + \delta_t + \varepsilon_{ijt}$$

where $M_{ijt}$ is a measure of childhood malaria infection. No data set has both childhood malaria infection data and adult income, and the research design is fundamentally at the period-of-birth $\times$ area-of-birth level, so I rewrite the equation above in aggregate form:

$$Y_{jt} = \tilde{\alpha} M_{jt} + \delta_j + \delta_t + \tilde{\varepsilon}_{jt}$$

I partition the cohorts into those born after the advent of the campaign and those who were already adults by the time the campaign started. I then difference the model along these lines, and take $M_{i,t-1}$ as an instrument for the decline in malaria following eradication. The resulting reduced form of this system is equation 3. Alternatively, one could have written the individual-level model with separate terms for individual and aggregate infection variables, the latter of which reflecting some spillover from peer infection to own human capital. But both of these effects would be subsumed into the \( \tilde{\alpha} \) coefficient on the ecological infection rate, and it is this composite coefficient that I seek to measure in the present study.}

5.1 United States

Areas in the U.S. with higher malaria burdens prior to the eradication efforts saw larger cross-cohort growth rates in income, as measured by the occupational proxies. These results are found in Table 2. Panel A contain estimates for the basic specification of equation 3, which includes a dummy for being born in the South plus the natural logarithm of state unskilled wages in 1899 from Lebergott (1964). The first two columns of the table report results using the measure of 1890 malaria mortality, while the remaining columns use alternative measures of malaria intensity. The malaria-ecology variables are those due to Gallup, Mellinger, and Sachs (1999a) and Hong (2007). The final measure is the malaria mortality per 100 thousand population, as reported by Maxcy (1923), who surveyed state health departments to obtain the data (which should be taken with a grain of salt because the death registration system was not fully functional in the South at that time).
If these noisy proxies of malaria are measured with independent errors, then the measurement-error bias in any one can be corrected by using the other three variables as instruments.\textsuperscript{15} Indeed, as seen in the second row of Panel A, the instrumented (2SLS) estimate is higher than the OLS estimate in almost every case. This correction is largest for the 1920 malaria measure, which was likely measured with the considerable error.

The estimates for malaria are not substantially affected by the inclusion of a number of additional control variables. Table 2, Panel B contains these results. The first row controls for additional state-of-birth-level measures of health, including fertility, infant mortality, and the proportion of deaths from various childhood diseases in 1890; late-1910s hookworm infection; state public-health spending and the number of doctors per capita in 1898; and the fraction of recruits rejected from service for health reasons by WWI-era Army physicians. The second row of Panel B shows the estimated effect of malaria when controlling for several education-related controls: the 1910 adult literacy rate, and the logarithmic change (\textit{circa} 1902–32) of teacher salaries, pupil/teacher ratios and total school expenditures. The third row, marked “Other”, includes a mixed basket of controls: male unemployment rates from 1930, the 1910 fraction black, and the 1910 fraction living in urban areas. The specification employed in the final row includes all of the above control variables simultaneously in the regression (which corresponds to the set of controls used to estimate equation 1 in Section 4 above). Finally, Figure 8 displays a scatter plot of the orthogonal component of cross-cohort income growth versus malaria (the 1890 measure), after having projected each variable onto the controls.

5.2 Brazil, Colombia, and Mexico

In Brazil and Mexico, malarious areas saw faster cross-cohort growth in income and literacy, but there is mixed evidence regarding differences in years of schooling. Table 3 reports the estimates for these two countries for a variety of control variables. Panel A contains estimates from ordinary least squares. The first row presents results from the basic specification, which contains just malaria, region-of-birth dummies, and the log of electrical capacity as a control for mean reversion.\textsuperscript{16} The next row includes estimates controlling for infant mortality as well. The third row includes controls for the sectoral composition of the labor force (at a one-digit level). (Sector is an important control variable because, for example, Latin American countries began policies of import substitution in

\textsuperscript{15}The assumption of independence of errors might seem inappropriate, particularly for the two ecology measures. However, similar results are obtained by using various subsets of the instruments, and accordingly, a Hausman/\textit{NR}\textsuperscript{2} test of the over-identifying restrictions fails to reject the null of identical parameter estimates in the second stage. Furthermore, similar results (not shown) are obtained using state-average temperature and altitude as instruments.

\textsuperscript{16}Some measure of income (as used for the United States) would be preferable as a mean-reversion control, but no suitable pre-campaign data were found for the full set of states. It is likely that electrical consumption is highly correlated with the level of development, however. According to data from the CIA (2006), gross domestic product per capita and electricity production per capita (in logs) have a correlation coefficient of 0.83 across countries.
the 1950s.) For the final row of Panel A, all the controls listed in Appendix C are included as regressors. The estimates for income are similar across control sets, albeit somewhat larger when I control for sector. Childhood malaria’s effect on literacy is consistently positive, although variable in its magnitude and statistical significance. Results for education are mixed, as per theoretical ambiguity. Panel B replicates specifications from Panel A, but corrects for measurement error using state-of-birth data on temperature, altitude, and the interaction of the two as instruments. Estimates using 2SLS are generally larger in magnitude, although the education results are imprecisely determined. The components of malaria and the outcome variables that are orthogonal to these controls are plotted in Figures 9 and 10.

Results from Colombia indicate that childhood exposure to malaria suppressed income. Cross-cohort growth in income, literacy, and education was higher in the areas with more perverse malaria ecology, as shown in Table 4. Basic specifications are shown in Panel A, which include malaria intensity and dummies for region of birth. In addition to the Poveda measure of malaria ecology from above, I also consider alternative measures of malaria intensity: the Mellinger variable described above and malaria cases registered by the Colombian eradication campaign in 1955 (SEM, 1957). The first row of Panel A presents OLS estimates, while the second row contains 2SLS estimates produced using as instruments the municipio’s average temperature, altitude, and the interaction of the two. Coefficient estimates on malaria are uniformly higher when instruments are used, which suggests attenuation bias in the OLS estimates. (Similar results, shown in Panel C, are found using alternative instrument sets, and Hausman tests do not reject the overidentification restrictions at conventional confidence levels.)

The 95/5-percentile comparison shows reduced-form magnitudes that are roughly similar across malaria measures. This is especially the case when temperature and altitude are used as instruments to correct for measurement error, which is likely large, in particular for cases notified. The 2SLS estimates indicate that malarious areas saw faster cross-cohort increases to the tune of four to seven percentage points of literacy, 0.4–0.8 years of schooling, and 0.09–0.17 log points of income. These estimates are robust to including a variety of controls for violent conflict, proxies of economic development, and other diseases. Panel B of Table 4 contains estimates with various control sets. The first row adds controls for conflict, including variables describing the intensity of the 1950s civil war in Colombia known as “La Violencia” and a variable indicating the concentration of land holdings (and therefore related to conflict arising during land reform). The second row reports estimates controlling for various types of economic activity: coffee-growing, mining, ranching, and manufacturing; as well as population density and indices describing the general level of development and quality of infrastructure. For the third row, controls for the presence of various helminth

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17See Garcia Montalvo and Reynal Querol (2006) for evidence on the importance of conflict in worsening the malaria situation in a panel of countries.
and (non-malarial) vector-borne diseases are included. The final row of the panel reports estimates controlling for all of the above controls simultaneously. Additionally, the residualized components (after projecting variables onto the full set of controls) of the cross-cohort changes and malaria ecology (using the instrumented Poveda measure) are shown in Figure 11.

6 Interpretation

In this section, I characterize the estimates from above in alternate units and consider several mechanisms for the results. First, expanding upon the reduced-form estimates above, I characterize the effects on adult income per malaria infection rate. Second, I present additional evidence that the results above are due to childhood morbidity from malaria.

6.1 Normalizing by the Probability of Childhood Infection

Data limitations required using heterogeneous measures of malaria above, but I constructed comparable reduced-form differences by comparing the most malarious to least malarious areas within each country. Representative values of these estimates are reported in the first row of Table 5, which run from 7 to 37 percent. A difficulty in interpreting these numbers, however, is that they are composed of two parts: (i) the effect on adult income of a given childhood malaria burden, and (ii) the magnitude of decline of the malaria burden following the eradication campaigns. The parameter (i) is of interest because it is portable: it is in units of income per infection rate, a number that can be applied to other situations with known infection rates. I therefore estimate the order of magnitude of (ii) and thereby can calculate the approximate effect on adult income of childhood malaria exposure in units of infection rates.

What was the range of pre-eradication malaria infection within each country? Molineaux (1988) reports on the WHO typology of malaria intensity (and associated malaria-infection rates among children): non-endemic (0%), hypoendemic (0-10%), mesoendemic (10-50%), hyperendemic (50-75%), and holoendemic (75-100%). Molineaux also reports estimates of the spatial distribution of different endemic zones throughout the world. (Both the typology and its associated geography are derived from the experience of many experts and do not simply reflect the opinion of that one author, however.) Taking the midpoint of the reported intervals, information about the types of endemicity within each country is used to estimate the cross-area differences in malaria burden prior to the campaigns. The pre-eradication malaria burden in the U.S. ranged from malaria free to mesoendemic, representing a within-country difference in malaria-infection rates of approximately 0.3. Areas within each of the three Latin American countries varied from essentially zero to hyperendemic, for a range of 0.625 in infection probability. (These are reported in the second row of Table 5.) Because infection rates were thought to have dropped precipitously in the decade
following the campaign, I take the pre-campaign level to be an adequate measure of the subsequent decline.\textsuperscript{18}

I estimate the effect of childhood malaria infection on adult wages to be substantial: being infected with malaria through childhood lead to a reduction in adult income of approximately 50 percent. I calculate this number by normalizing the reduced-form differences with the estimated decline in malaria. (Note that this procedure has the flavor of Indirect Least Squares.) These estimates are shown in the last row of Table 5. For Brazil, the estimated effect is higher for total income (0.62) than for earned income (0.45). In Mexico, the estimate for earned income is 0.43. For Colombia, the raw estimate is small (0.12), but this is probably because of the crudeness of the income score based on class of worker and one-digit industry. Because the between- and within-occupation components of the income changes induced by early-life malaria are most likely of the same sign, estimates using this score variable (as well as those for the U.S.) will be underestimates of the total effect on income. As a benchmark, I estimate the same income score for Brazil and obtain numbers 2–3 times smaller than the estimates for earned income, and 3–4 times smaller than those for total income. I report an adjusted number for Colombia (0.39), based on the total-income factor for Brazil. In the United States, the Duncan socioeconomic index shows a larger response to childhood malaria than the occupational income score. The latter variable is calibrated using total labor income, but only incorporates across-occupation changes in income. Accordingly, it is about 25% smaller than the effect on total income for Brazil. It is unclear whether the Duncan socioeconomic index is an under- or over-estimate of the full income effect, since the index effectively double counts schooling. I report it nevertheless for completeness.

6.2 Mechanisms

Schooling. Formal education had an important, but by no means dominant, role in the Latin-American results. (Lack of data prevent doing this computation for the U.S.) Using a standard return to schooling of 10% per year, I re-calculate the income effect of childhood exposure to malaria, but with years of schooling held constant. Because in Mexico formal education was estimated to have declined in response to the campaign, earned income would have been 10% higher (for a resulting effect size of 0.47 in units of log income per childhood infection rate) had schooling been fixed. In Brazil and Colombia, where schooling rose in response to childhood exposure to the campaign, the increase in education accounts for less than 25% and 10%, respectively, of the income results.

\textsuperscript{18}This is partly an assumption of necessity inasmuch as I have not found similar estimates of infection rates for the post-campaign period. Eradication was slightly less than complete in the decade following the campaign, so this will likely result in a downward bias of the estimates below. If, for example, infection dropped 95% instead of 100%, the effects on income reported below will be about 5% too low.
At conventional discount rates, these estimates indicate that eradication brought about an increase in the present discounted value of life-time income for the exposed cohorts. In Brazil and Colombia, the added schooling consumed at most two additional years at the beginning of their working-age lifetime, but income rose by around 40% in subsequent years. In Mexico, those who benefited from childhood exposure to eradication saw, on average, more income and fewer years of foregone earnings due to schooling investments.

Increases in the return to schooling may have contributed to the rise in income following the eradication campaigns, although the estimates of this channel are too imprecise to make definitive statements. To investigate this, I estimate Mincerian returns to schooling (RTS) for each cohort (again defined by year of birth × state of birth) for Mexico and Brazil. This procedure generates a panel data set of RTS, which I then analyze in a like manner to Section 4. For Mexico, estimates for the reduced-form effect of exposure on RTS hover around zero, but are imprecisely determined. When comparing the most to least malarious areas, the estimated differential effect has a 95% confidence interval of ±3 percentage points. At the upper extreme of this confidence band, it is possible to account, via increasing RTS × average years of schooling, for all of the effect of childhood malaria exposure on income. On the other hand, because of the imprecision of the estimates, changes in RTS could equally well account for none or even -100% of the estimated above. For Brazil, estimates of the effect of childhood malaria on RTS are also imprecise, such that the estimates can similarly account for both the observed effect on income as well as zero.

**Labor-market experience.** I consider the quantity of time worked and return to labor-market experience as possible mechanisms. Information on time worked are not available for a long enough range of cohorts is these data, so I employ an alternative strategy. The analysis for the U.S. was based on occupational indices of income calibrated to median total income in 1950. Using this same methodology, I constructed on occupational index of hours, which is constructed from the each occupation's median number of weeks worked last year × usual hours worked. Using the methodology of Section 4, I treat this as an outcome variable and find that childhood exposure to the anti-malaria campaign shifts workers into occupations with fewer, not more, average hours.

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19This test has certain limitations. This procedure, by comparing individuals with different terminal levels of attainment, estimates the average marginal effect of schooling in the sample (and how it changes following the anti-malaria campaign eradication). Absent distortions, the standard economic theory of schooling suggests that this effect should look like an interest rate, and that we should instead look for an effect along the quantity margin. (I.e., even if less exposure to malaria increased the return to schooling ceteris paribus, the quantity of schooling would adjust upwards until the return to additional schooling was back down to the rate of interest on borrowing.) However, the sign of the quantity response of schooling to childhood malaria is not an informative about the structural RTS because malaria also affects childhood wages. Moreover, it might be that the intervention had large effects on inframarginal returns to school investments. For example, the benefit might mostly accrue to elementary-school children without altering the marginal return at higher levels of education. A final problem is that the returns are estimated from the cross section, and no attempts are made to purge these estimates of endogeneity bias.
This suggests that childhood malaria’s impact on income was via the hourly wage rather than via labor supply. (This conclusion is confirmed by constructing occupational indices of wages rather than total income.)

On the other hand, increasing returns to labor-market experience was a modest component of the income results above. I repeat the analysis of Section 4, but do so separately for each age from 25–55. The estimated effect of childhood exposure is significantly differently from zero for men in their late twenties. This effect rises by about 30% by the time the worker reaches 55. Taken together, these facts suggest that childhood malaria depressed the return to labor-market experience, but that this mechanism accounts for only a modest fraction of total effect on income.

Other vector-borne diseases. The application of DDT most probably reduced the burden of other vector-borne diseases, but these diseases had minuscule prevalences relative to malaria. In 1962, the government of Colombia (DANE, 1963) reported 22 cases of yellow fever, and 167 cases of leishmaniasis. These diseases were dwarfed by the 21,245 cases of malaria reported in the same year. No systematic information is available for dengue, in part because prevalence was so low in those years that it was not a mandatory-notification disease. The last U.S. outbreak of yellow fever was in the 1880s, and mortality from dengue and leishmaniasis were so rare as to escape even being included in mortality statistics. No specific information on these diseases was found for Brazil and Mexico during the period of the anti-malaria campaign, but similar numbers to Colombia should be expected owing to their similar disease ecologies. Because the incidence of these other vector-borne diseases was so small, their simultaneous decline with malaria should not induce more than a small bias in the computations above.

Mortality selection. The eradication campaigns brought about a decline in malaria mortality as well as morbidity, but I argue that the change in mortality is most likely not responsible for the results of the present study. First, typically infectious-disease mortality is thought to differentially remove from a population the least healthy (and, for a positive health/income correlation, the least productive). Therefore, when the eradication campaigns reduced mortality, the most plausible composition effect would have reduced the income of the cohorts exposed to the eradication campaign as children. On the other hand, even if malaria killed off those who would have been more productive, this mechanism is probably not of the right order of magnitude to explain the cross-cohort rise in income. For example, suppose that malaria had previous killed off ten percent of children, and this non-surviving group would have earned 20% more than the rest of the population. Eradication of malaria would generate a 2% increase in income from earlier to later cohorts. Even if malaria killed 30% of children, and these non-survivors would have earned 30% more than the survivors, this would still only generate a cross-cohort difference of 9%. Furthermore, I show in the next paragraph that the income effects are weaker when I consider a strain that has higher
Comparing the *falciparum* and *vivax* strains. Within Colombia, most of the malarious areas were afflicted with *vivax* malaria. However, significant portions of the country suffered from elevated rates of *falciparum*, a malaria parasite associated with higher mortality and lower morbidity. To test the relative importances of mortality versus morbidity, I re-estimate the cross-cohort difference equation of Section 5, but with a new model: alongside the original measure of malaria, I include its interaction with the fraction of malaria due to *falciparum*. These results are found in Table 6, where a variety of specifications are presented. When compared with the estimates above, precision is lower, but the main effect of malaria intensity is generally positive and significant. (In the current specification, this term essentially measures the effect of the eradication of *vivax* malaria.) On the other hand, the *falciparum* interaction is almost always negative. Indeed, it is usually of such a magnitude that it would offset the main effect of malaria eradication in an area with 100% *falciparum*.

The timing of childhood exposure. The definition of childhood exposure that I used above assumed an effect of malaria infection that was uniform across youth. As seen above, the pattern of effective exposure to eradication that this generated across cohorts was a reasonable fit for the data. With this result, we can reject some alternative hypotheses about relative importance of exposure at various points in childhood. First, note that trend breaks were evident around birth years that precede the campaigns by 20 years or so. This fact rules out a model with disproportionately large effects of malaria infection *in utero* or during infancy. Second, this early trend break was indeed a trend break rather than a level shift. Because those same cohorts were adolescents at the time of the campaign, this suggests that the result is not driven exclusively by adolescent exposure.

A related concern is the effect of adult exposure, either contemporaneous or cumulative. While the data construction purges calendar-time effects that are common across the country, the eradication campaign does differentially benefit certain areas as a function of time. However, if the central mechanism were the *contemporaneous* effect on adult productivity, it would generate a trend break much earlier than the one seen above.20 Similarly, if cumulative adult exposure were the central mechanism, we would also expect an earlier break in the cohort trend. In addition, neither of these adult effects could explain the changes in literacy and schooling, which are almost exclusively childhood investments.

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20 In any event, I can remove effects for state of birth × post 1920 in the data-construction step and obtain a similar pattern of coefficients for the U.S. Note that, because the Latin American data are drawn from censuses of 1960 and thereafter, all cohorts have the same contemporaneous exposure to campaign in those three countries.
Spillovers. The present study considers outcomes at the cohort level, so any general-equilibrium effect within the cohort is built into the estimate. Moreover, if the healthier cohorts simply displaced older workers (because of ranking or signaling), we would expect a different time-series pattern to the coefficients in Section 4. A pure ranking/displacement mechanism would generate income differences by childhood exposure for those born within a generation of the intervention. But this mechanism would not affect the endpoints of the time series. Why? Consider the episode from the U.S. People born in 1820 never worked in the same labor market with cohorts exposed to the anti-malaria campaign as children. Similarly, people born in 1960 always worked alongside those fully exposed to the treatment. As seen in the Figure 4, the end points tend to be a bit closer together, but a gap remains. Similar patterns are seen for the Latin American countries, although the span of years covered in those samples is too short to completely separate these own versus spillover effects.

6.3 Extrapolations

The estimated impact of childhood malaria is large enough that it bears consideration in a macroeconomic context, although it is not so large that it can account for anything like the observed dispersion in income across areas. The log-income gap between the North and the South at 1900 was approximately 0.75. For a 10–20% infection rate in the South and an effect of childhood malaria on log total income of 0.62, we would expect a reduction in Southern incomes of approximately 0.06–0.12 log points. In other words, some 8–17% of this income gap could be attributed to malaria infection in the South. On the other hand, the 1950 difference in log GDP between the United States and the three Latin American countries was between 1.5 and 2. If these countries had 30–40% malaria infection rates among children, we would expect eradication to have reduced this gap by 0.19–0.25 in natural log terms, which would close 10–16% of the gap with the U.S.

The benefits estimated above played out across cohorts rather than time, so some time would have had to pass before the healthier cohorts filled out the working-age population. Indeed, only around 20% of the effect would have been realized by 1980, and the full gains will not be seen until almost 2010. For the econometrician working with standard output data from this transition period, there would appear to be a growth effect of malaria, although this is the result of time-aggregation bias.

A number of macroeconomic studies attempt to measure the impact of malaria on economic output across countries, but those estimates are too large to be plausibly generated by the childhood-exposure mechanism identified above. Examples of such work are by Gallup and Sachs (2001) and Sachs (2003). The latter study reports an estimate of -1.3, while the present study quantifies

\footnote{The approximate income figures cited in this subsection are from Barro and Sala-i-Martin (1999). The infection rates are estimated from Molineaux (1988).}
the childhood-exposure channel to be approximately 0.6, about 40% of the cross-country estimate. However, the Sachs estimates are in units of log(GDP) per fraction of population potentially exposed to malaria, in contrast with the results above, whose units are log(total income) per probability of childhood infection. Because the fraction exposed is less than the fraction infected, we need to inflate the former number by some amount. If the fully exposed countries have childhood infection rates around 0.6, then the Sachs estimate in my units is 2.16, and I can account for only around 25% of that result. On the other hand, those studies emphasize the importance of *falciparum*, whereas my results seem to work through *vivax*. Indeed, the analysis of regions in Colombia (shown in Table 6) suggests that, via the childhood-exposure mechanism, the productivity gains from reducing childhood exposure to *falciparum* malaria (e.g., in Africa) would be small.

7 Conclusions

This study considers the socioeconomic impact of the malaria-eradication campaigns in the United States (circa 1920), and in Brazil, Colombia, and Mexico (circa 1955). The goal is to measure how much childhood exposure to malaria depresses labor productivity.

Several factors combine to form the research design. The eradication campaigns studied happened because of advances in medical and public-health knowledge, which mitigates concerns about reverse causality of the timing of eradication efforts. Highly malarious areas saw large drops in their malaria incidence following the campaign. Furthermore, these gains against the disease were realized in approximately a decade. Finally, sufficient time has passed that we can evaluate its long-term consequences. Data from regional malaria eradication programs were collected and collated with publicly available census data.

In both absolute terms and relative to the comparison group of non-malarious areas, cohorts born after eradication had higher income and literacy as adults than the preceding generation. Mixed results are found for years of education, consistent with the economic theory of schooling (which compares returns with opportunity costs).

Another result of the present study is finding similar estimates of the effect of childhood malaria exposure on adult income across the four countries considered. This fact is remarkable particularly given the composition of the sample: one developed country and three others still on the path of development. Put another way, in spite of the differences in culture, institutions, and endowments among the countries studied, the effect of malaria was broadly similar.

The results above suggest potentially large benefits of public-health interventions in developing countries where malaria (especially the *vivax* strain) is still endemic today. Nevertheless, I show using a simple calculation that, while reducing malaria could bring substantial income gains to some countries, the estimated effect is approximately an order of magnitude too small to be useful.
in explaining the global income distribution. But, in any event, while much of the literature has focused on broad decompositions of income into institutions versus geography, one might argue that policy-minded social scientists should instead focus on the efficacy of specific interventions. Changing the geography or the colonial history of a country is impossible, and unfortunately the institutions literature has little to say about the complicated mess of intermediate variables that determine productivity. The present study quantifies the benefits of a particular health intervention and finds them to be substantial.

References


________ (2000). División Político-Administrativa de Colombia, Bogotá, Colombia: DANE.


Figure 1: Malaria Incidence Before and After the Eradication Campaigns

Panel A: Mortality per 100K Population, Southern United States

Panel B: Cases Notified per 100K Population, Colombia

Notes: Panel A plots the estimated malaria mortality per capita for the Southern region and bordering states. Because the death registration system was being phased in over the period, a regression model with state fixed effects is used to control for sample changes, and the time series is constructed from the year dummies in the regression, normalized to match the end-of-period data when all states were represented. (Census Bureau Vital Statistics, various years, and author’s calculations.) Panel B reports data on notified cases of malaria for Colombia (SEM, 1979).
Figure 2: Highly Infected Areas Saw Greater Declines in Malaria

Notes: The $y$ axis displays the estimated decrease in malaria mortality post-intervention. The $x$ axis is the pre-campaign malaria mortality rate. The 45-degree line represents complete eradication. Both variables are expressed per 100,000 population. United States data are reported in Maxcy (1923) and Vital Statistics (Census, 1933). Mexican data are drawn from Pesqueira (1957) and from the Mexican Anuario Estadístico (Dirección General de Estadística, 1960). SEM (1957) and the Colombian Anuario de Salubridad (DANE, 1968-70) are the sources for the Colombian data.
Figure 3: Childhood Exposure to Eradication Campaign

Notes: This graph displays the fraction of childhood that is exposed to a hypothetical (and instantaneous) campaign as a function of year of birth minus the start year of the campaign.
Figure 4: Cohort-Specific Relationship: States in the U.S.

Panel A: Occupational Income Score

Panel B: Duncan Socio-Economic Indicator

Notes: These graphics summarize regressions of income proxies on pre-eradication malaria-mortality rates (measured by the Census in 1890). The $y$ axis for each graphic plots the estimated cohort-specific coefficients on the state-level malaria measure. The $x$ axis is the cohort’s year of birth. Each cohort’s point estimate is marked with a dot. The dashed lines measure the approximate number of years of potential childhood exposure to the malaria-eradication activities in the South. For each year-of-birth cohort, OLS regressions coefficients are estimated on the cross section of states of birth. The state-of-birth average outcome is regressed onto malaria, Lebergott’s (1964) measure of 1899 wage levels, a dummy for the Southern region, and the various control variables described in Appendix C. Appendices A and B describe, respectively, the outcome variables and the malaria measure.
Figure 5: Cohort-Specific Relationship: States in Brazil

Notes: These graphics summarize regressions of the indicated socioeconomic outcomes on malaria-ecology rates (measured by Gallup, Mellinger, and Sachs, 1999a). The y axis for each graphic plots the estimated cohort-specific coefficients on the state-level malaria measure. The x axis is the cohort’s year of birth. Each cohort’s point estimate is marked with a dot. The dashed lines measure the approximate number of years of potential childhood exposure to the commencement of the malaria-eradication campaign in Latin America. For each year-of-birth cohort, OLS regressions coefficients are estimated on the cross section of states of birth. The state-of-birth average outcome is regressed onto malaria, region dummies, and the various control variables described in Appendix C. Appendices A and B describe, respectively, the outcome variables and the malaria measure.
Figure 6: Cohort-Specific Relationship: Municipios in Colombia

Notes: These graphics summarize regressions of the indicated socioeconomic outcomes on malaria-ecology rates (measured by Poveda, Graham, and Epstein (2000)). The y axis for each graphic plots the estimated cohort-specific coefficients on the municipio-level malaria measure. The x axis is the cohort’s year of birth. Each cohort’s point estimate is marked with a dot. The dashed lines measure the approximate number of years of potential childhood exposure to the commencement of the malaria-eradication campaign in Latin America. For each year-of-birth cohort, OLS regressions coefficients are estimated on the cross section of municipios of birth. The municipio-of-birth average outcome is regressed onto malaria, region dummies, and the various control variables described in Appendix C. Appendices A and B describe, respectively, the outcome variables and the malaria measure.
Notes: These graphics summarize regressions of the indicated socioeconomic outcomes on malaria-mortality rates (measured by Pesquiera (1957)). The $y$ axis for each graphic plots the estimated cohort-specific coefficients on the state-level malaria measure. The $x$ axis is the cohort’s year of birth. Each cohort’s point estimate is marked with a dot. The dashed lines measure the approximate number of years of potential childhood exposure to the commencement of the malaria-eradication campaign in Latin America. For each year-of-birth cohort, OLS regressions coefficients are estimated on the cross section of states of birth. The state-of-birth average outcome is regressed onto malaria, region dummies, and the various control variables described in Appendix C. Appendices A and B describe, respectively, the outcome variables and the malaria measure.
Figure 8: Cross-Cohort Differences versus Malaria: States in the U.S.

Panel A: Change in Occupational Income Score

Panel B: Change in Duncan Socio-Economic Indicator

Notes: Top panel displays results for the occupational income score, while the bottom panel uses the Duncan Socioeconomic Indicator. The y-axis are the changes in the indicated income proxy between cohorts born before 1895 and those born after 1925. The x-axis plots malaria mortality over total deaths in 1890. Appendices A and B describe, respectively, the outcome variables and the malaria measure. Both variables are residuals from having projected the original data onto a dummy for South, a 4th-order polynomial for Lebergott 1899 wage series, child-mortality rate in 1890, urbanization in 1910, adult literacy in 1910, doctors per capita in 1898, state public health spending in 1898, hookworm infection circa 1917, fraction black in 1910, unemployment rate in 1930, and the log change from 1905-25 in school-term length, pupil/teacher ratio, and teacher salary.
Figure 9: Cross-Cohort Differences versus Malaria: States in Brazil

Notes: The y-axis are the changes in the indicated socioeconomic variable between cohorts born before 1940 and those born after 1957. The x-axis plots Mellinger’s measure of malaria ecology. Appendices A and B describe, respectively, the outcome variables and the malaria measure. Both variables are residuals from having projected the original data onto the various control variables described in Appendix C.
Figure 10: Cross-Cohort Differences versus Malaria: States in Mexico

Notes: The y-axis are the changes in the indicated socioeconomic variable between cohorts born before 1940 and those born after 1957. The x-axis measures malaria-mortality rates (measured by Pesquiera, 1957). Appendices A and B describe, respectively, the outcome variables and the malaria measure. Both variables are residuals from having projected the original data onto the various control variables described in Appendix C.
Figure 11: Cross-Cohort Differences versus Malaria: Municipios in Colombia

Notes: The y-axis are the changes in the indicated socioeconomic variable between cohorts born before 1940 and those born after 1957. The x-axis plots the Poveda measure of malaria ecology as predicted using the instruments described in Table 4. Appendices A and B describe, respectively, the outcome variables and the malaria measure. Both variables are residuals from having projected the original data onto the various control variables described in Appendix C.
Table 1: Exposure to Malaria Eradication versus Trends

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<td>Duncan's Socioeconomic Index</td>
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**Panel A: United States**

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<td>48.862 ***</td>
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<td>55.248 ***</td>
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<td>(6.654)</td>
<td>(7.485)</td>
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**Panel B: Brazil**

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**Panel C: Colombia**

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**Panel D: Mexico**

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<tr>
<td>Years of Schooling</td>
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<tr>
<td>Log Earned Income</td>
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Notes: This table reports estimates of the childhood-exposure variable in equation 2 using OLS. The outcome variables used to construct the time series of $\beta_k$ are as indicated in each row. Robust (Huber-White) standard errors in parentheses. Single asterisk denotes statistical significance at the 90% level of confidence; double 95%; triple, 99%. Observations are weighted by the inverse of the coefficient's standard error. Reporting of additional terms suppressed. The terms in curly brackets report the point estimate multiplied by the difference between 95th and 5th percentile malaria intensity. For the United States, this number is also normalized by the average value of the relevant income proxy for white males born in the South between 1875 and 1895.
Table 2: Cross-Cohort Differences and Malaria: United States

<table>
<thead>
<tr>
<th>Dependent Variable:</th>
<th>Malaria Mortality (Fraction of Total), 1890</th>
<th>Malaria Ecology (Mellinger)</th>
<th>Malaria Ecology (Hong)</th>
<th>Malaria Mortality (per 100K Population), 1920</th>
</tr>
</thead>
<tbody>
<tr>
<td>Occupational Income Score</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
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<td>Duncan's Socioeconomic Index</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Specification:</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>OLS, Basic Specification</td>
<td>37.927 *** (11.101) (0.267) (2.040) (0.265) (4.737) (0.021) (0.036)</td>
<td>60.316 *** (21.311) (0.535) (4.737) (0.253) (4.737) (0.036) (0.074)</td>
<td>0.570 ** (0.267) (0.052) (0.265) (0.052) (0.052) (0.052) (0.052)</td>
<td>1.191 ** (0.535) (4.737) (0.253) (4.737) (0.052) (0.074) (0.074)</td>
</tr>
<tr>
<td>2SLS, Using the Other Three Proxies as Instruments</td>
<td>44.367 *** (14.238) (0.748) (3.813) (0.247) (0.301) (0.031) (0.053)</td>
<td>71.573 *** (24.199) (1.075) (8.205) (0.301) (0.301) (0.031) (0.053)</td>
<td>1.312 * (0.748) (3.813) (0.247) (0.301) (0.031) (0.053)</td>
<td>2.064 * (1.075) (8.205) (0.301) (0.301) (0.031) (0.053)</td>
</tr>
<tr>
<td>Additional Controls:</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Health</td>
<td>33.897 *** (9.733) (0.183) (2.506) (0.256) (0.025) (0.044)</td>
<td>63.480 *** (20.610) (0.346) (5.092) (0.025) (0.044)</td>
<td>0.483 *** (0.183) (0.346) (2.506) (0.256) (0.025) (0.044)</td>
<td>1.078 *** (0.346) (5.092) (0.025) (0.044)</td>
</tr>
<tr>
<td>Education</td>
<td>44.825 *** (12.240) (0.268) (2.093) (0.093) (0.024) (0.046)</td>
<td>59.306 *** (23.279) (0.412) (5.067) (0.093) (0.024) (0.046)</td>
<td>0.552 ** (0.268) (2.093) (0.093) (0.024) (0.046)</td>
<td>1.080 *** (0.412) (5.067) (0.093) (0.024) (0.046)</td>
</tr>
<tr>
<td>Other</td>
<td>30.118 *** (11.400) (0.162) (2.083) (0.083) (0.038) (0.045)</td>
<td>45.827 *** (18.134) (0.367) (4.751) (0.083) (0.038) (0.045)</td>
<td>0.388 ** (0.162) (2.083) (0.083) (0.038) (0.045)</td>
<td>1.050 *** (0.367) (4.751) (0.083) (0.038) (0.045)</td>
</tr>
<tr>
<td>Full Controls</td>
<td>33.392 *** (13.844) (0.236) (3.280) (0.098) (0.030) (0.056)</td>
<td>59.257 *** (29.103) (0.473) (7.088) (0.098) (0.030) (0.056)</td>
<td>0.385 (0.236) (3.280) (0.098) (0.030) (0.056)</td>
<td>0.985 ** (0.473) (7.088) (0.098) (0.030) (0.056)</td>
</tr>
</tbody>
</table>

Notes: This table reports estimates of equation 3 using OLS and 2SLS. The units of observation are U.S. states. The dependent variables are as indicated in the column headings. Robust (Huber-White) standard errors in parentheses. Single asterisk denotes statistical significance at the 90% level of confidence; double 95%; triple, 99%. Reporting of constant term suppressed. Unexposed cohorts are those born before 1890 and fully exposed cohorts are those born after 1920. Cohorts are determined based on state of birth. The universe for the base sample consists of the native-born white population between the ages of 25 and 55 (15–55 for literacy) in the 1880–2000 census microdata from the IPUMS and NAPP databases. The terms in curly brackets report the point estimate multiplied by the difference between the 95th and 5th percentile malaria intensity and normalized by the average value of the relevant income proxy for white males born in the South between 1875 and 1895. The specification for the basic results includes the malaria variable, a dummy for Southern birthplace, and the Lebergott (1964) measure of average unskilled wage in the state of birth. Appendices A and B describe, respectively, the outcome variables and malaria measures. The additional controls are described in the text and Appendix C.
### Table 3: Cross-Cohort Differences and Malaria: Brazil and Mexico

<table>
<thead>
<tr>
<th>Dependent Variables: Differences across Cohorts in...</th>
<th>Brazilian States (N=24)</th>
<th>Mexican States (N=32)</th>
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<tr>
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<td>Literacy</td>
<td>Education</td>
</tr>
<tr>
<td><strong>Panel A: Estimates using Ordinary Least Squares</strong></td>
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<td></td>
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<tr>
<td>Basic</td>
<td>0.012</td>
<td>0.106</td>
</tr>
<tr>
<td></td>
<td>(0.012)</td>
<td>(0.116)</td>
</tr>
<tr>
<td></td>
<td>[0.063]</td>
<td>[0.555]</td>
</tr>
<tr>
<td>Include Infant Mortality</td>
<td>0.012</td>
<td>0.110</td>
</tr>
<tr>
<td></td>
<td>(0.012)</td>
<td>(0.111)</td>
</tr>
<tr>
<td></td>
<td>[0.063]</td>
<td>[0.576]</td>
</tr>
<tr>
<td>Include Sectorial Shares</td>
<td>0.025 ***</td>
<td>0.246 **</td>
</tr>
<tr>
<td></td>
<td>(0.008)</td>
<td>(0.114)</td>
</tr>
<tr>
<td></td>
<td>[0.131]</td>
<td>[1.288]</td>
</tr>
<tr>
<td>Full Controls</td>
<td>0.028 ***</td>
<td>0.190 **</td>
</tr>
<tr>
<td></td>
<td>(0.008)</td>
<td>(0.093)</td>
</tr>
<tr>
<td></td>
<td>[0.147]</td>
<td>[0.995]</td>
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<tr>
<td><strong>Panel B: Estimates using Two-Stage Least Squares (Temperature and Altitude Instruments)</strong></td>
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<tr>
<td>Basic</td>
<td>0.043</td>
<td>-0.259</td>
</tr>
<tr>
<td></td>
<td>(0.041)</td>
<td>(0.413)</td>
</tr>
<tr>
<td></td>
<td>[0.225]</td>
<td>[-1.356]</td>
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<tr>
<td>Full Controls</td>
<td>0.041 *</td>
<td>0.049</td>
</tr>
<tr>
<td></td>
<td>(0.023)</td>
<td>(0.187)</td>
</tr>
<tr>
<td></td>
<td>[0.215]</td>
<td>[0.257]</td>
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</table>

Notes: This table reports estimates of malaria in equation 3 using OLS and 2SLS. The units of observation are Brazilian and Mexican states. The dependent variables are as indicated in the column headings. Robust (Huber-White) standard errors in parentheses. Single asterisk denotes statistical significance at the 90% level of confidence; double, 95%; triple, 99%. The terms in curly brackets report the point estimate multiplied by the difference between 95th and 5th percentile malaria intensity. Reporting of constant term suppressed. Unexposed cohorts are those born before 1940 and fully exposed cohorts are those born after 1957. Cohorts are determined based on state of birth. The universe for the base sample consists of the native-born population between the ages of 25 and 55 (15–55 for literacy) in the 1960–2000 census microdata from the IPUMS. The malaria measure for Brazil is Mellinger’s ecology variable, while for Mexico it is malaria mortality circa 1950 (Pesqueira, 1957). All regressions include dummies for region of birth and log electrical capacity as a proxy for economic development. Excluded instruments in Panel B are state-of-birth average temperature, altitude, and the interaction of the two. Appendices A and B describe, respectively, the outcome variables and malaria measures. The additional controls are described in the text and Appendix C.
Dependent Variables: Differences across Cohorts in...

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<td>Literacy</td>
<td>Years of Schooling</td>
<td>Income Index</td>
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<td>OLS, Basic Specification</td>
<td>0.089 ***</td>
<td>0.425 *</td>
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<td>Conflict</td>
<td>0.081 ***</td>
<td>0.442 *</td>
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<td>Economic Activity</td>
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<td>0.489 **</td>
<td>0.145 ***</td>
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<td>0.113</td>
<td>0.764 *</td>
<td>0.258 ***</td>
</tr>
<tr>
<td>All of the Above Instruments</td>
<td>0.124</td>
<td>0.816 ***</td>
<td>0.233</td>
</tr>
</tbody>
</table>

Notes: This table reports estimates of malaria in equation 3 using OLS and 2SLS for the indicated dependent variables. The units of observation are Colombian municipios. The malaria variables are as indicated in the column headings. Robust (Huber-White) standard errors in parentheses. Single asterisk denotes statistical significance at the 90% level of confidence; double 95%; triple, 99%. To present results on a similar scale, the terms in curly brackets reported in Panel A are the point estimates multiplied by the 95-5-percentile difference across municipios in the malaria variable. Reporting of additional estimates is suppressed. Exunexposed cohorts are those born before 1940 and fully exposed cohorts are those born after 1957. Cohorts are determined based on municipio of birth. The universe for the regressions include dummies for region of birth. Excluded instruments for 2SLS in Panel B are municipio-of-birth average temperature, altitude, and the interaction of the two. Appendices A and B describe, respectively, the outcome variables and malaria measures. The additional controls and instruments are described in the text and Appendix C.
Table 5: Approximate Effects on Adult Income per Probability of Childhood Malaria Infection

<table>
<thead>
<tr>
<th>Dependent Variables:</th>
<th>United States</th>
<th>Brazil</th>
<th>Colombia</th>
<th>Mexico</th>
</tr>
</thead>
<tbody>
<tr>
<td>Occupational Income Score</td>
<td>0.14</td>
<td>0.37</td>
<td>0.07</td>
<td>0.47</td>
</tr>
<tr>
<td>Duncan's Index</td>
<td>0.18</td>
<td>0.27</td>
<td>0.26</td>
<td>0.60</td>
</tr>
<tr>
<td>Log Total Income</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Log Earned Income</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Industrial Income Score</td>
<td></td>
<td></td>
<td>0.11</td>
<td></td>
</tr>
<tr>
<td>Log Earned Income</td>
<td></td>
<td></td>
<td>0.41</td>
<td></td>
</tr>
</tbody>
</table>

Maximal Endemcity (and Malaria Infection Rate)

<table>
<thead>
<tr>
<th></th>
<th>Mesoendemic</th>
<th>Hyperendemic</th>
<th>Hyperendemic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Income Effect per Probability of Childhood Infection</td>
<td>0.37 (0.625)</td>
<td>0.59 (0.625)</td>
<td>0.11 (0.39, adjusted)</td>
</tr>
</tbody>
</table>

Notes: The reduced-form differences are taken from Tables 2, 3, and 4, for the indicated outcomes. Reported are averages of the OLS point estimates, multiplied by the difference between 95th and 5th percentile malaria intensity. For the United States, these numbers are also normalized by the average value of the relevant income proxy for white males born in the South between 1875 and 1895. Maximal endemcity levels and approximate malaria-infection rates are according to Molineaux (1988, p. 988 and Figure 35.10). Note that these numbers refer to pre-eradication malaria burdens for children. The effect on adult income per probability of childhood infection is the reduced-form difference divided by the estimated pre-eradication infection rate for malaria.
Table 6: Differences in the Response to *falciparum* versus *vivax* Malaria in Colombia

<table>
<thead>
<tr>
<th>Dependent Variables: Differences across Cohorts in...</th>
<th>Malaria Ecology (Poveda)</th>
<th>Malaria Ecology (Mellinger)</th>
<th>Cases Notified, 1955, per 100K Pop.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Literacy</td>
<td>Years of Schooling</td>
<td>Income Index</td>
</tr>
<tr>
<td>Independent Variables:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Malaria Intensity</td>
<td>0.088</td>
<td>1.219 ***</td>
<td>0.169 ***</td>
</tr>
<tr>
<td></td>
<td>(0.072)</td>
<td>(0.412)</td>
<td>(0.057)</td>
</tr>
<tr>
<td>Malaria Intensity x Fraction of <em>falciparum</em> Infection in Depto.</td>
<td>0.004</td>
<td>-2.521 **</td>
<td>-0.022</td>
</tr>
<tr>
<td></td>
<td>(0.198)</td>
<td>(1.084)</td>
<td>(0.157)</td>
</tr>
<tr>
<td>Malaria Intensity</td>
<td>0.017</td>
<td>0.977 **</td>
<td>0.165 ***</td>
</tr>
<tr>
<td></td>
<td>(0.051)</td>
<td>(0.421)</td>
<td>(0.052)</td>
</tr>
<tr>
<td>Malaria Intensity x Fraction of <em>falciparum</em> Infection in Depto.</td>
<td>-0.008</td>
<td>-1.783 *</td>
<td>-0.009</td>
</tr>
<tr>
<td></td>
<td>(0.131)</td>
<td>(1.058)</td>
<td>(0.130)</td>
</tr>
<tr>
<td>Malaria Intensity</td>
<td>0.108</td>
<td>2.120 ***</td>
<td>0.260 ***</td>
</tr>
<tr>
<td></td>
<td>(0.084)</td>
<td>(0.524)</td>
<td>(0.069)</td>
</tr>
<tr>
<td>Malaria Intensity x Fraction of <em>falciparum</em> Infection in Depto.</td>
<td>-0.048</td>
<td>-3.222 ***</td>
<td>-0.043</td>
</tr>
<tr>
<td></td>
<td>(0.211)</td>
<td>(1.174)</td>
<td>(0.168)</td>
</tr>
<tr>
<td>Malaria Intensity</td>
<td>0.056</td>
<td>2.154 ***</td>
<td>0.349 ***</td>
</tr>
<tr>
<td></td>
<td>(0.072)</td>
<td>(0.583)</td>
<td>(0.078)</td>
</tr>
<tr>
<td>Malaria Intensity x Fraction of <em>falciparum</em> Infection in Depto.</td>
<td>-0.048</td>
<td>-2.557 **</td>
<td>-0.044</td>
</tr>
<tr>
<td></td>
<td>(0.136)</td>
<td>(1.151)</td>
<td>(0.141)</td>
</tr>
</tbody>
</table>

Notes: This table reports estimates of malaria in equation 3 using OLS and 2SLS for the indicated dependent variables, plus an interaction term of malaria intensity with the fraction of malaria infections in the departamento that were attributed to the *falciparum* strain (a more lethal variety of malaria). (The *falciparum* data are drawn from SEM (1957).) The units of observation are Colombian municipios. The malaria variables are as indicated in the column headings. Robust (Huber-White) standard errors in parentheses. Single asterisk denotes statistical significance at the 90% level of confidence; double 95%; triple, 99%. Reporting of additional estimates is suppressed. Unexposed cohorts are those born before 1940 and fully exposed cohorts are those born after 1957. Cohorts are determined based on year and municipio of birth. The universe for the base sample consists of the native-born population between the ages of 25 and 55 (15–55 for literacy) in the 1973 and 1993 census microdata from the IPUMS. All regressions include dummies for region of birth. Excluded instruments for 2SLS in Panels C and D are municipio-of-birth average temperature, altitude, the interaction of the two, fraction of *falciparum* malaria by departamento of birth, and the interaction of the first three variable with *falciparum*. The additional controls are described in the text and Appendix C.
A Construction of the Cohort-Level Data

The micro data for the RC analysis are drawn primarily from the IPUMS data for the United States, Brazil, Colombia, and Mexico. For each country, these data are used to construct a panel of income by year and area of birth. The cohort-level outcomes are constructed as follows.

1. The microdata are first pooled together.

2. The individual-level outcome variable are projected onto dummies for year-of-birth × Census year, i.e. I run the following regression:

\[ y_{itk} = \delta_{tk} + \epsilon_{itk} \]

for individual \( i \) in cohort \( k \) when observed in census year \( t \). This regression absorbs all cohort, age, and period effects that are common for the whole country.

3. I then define cells for each combination of year of birth and area of birth. Within each cell, I compute the average of the estimated residuals (the \( \hat{\epsilon}_{itk} \)). Because these averages are constructed with differing degrees of precision, I also compute the square root of the cell sizes to use as weights when estimating equation 1.

4. I repeat this process separately for each outcome variable.

These average outcomes by cohort form the dependent variables used in the present study.

A.1 Details for the United States Sample

The underlying sample used for the United States consists of native-born white males in the age range [25,60] in the 1900–1990 IPUMS microdata or in the 1880 microdata from the North Atlantic Population Project (NAPP, 2004). (These data were last accessed November 14, 2005.) This results in a data set with year-of-birth cohorts from 1825 to 1965. The original micro-level variables are defined as follows:

- **Occupational income score.** The occupational income score is an indicator of income by disaggregated occupational categories. It was calibrated using data from the 1950 Census, and is the average by occupation of all reported labor earnings. See Ruggles and Sobek (1997) for further details.

- **Duncan socio-economic index.** This measure is a weighted average of earnings and education among males within each occupation. The weights are based on analysis by Duncan (1961) who regressed a measure of perceived prestige of several occupations on its average income and education. This measure serves to proxy for both the income and skill requirements in each occupation. It was similarly calibrated using data from the 1950 Census.

For the majority of the years of birth, I can compute average income proxies for all of the 51 states plus the District of Columbia. The availability of state-level malaria data and the control variables restricts the sample further to 46 states of birth. Alaska, Colorado, the District of Colombia, Hawaii, and Oklahoma are excluded because of missing data for at least one of the other independent variables. This leaves 46 states of birth in the base sample.

There are a number of cohorts born before 1885 for which as few as 37 states of birth are represented. (See Appendix Figure A–1.) For those born between 1855 and 1885, this appears to be due to small samples, because, while the NAPP data are a 100% sample for 1880, there are no microdata for 1890 and 1900 IPUMS data are only a 1% sample. On the other hand, for the 1843-1855 birth cohorts, all but two of the years have all 46 states represented. Nevertheless, even with the 100% sample from 1880, there are as many as six states per year missing for those cohorts born before 1843. A number of the territories (all of which would later become states) were being first settled by people of European descent during the first half of the 19th century, and it is quite possible that, in certain years, no one eligible to be enumerated was born in some territories. (Untaxed Indians were not counted in the censuses.) Note that I use the term state above to refer to states or territories. Territories were valid areas of birth in the earlier censuses, and are coded in the same way as if they had been states.
While this procedure generates an unbalanced panel, results are similar when using a balanced panel with only those states of birth with the maximum of 141 valid observations. A comparison of the cohort-specific estimates from the balanced and unbalanced panels shows high correlation (over 0.96, for example, in the case of the full-controls specification for the occupational income score).

A.2 Details for the Brazilian Sample

The underlying sample used for Brazil consists of native males in the age range $[15,60]$ in the 1960–2000 IPUMS microdata. (These data were last accessed April 7, 2006.) This results in a data set with year-of-birth cohorts from 1905 to 1984. See Appendix Figure A–2 for sample statistics by year of birth.

State of birth is available for these samples. Brazilian states (and several territories that were to become states) were, by and large, consistently defined over the course of the sample. Those few that were not were merged together to reflect administrative divisions in the early 1950s. Specifically, I merged Rondônia into Guaporé, Roraima into Rio Branco, Tocantins into Goias, Fernando de Noronha into Pernambuco, Serra do Aimores into Minas Gerais, and Mato Grosso do Sul into Mato Grosso.

The original micro-level variables are as follows:

- **Literacy.** A binary variable individual measuring whether an individual can read and write at least a simple note.

- **Years of Schooling.** Numbers of years of education corresponding to highest grade completed. Non-numeric responses (e.g., “some secondary”) are mapped onto the midpoints of the appropriate intervals.

- **Total Income.** Records the total personal income from all sources in the prior month. In the empirical work above, this variable is treated in natural logs. This variable is intervalled in the 1960 census, and their midpoints are used in translating the data into income.

- **Earned Income.** Records the personal income from their labor (wages, business, or farm) in the prior month. In the empirical work above, this variable is treated in natural logs.

A.3 Details for the Colombian Sample

The underlying sample used for Colombia consists of native males in the age range $[15,60]$ in the 1973 and 1991 IPUMS microdata. (These data were last accessed April 10, 2006.) This results in a data set with year-of-birth cohorts from 1918 to 1977. See Appendix Figure A–3 for sample statistics by year of birth.

Area of birth is available in these samples at the level of departamento and municipio. The departamento is a first-order administrative division, similar to a state, while the municipio is a second-order division, similar to a county in the United States. A cohort’s municipio of birth is used in the present study to construct a proxy for childhood exposure to malaria. Colombia contains over one thousand municipios in the present day, but, to preserve confidentiality in the the IPUMS data, some of the smaller municipios are aggregated into larger groupings. This results in over 500 unique codes for area of birth, and I refer to these units simply as “municipios” in the text. Because municipal boundaries change over time, maps (SEM, 1957) and other administrative information (DANE, 2000) were used to relate data observed at various points in time onto the IPUMS recode of municipio.

The original micro-level variables are as follows:

- **Literacy.** A binary variable individual measuring whether an individual can read and write.

- **Years of Schooling.** Numbers of years of education corresponding to highest grade completed. Non-numeric responses (e.g., “some secondary”) are mapped onto the midpoints of the appropriate intervals.

- **Industrial Income Score.** The industrial income score is an indicator of income by industry and class of worker. It was calibrated using data from the Brazilian and Mexican censuses for all available years.
To remove census-year times country effects, the starting point for this variable is the residualized total income (\( \hat{\epsilon}_{itk} \)) described above, which is then averaged by industry and matched onto the Colombian sample. Because of the way this score is constructed, the variable is measured in natural logs. (Total income is available in the 1973 Colombian census, but the range of years of birth that these data cover is too limited.)

A.4 Details for the Mexican Sample

The underlying sample used for Mexico consists of native males in the age range \([15,60]\) in the 1960–2000 IPUMS microdata. (These data were last accessed April 7, 2006.) This results in a data set with year-of-birth cohorts from 1905 to 1984. See Appendix Figure A–4 for sample statistics by year of birth.

State of birth is available for these samples. Mexican states (some of which were territories early on) were defined consistently throughout the sample period.

The original micro-level variables are as follows:

- **Literacy.** A binary variable individual measuring whether an individual can read and write.

- **Years of Schooling.** Numbers of years of education corresponding to highest grade completed. Non-numeric responses (e.g., “some secondary”) are mapped onto the midpoints of the appropriate intervals.

- **Earned Income.** Records the personal income from their labor (wages, business, or farm) in the prior month. In the empirical work above, this variable is treated in natural logs. (Total income is available in certain years of the Mexican censuses, but the range of years of birth that these data cover is inappropriate for the analysis.)
Appendix Figure A – 1: Sample Statistics for the U.S. Sample

These graphs report additional summary statistics by year of birth for the $\hat{\beta}_t$ reported in Figure 4 in the subplot labeled “Occupational Income Score.”

Appendix Figure A – 2: Sample Statistics for the Brazilian Sample

These graphs report additional summary statistics by year of birth for the $\hat{\beta}_t$ reported in Figure 5 in the subplot labeled “Total Income”.

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Appendix Figure A – 3: Sample Statistics for the Colombian Sample

These graphs report additional summary statistics by year of birth for the $\hat{\beta}_t$ reported in Figure 6 in the subplot labeled “Income Score”.

Appendix Figure A – 4: Sample Statistics for the Mexican Sample

These graphs report additional summary statistics by year of birth for the $\hat{\beta}_t$ reported in Figure 7 in the subplot labeled “Earned Income”.

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B  Sources and Construction of the Malaria Data

Sources are indicated in parentheses at the end of each item.

- **United States.** Malaria mortality expressed a fraction of total mortality. This was measured in the 1890 Census as refers to the proceeding year. I normalize by total mortality in the state to filter any factor in the underreporting that is common to malaria and total mortality. These data were collected by Census enumerators. (Bureau of the Census, 1894.)

  As a specification check, I use three alternative measures of malaria intensity. An additional measure of malaria mortality refers to 1919–21 and is drawn from Maxcy (1923), who surveyed state departments of health. Because the death-registration system was not yet completely operational at the time of the Maxcy’s survey, these data are to be taken cum grano salis. No official measure of total mortality was available for the period, so I scale the data by population instead. The remaining two malaria variables are indices based on geographic and climatic factors. The first index (referred to as “Mellinger” in the text) of malaria ecology is computed using information on climate and local vectorial capacity. The construction of these data are described in Gallup, Mellinger, and Sachs (1999a). The source data were provided as raster data in one-degree grids. A GIS program was used to extract average malaria ecology by state. (Maxcy, 1924; Andrew Mellinger, private communication, and author’s calculations; Sok-Chul Hong, private communication, and author’s calculations.)

- **Brazil.** An index of malaria ecology, computed using information on climate and local vectorial capacity. The construction of these data are described in Gallup, Mellinger, and Sachs (1999a). The source data were provided as raster data in one-degree grids. A GIS program was used to extract average malaria ecology by state. (Andrew Mellinger, private communication, and author’s calculations.)

- **Colombia.** Two measures of ecology are used, as well as one measure of morbidity. The Poveda measure is an index of malaria ecology based on climatic factors, described by Poveda, Graham, Epstein, Rojas, Quiñones, Darío Vélez, and Martens (2000).

  A map in that study displaying the computed survival probability of \( p. \) vivax (Fig 6.5) was digitized and fed into a GIS program, which was then used to construct averages by municipio. The Mellinger measure of malaria ecology is the same as that used for Brazil, and was averaged by municipio in a GIS program (the “Spatial Analyst” toolbox within ArcView). Glenn Hyman of the Centro Internacional de Agricultura Tropical shared data on the Colombian municipio boundaries. Malaria cases notified per capita were drawn from the reports of the Servicio Nacional de Erradicación de la Malaria (SEM) and refer to 1956. (Poveda et al., 2000; Andrew Mellinger, private communication; Jonnes and Bell, 1997; SEM, 1957; and author’s calculations.)

  To account for measurement error in the above variables, I also construct climate-based instruments. The set of instruments consists of the municipio’s temperature, altitude and the interaction of the two. The temperature and altitude data are from records prior to 1960. Another proxy for climate is the fraction of each municipio within particular Holdridge climate zones. Those relevant for the areas under study are the following: cool temperate, warm temperate, subtropical dry, subtropical wet, tropical dry, and tropical wet. These data come from a GIS file provided by the Center for International Development at Harvard University, and were computed by municipio in a GIS program (the “spatial join” in ArcView). (Banco de la República, 1964; Gallup, Mellinger, and Sachs 1999b; and author’s calculations.)

- **Mexico.** Malaria mortality by state, expressed in per capita terms. (Pesqueira, 1957.)
Figure B – 1: Malaria Intensity by State in the United States

Notes: Displays a map of the ratio of malaria mortality to total mortality by state circa 1890. Source: Bureau of the Census (1894). Darker colors indicate more malaria.

Figure B – 2: Malaria Intensity by State in Brazil

Notes: Displays a map of an index of malaria ecology as constructed by Gallup, Mellinger, and Sachs (1999a). Darker colors indicate climatic and geographic conditions more conducive to the transmission of malaria.
Figure B – 3: Malaria Intensity by Municipio in Colombia

Notes: Displays a map of an index of malaria ecology as constructed by Gallup, Mellinger, and Sachs (1999a). Darker colors indicate climatic and geographic conditions more conducive to the transmission of malaria.

Figure B – 4: Malaria Intensity by State in Mexico

Displays a map of malaria mortality per capita, circa 1950. Source: Pesqueira (1957). Darker colors indicate more malaria.
C  Control Variables

Control variables for the United States:

- **Average wage, 1899.** I input the average monthly earnings (with board) for farm laborers by state in 1899. Various other wage measures are summarized by the same source, but are generally not available for a complete set of states. (Lebergott, 1964, Table A–24.)

- **Dummy for being born in the South.**

- **Doctors per capita, 1898.** Number of physicians per 1,000 inhabitants of each state. The primary source is listed as Polk’s *Register of Physicians*, 1898. (Abbott, 1900.)

- **State public-health spending, 1898.** Per capita appropriations, by state, for state boards of health in 1898. Primary sources include the annual reports of state boards of health, state appropriations laws, and correspondence with the secretaries of the boards of health. (Abbott, 1900.)

- **Infant mortality, 1890.** The estimates of infant mortality are constructed from published tabulations. Table 3 in Part III contains enumerated deaths of children under one year of age. I scale this number by the estimated birth rate (Part I, page 482) times the female population (Part I, Table 2). The rate from 1890 was used because child-mortality data are not available comprehensively for the years 1900–1932, during which time the death-registration system was established. The 1890 mortality data were collected by Census enumerators. (Census, 1894.)

- **Recruits for World War I found rejected for military service because of health “defects,” 1917-1919.** The fraction of recruits by state who were rejected by army physicians for physical defects. (Love and Davenport, 1920.)

- **Hookworm Infection.** Computed from examinations of army recruits. (Kofoid and Tucker, 1921)

- **Mortality from other diseases.** Separate variables are constructed for the following eight causes of death: scarlet fever, measles, whooping cough, diphtheria/croup, typhoid fever, diarrheal diseases, and pneumonia. Data are expressed as the fraction of total mortality in 1890. (Census, 1894.)

- **Fertility rate, 1890.** The estimated birth rate (from Part I, page 482). (Census, 1894.)

- **Log change in School Term Length, c. 1902–1932.** Average length of school term, in weeks. (Annual reports of the federal Commissioner of Education, 1905-1932.)

- **Log change in Average Monthly Salaries for Teachers, c. 1902–1932.** (Annual reports of the federal Commissioner of Education, 1905-1932.)

- **Log change in Pupil/Teacher Ratio, c. 1902–1932.** Average attendance divided by number of teachers. (Annual reports of the federal Commissioner of Education, 1905-1932.)

- **Log change in School Expenditure, c. 1902–1932.** (Annual reports of the federal Commissioner of Education, 1905-1932.)

- **Adult literacy rate.** These data were compiled at the state level and come from the 1910 Census. Adult literacy refers to males of voting age. (ICPSR #3.)

- **Population urban.** From Census tabulations measuring the population residing in metro areas in 1910. (ICPSR #3)

- **Fraction black.** From tabulations of the 1910 Census. (ICPSR #3)

- **Male unemployment rate.** From tabulations of the 1930 Census. (ICPSR #3.)

Control variables for the Brazilian states:

- **Region dummies.** North (Norte and Nordeste) and South (Centro-Oeste, Sudeste, and Sul).
• **Population Density.** Population per square kilometer in 1950. (IBGE, 1950 and 1951.)

• **Infant mortality.** Number of infant deaths in the municipio of the state capital, scaled by the estimated birth rate, which is computed from data for the whole state. (IBGE, 1951.)

• **Log of Electricity Capacity.** Measured circa 1950. Original data in kilowatts. (IBGE, 1950.)

• **Fraction of population economically active.** Measured for population ten years and older for 1950. (IBGE, 1950.)

• **Shares of labor force by sector.** Fraction of economically active population in each of the following sectors: agriculture, extractive industries, manufacturing, transportation, and services. Measured for population ten years and older for 1950. (IBGE, 1950.)

**Control variables for Colombian municipios:**

• **Region dummies.** The regions are as follows: Central, Bogota, Pacifico Norte, Eje Cafetero, Andina Norte, Andina Sur, Pacifico Sur, Caribe, Orinoquia, and Amazonia.

• **“La Violencia”**. A qualitative variable (ranging from 1 to 3) indicating the intensity of violence in the Colombian civil war known locally at “La Violencia”. The data are taken from Oquist, who classified conflict intensity decomposed by municipio and sub-period: before 1955, when the violence was largely in population centers, and 1955 and after, when the conflict was more likely to take place in the countryside. (Oquist, 1976.)

• **High Concentration “Minifundista”**. Binary variable indicating the presence of small-land holders or minifundistas, as opposed to large land holders or urban areas. The reference period is the 1950s, although land-holding patterns were persistent historically. To construct municipio-level data, the map was digitized and georeferenced. Digital data on municipio boundaries, provided under special agreement from the Centro Internacional de Agricultura Tropical (CIAT), was overlaid on the map and municipios were coded dichotomously as indicated by the map. The municipio boundaries of the CIAT data refer to 1993, and therefore these mapped back onto 1950s entities. (Banco de la República, 1964 (map 57); Jonnes and Bell, 1997; DANE, 2000; author’s calculations.)

• **Coffee-growing Region.** Binary variable indicating the presence of coffee cultivation. The reference period is 1960. Municipio-level data were created using the process described above for the “minifundista” variable. (Banco de la República, 1964, map 38.)

• **Coal Mining Region.** Dummy indicating the presence of actively exploited coal deposits, circa 1960. Municipio-level data were created using the process described above for the “minifundista” variable. (Banco de la República, 1964, map 22.)

• **Expansion of Ranching.** Areas identified for possible expansion of ranching in 1960. Municipio-level data were created using the process described above for the “minifundista” variable. (Banco de la República, 1964, map 55.)

• **Infrastructure/Market Access.** An index variable for the ease of transport to major markets or seaports from the area, based on infrastructure in circa 1960. Six (ordered) categories are used, following the map’s categorization. Municipio-level data were created using the process described above for the “minifundista” variable. (Banco de la República, 1964.)

• **Level of development.** An index variable for the general level of economic development of the area (“nivel de vida”), circa 1960. Six (ordered) categories are used, following the map’s categorization. Municipio-level data were created using the process described above for the “minifundista” variable. (Banco de la República, 1964, map 59.)

• **Manufacturing employment per capita.** Computed by municipio from the 1945 Colombian census of manufacturing. (Dirección Nacional de Estadística, 1947.)
• **Log Population Density.** Computed using the 1951 population. The land area was constructed from digital maps of *circa* 1993 municipio boundaries, and the municipios were re-aggregated to account for subdivision of entities. (Dirección Nacional de Estadística, 1947; Joffres and Bell, 1997; DANE, 2001.)

• **Disease controls.** The fractions of territory within each municipio in which transmission of the following diseases occurs: leishmaniasis, yellow fever, hookworm, and non-hookworm helminth diseases. The first three categories are vector-borne diseases and could themselves have been affected by the campaign. The category of non-hookworm helminths represents an aggregate of numerous types of helminths. The underlying geographic data are defined with a fairly broad brushstroke, and as a result this is almost a dichotomous variable by municipio. These data come from a GIS file provided by the Center for International Development at Harvard University, and were computed by municipio in a GIS program (the “spatial join” in ArcView). (Gallup, Mellinger, and Sachs, 1999b; and author’s calculations.)

**Control variables for the Mexican states:**

• **Region dummies.** “Norte”, “Centro” and “Sur”, as per the divisions in the 1960 *Anuario Estadístico.* (Dirección General de Estadística, 1960.)

• **Population Density.** Population per square kilometer in 1950. (Dirección General de Estadística, 1952a and 1952b.)

• **Infant mortality.** Rate per 1,000 births. Data refer to 1950. (Coordinación General de los Servicios Nacionales de Estadística, Geografía e Informática, 1981.)

• **Log of Electricity Capacity.** Measured *circa* 1950. Original data in kilowatts. (Dirección General de Estadística, 1952b.)

• **Fraction of pop economically active.** Measured for population 12 years and older for 1950. (Dirección General de Estadística, 1952b.)

• **Shares of labor force by sector.** Fraction of economically active population in each of the following sectors: agriculture, extractive industries, manufacturing, transportation, and services. Measured for population 12 years and older for 1950. (Dirección General de Estadística, 1952b.)

• **Household income GINIs.** GINI coefficients were constructed from the 1950 census, which included tabulations by state of the distribution of families by household income. (Dirección General de Estadística, 1952b.)
Appendix D: Panel Estimates of Childhood Exposure

<table>
<thead>
<tr>
<th>Dependent Variables: Income Measures by Cohort</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Panel A: United States</strong></td>
</tr>
<tr>
<td>Occupational Income Score</td>
</tr>
<tr>
<td>27.936 ***</td>
</tr>
<tr>
<td>(5.528)</td>
</tr>
<tr>
<td>[0.106]</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Panel B: Brazil</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Literacy</td>
</tr>
<tr>
<td>0.030 ***</td>
</tr>
<tr>
<td>(0.005)</td>
</tr>
<tr>
<td>[0.156]</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Panel C: Colombia</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Literacy</td>
</tr>
<tr>
<td>0.209 ***</td>
</tr>
<tr>
<td>(0.032)</td>
</tr>
<tr>
<td>[0.083]</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Panel D: Mexico</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Literacy</td>
</tr>
<tr>
<td>0.047 ***</td>
</tr>
<tr>
<td>(0.011)</td>
</tr>
<tr>
<td>[0.150]</td>
</tr>
</tbody>
</table>

Notes: Each cell reports estimates, from a separate regression, of the childhood-exposure variable times pre-campaign malaria intensity using OLS. The outcome variables are as indicated in each Panel’s column heading. The malaria and control variables, which enter the specification interacted with potential exposure, are described in Appendices B and C. Unlike the results in the main text, these parameters are estimated directly from panel data structured by year of birth and area of birth, the construction of which is described in Appendix A. The following equation is estimated:

\[ Y_{jk} = \tilde{\beta} M_j \times \text{Exp}_k + \delta_k + \delta_j + \sum_i x_i^j \times \text{Exp}_k \times \gamma_i + \nu_{jk} \]

in which \( \text{Exp}_k \) is potential exposure to the malaria-eradication campaign (defined above), \( M_j \) is the pre-campaign malaria intensity in area of birth \( j \), the \( x^i \) are the other state-of-birth controls, and the \( \delta_k \) and \( \delta_j \) are fixed effects for year of birth and area of birth, respectively. The table reports estimates of \( \tilde{\beta} \). Standard errors, shown in parentheses, are clustered on area of birth. Single asterisk denotes statistical significance at the 90% level of confidence; double 95%; triple, 99%. Observations are weighted by the square root of the size of the cell used to construct the cohort average. Reporting of additional terms suppressed. The terms in curly brackets report the point estimate multiplied by the difference between 95th and 5th percentile malaria intensity. For the United States, this number is also normalized by the average value of the relevant income proxy for white males born in the South between 1875 and 1895.