Early Life Exposure to Malaria and Economic Development*

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Abstract

This paper investigates the long-term relationship between early life exposure to malaria and adult socioeconomic outcomes in Brazil. The identification strategy relies on exogenous variation in the risk of malaria outbreaks in different states and seasons of the year to identify early life exposure according to the timing and location of birth. Furthermore, Brazil has undergone a successful campaign of malaria eradication during the 1950s, which allows for employing a differences-in-differences design to compare outcomes of birth cohorts born just prior to and just after eradication. I find consistent negative treatment effects of in utero exposure on years of education and on income levels and the effects are stronger for exposure during the first trimester of pregnancy than during other periods of gestation. Additionally, consistent with previous findings, men are more likely to exhibit larger long-term effects. I find no significant treatment effects of early life exposure to malaria on fertility and no significant differences in socioeconomic conditions of more exposed individuals born after eradication campaign relative to less exposed ones.

Keywords: Malaria, Exposure, Economic Development
JEL Codes: I15, I25, J13, J31

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1 Introduction

How important are early life health conditions for adult outcomes? The "fetal origins hypothesis" suggests that initial health endowments might have important and persistent effects on long-term health and other socioeconomic variables (Almond and Currie, 2011). However, addressing the causal relationship between fetal health and lifetime economic conditions is challenging because other factors, such as unobserved socioeconomic characteristics, might affect initial health conditions in the months between conception and birth.

This paper investigates the effect of prenatal exposure to malaria and long-term adult outcomes in Brazil. The identification strategy employed by this paper relies on exogenous variation in the risk of outbreaks of the disease in different states and seasons of the year to identify early life exposure according to the timing and location of birth. I use this measure of exposure to estimate the causal effects of early life health environment on long-term socioeconomic conditions, including education, income, and fertility, for different birth cohorts. In addition to this fixed effects model, I use a differences-in-differences approach to compare outcomes of birth cohorts born prior to and after Brazil's successful 1950s campaign of malaria eradication.

Malaria is a tropical disease caused by *Plasmodium* parasites, spread to humans through the bites of the female *Anopheles* mosquito, or malaria vector. According to the World Health Organization (WHO),[1] in 2016, 216 million cases of malaria and an estimated 445 thousand deaths were reported. Cases are concentrated in tropical areas, reaching subtropical regions in five continents. Areas with temperate climates also experience outbreaks of the disease, depending on the season. The largest fraction of malaria cases occurs in Sub-Saharan Africa; in 2016, 14 sub-Saharan countries (and India) accounted alone for 80 percent of the malaria burden worldwide.[2] In Brazil, after the eradication efforts throughout the 1950s, malaria is under control in most states outside of the Legal Amazon region;[3] in 2017, the region accounted for approximately 99.7 percent of the

[1] WHO (2018)

[2] WHO (2018)

[3] Prior to the Eradication Campaign, malaria was a widespread public health hazard. The governmental efforts to fight the disease essentially dropped malaria rates to approximately zero in states outside the Legal Amazon, which includes the states of the North region (Acre, Amazonas, Amapa, Pará, Rondônia, Roraima, and Tocantins), two states

194,425 reported cases.[4]

Malaria destroys red blood cells, depriving the distribution of oxygen and nutrients to the body tissue. Its effects on long-term health and cognitive development, however, are most powerful when exposure occurs during the first periods of life, whereby fetal development is hampered, leading to long-lasting consequences (Barreca, 2010). For instance, placental malaria (infection from the mother to the placenta) induces changes in the placental structure and functioning, which is associated with fetal growth restrictions (Umbers et al., 2011).[5] Additionally, pregnant women are relatively more susceptible to malaria infections compared to non-pregnant women, especially in first- (primigravidae) and second-order pregnancies (Menendez, 1995; Lucas, 2013).

The identification strategy proposed by this paper relies on two main sources of exogenous variations to estimate the long-term consequences of early life exposure to the disease. The first is given by differential transmission risks across months and states, which jointly determine heterogeneous levels of exposure across individuals within birth cohorts. The second source of variation is represented by the sharp decline in the overall scale of malaria transmission due to the eradication efforts during the 1950s. I start by constructing a measure of malaria risk using disaggregated data on the frequency of reported number of cases of malaria at the state-month cell level. By observing individuals' adult socioeconomic outcomes and their respective state, month, and year of birth, I am able to link each individual's socioeconomic later-life conditions to the degree of exposure faced during early life.

I follow two different estimation strategies to address the in utero effects of exposure to malaria. In the first strategy, I estimate the above relationship for individuals born shortly before the sharp decline in malaria rates. As a falsification test, I conduct the same analysis for the subsequent birth cohort, in which early life exposure is dramatically shortened after the reduction in the scale of transmission. By construction, the exposure difference between pre- and post-eradication cohorts

in the Northeast region (Maranhão and Piauí), and one state in the Mid-west region (Mato Grosso). These states are dropped from the sample.

[4]Ministry of Health – DATASUS data.

[5]Fetal growth restriction is defined as a condition in which babies are unable to develop to their genetically predetermined potential. This condition might lead to premature deliveries, low birth weight, and direct long-term health consequences, such as hypertension and vascular disorders (Matteelli et al., 1997; Umbers et al., 2011).

is the degree of overall malaria burden during the early periods of life. If the exposure to malaria during intrauterine life is important for lifetime outcomes, its effects should be seen in the estimations of the pre-eradication birth cohort sample and not in the estimations of the post-eradication birth cohort sample.

In the second estimation strategy, I exploit the exogenous variation in the scale of the malaria burden across states in Brazil to employ a differences-in-differences strategy. I estimate the effects of early life exposure to malaria across birth cohorts before and after the eradication campaign, thereby controlling for unobserved invariant characteristics that might be correlated with exposure levels. In particular, I interact the exposure variable with an indicator for being born after the eradication era to test whether positive health shocks lead to significant gains in long-term socioeconomic conditions for treated individuals.

In the analysis that follows, I consider different critical periods of early life exposure – according to trimesters of pregnancy – to test for heterogeneous treatment effects during different periods of exposure. In the baseline specification, I construct a measure of average in utero exposure, by simply taking the average risk of malaria transmission of the nine months prior to the birth of the individual. Second, I construct analogous measures of exposure for each trimester of pregnancy to test whether the timing of transmission is an important factor that determines long-term outcomes.

I find consistently negative effects of early life exposure to malaria on educational attainment and personal income for individuals born shortly before the eradication campaign. OLS estimates suggest that, on average, an increase in the in utero exposure to malaria from the fifth to the ninety-fifth percentile of the transmission probability distribution reduces educational attainment by nearly 2 years, and leads to reductions in hourly income by approximately 24 percent. The results also indicate heterogeneous treatment effects on different periods of early life, with the largest effects stemming from exposure to the disease during the first three months of intrauterine life. I do not find consistent treatment effects on fertility choices. Additionally, differences-in-differences estimates indicate that, relative to less exposure, more exposed individuals during intrauterine life born on the post-eradication cohort have, on average, between 0.611 and 3.363 more years of ed-

ucation and fertility declines by a factor between 0.243 and 1.92 when compared to individuals members of the pre-eradication birth cohort.

I discuss different sources of potential biases of my estimation strategy in the sensitivity analysis as follows: First, I address the treatment effects for men versus women; I find larger effects on educational attainment and income for men. A possible explanation for this result is that male fetuses are biologically more vulnerable than female ones (Kraemer, 2000; Low, 2015; Waldron, 1983). Thus, higher relative exposure of male individuals would lead to a larger impact on subsequent health. Second, I show that mortality selection – potentially caused by the death of the weakest fetuses after exposure – is not a factor leading to biased estimates. In order to formally test this hypothesis, I construct a dataset identifying childbearing-age women during the pre-eradication era, and I estimate the effects of malaria risk on the likelihood of stillbirths for women who were pregnant during that time. I find no relationship between exposure to malaria and in utero mortality. Third, I show that the results are not likely to be driven by selective migration. I construct two different datasets, one for movers and one for non-movers, and show that estimation results for both samples are qualitatively similar. I also test for different timings of birth to alleviate concerns of birth cohort misclassification in identifying pre- and post-eradications individuals. The results are robust to a range of different specifications according to the timing of exposure.[6]

Finally, I address the potential concern of measurement error in the risk of transmission variable by instrumenting the malaria risk with climatic conditions that are associated with the biological life-cycle characteristics of parasite and vector population distributions. The instrumental variables (IV) results suggest a larger treatment effect of first trimester exposure on education; the effects on income are only significant for the bottom decile of the distribution. Moreover, the IV results indicate no significant treatment effects on fertility and no significant differences in outcomes between more exposed individuals born after the campaign relative to less exposed ones.

The present paper contributes to the literature on the long-term effects of early life health shocks in at least two important ways. First and foremost, this paper is the first to explore hetero-

[6]However, the effects on income are somewhat imprecisely estimated for some specifications.

geneous effects of timing of in utero exposure to malaria on economic outcomes.[7] The identification strategy employed by this paper compares individuals on two dimensions: across geographic location and across month of birth for different birth cohorts, which allows me to test for differential effects on different critical periods of gestation. Second, the identification strategy arguably accounts for any pre-existing heterogeneous regional trends across states, since the disaggregated nature of the exposure measure allows me to explore monthly variations in exposure within both states and birth years. Previous studies usually identify exposure by geographical differences in endemicity rates across only birth cohorts, which might not fully account for pre-trends in the outcome variables possibly associated with the timing and effects of variations in the disease burden.

The analysis of the Brazilian experience with malaria provides a relevant setting for the purposes of the paper for four main reasons. First, it provides a unique opportunity to address the paper's main question of the effects of exposure to malaria because Brazil underwent a sharp and almost instantaneous decline in malaria rates after the eradication campaign efforts, with the application of DDT spraying inside houses and anti-malarial drugs distribution. This feature of the historical path of the incidence of malaria in Brazil allows me to test for the in utero effects of exposure to the disease among cohorts born shortly before and after the sharp decline in the overall exposure. An important attribute of the worldwide campaign is that its enactment was possible after the discovery of the insecticidal properties of DDT, and therefore, alleviates concerns about the exogeneity of the campaign to the outcome variables analyzed. Second, malaria infections during pregnancy affect fetuses in ways that can lead to long-term health consequences, as discussed above; therefore, fetal exposure to malaria can serve as a proxy for early life endowment differences. Third, detailed information on individual-level socioeconomic indicators, such as educational attainment, income, fertility, and migration, as well as state-level malaria rates for a large period, are publicly available for Brazil. Fourth, the type of malaria parasite prevailing in Brazil, *Plasmodium vivax*, is rarely lethal, which mitigates potential mortality selection issues.[8]

[7] Epidemiological studies have also addressed the consequences of different timing of malaria infection during pregnancy. However, these studies do not fully account for confounding factors and the small sample sizes that frequently are used make it difficult to generalize the findings.

[8] I provide a detailed analysis of mortality selection issue in section 6.3, when discussing alternative specifications

The paper is structured in seven sections, including this introduction, as follows: Section 2 discusses the related literature. Section 3 presents some background information about malaria transmission and the history of malaria in Brazil. Section 4 presents the data, and discusses some conceptual definitions are discussed. Section 5, provides detailed discussion of the research design implemented in this paper. Section 6 provides the main results, and further analyzes alternative explanations that could potentially drive the results. Section 7 concludes.

2 Related Literature

This paper relates to the empirical literature of long-term effects of early life environmental shocks on health and socioeconomic outcomes. The "fetal origins hypothesis", proposed by this strand of the epidemiological and economic literatures, assesses the relative importance of the persistence of in-utero conditions to subsequent health and mortality (Almond and Currie, 2011). This hypothesis suggests that environmental shocks on health are most powerful during the early periods of life, when the organs and body systems are not fully developed.

Different levels of exposure to health shocks in critical periods of early life may lead to large differences in adult health and socioeconomic conditions. In his seminal study, Barker (1998) finds a strictly negative correlation between fetal nutrition and later-life mortality caused by heart disease, by linking health conditions in different locations in Britain from the 1901–1910 period to adult mortality in these locations between 1968 and 1978. Other studies have also found strong statistical associations between early-life conditions and long-term outcomes. For example, increases in longevity observed in United States in the nineteenth and twentieth centuries can partially be attributed to early childhood environmental conditions (Costa, 2000, 2003; Costa and Lahey, 2005). Accordingly, low birth weight is usually associated with lower achievements later in life (Hack et al., 2002; Case et al., 2005; Black et al., 2007; Currie and Moretti, 2007).

Other studies have incorporated robust empirical strategies to isolate the causal long-run con-

of the model.

sequences of early-life conditions, taking into account other confounding factors that might cause bias to the analysis.[9] These studies frequently rely on exogenous short-term shocks that affect the population under study in an heterogeneous manner to identify the exposure effects. Individuals who had been exposed in the first trimester of intrautrine life to the Dutch "Hunger Winter" of 1944, caused by the cut in the food supply after the Nazi occupation of the Netherlands, are found to be more likely to develop obesity and other chronic diseases during adulthood (Stein et al., 1975; Roseboom et al., 2011). In the same vein, Almond (2006) and Almond and Mazumder (2005) find that individuals who experienced more exposure to the 1918 Influenza Pandemic in United States – both across timing of birth and state of birth – have less education and income, as well as worse adult health. Chen and Zhou (2007) and Meng and Qian (2006) find long-term health and socioeconomic effects of cohorts born during the Chinese Great Famine, whereas fasting during pregnancy to observe Ramadan in Islamic religious groups lead to lower later-life health and socioeconomic conditions (Almond and Mazumder, 2011; Van Ewijk, 2011).

Exposure to infectious diseases, in general, may affect fetal development, given that maternal energy might be diverged from the fetus to combat spread of disease (Almond and Currie, 2011). Costa and Lahey (2005) explore quarter of birth variation as a proxy for early life environment in order to identify the effects of exposure to seasonal infectious diseases on mortality rates. Barreca (2010) finds strong effects of malaria reduction in United States on schooling attainment, by instrumenting malaria transmission with climatic factors such as temperature and rainfall.

Some other studies have explored exogenous effects of eradication efforts to reduce different epidemic diseases – such as hookworm and malaria – to test whether larger gains in socioeconomic outcomes occurred among cohorts born in formerly more endemic locations than those in lower endemic areas before and after the eradication. Using this methodology, Bleakley (2007) found evidence of increases in school enrollment, attendance, and literacy rates following the successful eradication of hookworm disease in the American South, whereas other studies such as Lucas (2010), Cutler et al. (2010), Burlando (2012), and Bleakley (2010b), find positive effects of malaria

[9]Currie and Vogl (2013) provide a thorough review of the empirical evidence for the "fetal origins hypothesis" in the developing economies context.

eradication on human capital accumulation and other adult outcomes.[10]

3 Background

3.1 The Disease

The malaria parasite is a micro-organism that belongs to the genus *Plasmodium*. The natural ecology of malaria involves the successive infection of humans and a vector, the female *Anopheles* mosquito.[11] In humans, the parasites initially affect liver cells, subsequently spreading to the red blood cells. In this stage, the symptoms of the infection start to develop.[12] Blood stage parasites in a form named *gametocytes* are responsible for the parasite's growth and multiplication when present on the blood meal taken from the *Anopheles* mosquito. The infected adult female mosquito, then, lays eggs in stagnant water reservoirs. The development of the eggs into larvae, then pupae, to reach adulthood is aquatic and takes approximately 9-12 days, depending on the species, in tropical areas. Once adulthood is reached, the aquatic phase is finished, and the adult female mosquito is then, ready to serve as host to the malaria parasite.[13]

The transmission of malaria is highly sensitive to climatic variation. Rainfall is important due to its effects on the mosquito life-cycle, whereas temperature regulates malaria transmission (Martens et al., 1995). First, rainfall provides "breeding sites" for the female *anopheles* mosquitoes to lay their eggs. The continuation of the rainfall may contribute to the development of the larvae and pupae into adulthood. Temperature also plays an important role in this process. The aquatic development stages of the mosquito occurs more efficiently in temperatures between $16°C$ to $28°C$

[10]Other studies have analyzed the effects of exposure to malaria on a range of different outcomes. For example, Venkataramani (2012) uses the Mexican eradication effort to estimate the impact of the malaria reduction on cognition in adults. Klejnstrup et al. (2018) use district-level variation in malaria rates together with declining overall transmission in Tanzania to estimate the effect of early-life exposure to malaria on children's test scores for cognitive skills. None of these studies, however, explores seasonal variations in malaria transmission to identify the impact of the timing of birth on long-term outcomes.

[11]Only some species of the *Anopheles* mosquitoes are antropophilic (feeds on humans). In Brazil, there are more than 50 species, including the *Anopheles darlingi* and *Anopheles gambiae*, which have bitting preferences at the outdoors, making them highly effective vectors in the malaria transmission.

[12]Common symptoms include vomits, diarrhea, acute fever, shivering, anemia.

[13]On average, an adult *Anopheles* female mosquito can live for approximately 1-2 weeks.

(Cervellati et al., 2017). Moreover, survival of adult mosquitoes also depends on temperature and rainfall, as well as humidity. Warmer temperatures decreases the incubation period ("extrinsic" cycle) of the parasite inside the infected female *Anopheles*, increasing the chances of transmission. Temperatures below $15°C$ for *Plasmodium vivax* and $20°C$ for *Plasmodium falciparum* break the extrinsic cycle, hindering the transmission.[14]

The Extrinsic incubation period ranges from 10 to 21 days, whereas the intrinsic incubation period ranges from 7 to 30 days, on average, depending on the parasite species[15]. For the *P. vivax*, the range is 12 to 17 days, whereas for the *P.falciparum* and the *P. malariae*, the ranges are from 9 to 14 and 18 to 40 days, respectively (Brasil et al., 2011).

Taken together, this complex relationship between temperature and rainfall with the transmission cycle of malaria implies that outbreaks of malaria transmission are associated with a particular combination of the wet season and sufficiently high temperatures during the year; i.e., malaria transmission is characterized by a clear-cut seasonal component. I make use of this important mechanism in the transmission to identify the exposure during the first periods of life.

3.2 Malaria history in Brazil

Malaria was introduced in Brazil during the colonial times by African slaves, who carried the *Plasmodium falciparum* parasite, around 1560 (Griffing et al., 2015).[16] There were two main factors that triggered malaria transmission and spread throughout the country during late eighteenth and early nineteenth centuries. The boom of the rubber industry in the North region, where the geographic and climatic conditions for the survival of the *anopheles* mosquito are ideal, induced a massive influx of workers with a lack of immunity, causing a serious health hazard risk to the region and the country. Second, the precarious conditions of infrastructure constructions (mainly

[14] See https://www.cdc.gov/malaria/about/biology/index.html, accessed 08/30/2018.

[15] https://www.cdc.gov/malaria/about/biology/index.html, accessed 08/30/2018.

[16] The timing of the *Plasmodium vivax* parasite's arrival is not agreed on in the literature. One hypothesis suggests that the introduction of *Plasmodium vivax* in South America occurred around 15,000–30,000 years ago; another suggests that the introduction took place "multiple times from a now extinct European P. vivax population less than 500 years ago" (Griffing et al., 2015)

railroads connecting various parts of the country) contributed to the proliferation of the disease. During the early 1990s, Brazilian government aimed at expanding infrastructure in many remote areas and crews working on these projects fell victim to the disease, causing several construction sites to be interrupted or abandoned. Around that period, malaria was widespread in Brazil, with approximately 60 million cases – representing roughly 50 percent of the Brazilian population - being reported each year (Coura et al., 2006).

Early efforts to combat malaria were usually restricted to specific cases, especially on large public works projects. The common measures were mainly palliative, including the intake of quinine, an anti-malarial drug; the application of insecticides on water deposits near domiciles; drainage of water banks; distribution of bed nets, and destruction of mosquitoes breeding sites. [17] The increasingly health hazard caused by malaria on public infrastructure projects and among the poor population, mainly in North and Northeast regions, compelled some of the most prominent malarial experts in Brazil to urge for a centralized effort by the federal government to halt transmission of the disease (Griffing et al., 2015).

The National Department of Public Health (DNSP) was created in 1920 in an effort to control infectious diseases such as hookworm, Chagas disease and malaria. In a joint effort with the state governments of Rio de Janeiro and São Paulo and local-level administrations, the Rockefeller Foundation made important contributions to the study of malaria, advancing several contributions to the understanding of the type of tropical transmission of malaria in Brazil compared to the observed in the U.S. South. However, due to funding limitations and diverging views around the subject, its efforts were discontinued in 1929.

Until 1930, the dominant vector responsible for transmitting malaria was the *Anopheles darlingi*. In that year, the vector *Anopheles gambie* was introduced to Natal, a coastal city in the Northeast region, from Dakar, Senegal, most likely due the presence of adult mosquitoes on airplanes and ships. In the years that followed, the Northeast region experienced an outbreak that

[17] Compliance to the treatment were often an issue among the workers. Anecdotal evidences of the lack of compliance are numerous. For example, during the construction of the dam of the Xerém river, one worker after being fired for not taking the drug, assaulted the staff member responsible for administering the quinine and was killed the next day (Griffing et al., 2015).

stemmed from the internal migration of the vectors carried either by car or boats. During an eight-month period of 1938, 15,000 cases and 14,000 deaths were reported in the state of Rio Grande do Norte, and 40,000 infections with 20,000 deaths were reported in the state of Ceará. The following year, the Northeast Malaria Service was created to control the spread of the disease in the region. With the financial and organizational support of the Rockefeller Foundation, the service was able to reduce the number of infections by a considerable rate in a relatively short period of time. The eradication efforts relied largely on insecticides, used inside residences and transportation vehicles, and on larvicides used to treat bodies of water that served as mosquito breeding sites. The number of cases fell from 30,000 in January 1940 to 400 in September of the same year. During the 1940s, there were around 4–6 million cases of malaria among a population of 45 million people. More than half of these cases were outside the Amazon region. In 1941, it was created the National Malaria Service (SNM), which centralized the efforts to study and fight malaria in the country. The main method of control was deforestation (Griffing et al., 2015).

The insecticide Dichlorodiphenyltrichloroethane, commonly known as DDT, was first used in the anti-malarial campaign in an organized way in 1945; its success led to its widespread use in most of the states in the North region. By 1954, DDT spraying covered areas occupied by around 3 million people in all states, except São Paulo, which was incorporated into the campaign in 1959. In 1957, the SNM was renamed the Malaria Eradication Campaign (CEM), in response to the Malaria Eradication Program that the World Health Organization (WHO) had established in 1955. At this time, measures taken to control malaria also incorporate the use of chloroquine (CQ), which doctors prescribed to targeted individual patients with the disease.

In September 1965, the Brazilian federal government adopted WHO guidelines established, which consisted of spraying DDT inside the residences in infected areas, and prescribing anti-malarial drugs to patients with the disease. The CEM was successful in eliminating malaria in four out of the five Brazilian regions, with the exception of the North. An increase in the number of malaria tests and a reduction in the number of positive cases occurred simultaneously (Griffing et al., 2015). The failure of the CEM in the North region was primarily given by the resistance

of the *P. falciparum* to CQ, lack of health and social infrastructure and a large number of people susceptible to the disease who were engaged in agricultural, mining, and rubber industries. The CEM was suspended in 1970 due to criticisms with respect to the administrative organization of the campaign and also given the relatively low and concentrated number of infections.

During the 1970s, the federal government created incentives for migration into the Amazon region; in order to populate the area, the government provided subsidies for small farmers and miners, and for infrastructure projects such as the construction of roads connecting the North region with the rest of Brazil. As a result, between 1970 and 1980, around 1 million people migrated to the North region. During this time, resources devoted to malaria were largely reduced. The malaria eradication campaign integrated with other public health programs, such as the smallpox eradication program in 1971, the action against meningococcal meningitis and Chagas disease in 1975, and the campaign against leishmaniasis and leprosy in 1976. Although the vast majority of malaria cases were confined in the North region, internal migration together with the discontinuation of the programs specifically targeting malaria caused a subsequent increase in the number of infections throughout the country, which registered some 50,000 cases. Subsequent efforts were then made to eliminate or control the transmission of the disease, with localized administration of the resources dramatically reducing the number of cases outside of the Legal Amazon.

4 Data

The micro-level data for the individual-level analysis come from the annual Brazilian National Household Sample Survey (PNAD), which is designed to be representative of the demographic and socioeconomic characteristics of the Brazilian population's demographic and socioeconomic characteristics such as race, gender, education, and labor market outcomes. I use the waves from 1992 to 2015 to construct a sample of individuals aged 23 to 65; the sample includes birth cohorts from 1926 to 1999. Individuals are identified in the sample by location of birth rather than current residence. This formulation allows for a clearer interpretation of the findings, since results iden-

tifying individuals by location of residence would be contaminated by selective migration issues. Additionally, information on month and year of birth for the sampled individuals allows me to link individuals' outcomes to specific conditions at the time and location of birth.

I combine the micro-level data from various PNAD samples with state-level malaria rates to build the data set used in this paper. To construct the malaria transmission risk, I use the monthly average number of reported cases of malaria at the state level from 2007 to 2017.[18] The units of observation are expressed in terms of individual - month of exposure - year of birth - state of birth cells. The month of exposure captures the risk of malaria transmission across months of the year, whereas year of birth links individuals to the prevailing scale of transmission. Figure 1 shows the average risk of malaria transmission across states in Brazil. In the figure, each point corresponds to a different state - month observation. The figure highlights some degree of heterogeneity in transmission in both dimensions. Given the enormous geographic size of Brazil, climatic conditions and, thus, malaria transmissions, may vary substantially by state.[19]

Figure 2 displays the distribution of malaria cases across states in Brazil in 1959, the first year in which the Ministry of Health began providing state-level malaria burden reports). As the figure shows, the Legal Amazon is the area with the highest incidence; some degree of heterogeneity surfaces in the remaining states.

In addition to the monthly variation in malaria risk, I use the exogenous decline in the overall scale of transmission after the eradication efforts started in 1957. The magnitude of the reduction is shown in figure 3. The first two observations, from Griffing et al. (2015), correspond to estimates of malaria country-wide malaria cases prior to the campaign era. The remaining data points are from Ministry of Health, which records data on the number of cases reported annually, starting from 1959.

In the empirical analysis I consider individuals born shortly before and shortly after the sharp decline in malaria transmission to estimate the effects of early life exposure to malaria on socioeco-

[18] The data are provided by the Brazilian Ministry of Health – DATASUS. See Appendix A for a detailed description of the data.

[19] Spikes in the reported number of cases frequently happen during wet seasons, which vary by regions in Brazil.

nomic conditions. This corresponds to 1959 and 1960 birth cohorts. Lack of detailed information about the intensity and distribution of the transmission prior to the campaign at the state-level prevents the comparison of a larger number of cohorts.[20]

5 Research Design

The first aspect of malaria transmission that this paper explores is its seasonal variations related to specific climatic conditions. Exposure to malaria is largely affected by variations in temperature and precipitation throughout the year. Therefore, I make use of the differential timing of exposure to malaria during early periods of life to assess the long-term effects on adult socio-economic outcomes. The second feature of the research design makes use of the exogenous sharp decline in malaria transmission after the enactment of the Malaria Eradication Campaign in 1957, to assess the effects of differences in exposure of cohorts born shortly before and immediately after the reduction.

The baseline specification identifies exposure to malaria as the average risk of transmission across the months of the year and state in which the individuals were exposed while in utero. For example, consider a given individual, born in month m and state s; her exposure is given by the average risk of the months $m-1$ through $m-9$ for state s. More formally, the exposure variable is given by the total risk of transmission, defined as the sum of the average number of reported cases per 1,000 inhabitants for a given month-state cell, throughout the period analyzed. The constructed measure to represent in utero exposure of individuals born in month m and state s is given by

$$Exposure_{ms} = \sum_{k=m-9}^{m-1} \sigma_k^s,$$

where σ_k^s denotes the average risk of transmission in month k and state s. Therefore, to determine the exposure variable, I observe month of birth and identify the nine months in which each indi-

[20] In the sensitivity analysis I consider larger samples of birth cohorts to alleviate concerns related to the intensity of transmission prior to the eradication era.

vidual was exposed to malaria while in utero.[21] The baseline equation to be estimated is given by

$$Outcome_{ismy} = \beta Exposure_{ms} + \alpha_c + \gamma_s + X'_{imsy}\phi + \varepsilon_{imsy}, \tag{1}$$

where $Exposure_{ms}$ is defined as the risk of transmission at a given month of birth m and state of birth s. I estimate equation 1 for two consecutive birth cohorts: 1959 (pre-eradication) and 1960 (post-eradication). Individuals born in 1959, in general, were largely exposed to malaria transmission while in utero, while postnatal exposure was limited. As figure 3 shows, by 1959, incidence of malaria had already consistently declined. Individuals born in 1960, on the other hand, were relatively less exposed throughout their early life period. Any effect of malaria exposure during pregnancy should be seen in the estimation of equation 1 for the pre-eradication birth cohort, while the treatment effect of early life exposure on individuals members of the post-eradication birth cohort – the placebo birth cohort group – is expected to be very small (if any). The rate of transmission plummeted from an estimated 97 cases per 1,000 inhabitants per year, in 1953, to only 0.63 cases per 1,000 inhabitants per year in 1959.[22] Assuming a linear path in the yearly reduction along this period, malaria rates would have been around 16.71 cases per 1,000 inhabitants per year in 1958; this corresponds to 26.5 times the intensity of exposure in the following year.[23] Therefore, the large estimated decline in the malaria burden between these two consecutive years is of extreme relevance to compare outcomes of cohorts whose in utero exposure to malaria was dramatically different.

I abstract from the complication of partial exposure and consider all individuals members of the 1959 birth cohort to be fully exposed while in utero, while all members of birth cohort 1960

[21] Ideally, the measure of risk of malaria transmission would rely on average monthly malaria cases when exposure to malaria was greatest, that is, during the period prior to the eradication era. However, due to data limitations, monthly malaria cases can only be observed for the period 2007–2017. The implicit assumption carried out throughout the analysis, then, is that relative risk across month - state cells is constant. The implication of this assumption is that the eradication efforts did not affect the relative intensity of the monthly variation in malaria transmission. Its only effect was through the decline in the scale of overall transmission throughout the country.

[22] See figure 3.

[23] However, considering the time of personnel training, and bureaucratic delays in the initial stage of the eradication, the decline in the malaria burden from 1958 to 1959 is likely to be larger.

are considered not exposed.[24] I make this simplifying assumption for two main reasons. First, due data limitations, I cannot establish the exact reduction on malaria rates prior to the eradication era. Second, the results are robust to restricting the date of birth to specific month brackets around the years of 1959 and 1960.[25] Moreover, I use broader definitions of birth cohorts for the analysis to ensure that pre-eradication birth cohorts were indeed exposed. Specifically, I consider the effects of early life exposure for individuals born between 1957 and 1959 (or alternatively between 1958 and 1959) as the pre-eradication sample, and individuals born between 1961 and 1962 (or between 1961 and 1963) as the post-eradication sample.

In addition to estimating equation 1 separately for each birth cohort, I employ a DID strategy to ask whether more exposed individuals, according to their month and state of birth, experienced larger gains in long-run socioeconomic outcomes, compared to less exposed individuals. The DID strategy accounts for time-invariant unobservable regional characteristics, such as geographic factors, that might be correlated with the outcomes. Therefore, the second specification can be estimated by the equation

$$Outcome_{ismy} = \beta Exposure_{ms} \times Post + \alpha_c + \gamma_s + X'_{ismy} \times Post\phi + \varepsilon_{ismy}, \tag{2}$$

where the variables are expressed as before with the addition of the term $Post$, which indicates whether the individual belongs to the pre- or post-eradication birth cohort. Control variables are allowed to vary between cohorts. The β coefficient captures any relative gains in outcomes by more exposed individuals members of the post-eradication birth cohort. Suppose, for simplicity, that exposure is given by a binary treatment: exposed or not exposed. In this sense, the DID analysis would compare outcomes of both groups across birth cohorts to see whether after the decline in malaria, the treatment group (those who benefited from the campaign) exhibited relatively larger improvements in outcomes across time compared to the control group. Even though the treatment

[24] For example, individuals born at the beginning of 1959 were more exposed than individuals born at the end of that year. By the same token, individuals born in the initial months of 1960 are likely to have been exposed while in utero.

[25] However, for specifications in which I impose strong sample size restrictions, the results become imprecisely estimated, despite the large effects suggested by the estimated coefficients. Results are available upon request.

is a continuous variable in the analysis, the interpretation of the effects follows this same basic idea.

I consider risk of malaria transmission on different periods of early life to account for potential heterogeneous long-term effects on different stages of life. Conceptually, the timing of malaria infection might lead to different patterns of subsequent health conditions. Early gestational malaria, occurring within the first 18 weeks following conception, is associated with symmetric growth retardation, thereby causing permanent neurological consequences for the infant.[26] However, asymmetric growth retardations, which are associated with infections at later stages, may also also lead to ill effects, such as low birth weight, increasing the likelihood of long-term repercussions. Some authors, for example, point out to the relatively higher risk of malaria infection during the second trimester of pregnancy, a critical period of gestation (Singh et al., 1999; Desai et al., 2007). Moreover, Luxemburger et al. (2001) find low birth weight, and other sequelae, after infections during the third trimester of pregnancy. However, epidemiological studies addressing the consequences of timing of infection during pregnancy lack longitudinal observations on parasite prevalence throughout pregnancy (Umbers et al., 2011). The research design followed by this paper, thus, remains agnostic as to what are the most critical periods of exposure to malaria and explores the long-term effects on a range of different early life periods.

In light of the potential heterogeneous treatment effects during different periods of early life, I employ the same strategy to define early life exposure at the trimester level. In particular, I construct an exposure variable for each of the three trimesters of pregnancy as the average risk of transmission during the corresponding three months of exposure as

$$Exposure_{ms}^{T} = \sum_{i=m-k}^{m-n} \sigma_i^s,$$

where $k = 9, 6, 3$ and $n = 7, 4, 1$ whenever $T = 1, 2, 3$, respectively. The trimester exposure

[26] Epidemiological studies have found severe consequences for later-life health after deteriorations of initial health stock. Ravelli et al. (1976) found that exposure to famine on the first trimester of pregnancy leads to large effects on obesity rates after reaching adulthood. Moreover, McAlister (2007) has found long-term defects such as heart disease and deafness after pregnant women are infected with Rubella during the first trimester.

analysis, then, can be represented by

$$Outcome_{ismy} = \beta Exposure_{ms}^{T} + \alpha_c + \gamma_s + X'_{imsy}\phi + \varepsilon_{imsy}, \tag{3}$$

where the variable definitions are as above. I estimate equation 3 separately for each trimester of exposure to address the effects of malaria exposure. Additionally, I compare long-term socioeconomic outcomes of both pre- and post-eradication cohorts by estimating a DID model along the lines of equation 2 above.

The empirical strategy described above has some limitations that are worth mentioning. First, I cannot specifically ascertain which specific mechanisms are being captured by the results. Since I do not observe the individual-level incidence of malaria, the effects might be through infections of the parents, relatives, or neighbors. The identification strategy can, thus, be characterized as an intent-to-treat analysis. Additionally, general equilibrium effects might also be at play. For instance, assuming that decline in malaria rates lead to higher human capital, the rate of return to these investments would increase among more exposed individuals. These effects might also spill over to non-exposed individuals. The net general equilibrium effect in this situation is uncertain. Third, malaria infections might cause the death of the weakest individuals, causing a selection problem in the analysis, with the parameters being estimated with an upward bias. I postpone the discussion of mortality selection to section 6.3.

6 Results and Discussion

In general, average socioeconomic outcomes across pre- and post-eradication samples are similar. Table 1 shows the descriptive statistics of both periods, broken down by gender. Women accumulate more education than men in both periods, whereas men attain consistently higher incomes. The average number of children is 2.69 in the 1959 sample and 2.64 in the 1960 sample, as shown in panel A. Panel B displays demographic characteristics such as race, fraction of migrants, and fraction of individuals residing in rural areas. The table shows consistent numbers across gender

and cohorts. Panel C presents average information about state infant mortality and GDP per capita.

The results of the estimation are shown below according to different periods of exposure to malaria. The first set of results addresses in utero exposure as the average risk of malaria transmission throughout the pregnancy period. Next, I address timing of exposure in different trimesters of the pregnancy to account for heterogeneous effects of exposure in different critical periods of gestation. Finally, I consider a range of potential issues that might lead to inconsistent estimates of my main specification.

6.1 Baseline Results

The first analysis of the connection between early life exposure to malaria and long-term adult outcomes compares cohorts born shortly before and shortly after the large decline in malaria rates throughout the country. If early life exposure to malaria affects long-term outcomes, the baseline estimation in equation 1 should display stronger effects on the pre period. Since the sample is restricted to only two consecutive birth cohorts, any differences in long-term outcomes can be attributed to the effects of changes in the early-life environment.

Panel A of table 2 shows the estimation results for the 1959 birth cohort sample. All coefficients are from regressions using the full set of controls and state-of-birth fixed effects. The results suggest a strong and statistically significant effect of early life exposure to malaria on educational attainment for the pre-eradication birth cohort sample. Individuals in utero in months with higher risk of exposure to malaria, on average, have less education than less-exposed individuals during the same period. The long-term effects of early life exposure on income are somewhat less precise, but still statistically significant and negative. In utero exposure does not seem to have any effect on fertility. Panel B provides the same estimation carried out in Panel A for the 1960 birth cohort. Individuals born in the subsequent year, when malaria rates have largely declined, do not show any statistically significant differences in any of the analyzed outcomes according to their early life exposure, as expected. By 1960, malaria rates had already reached their lowest levels, causing no effect on socioeconomic outcomes. In panel C, a DID design compares the gains in outcomes

for the pre- and post-eradication birth cohorts. In particular, I interact the treatment variable – in utero exposure – with a dummy variable $Post$ indicating whether the individual belongs to the post-eradication birth cohort. The results show that more-exposed cohorts born in the post period saw larger gains in years of education compared to less-exposed cohorts. The results in column (2) show positive, though not statistically significant, gains in income for the more-exposed cohorts born in the post-eradication period. The effects on fertility are negative but also statistically significant.

In general, the results suggest long-term effects of in utero exposure to malaria on educational attainment and income for the cohorts born before the eradication era. The effects seem to stem from exposure during gestation; otherwise we would see similar results for the 1960 birth cohort. The results further indicate that the gains in education from the more-exposed cohorts were not followed by any gains in income. Moreover, women more exposed to malaria during early life do not exhibit any differences in terms of fertility, compared to the non-exposed women.

6.2 Trimester of Exposure Results

According to some epidemiological studies, infections in different timing of fetal development might lead to different paths of effects on cognition and health development (Smith, 2004; Menendez, 1995; Matteelli et al., 1997; Umbers et al., 2011). In this section, I consider exposure in all three trimesters of pregnancy to check for heterogeneous effects of timing of exposure. Table 3 shows the estimation of equation 1 for exposure in each trimester of pregnancy for both samples (1959 and 1960). The results indicate a strong effect of exposure during the first three months of pregnancy on the long-run outcomes in the 1959 birth cohort. In general, higher in utero exposure to malaria during first trimester leads to lower educational attainment, lower income, and higher fertility.[27] The point estimates are qualitatively similar to the ones obtained in the previous results for the overall in utero exposure. The treatment effect is somewhat smaller for the educational

[27]The positive effect on fertility might be explained by the lower opportunity costs associated with the reduced achievements in the labor market.

attainment and roughly the same for income. The fertility result, however, indicates that exposure to malaria early in gestation might have long-term consequences. In fact, fetal growth restrictions caused by malaria infections might have more severe consequences during the first trimester of pregnancy, when the impairment of fetal development is made in a symmetric manner. Fetuses with symmetric growth restrictions are more likely to develop permanent neurological sequalae (Umbers et al., 2011).

Exposure to malaria in the second trimester of pregnancy seems to also have negative impact on education attainment, albeit a smaller treatment effect when compared to exposure during the first trimester. The results do not indicate any effects of on income or fertility linked to infections during the second trimester. My second-trimester results are in line with Desai et al. (2007) and Singh et al. (1999), who conclude that malaria infections during the second trimester may lead to greater likelihood of low birth weights and pre-mature births. Additionally, third trimester malaria exposure does not seem to be associated with long-term consequences for any of the analyzed outcomes. The results for the post eradication sample indicate no exposure effects on socioeconomic outcomes, except for income, during the first trimester, as shown in column 5 of table 3.[28]

Next, I analyze the DID results by trimester of exposure. Again, the results indicate that the effects of changes in the malaria burden are more pronounced during first periods of intrauterine life. Table 4 provides the results of equation 2 for each trimester. Relative to less exposure, more-exposed individuals during first and second trimesters of pregnancy, born on post-eradication cohort, experienced larger gains in educational attainment compared to the pre-eradication cohort.[29] Moreover, the DID results suggest no relative gains in income for the more-exposed individuals members of the post-eradication birth cohort. Finally, the fertility DID analysis shows a larger decline in number of children for the more-exposed cohort compared to less exposure.

The analysis carried out in this and in the previous sections suggests that early life exposure to malaria might lead to long-term consequences on adult socioeconomic outcomes. Although the

[28]This result might seem puzzling at first, but as I discuss in section 6.3.5, some groups of people born in 1960 might display some degree of residual early-life exposure, given that reductions in malaria burden were not instantaneous (although very rapidly).

[29]The second trimester education results are only significant at the 10 percent level.

exact mechanism of this relationship is not explicitly tested, it is clear that shocks to the initial health endowment cause long-term health problems. Poor adult health might seem the main channel through which early life malaria exposure affect educational attainment, income and fertility decisions.

6.3 Sensitivity Analysis

In this section, I discuss some of the possible alternative answers or mechanisms that might lead to the results obtained in the previous section. First, I separate the effects of early life exposure to malaria on men and women. Second, I address the issue of mortality selection by analyzing the exposure effects on the likelihood of stillbirths for childbearing-age women during the period in which the disease was endemic. A different possible bias in the previous section's results relates to selection in migration patterns across states in Brazil. To address this issue, I separately estimate the exposure effects on two subsamples: movers and non-movers. I also address the issue of endogeneity in the exposure variable by instrumenting the risk of infection with specific climatic conditions necessary for malaria outbreaks to occur. Finally, I test for different timings of birth in defining exposure across cohorts.

6.3.1 Men vs. Women

Are there any differences in the long-run effects of early life exposure to malaria between men and women? Some studies in the literature on long-run impacts of early life health have noted systematic heterogeneous effects across genders (Almond and Mazumder, 2011; Banerjee et al., 2010; Cutler et al., 2010; Bhalotra and Venkataramani, 2015; Venkataramani, 2012).

In order to address this issue, I estimate equations 1 and 2 separately for men and women. Results are displayed in table 5. Consistent with previous findings, I do observe stronger and more precisely estimated effects for men when considering in utero exposure for the 1959 birth cohort, in panel A. For the placebo birth cohort, in panel B, no treatment effects, either on education or income, are observed for ether men or women. Relative gains in education are seen, however, for

both gender groups, in panel C. Relative to the less exposed, members of the 1960 birth cohort who were relatively more exposed to malaria, on average, spend more time in school, compared to the previous birth cohort. The results do not indicate any relative difference in income or fertility for either men or women across birth cohorts.[30]

A possible explanation for why I find statistically significant results only for men in the fixed effects model is that male fetuses are biologically more vulnerable than female ones. Thus, higher exposure of male individuals would lead to a higher likelihood of greater morbidity and larger impacts on subsequent health. This hypothesis is consistent with medical findings, suggesting stronger negative health shocks effects on men versus women (Kraemer, 2000; Low, 2015; Waldron, 1983).[31]

As for the DID model results, notice that women spend more years in school than men, on average (see table 1). If the returns to human capital are higher for women in the labor market, the results that the educational benefits from the eradication of malaria are stronger for women, as shown in panel C, are consistent with the hypothesis that women benefited relatively more from the decline in the overall exposure to malaria transmission.

6.3.2 Selective Mortality

If the most vulnerable and weakest individuals do not survive in utero exposure to malaria, the previous results are potentially biased due to selection on mortality. Early life exposure to health shocks affect both the unobserved distribution of initial health endowments and the health threshold level at which survival to infancy occurs. Whenever mortality increases due to negative health shocks (such as early life exposure to malaria), the initial health distribution shifts, which implies lower lifetime health. However, when the health shocks lead to greater fetal mortality, then the implication for general subsequent health is the opposite: the fetuses that survive to infancy will likely be healthier than if the in utero health shock had not caused greater mortality rates. If the

[30] Notice that the fertility results are the same as the ones observed and discussed in table 1, since the data only reports fertility outcomes among women.

[31] Venkataramani (2012) discusses his findings of stronger effects of malaria on cognition for men in Mexico in light of this hypothesis.

latter effect prevails, the results would be biased against my estimation results.

The concern that mortality selection is a potential source of bias on my results are alleviated by the fact that the most prevalent parasite in Brazil, the *P. vixax*, rarely causes death, compared to the *P. falciparum*, which is commonly found in Sub-Saharan Africa.[32] I address the issue more formally by testing whether more exposed women at childbearing age during the pre-eradication era had higher likelihood of miscarriages or stillbirths. In particular, I use number of deceased children while in utero as the dependent variable, and I use a state-level ecology-based index[33] that captures the stability and strength of malaria transmission as the main independent variable.[34] The results show no difference between in utero mortality across different levels of exposure to malaria. I further test for differential effects on male and female fetuses, and for the probability of stillbirths associated with different malaria risks. The results indicate no relationship between malaria burden and mortality differences among women who experienced greater or lesser levels of exposure to the disease.[35]

6.3.3 Selective Migration

Another potential source of bias might be attributed to selection on migratory patterns in Brazil. It is possible that the most privileged (high-skill) individuals migrate to areas in which the malaria burden is low. I rule out this potential effect in my analysis because I identify individuals according to their location of birth rather than to their location of residence. To more formally address this potential bias, I construct two individual-level datasets for movers and non-movers and estimate the effects of early life exposure to malaria separately for each sample.

Table 6 contains the OLS results for the 1959 and 1960 birth cohorts, as well as DID estimations comparing differences in outcomes pre- and post-eradication periods. Panel A shows a negative

[32] *P. falciparum* is also common in the Amazon region in Brazil. However, this is not a concern since I drop Legal Amazon states from the analysis.

[33] See Appendix A for further details on the construction of the malaria index.

[34] The estimation uses the same set of controls as in the other results, with the lone difference being that I use a higher-order birth cohorts in the sample. Particularly, I consider female individuals born prior to 1944, which indicates being at a childbearing age during pre-eradication. I am able, therefore, to control for state-level fixed effects, as well as for birth-cohort fixed effects and linear trends.

[35] Selective mortality results are available upon request.

and statistically significant treatment effect on educational attainment for both movers and non-movers born in 1959 (although the effect is somewhat stronger for the mover sample). Moreover, the treatment effect on income and fertility only seem to be significant for the non-mover sample. As expected, I do not find any treatment effects on the long-run outcomes for the placebo cohort, as shown in panel B.

In panel C, the DID estimation shows qualitatively similar results across movers and non-movers. The educational attainment gains for more-exposed individuals compared to less-exposed was higher among the movers. Moreover, I find no associated gains in income on either sample. Finally, the larger decline in fertility after the reduction of malaria burden among the more exposed cohorts is observed among movers.

In general, the results described in this section, allied to the fact that I observe individuals' state of birth rather than residence, suggest that selective mortality is not a first-order factor driving the results. Although the treatment effects are larger for the movers sample in some specifications, the qualitative implications of both estimations are similar.

6.3.4 Endogeneity of Exposure: Instrumental Variables Estimation

One potential concern in my analysis is that the constructed exposure variable may suffer from measurement error, and thereby may not be able to fully account for truly exogenous variations in the risk of malaria transmission. One example of a possible bias resulting from measurement error is that higher-income states are more capable of diagnosing and reporting malaria than lower-income states with poorer health institutions. Alternatively, malaria can be misreported when mistakenly diagnosed as a different disease.[36] If this underreporting is associated with regional characteristics, such as average educational levels, my results would suffer from attenuation bias. In this section I discuss the sensitivity of my results to such potential measurement error by instrumenting the risk of malaria transmission with specific climatic factors that affect the distribution and survival of both the mosquito and parasite of malaria.[37]

[36] The most common symptoms of malaria are similar to other diseases caused by viruses, such as the Flu.

[37] If the measurement error is classical, the instrumental variables approach will produce consistent estimates.

I follow Tanser et al. (2003) and identify short-term exposure to malaria as a function of monthly climatic variations, which reflect the intrinsic relationship between vector and parasite development with temperature and precipitation. Malaria transmission is highly sensitive to climatic variations within the year. Warm temperatures are needed for survival of both parasite and vector. Additionally, rainfall augments opportunities for the proliferation of the *Anopheles* mosquitoes by creating breeding sites where eggs can be deposited.[38]

I identify months in which malaria transmissions are likely the highest throughout the year according to climatic variations that satisfy the following conditions:[39]

1. Average monthly rainfall in the last three months is at least $60mm/m^2$;
2. Rainfall in at least one of the past three months is at least $80mm/m^2$;
3. The average monthly temperature in the past three months exceeds $19.5°C+$ Standard Deviation of average temperature in the past 12 months.

The Two Stage Least Squares (2SLS) estimation procedure for each trimester of exposure results is constructed as follows: In the first stage, I estimate the relationship between exposure, as measured by the monthly risk of malaria transmission, and a set of variables indicating whether each month of exposure satisfies each of the above conditions.[40] The first stage relationship between malaria transmission risk and climatic conditions can be expressed as

[38] See Section 3 for further discussion on the relationship between short-term malaria outbreaks and climatic variation.

[39] Cervellati et al. (2017), in a recent paper, explores the causal effects of short-term malaria outbreaks on the escalation of violent conflicts within Sub-Saharan Africa using the same conditions as above, with the inclusion of the additional restriction that the average temperature does not fall below $5°C$ in the previous 12 months. However, this restriction is non-binding for all data points in my sample.

[40] I forgo the in overall in utero exposure analysis since the required number of exogenous regressors and excluded instruments for such analysis would be greater than the number of clusters of the standard errors, which implies that the usual overidentification tests and the first stage F-statistics cannot be computed, since the covariance matrix of orthogonality conditions would not have full rank.

$$
\begin{aligned}
Exposure^{T}_{sm} = \alpha + \beta_1 I^{s}_{1m-k} + \beta_2 I^{s}_{1m-k-1} + \beta_3 I^{s}_{1m-k-2} \\
+\gamma_1 I^{s}_{2m-k} + \gamma_2 I^{s}_{2m-k-1} + \gamma_3 I^{s}_{2m-k-2} \\
+\psi_1 I^{s}_{3m-k} + \psi_2 I^{s}_{3m-k-1} + \psi_3 I^{s}_{3m-k-2}
\end{aligned} \tag{4}
$$

where, as before, I define $T = 1, 2, 3$ to represent the trimesters of exposure, and $k = 9, 6, 3$ whenever $T = 1, 2, 3$, respectively. I^{s}_{1}, I^{s}_{2}, and I^{s}_{3} are indicator variables that denote whether conditions 1, 2, and 3 above are satisfied for each month of exposure and state s, respectively.[41] The second stage regression equation is given by

$$
Outcome_{ismy} = \beta \widehat{Exposure}^{T}_{ms} + \alpha_c + \gamma_s + X'_{imsy}\phi + \varepsilon_{imsy}, \tag{5}
$$

, where the variables in the above equation are given as before, and $\widehat{Exposure}^{T}_{ms}$ is given by the estimated relationship in equation 5. I also estimate equation 2 by 2SLS, using the climatic variables described above as excluded instruments. Particularly, I interact each excluded instrument with the indicator $Post$ in the first stage. The second stage is estimated by using predicted $Exposure$ obtained in the first stage.[42]

Table 7 describes the second stage results of the IV estimation. I provide the OLS results from previous sections in order to compare the differences in the estimated coefficients. The 1959 sample results are shown in columns 1 through 6. In controlling for measurement error in the dependent variable, the 2SLS estimation produces more significant treatment effects for first-trimester exposure on educational attainment, which is measured by years of education. The estimated coefficient is larger in magnitude than simple OLS estimates and statistically significant at the 1 percent confidence level. This result provides further support for the observations that first-trimester exposure might cause long-lasting consequences. Despite OLS estimates suggest significant treatment

[41] In addition to the specified monthly climatic variables, the first stage estimation adds the full control variables set and state fixed effects as in the OLS estimation discussed above.

[42] Appendix B provides the results of the first stage estimation as well as reduced form estimates, relating outcome variables and excluded instruments.

effects of first-trimester exposure on both income and fertility, the 2SLS results do not seem to corroborate these results. It is possible that OLS estimates are capturing some of the misreporting effects or regional unobserved characteristics associated with the health environment. However, I then conduct the analysis for different income percentiles to capture heterogeneous effects of exposure on the income distribution; this analysis shows negative and statistically significant effects for bottom 10 percent, suggesting that, among low-income families, exposure to malaria during the first trimester of pregnancy leads to long-term effects on income. In particular, restricting the sample to the bottom 10 percent of the income distribution, the point estimate of the first trimester exposure effect on personal income is -28.860, with p-value 0.044. The 2SLS results, furthermore, suggests no significant effect on the remaining outcomes of the analysis as the result of in utero exposure during the second or third trimester.

Columns 7 through 12 of table 7 shows the effects of in utero exposure for the 1960 (placebo) sample. Contrary to the OLS results, the 2SLS estimates indicate a statistically significant negative treatment effect on education and income, which potentially contradicts the previous findings that individuals in the post-eradication sample were not exposed to the treatment effect because malaria had declined precipitously in 1959. However, notice that individuals born in 1960 could potentially be somewhat exposed to higher malaria, depending on the distribution of the birth dates across individuals from that sample. Moreover, the point estimates are consistently smaller and less significant than those from the 1959 sample. To address this issue more precisely, I re-estimate the exposure effects for a different sample, considering individuals who were born in subsequent years, 1961 and 1962; this guarantees no overlap in the scale of different exposure. The results validate the previous findings, indicating no effect of early life exposure on long-term outcomes.[43] For each of the regression results displayed in table 7 I present the F statistics of the first stage to assess the inclusion restriction assumption of the IV model. For all estimations, the F statistics are large enough to reject the hypothesis of weak instruments.[44]

Table 8 provides the DID results for trimester of exposure, using the climatic variables as

[43] Results are available upon request.

[44] All regressions F statistics are above the Stock and Yogo (2005) critical value for a test of maximal size 0.05.

excluded instruments. Once again, I provide OLS estimates to compare with the 2SLS results. I find no gains in socioeconomic outcomes by exposed individuals born in the post eradication year compared to individuals born in pre-eradication era.

Collectively, the results of this section further underscores the long-term effects of in utero exposure to malaria on human capital accumulation. The findings suggests that the timing of exposure is paramount, and that the critical period of exposure is very early, during the initial three months of fetal development. The strong effects observed on educational attainment might suggest that first-trimester infections, by delaying or hampering brain development, might affect cognition, leading to worse educational achievement. The observed treatment effect of early life exposure to malaria only on lower-income groups can partially be explained by the fact that higher-income individuals have better opportunities and resources to help compensate for the potential detrimental effects of the exposure. On a theoretical note, Bleakley (2010a) points out that the economic decisions about human capital investments depend on both the discounted benefits of one additional year in school (higher adult productivity) and costs (either in the form of implicit opportunity costs or forgone wages in the labor market). Lower levels of initial physical health endowments or cognitive abilities might negatively affect both benefits. That is they could lead to both a decline in productivity due to lower health, and to a decline in opportunity cost of schooling, due, perhaps, to a decline in the potential forgone wages. The net effect is ambiguous, in theory. Thus, the effect of the health shock on education and income might not be associated.

6.3.5 Exploring Different Timing of Birth

Throughout the analysis above, I assume full exposure for individuals born in 1959 and no exposure for individuals born in 1960. However, since timing of birth is a continuous variable, I might be misclassifying individuals according to the degree of exposure.[45] Data limitations preclude such analysis, given the disparity in the frequency of the exposure (yearly) and individual-level birth dates observations (daily). Although I am not able to correctly identify individuals' exposure

[45]This is probably the case, since I still find some treatment effect on the IV estimation for the 1960 birth cohort sample.

according to their exact timing of birth, the data allow me to test different ranges of birth dates for categorizing early life exposure. For instance, in one of the specifications I consider the (fully exposed) sample of those individuals born before the last quarter of 1959, which assures no postnatal exposure in that year. The results for this specification suggest stronger and statistically significant treatment effect of early life exposure. Additionally, testing for different cohorts (such as 1961, 1962, etc.) as the placebo treatment does not alter the main results of no treatment effects.

One might also be skeptical about the choice of the year of 1959 to identify the pre-eradication birth cohort, as it is possible that the effects of the campaign have happened prior to that year. If that is the case, my results above would be capturing a different source of negative shock, not malaria, on early life exposure. To address this issue, I add older cohorts to the pre-eradication sample to ensure that the effects observed for the 1959 birth cohort are indeed due to malaria exposure, given that the malaria burden before 1959 was likely to be more pronounced. By the same token, I add younger cohorts to the post-eradication sample to establish lower levels of exposure at the other end. In particular, I consider as the exposed cohort those individuals born between 1958 and 1959 (or between 1957 and 1959) and the non-exposed cohort those individuals born between 1961 and 1962 (or between 1961 and 1963). This particularly narrow choice of years is due to the fact that broader birth year ranges would likely be contaminated by changes in the economic environment.[46]

The OLS results for educational attainment and fertility choices considering this broader birth cohort sample do not contradict the main qualitative implications of the baseline specification. The treatment effects on income, however, seem to be significant only for the bottom quartile of the income distribution. Moreover, the IV results become imprecisely estimated and the magnitude of the coefficients drops to values similar to the OLS estimates. Further analysis is needed to assess the robustness of the findings to longer horizon estimates.

[46]For example, in 1964, Brazil underwent a series of political events that led to a military coup d'état, which affected society in ways that would likely be correlated with the outcome variables in the analysis.

6.4 Discussion and Interpretation of the Findings

An important aspect of the identification strategy in the present paper is the precise and clear definition of early life exposure, in terms of the relative frequency of reported number of malaria cases. This specific representation of exposure allows for an interpretation of the coefficients in terms of direct probability of treatment or risk of being infected with malaria.[47]

Although my early life exposure variable carries a straightforward probability meaning for the interpretation of the above results, notice that it only captures the relative risk of malaria across month-state cells. In order to directly interpret the coefficients of the above results, and to assess the magnitude of the effects, one might need to adjust for the scale of the transmission in different periods. However, as discussed above, lack of detailed information on the period analyzed does not allow for a direct assessment of such magnitude. Throughout the analysis, I assume that relative malaria incidence across months and states are constant. This assumption does not seem to be unrealistic, since within-year malaria variations are mostly determined by seasonal climatic conditions, which are fairly predictable through time.

The overall results suggest that exposure to malaria during early life causes long-term consequences for adult outcomes. In the baseline specification, OLS results indicate a negative effect of in utero exposure to malaria on years of education and personal income on exposed cohorts (born in 1959). The point estimate for the effect of in utero exposure on educational attainment is -20.77. What does this number represent? The difference between the ninety-fifth and fifth percentiles of the in utero risk is 0.089. An increase in malaria risk of this magnitude would induce an effect of 0.089 times the estimated coefficient. Therefore, an increase in exposure, as measured by the risk of infection, from the fifth percentile to the ninety-fifth percentile of risk distribution leads to, on average, 1.858 fewer years of education. The effect might vary between -0.766 and -2.950, considering the interval estimation of the coefficient(-32.986 and -8.563). Carrying out the same computations for the income coefficient (-2.712), an increase of malaria risk from the fifth

[47]Most studies, in the absence of endemicity data, use proxies such as malaria indexes capturing the geographic distribution of malaria risk (Bleakley, 2010b; Cutler et al., 2010). Estimated coefficients in these settings poses a challenge to the interpretation of the obtained results.

percentile to the ninety-fifth percentile of the risk distribution would generate a loss between 5.3 percent and 43.1 percent in hourly personal income.

The results also indicate the importance of timing of in utero exposure for long-term outcomes for the individuals members of the exposed birth cohort. In particular, exposure during the first trimester of pregnancy affects education, income, and fertility, according to the OLS estimates. How large are these effects? Again, using the above procedure, OLS results indicate that a rise in risk of exposure from the fifth percentile (0.003) to the ninety-fifth percentile (0.036) induces a decrease in education of between 0.064 and 0.996 years, and a reduction in personal hourly income of between 1.4 percent and 16.5 percent. Among women, an increase of 0.032 (fifth percentile to ninety-fifth percentile) first-semester risk of exposure leads to an average increase in the number of children between 0.0149 and 0.149. Accounting for attenuation bias caused by measurement error in the exposure measure, the instrumental variables estimation suggests similar treatment effects of first trimester exposure on educational attainment: an increase in exposure from the fifth percentile to the ninety-fifth percentile in risk of transmission causes, on average, a reduction of between 0.399 and 2.736 years of education.

The strategy employed to estimate the long-run effects of early life exposure to malaria can be related to the one employed by Barreca (2010) to assess the long-run effects of malaria exposure in United States prior to the eradication efforts; he also targets in utero exposure, and he explores climatic variations on malaria transmission. His findings suggest that 10 additional malaria deaths per 1,000 inhabitants cause a reduction of 0.4 in years of education and 13 percent in income for the exposed individuals; these findings are fairly consistent with the range of my estimates for the first-trimester effects. Despite some similarities between the strategies, there are some important differences that suggest caution when comparing our results. First, he uses yearly malaria deaths as a measure of malaria incidence, whereas I rely on monthly malaria reported cases to employ a narrower definition of exposure. Therefore, my results complement his findings in suggesting that exposure during the first trimester carries the most serious long-term development risk.

The second approach compares outcomes of individuals born in pre- and post-eradication years

according to exposure degree. Although OLS estimates suggest positive treatment effects on education and negative treatment effects on fertility, instrumental variables estimations call into question the validity of these results. Thus, the findings of this paper suggest no considerable improvements in socioeconomic conditions for those born after eradication compared to individuals born before eradication. The pre- and post-eradication comparison shares some similarities with the strategy employed by Bleakley (2010b), Cutler et al. (2010), Lucas (2010, 2013), Burlando (2012), and Venkataramani (2012). All of them explore the effects of malaria reduction caused by eradication campaigns. In my specification, I add another layer to the exposure measure, month of birth, to account for differences in pre-trends prior to the treatment. In general, these studies find significant gains in (personal or household) income and mixed results for educational attainment. After accounting for seasonal exposure, my estimates do not seem to confirm previous findings, as I do not observe any significant treatment effect in my sample.[48]

7 Conclusions

Differences in initial health stocks across individuals and locations are associated with lifetime socioeconomic conditions. Causal interpretation of this connection requires a carefully designed research identification strategy, since unobserved socioeconomic differences across different groups of people might otherwhise overstate the relative importance of early life health. From a theoretical perspective, initial health might affect the path of health capital, which determine different dimensions of one's capabilities, such as educational attainment, labor productivity, and fertility. Establishing and quantifying this relationship might help policymakers in the elaboration of developmental policies to improve living standards in areas in which adverse health environment, such as the presence of infectious diseases, exposure to pollution, or a lack of basic sanitation, might

[48]However, this lack of treatment effects does not contradict the results previously found in the literature. First, I consider the effects of exposure in the months prior to birth, whereas the other studies consider exposure to malaria during infancy or adolescence. Second, as discussed above, I explore timing of birth within treated and non-treated cohorts. whereas previous studies consider a single dimension of exposure, which is being born prior to or after the eradication era. Finally, I only compare two consecutive birth cohorts (in most specifications) in order to address the in utero exposure effects, while previous studies construct a higher order cohort-based sample.

disproportionately affect highly exposed pregnant women and individuals during their early life.

This paper explores two sources of variations in malaria risk in Brazil as proxies for exogenous shocks on initial health, which affect otherwise similar individuals in heterogeneous ways. The first source of variation relies on the month of birth to identify degrees of exposure according to seasonal variations in malaria transmission risk. Individuals who are potentially at greatest risk of experiencing long-term effects are those whose gestation periods that encompassed a longer period with climatic conditions favoring mosquito and vector proliferation; by contrast, individuals in the same locations but whose gestation period encompassed a shorter period of such conditions face comparatively lower risks of long-term effects. The second source of variation is based on the large and rapid overall decline in malaria burden in Brazil after governmental eradication efforts.

The results found on this paper suggest that initial health plays an important role in adult human capital accumulation and income. In practical terms, accounting for other factors, such as demographic characteristics and state-level attributes, a given individual born in either January or December in the state of Paraíba, in the Northeast region (fifth percentile of the malaria risk distribution), would attain roughly three more years of education, and earn an income that is 40 percent higher than an individual born in April in the State of Goiás, Mid-West (ninety-fifth percentile of the malaria risk distribution).

An additional feature of the findings is that first trimester of pregnancy seems to be the most critical period; malaria infections that occur in this period have the largest effects. One possible theoretical explanation for this result is that fetal growth restrictions during the very beginning of life affect nervous system development in a very strong and permanent manner. Therefore, policies devoted to improve socioeconomic conditions in afflicted areas should target pregnant women, specially those in early stages of gestation.

Finally, although the present paper consistently finds negative long-term effects of early life exposure among the birth cohort born before Brazil's malaria eradication campaign, it does not find significant gains in outcomes among pre- and post-eradication cohorts according to their degree of exposure. It seems that the differences in outcomes across consecutive cohorts (1959 and 1960)

are not sufficient to identify any substantial relative gains from treated individuals. Moreover, estimation results for older cohorts do not seem to robustly confirm the results for personal income.

Further research in this area might help shed light on the specific mechanisms that lead to the results obtained by this paper. For example, one might estimate the in utero effects of exposure to malaria transmission on general and specific health, such as likelihood of developing different morbidities, in adulthood. Moreover, since the effects of malaria on educational attainment are thought to be the result of impaired cognitive development, one could potentially observe effects of exposure on cognition test scores across school-aged children. Finally, testing the specific relationship of this paper in different contexts, with different data sets and a range of different outcome variables might help testing the external validity of this study.

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Figures

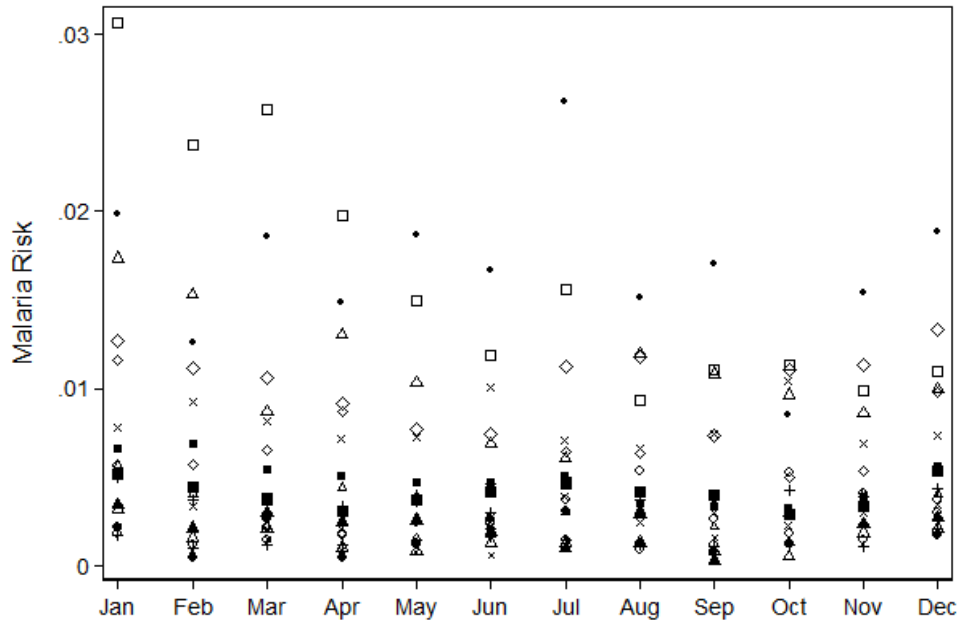

Figure 1: State-Level Monthly Average Malaria Cases per 1,000 inhabitants (2007–2017)

Source: DATASUS – Ministry of Health

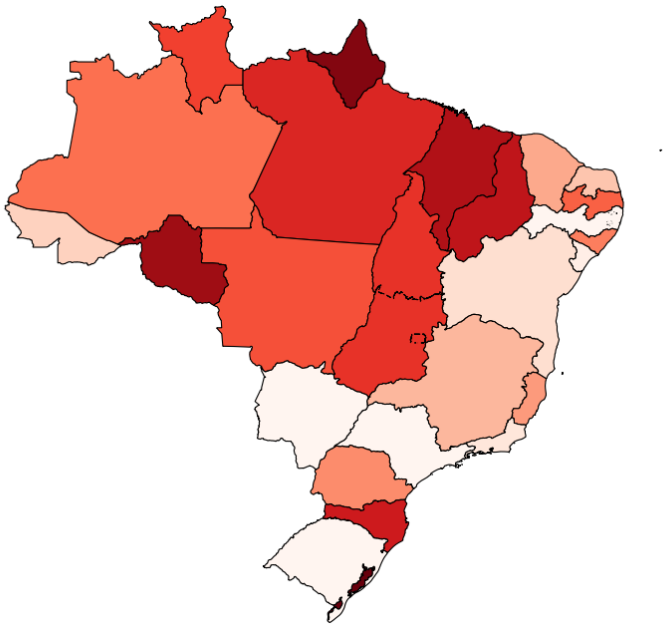

Figure 2: State-Level Average Malaria Cases – 1959

Source: DATASUS – Ministry of Health

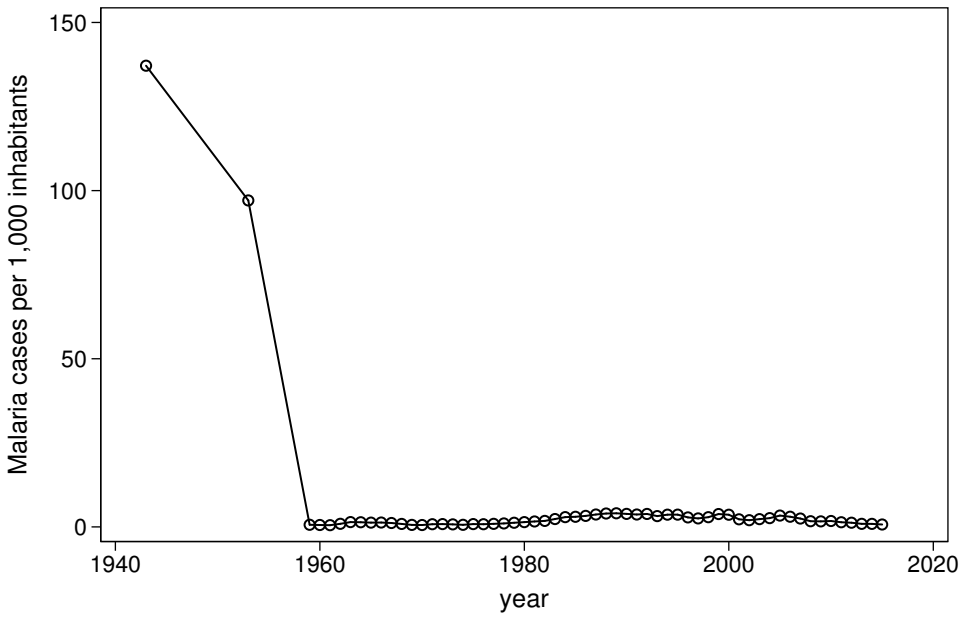

Figure 3: State-Level Monthly Average Malaria Cases per 1,000 inhabitants (2007–2017)

Source: DATASUS – Ministry of Health and Griffing et al. (2015).

Tables

Table 1: Summary Statistics

	Pre-Eradication Sample (1959)				Post-Eradication Sample (1960)			
	Men		Women		Men		Women	
	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.
Panel A. Outcome variables								
Years of Education	6.78	4.53	7.54	4.72	6.75	4.54	7.53	4.70
Income	9.86	16.02	7.06	12.51	9.40	15.54	6.73	11.40
Fertility	.	.	2.69	2.14	.	.	2.64	2.11
Panel B. Demographic Characteristics								
White	0.55	0.50	0.56	0.50	0.54	0.50	0.55	0.50
Indigenous	0.00	0.05	0.00	0.05	0.00	0.04	0.00	0.05
Black	0.07	0.25	0.07	0.26	0.07	0.26	0.07	0.25
Brown	0.38	0.48	0.36	0.48	0.38	0.49	0.38	0.48
Mover	0.23	0.42	0.22	0.41	0.22	v0.42	0.22	0.41
Rural	0.17	0.38	0.16	0.36	0.18	0.39	0.16	0.37
Panel C. State-level Controls								
Infant Mortality	0.25	0.10	0.25	0.10	0.25	0.10	0.25	0.09
GDP per capita	15.68	6.79	15.49	6.77	15.47	6.90	15.35	6.90
Number of Observations	36421		28124		40806		31509	
Number of States	18							

Notes. Means and standard deviations are weighted using sample weights. Sample includes Brazilian residents aged 23–65 and excludes individuals born in Legal Amazon states. Panel A reports average and standard deviation of outcome variables in the 1959 and 1960 birth cohorts. Panel B reports demographic characteristics of the sample, including race, the proportion of migrants, and the proportion of individuals living in rural areas.

Table 2: Early Life Exposure to Malaria OLS Results – In utero Exposure

	Education (1)	Income (2)	Fertility (3)
In utero Exposure			
	Panel A. Pre-Eradication Sample (1959)		
Treatment Effect	-20.77^{***}	-2.712^{**}	5.003
	(5.812)	(1.007)	(3.140)
Observations	29,677	27,936	13,115
R-squared	0.209	0.183	0.130
Full Set of Controls	Yes	Yes	Yes
State Fixed Effects	Yes	Yes	Yes
Linear Trends	No	No	No
	Panel B. Post-Eradication Sample (1960)		
Treatment Effect	1.602	-1.608	-7.154^{*}
	(6.938)	(1.377)	(3.483)
Observations	33,116	31,127	14,743
R-squared	0.209	0.194	0.120
Full Set of Controls	Yes	Yes	Yes
State Fixed Effects	Yes	Yes	Yes
Linear Trends	No	No	No
	Panel C. Differences-in-Differences Results		
Treatment Effect×Post	22.38^{***}	1.102	-12.16^{**}
	(7.376)	(1.203)	(4.486)
Observations	62,793	59,063	27,828
R-squared	0.209	0.189	0.125
Full Set of Controls	Yes	Yes	Yes
Birth Cohort Fixed Effects	Yes	Yes	Yes
State Fixed Effects	Yes	Yes	Yes
Linear Trends	Yes	Yes	Yes

Notes. OLS estimates with robust standard errors clustered at the state level in parentheses. Sample weights are used. The sample includes individuals aged 23-65 years old in years 1959 and 1960. In Panels A and B, treatment is identified by the average in utero exposure, expressed in terms of state-level monthly average cases. In Panel C, the treatment is interacted with a variable indicating whether the individual belongs to Pre or Post eradication birth cohort. Legal Amazon is excluded. Individual controls include gender and race, whereas region control includes whether the individual lives in a rural area, state GDP per capita, infant mortality, whether the individual is a migrant. Panels A and B estimations also include latitude and longitude as additional controls. The fertility analysis restricts the sample to female individuals only. *** p<0.01, ** p<0.05, * p<0.1

Table 3: Early Life Exposure to Malaria OLS Results – Trimester Exposure

	Pre-Eradication Sample (1959)			Post-Eradication Sample (1960)		
	Education (1)	Income (2)	Fertility (3)	Education (4)	Income (5)	Fertility (6)
First Trimester	-16.31^{**}	-2.766^{**}	14.30^{***}	1.684	-2.423^{**}	-4.766
	(6.827)	(1.104)	(4.633)	(6.054)	(1.048)	(5.204)
Second Trimester	-12.20^{**}	-0.788	-9.102	-1.506	0.691	-7.228
	(5.279)	(1.007)	(5.931)	(6.295)	(0.893)	(5.228)
Third Trimester	7.159	0.766	-0.347	1.457	0.239	5.516
	(9.330)	(0.839)	(4.060)	(6.155)	(1.438)	(3.496)
Observations	29,677	27,936	13,115	33,116	31,127	14,713
Full set of controls	Yes	Yes	Yes	Yes	Yes	Yes
State Fixed Effects	Yes	Yes	Yes	Yes	Yes	Yes

OLS estimates with robust standard errors clustered at the state level in parentheses. Sample weights are used. The sample includes individuals aged 23-65 years old in years 1959 and 1960. Treatment is identified by the average risk of exposure, expressed in terms of state-level monthly average cases. Legal Amazon is excluded. Individual controls include gender and race, whereas region control includes whether the individual lives in a rural area, state GDP per capita, infant mortality, whether the individual is a migrant. The fertility analysis restricts the sample to female individuals only. *** p<0.01, ** p<0.05, * p<0.1

Table 4: Early Life Exposure to Malaria DID Results – Trimester Exposure

	Education (1)	Income (2)	Fertility (3)
First Trimester Exposure $\times$ $Post$	18.00^{***}	0.343	-19.07^{**}
	(5.670)	(1.726)	(8.397)
Second Trimester Exposure $\times$ $Post$	10.69	1.497	1.874
	(6.244)	(1.529)	(9.908)
Third Trimester Exposure $\times$ $Post$	-5.703	-0.527	5.863
	(7.165)	(1.384)	(3.984)
Observations	62,793	59,063	27,828
Full set of controls	Yes	Yes	Yes
State Fixed Effects	Yes	Yes	Yes
Linear Trends	Yes	Yes	Yes

OLS estimates with robust standard errors clustered at the state level in parentheses. Sample weights are used. The sample includes individuals aged 23-65 years old in years 1959 and 1960. Treatment is identified by the average risk of exposure, expressed in terms of state-level monthly average cases. The treatment is interacted with a variable indicating whether the individual belongs to the Post eradication birth cohort. Legal Amazon is excluded. Individual controls are the same as in previous table. *** p<0.01, ** p<0.05, * p<0.1

Table 5: Early Life Exposure to Malaria OLS Results – Men vs Women

	Men		Women		
	Education (1)	Income (2)	Education (3)	Income (4)	Fertility (5)
In utero Exposure					
			Panel A. Pre-Eradication Sample (1959)		
Treatment Effect	-22.94^{***}	-2.762^{**}	-16.59	-2.550	5.003
	(5.531)	(0.978)	(12.71)	(1.955)	(3.140)
Observations	16,606	16,401	13,071	11,535	13,115
R-squared	0.223	0.212	0.189	0.127	0.130
Birth Cohort Fixed Effects	Yes	Yes	Yes	Yes	Yes
State Fixed Effects	Yes	Yes	Yes	Yes	Yes
Linear Trends	No	No	No	No	No
			Panel B. Post-Eradication Sample (1960)		
Treatment Effect	-6.305	-1.413	10.14	-2.173	-7.154^{*}
	(4.485)	(1.407)	(11.81)	(1.890)	(3.483)
Observations	18,458	18,175	14,658	12,952	14,713
R-squared	0.232	0.231	0.176	0.132	0.120
Birth Cohort Fixed Effects	Yes	Yes	Yes	Yes	Yes
State Fixed Effects	Yes	Yes	Yes	Yes	Yes
Linear Trends	No	No	No	No	No
			Panel C. Differences-in-Differences Results		
Treatment Effect×Post	13.64^{***}	1.349	26.73^{*}	0.378	-12.16^{**}
	(5.510)	(1.801)	(14.59)	(1.804)	(4.486)
Observations	35,064	34,576	27,729	24,487	27,828
R-squared	0.228	0.222	0.182	0.130	0.125
Birth Cohort Fixed Effects	Yes	Yes	Yes	Yes	Yes
State Fixed Effects	Yes	Yes	Yes	Yes	Yes
Linear Trends	Yes	Yes	Yes	Yes	Yes

Notes. OLS estimates with robust standard errors clustered at the state level in parentheses. Sample weights are used. The sample includes individuals aged 23-65 years old in years 1959 and 1960. In Panels A and B, treatment is identified by the average in utero exposure, expressed in terms of state-level monthly average cases. In Panel C, the treatment is interacted with a variable indicating whether the individual belongs to Pre or Post eradication birth cohort. Legal Amazon is excluded. Individual controls include gender and race, whereas region control includes whether the individual lives in a rural area, state GDP per capita, infant mortality, whether the individual is a migrant. Panels A and B estimations also include latitude and longitude as additional controls. The fertility analysis restricts the sample to female individuals only. *** p<0.01, ** p<0.05, * p<0.1

Table 6: Early Life Exposure to Malaria OLS Results – Movers vs Non-Movers

	Non-Movers			Movers		
	Education (1)	Income (2)	Fertility (3)	Education (1)	Income (2)	Fertility (3)
In utero Exposure						
	Panel A. Pre-Eradication Sample (1959)					
Treatment Effect	-18.40^{***}	-3.282^{***}	7.338^{**}	-30.17^{**}	-1.992	2.012
	(5.865)	(0.978)	(3.382)	(11.83)	(2.237)	(9.296)
Observations	22,883	21,459	10,252	6,794	6,477	2,863
R-squared	0.230	0.204	0.153	0.152	0.123	0.057
Full Set of Controls	Yes	Yes	Yes	Yes	Yes	Yes
State Fixed Effects	Yes	Yes	Yes	Yes	Yes	Yes
Linear Trends	No	No	No	No	No	No
	Panel B. Post-Eradication Sample (1960)					
Treatment Effect	-1.384	-2.460	-5.562	17.77	1.844	-11.74
	(6.207)	(2.057)	(5.422)	(12.32)	(1.990)	(11.98)
Observations	25,717	24,088	11,475	7,399	7,039	3,238
R-squared	0.225	0.216	0.139	0.161	0.124	0.052
Full Set of Controls	Yes	Yes	Yes	Yes	Yes	Yes
State Fixed Effects	Yes	Yes	Yes	Yes	Yes	Yes
Linear Trends	No	No	No	No	No	No
	Panel C. Differences-in-Differences Results					
Treatment Effect×Post	17.01^{**}	0.822	-12.90^{**}	47.94^{***}	3.836	-13.75
	(7.120)	(1.583)	(5.964)	(13.40)	(2.384)	(10.26)
Observations	48,600	45,547	21,727	14,193	13,516	6,101
R-squared	0.227	0.211	0.146	0.157	0.123	0.055
Full Set of Controls	Yes	Yes	Yes	Yes	Yes	Yes
Birth Cohort Fixed Effects	Yes	Yes	Yes	Yes	Yes	Yes
State Fixed Effects	Yes	Yes	Yes	Yes	Yes	Yes
Linear Trends	Yes	Yes	Yes	Yes	Yes	Yes

Notes. OLS estimates with robust standard errors clustered at the state level in parentheses. Sample weights are used. The sample includes individuals aged 23-65 years old in years 1959 and 1960. In Panels A and B, treatment is identified by the average in utero exposure, expressed in terms of state-level monthly average cases. In Panel C, the treatment is interacted with a variable indicating whether the individual belongs to Pre or Post eradication birth cohort. Legal Amazon is excluded. Individual controls include gender and race, whereas region control includes whether the individual lives in a rural area, state GDP per capita, infant mortality, whether the individual is a migrant. Panels A and B estimations also include latitude and longitude as additional controls. The fertility analysis restricts the sample to female individuals only. *** p<0.01, ** p<0.05, * p<0.1

Table 7: Early Life Exposure to Malaria IV Results

	Pre-Eradication Sample (1959)						Post-Eradication Sample (1960)					
	Education		Income		Fertility		Education		Income		Fertility	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
	OLS	2SLS	OLS	2SLS	OLS	2SLS	OLS	2SLS	OLS	2SLS	OLS	2SLS
First Trimester	−16.31**	−48.24***	−2.766**	3.723	14.30***	-9.124	1.684	−27.59*	−2.423**	−8.220**	-4.766	-1.639
	(6.827)	(18.35)	(1.104)	(7.182)	(4.633)	(19.17)	(6.054)	(14.32)	(1.048)	(3.332)	(5.204)	(6.364)
First stage F statistic		173.81		219.27		198.21		46.63		60.00		66.65
Second Trimester	−12.20**	12.44	-0.788	-0.690	-9.102	-0.178	-1.506	13.39	0.691	9.880*	-7.228	-10.45
	(5.279)	(24.63)	(1.007)	(5.016)	(5.931)	(19.33)	(6.295)	(13.42)	(0.893)	(5.371)	(5.228)	(16.05)
First stage F statistic		188.71		150.88		560.87		192.54		126.89		191.38
Third Trimester	7.159	25.02	0.766	-1.023	-0.347	-0.0173	1.457	11.94	0.239	6.184	5.516	23.08*
	(9.330)	(19.67)	(0.839)	(2.361)	(4.060)	(19.90)	(6.155)	(26.25)	(1.438)	(4.399)	(3.496)	(13.35)
First stage F statistic		206.96		198.17		177.78		73.28		98.54		50.74
Observations	29,677		27,936		13,115		33,116		31,127		14,713	
Full set of controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
State Fixed Effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes

2SLS estimates with robust standard errors clustered at the state level in parentheses. Sample weights are used. The sample includes individuals aged 23-65 years old in years 1959 and 1960. Treatment is identified by the average risk of exposure, expressed in terms of state-level monthly average cases. Legal Amazon is excluded. Individual controls include gender and race, whereas region control includes whether the individual lives in a rural area, state GDP per capita, infant mortality, whether the individual is a migrant. The fertility analysis restricts the sample to female individuals only. *** p<0.01, ** p<0.05, * p<0.1

Table 8: Early Life Exposure to Malaria DID IV Results – Trimester Exposure

	Education		Income		Fertility	
	(1)	(2)	(3)	(4)	(5)	(6)
	OLS	2SLS	OLS	2SLS	OLS	2SLS
First Trimester Exposure × $Post$	30.73***	-19.66	2.135	-8.842	-20.96*	15.65
	(8.303)	(19.33)	(1.592)	(7.691)	(10.02)	(15.08)
First stage F statistic		118.11		140.77		2102.71
Second Trimester Exposure × $Post$	14.05*	-6.953	-0.530	0.0838	5.484	19.79
	(6.884)	(19.94)	(1.733)	(4.173)	(6.239)	(20.38)
First stage F statistic		50229.49		17244.46		4.9e+05
Third Trimester Exposure × $Post$	-11.90	-19.92	1.298	-5.617	5.677	15.66
	(12.92)	(22.56)	(2.324)	(6.941)	(7.230)	(11.30)
First stage F statistic		2078.87		65778.05		7.2e+05
Observations	60,904		57,440		26,904	
Full set of controls	Yes	Yes	Yes	Yes	Yes	Yes
State Fixed Effects	Yes	Yes	Yes	Yes	Yes	Yes
Linear Trends	Yes	Yes	Yes	Yes	Yes	Yes

2SLS estimates with robust standard errors clustered at the state level in parentheses. Sample weights are used. The sample includes individuals aged 23-65 years old in years 1959 and 1960. Treatment is identified by the average risk of exposure, expressed in terms of state-level monthly average cases. The treatment is interacted with a variable indicating whether the individual belongs to the Post eradication birth cohort. Legal Amazon is excluded. Individual controls include gender and race, whereas region control includes whether the individual lives in a rural area, state GDP per capita, infant mortality, whether the individual is a migrant. The fertility analysis restricts the sample to female individuals only. *** p<0.01, ** p<0.05, * p<0.1

Appendix A. Data sources and Details

Malaria Variables

Malaria Risk

The malaria risk variable is constructed using malaria reported cases at the month-state level for the period between 2007–2017. In order to construct the variable at a per capita basis, I collect state population data from IPEADATA[49]. The malaria risk variable is number of reported cases per 1,000 inhabitants for a given state and month.[50]

Malaria Index

The mortality selection estimation makes use of a state-level time-invariant measure of malaria transmission, which is constructed by using the ecology-based spatial Malaria Stability Index proposed by Kiszewski et al. (2004). The index captures geographical distribution of the dominant *Anopheles* mosquito as well as information on climatic and geographic conditions that contribute to the survival and spread of vector and parasite. Using a Geographic Information Sytem (GIS) software, I overlay a Brazilian map with state boundaries over the global spatial Malaria Index map and generate state-level averages of the index.

Individual-level outcomes and controls

Education

The education variable used throughout the analysis is number of years of education which corresponds to the highest grade completed. The primary source is the Brazilian National Household Sample Survey (PNAD).

Income

Income is given by the hourly earned income from main activity in the previous week. In the survey, income is reported at a monthly basis. I transform monthly income to hourly income by multiplying reported hours worked weekly by 52 (total number of weeks in a year) and divide by 12 (total number of months). I drop from the analysis hourly income higher than R$ 300,00. In the analysis, I use the natural log of income. The primary data source is the Brazilian National Household Sample Survey (PNAD).

[49] http://www.ipeadata.gov.br/Default.aspx Accessed on 10/17/2018.

[50] Population data is given at an yearly basis. I use the same constant population for each month in a given year.

Fertility

The fertility variable is given by the number of reported children by female respondent. The variable is constructed by adding the reported number of male and female children who live in the household, the number of male and female children who do not live in the household, and the number of male and female children who have died. The primary data source is the Brazilian National Household Sample Survey (PNAD).

Demographic control variables

Gender: dummy variable indicating whether the individual is a female.
Race: dummy variable indicating whether the individual is non-white.
Mover: dummy variable indicating whether the individual resides in the state of birth.

State-level controls

GDP per capita

State GDP per capita is constructed by state GDP, at 2000 prices, divided by the population. The state GDP per capita data is matched with individual's state of birth and survey year. The primary source is the IPEADATA.

Infant mortality

Reports the number of infant deaths by state. Data source is DATASUS – Ministry of Health. To construct infant mortality per capita I use population data from IPEADATA. The infant mortality per capita data is matched with individual's state of birth and survey year.

Latitude and Longitude

Latitude and longitude jointly report the geo-referenced coordinate of the centroid of the state. The data source is from the GADM database.[51]

Climatic Variables

In the Instrumental Variables estimation, I use average monthly temperature and precipitation as excluded instruments for the malaria transmission variable. The data source is WorldClim – Global Climate Data (v.2), available at http://worldclim.org/version2. The climatic data set

[51] www.gadm.org, version 2.5, July 2015.

is based on a raster file with 10 km^2 resolution cells, with mean monthly temperature and precipitation for the period 1970–2000. I construct state-level monthly averages by overlaying a Brazilian georeferenced map with state boundaries with the help of a GIS software.

Appendix B. Instrumental Variables First Stage Results

Table B1: Instrumental Variables Estimation – First Stage Reults

	First Trimester (1)	Second Trimester (2)	Third Trimester (3)
Prec $> 60mm^2$ last three months			
Month 1	-8.92e-05*	-1.99e-05	4.69e-05
	(5.38e-05)	(5.81e-05)	(5.99e-05)
Month 2	0.000259***	0.000171***	0.000178***
	(6.03e-05)	(6.64e-05)	(6.84e-05)
Month 3	0.000609***	0.000641***	0.000699***
	(6.95e-05)	(7.10e-05)	(7.01e-05)
Prec $> 80mm^2$ at least one of the last three months			
Month 1	0.000449***	0.000559***	0.000481***
	(6.35e-05)	(6.24e-05)	(6.62e-05)
Month 2	−0.000716***	−0.000892***	−0.000906***
	(7.36e-05)	(7.73e-05)	(8.08e-05)
Month 3	−0.000832***	−0.000911***	−0.000856***
	(6.40e-05)	(6.62e-05)	(6.79e-05)
Temp $> 19.5°C+$ std. dev. last 12 months			
Month 1	0.00115***	0.00111***	0.00104***
	(6.45e-05)	(6.57e-05)	(6.44e-05)
Month 2	0.000875***	0.00115***	0.00114***
	(7.33e-05)	(8.30e-05)	(8.68e-05)
Month 3	0.00201***	0.00192***	0.00195***
	(8.07e-05)	(8.52e-05)	(8.90e-05)
Observations	29,793	29,793	29,793

OLS estimates with robust standard errors clustered at the state level in parentheses. Sample weights are used. The sample includes individuals aged 23-65 years old in years 1959 and 1960. The first stage of the instrumental variables analysis instruments the average risk of exposure, expressed in terms of state-level monthly average cases, with indicator variables expressing necessary climatic conditions for malaria outbreaks. The first indicator is whether the month (or the previous two) has an average precipitation of at least $60mm^2$. The second indicator refers to whether at least one of the last three months experienced an average precipitation of at least $80mm^2$. The third variable indicates whether average monthly temperature exceeds $19.5°C+$ standard deviation of the average temperature in the previous 12 months. Legal Amazon is excluded. Individual controls include gender and race, whereas region control includes whether the individual lives in a rural area, state GDP per capita, infant mortality, whether the individual is a migrant. *** p<0.01, ** p<0.05, * p<0.1

Table B2: Instrumental Variables Estimation – Reduced Form First Trimester Results

	Pre-Eradication Sample (1959)			Post-Eradication Sample (1960)		
	Education (1)	Income (2)	Fertility (3)	Education (4)	Income (5)	Fertility (6)
Prec $> 60mm^2$ last three months						
Month 1	-0.0911	-0.0243	0.0780	-0.0717	-0.0239	-0.0160
	(0.0830)	(0.0188)	(0.0926)	(0.0441)	(0.0189)	(0.0729)
Month 2	-0.153^{*}	-0.0407^{**}	-0.0610	-0.110	-0.0104	-0.0221
	(0.0760)	(0.0169)	(0.0782)	(0.117)	(0.0201)	(0.0789)
Month 3	-0.00968	-0.0110	0.0918	0.164	0.00617	0.0905
	(0.0911)	(0.0244)	(0.0897)	(0.0991)	(0.0197)	(0.110)
Prec $> 80mm^2$ at least one of the last three months						
Month 1	0.102	0.0398^{*}	-0.0664	-0.0581	-0.00791	0.0373
	(0.0954)	(0.0207)	(0.0656)	(0.104)	(0.0215)	(0.0778)
Month 2	-0.0814	-0.0386^{**}	-0.0655	-0.00463	0.00868	-0.0647
	(0.0675)	(0.0160)	(0.113)	(0.109)	(0.0176)	(0.100)
Month 3	0.189^{**}	0.0410^{**}	-0.0107	0.00761	0.0230	0.0207
	(0.0789)	(0.0167)	(0.0964)	(0.0695)	(0.0159)	(0.0668)
Temp $> 19.5°C+$ std. dev. last 12 months						
Month 1	0.202^{**}	0.0424	-0.0236	0.166^{*}	0.00737	-0.00822
	(0.0839)	(0.0252)	(0.0496)	(0.0867)	(0.0414)	(0.0904)
Month 2	-0.201^{**}	-0.00740	0.0404	-0.0689	-0.0216	0.0985
	(0.0972)	(0.0378)	(0.133)	(0.0922)	(0.0348)	(0.0740)
Month 3	-0.0383	0.0298^{**}	-0.0525	-0.107	-0.00488	-0.176^{*}
	(0.0986)	(0.0140)	(0.0860)	(0.123)	(0.0235)	(0.100)
Observations	33,172	31,199	14,705	37,309	34,971	16,636

OLS estimates with robust standard errors in parentheses. Sample weights are used. The sample includes individuals aged 23-65 years old in years 1959 and 1960. The reduced form estimates show the regression results of the outcome variables on the climatic instruments of the first stage analysis. Legal Amazon is excluded. Individual controls include gender and race, whereas region control includes whether the individual lives in a rural area, state GDP per capita, infant mortality, whether the individual is a migrant. *** p<0.01, ** p<0.05, * p<0.1

Table B3: Instrumental Variables Estimation – Reduced Form Second Trimester Results

	Pre-Eradication Sample (1959)			Post-Eradication Sample (1960)		
	Education (1)	Income (2)	Fertility (3)	Education (4)	Income (5)	Fertility (6)
Prec $> 60mm^2$ last three months						
Month 1	-0.0946	-0.00248	0.0784	-0.0162	-0.00636	-0.00521
	(0.0685)	(0.0169)	(0.0928)	(0.0708)	(0.0202)	(0.0671)
Month 2	0.235^{*}	0.0188	0.0537	-0.0352	-0.0152	0.0745
	(0.115)	(0.0231)	(0.0813)	(0.0955)	(0.0224)	(0.0628)
Month 3	-0.0411	-0.000856	-0.00312	-0.0129	0.0138	-0.00238
	(0.0936)	(0.0212)	(0.110)	(0.0679)	(0.0187)	(0.0586)
Prec $> 80mm^2$ at least one of the last three months						
Month 1	0.0329	-0.0128	-0.0842	0.184^{*}	0.0310^{**}	0.0756
	(0.176)	(0.0401)	(0.0666)	(0.0988)	(0.0131)	(0.0578)
Month 2	-0.165	-0.00357	0.0438	0.0257	0.0127	-0.0855
	(0.106)	(0.0223)	(0.0948)	(0.118)	(0.0201)	(0.0614)
Month 3	0.159^{**}	0.0295	-0.0484	-0.00101	-0.00567	0.0213
	(0.0651)	(0.0186)	(0.0595)	(0.0661)	(0.0190)	(0.0824)
Temp $> 19.5°C+$ std. dev. last 12 months						
Month 1	-0.245^{***}	0.00812	-0.0120	-0.282^{***}	-0.0258	-0.122
	(0.0624)	(0.0160)	(0.0711)	(0.0957)	(0.0185)	(0.0908)
Month 2	0.168^{***}	0.0318	-0.198^{***}	0.0703	0.0152	-0.0843
	(0.0467)	(0.0212)	(0.0436)	(0.0451)	(0.0238)	(0.0791)
Month 3	-0.0796	-0.0597^{***}	0.191^{***}	0.0851	0.0273	0.0672
	(0.0493)	(0.0126)	(0.0687)	(0.0565)	(0.0214)	(0.0541)
Observations	33,172	31,199	14,705	37,309	34,971	16,636

OLS estimates with robust standard errors in parentheses. Sample weights are used. The sample includes individuals aged 23-65 years old in years 1959 and 1960. The reduced form estimates show the regression results of the outcome variables on the climatic instruments of the first stage analysis. Legal Amazon is excluded. Individual controls include gender and race, whereas region control includes whether the individual lives in a rural area, state GDP per capita, infant mortality, whether the individual is a migrant. *** p<0.01, ** p<0.05, * p<0.1

Table B4: Instrumental Variables Estimation – Reduced Form Third Trimester Results

	Pre-Eradication Sample (1959)			Post-Eradication Sample (1960)		
	Education (1)	Income (2)	Fertility (3)	Education (4)	Income (5)	Fertility (6)
Prec $> 60mm^2$ last three months						
Month 1	0.0960	0.00898	0328	0.0408	0.0405^{**}	-0.0506
	(0.0601)	(0.0190)	(0.0809)	(0.0682)	(0.0191)	(0.0461)
Month 2	-0.0561	0.0332	-0.151^{*}	0.141^{**}	0.00959	-0.0412
	(0.138)	(0.0233)	(0.0752)	(0.0567	(0.0161)	(0.0993)
Month 3	0.0385	-0.0274^{*}	0.0376	-0.0422	-0.00108	-0.0319
	(0.0514)	(0.0147)	(0.0535)	(0.0647)	(0.0168)	(0.0559)
Prec $> 80mm^2$ at least one of the last three months						
Month 1	-0.00870	0.00488	-0.0461	0.0104	0.0102	0.0800
	(0.0750)	(0.0155)	(0.0570)	(0.0697)	(0.0175)	(0.0605)
Month 2	-0.0783	0.00170	0.192^{*}	-0.147	-0.0456^{**}	-0.0101
	(0.103)	(0.0295)	(0.105)	(0.107)	(0.0210)	(0.0547)
Month 3	0.133	0.00513	-0.0875	0.0965	0.0102	-0.0285
	(0.115)	(0.0185)	(0.0643)	(0.116)	(0.0199)	(0.0603)
Temp $> 19.5°C+$ std. dev. last 12 months						
Month 1	0.132	-0.0432^{*}	0.0643	-0.0120	-0.00734	0.209^{***}
	(0.142)	(0.0220)	(0.0430)	(0.0932)	(0.0154)	(0.0579)
Month 2	0.0742	0.0127	0.0551	0.0841	0.0355	-0.186^{**}
	(0.134)	(0.0224)	(0.0727)	(0.102)	(0.0219)	(0.0703)
Month 3	-0.104^{*}	-0.00246	-0.0443	-0.104	-0.0297	0.167^{***}
	(0.0573)	(0.0194)	(0.0306)	(0.0788)	(0.0344)	(0.0597)
Observations	33,172	31,199	14,705	37,309	34,971	16,636

OLS estimates with robust standard errors in parentheses. Sample weights are used. The sample includes individuals aged 23-65 years old in years 1959 and 1960. The reduced form estimates show the regression results of the outcome variables on the climatic instruments of the first stage analysis. Legal Amazon is excluded. Individual controls include gender and race, whereas region control includes whether the individual lives in a rural area, state GDP per capita, infant mortality, whether the individual is a migrant. *** p<0.01, ** p<0.05, * p<0.1