Wrong Place, Wrong Time: The Long-Run Effects of In-Utero Exposure to Malaria on Educational Attainment*

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Job Market Paper

Abstract

This paper investigates the long-term relationship between early life exposure to malaria and human capital accumulation in Brazil. The identification strategy exploits variation in exposure to malaria induced by environmental and climatic factors that identify exogenous in utero exposure according to location and timing of birth of cohorts born around a health policy aiming to eradicate malaria in Brazil. This novel source of identification allows me to distinguish exposure across different critical periods of early life, such as different trimesters of gestation, as well as control for unobserved regional-level factors that might correlate with the geographic distribution of malaria across locations. I find negative treatment effects on the number of years of education and primary completion rates The effects are stronger for exposure during the first trimester of pregnancy than during other periods of gestation. Effective anti-malaria policies can, thus, be an important factor contributing to reducing the educational inequality by targeting pregnant women, especially those in their first months of gestation.

Keywords: Malaria, Human Capital, Public Health
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1 Introduction

Malaria is a vector-borne disease with distribution and prevalence depending, to a certain extent, on geographic and environmental conditions. It is disproportionately concentrated in humid and tropical climate locations, wherein approximately half of the world population is at risk (WHO, 2019). Malaria infections destroy red blood cells, depriving the distribution of oxygen and nutrients to the body tissue. The long-lasting consequences to 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 to cognition (Barreca, 2010). Understanding such relationships and estimating the impacts of differential exposure to malaria on socioeconomic conditions are of extreme relevance to devise effective antimalarial interventions by identifying and targeting the most vulnerable population groups.

Estimating the long-term impact of changes in early life health distribution can be challenging because of omitted factors, such as unobserved underlying socioeconomic conditions, that might confound the results. Previous studies have addressed long-term socioeconomic consequences of malaria exposure by making use of exogenous declines in malaria rates after eradication campaigns or by exploring relationships between malaria transmission and climatic conditions across different geographic locations and years. These studies, however, do not fully account for predetermined unobserved heterogeneity across units of observation by identifying the degree of exposure based on regional differences in malaria intensity alone. The geographic distribution of malaria is endogenous to regional (potentially unobservable) characteristics, including local-level state capacity as well as community- and individual-level attributes that might contribute to the success or failure of efforts to controlling the disease.

To answer whether in utero exposure to malaria affects human capital accumulation, accounting for the potential regional endogeneity in malaria burden, this paper relies on two main sources of exogenous variations in its transmission rates in Brazil that jointly identify the extent of the exposure during early life. The first is given by differential transmission risks across months and states, which determine heterogeneous levels of exposure across individuals within birth cohorts. Identifying exposure according to both location and month of birth serves a dual purpose for my analysis: first, it allows me to control for any unobserved regional effects, such as

national policies that might heterogeneously affect individuals according to geographic location; second, I can ascertain the timing of individual-level exposure according to different periods of gestation, which allows me to test for heterogeneous treatment effects along the in utero life.

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, with the application of DDT spraying inside houses and anti-malarial drugs distribution.[1] This abrupt and arguably exogenous variation in the overall malaria transmission rates in Brazil provides a unique opportunity to isolate the magnitude of the average effect of in utero exposure to malaria from the effects of prolonged exposure during childhood and adulthood by identifying individuals born at different moments of the campaign roll-out. This paper, thus, sheds light on the potential role of directed efforts to fight malaria in endemic areas by identifying the critical periods of in utero exposure and their consequences for economic outcomes.[2]

I find consistently negative effects of early life exposure to malaria on educational attainment for relatively more exposed individuals, born shortly before malaria rates have reached their lowest levels. 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 0.17 years and leads to reductions of approximately 1.59 percentage points in the probability of primary completion and 2.16 percentage points in the probability of secondary completion.

In the subsequent analysis, 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. 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.[3] However, asymmetric growth retardations,

[1] 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.

[2] 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.

[3] Epidemiological studies have found severe consequences for later-life health after deterioration of initial health stock. Ravelli et al. (1976) found that exposure to famine on the first trimester of pregnancy leads to large effects on

which are associated with infections at later stages, may also lead to ill effects, such as low birth weight, increasing the likelihood of long-term repercussions. Some studies, 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). Additionally, Luxemburger et al. (2001) find low birth weight, and other sequelae, after infections during the third trimester of pregnancy. 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. The results of this analysis indicate heterogeneous treatment effects according to the timing of exposure, with the largest effects stemming from exposure to the disease during the first months of intrauterine life.

This paper broadly 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 literature, 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. 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 (Costa, 2000, 2003; Costa and Lahey, 2005; Hack et al., 2002; Case et al., 2005; Black et al., 2007; Currie and Moretti, 2007), while others have incorporated robust empirical strategies by identifying exogenous shocks to the early life environment, including the Dutch "Hunger Winter" of 1944 (Stein et al., 1975; Roseboom et al., 2011), the 1918 Influenza Pandemic (Almond, 2006; Almond and Mazumder, 2005; Lin and Liu, 2014), the Chinese Great Famine (Chen and Zhou, 2007; Meng and Qian, 2009), and observation of Ramadan among Islamic religious groups (Almond and Mazumder, 2011; Van Ewijk, 2011).[4]

obesity rates after reaching adulthood. Moreover, McAlister (2007) has found long-term defects such as heart disease and deafness after pregnant women being infected with Rubella during the first trimester.

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

This paper is more closely connected to the literature exploring the potential negative effects of early life disease environment. 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. Relying on historical records of malarial fevers among U.S soldiers across the country, combined with environmental factors that determine malaria prevalence, Hong (2007) finds that early life exposure to malaria is associated with lower health among Union Army soldiers during the 1850s. Barreca (2010) and Burlando (2015) find effects of malaria transmission in United States and Ethiopia, respectively, on schooling attainment by instrumenting malaria transmission with geographic factors such as temperature, rainfall, and topography. Rawlings (2016) identifies selection effects from in utero exposure to epidemic malaria in two states in Brazil.

This paper is also related to some other studies that 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), and Bleakley (2010b), find positive effects of malaria eradication on human capital accumulation and other adult outcomes. 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. Additionally, the estimation strategy pursued by these studies might not adequately control for potential unobserved geographic heterogeneity in treated units.

In identifying in utero exposure to malaria according to both geographic and seasonal variations in different years, this paper contributes to the aforementioned literature in at least two important ways. First, by testing for heterogeneous effects of early life exposure according to different periods of intrauterine life, this paper documents important evidence on the most critical periods of vulnerability to health insults. The evidence in this paper provides relevant information for informed policies to improve educational attainment in malaria ridden areas by identifying the most needed groups: pregnant women in their initial stages of gestation. Second, this paper takes a step further to cleanly identify exposure by controlling for unobserved factors that might be associated with the geographic distribution of malaria and finds that the effects of in utero exposure malaria are, although significant, less strong than previously found elsewhere.

The paper is structured in five sections, including this introduction, as follows: Section 2 presents some background information about malaria transmission and the history of malaria in Brazil. Section 3 details the data and some conceptual definitions as well as the research design implemented in this paper. Section 4 exposes the main results, and further expands the analysis to exploit the long-run effects on other socioeconomic outcomes as well as potential heterogeneity associated with exposure. Section 5 concludes.

2 Background

2.1 Biological Aspects of 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.[5] 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.[6] 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

[5] 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.

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

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.[7]

The transmission of malaria is highly sensitive to climatic variation. Rainfall is important due to its effects on the mosquito life-cycle. 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 (Martens et al., 1995). The aquatic development stages of the mosquito occurs more efficiently in temperatures between 16°C to 28°C (Cervellati et al., 2018). Moreover, survival of adult mosquitoes also depends on temperature and rainfall, as well as humidity. Warmer temperatures decrease 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.[8]

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.

2.2 The Importance of the Timing of Malaria Infections for Subsequent Development

Disentangling the importance of in utero malaria infections relative to post-natal and childhood exposure is empirically challenging because it is rarely possible to identify individuals whose exposure to malaria happened only after birth or those who faced exposure while in utero only. Furthermore, in utero infections are associated with higher susceptibility to subsequent infections due to immunity impairments, rendering comparison even more difficult (Moya-Alvarez et al., 2014). Most of the evidence relating pre-natal and post-natal consequences of malaria infections

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

[8] 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. 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). See https://www.cdc.gov/malaria/about/biology/index.html, accessed 08/30/2018, for detailed information about the biologic aspects of malaria.

come from the medical literature, stressing the relative importance of fetal development and its long-term associations with cognitive and immunological systems development. Evidence of the effects of childhood malaria infections on children's educational and cognitive outcomes is abundant. For example, in a survey paper, Fernando et al. (2010) point out to the established stylized fact that early childhood malaria reduces school attainment and cognitive capacity. Jukes et al. (2006), in a controlled trial setting among children under the age of 5, find that children who received a preventive treatment during malaria seasons exhibited improved school performance (0.52 grades) relative to a control group. Boivin (2002) finds that children with episodes of cerebral malaria have lower cognitive ability compared to healthier children. Boivin et al. (2007) and John et al. (2008) find some short-run effects of cerebral malaria on cognition in Uganda, while Bangirana et al. (2006) discusses some coping strategies to deal with cognitive losses and their associated socioeconomic barriers.

Among the studies concentrating on pre-natal malaria infections, some stress the importance of the intrinsic association of malaria infections to low birth weights, intrauterine growth retardations, premature birth delivery, and fetal anemia (Singh et al., 1999; Holding and Snow, 2001; Luxemburger et al., 2001; Desai et al., 2007; Umbers et al., 2011; Moya-Alvarez et al., 2014). During the in-utero phase of fetal development, cognitive functions can permanently be altered after placental infections (Barker, 1998; Smith, 2004; Almond and Currie, 2011; McDonald et al., 2013). Mireku et al. (2015) show that infants born from mothers who had at least one hookworm infection exhibit lower cognitive and motor skills, while Adam et al. (2011) find that placental malaria is associated with a higher risk of preeclampsia and Brabin (1991) finds increased risk of low birth weight among individuals born in endemic regions.

In general, the medical studies addressing the impact of both pre-natal and childhood exposure to malaria suggest significant short- and long-run effects on cognitive and behavioral outcomes. The paucity of specific comparative studies, the limited external validity of the existing ones, and the importance of in utero infections to post-natal risk, however, pose an empirical challenge in separating out the relative relevance of the different timings of exposure. This study aims at assessing the independent effect in utero exposure to long-run educational outcomes by leveraging on a natural experiment that allows me to identify heterogeneous degree of exposure according to timing and location of birth.

2.3 Malaria history in Brazil

Malaria is believed to have been introduced in Brazil during the colonial times around 1560 and its spread throughout the country was triggered by the precarious conditions of infrastructure constructions (mainly railroads connecting various parts of the country) and the influx of the population to the Amazon region, where the geographic and climatic conditions for the survival of the *anopheles* mosquito are ideal, with the boom of the rubber industry (Griffing et al., 2015).

Early efforts to combat malaria were usually done at a case-by-case basis, with special attention to large public works projects.[9] The common measures were mainly palliative, including the intake of chloroquine, 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. 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, which started in 1941 with the creation of the National Malaria Service, the SNM (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. Indoor DDT spraying and the distribution of antimalarial drugs among the population were the main actions taken by CEM at the prevention and treatment of malaria. Although numbers related to the cost and benefits of the nationwide campaign are scarce, Akhavan et al. (1999) find that, for a program targeting to control malaria in the Amazon basin, the overall cost-effectiveness was around US$69 per Disability-Adjusted Life Years, whereby case treatment seems to be the most-cost effective way to tackle malaria compared to vector-control. Figure 1

[9] During the early 1900s, 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).

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.

The CEM was successful in eliminating malaria in four out of the five Brazilian regions, with the exception of the North. The failure of the CEM in the North region was primarily given by the resistance of the *P. falciparum* to chloroquine, 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.[10] 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.

3 Data and Empirical Strategy

The micro-level data for the individual-level analysis come from the annual Brazilian National Household Sample Survey (PNAD). 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.[11] Importantly, 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 identifying individuals by location of residence would be contaminated by selective migration issues.[12] 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.[13]

[10]Malaria cases in the Amazon basin rose steadily until 1989, reaching about 500,000 cases per year. The number of cases only started to halt by the mid-1990s, after the World Bank's support to the national government's control effort in the region (Akhavan et al., 1999).

[11]I exclude individuals born in the Amazon region because the eradication campaign was not successful in achieving the same dramatic decline in malaria rates as in the rest of the country due to the region-specific environmental and demographic conditions that favor the stability and proliferation of both malaria vector and parasite. The lack of variation of exposure across different cohorts during the eradication period would not allow for a clear comparison of treated versus non-treated cohorts. The results including individuals born in the Legal Amazon region are similar to the ones obtained throughout the paper and are available upon request.

[12]It is possible that the most privileged (high-skilled) individuals migrate to areas in which the malaria burden is low. This selection channel is, thus, shut down in my analysis, in contrast with other studies identifying exposure according to location of residence due to lack of precise location of birth data.

[13]Since the data on date of birth and age is self-reported, age heaping might affect my estimates if misreporting is systematically associated with individual socioeconomic characteristics. In appendix B2, I show that age heaping is not an important factor driving my results by analyzing the age distribution in my data according to socioeconomic status.

I combine the micro-level data from various PNAD samples with state-level malaria transmission 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.[14] The units of observation are expressed in terms of individual, month of exposure, year of birth, and 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 2 shows the average risk of malaria transmission across states in Brazil. The figure highlights the degree of heterogeneity in transmission in both state and month dimensions. Given the enormous geographic size of Brazil, climatic conditions and, thus, malaria transmissions, may vary substantially by state and seasons of the year.[15]

In addition to the monthly variation in malaria risk by state, 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 observation, from Griffing et al. (2015), corresponds 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.

Research Design

Taken together, figures 2 and 3 show the relevant variation of malaria transmission to identifying in utero exposure to malaria. The first aspect of malaria transmission that this paper explores is its seasonal variation related to specific climatic conditions, as figure 2 displays. I make use of the differential timing of exposure to malaria during early periods of life to assess the long-term effects on adult human capital and socio-economic outcomes. The additional source of variation in malaria burden according to the seasonality of the transmission is a step toward controlling for unobservable behavioral responses to the incidence of the disease. Castilla and Sawyer (1993), for example, find that socioeconomic status and knowledge of malaria transmission play crucial roles in its prevention and control. Therefore, areas with higher average socioeconomic status and

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

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

stronger adherence to protective measures are likely to be more successful in avoiding infections than otherwise similar localities.[16]

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, highlighted in figure 3, to assess the effects of differences in exposure across cohorts born at different levels of overall malaria rates.[17]

The baseline specification identifies exposure to malaria as the average risk of transmission across the months, year, and state in which the individuals were exposed while in utero. For example, consider a given individual, born in month m, year y, and state s; her exposure is given by the average risk associated with months $m-9$ through $m-1$ in state s and year y.[18]

I construct the main independent variable in the analysis by combining the 2007–2017 state-month-level reported malaria cases data with state-level data available from 1959 onward, based on some assumptions about the dynamics of the state-month level malaria transmission through time.[19] Formally, the exposure variable can be decomposed in two main components: the overall nation-wide scale of transmission and the relative intensity at the month-state level, in the following way:

$$\text{Exposure}_{sky} \approx \text{SCALE}_{sy} \times \text{REL}_{sk}, \tag{1}$$

where Exposure_{sky}, the average exposure in month k of year y for each state s, is defined by the decomposition of the overall scale effect, SCALE_{sy}, measured as overall total number of cases in year y, and the relative intensity of malaria transmission across months and states, REL_{sk}, which

[16] Vosti (1990), on the other hand, finds little correlation between socioeconomic status and malaria incidence when controlling for vector-related risk factors among a group of mining workers in Northern Brazil.

[17] A potential concern is that the eradication efforts might also have reduced the incidence of other vector-borne diseases such as yellow fever and dengue. Therefore, it is possible that my estimates are picking up the effects of other vector-borne diseases besides malaria. Appendix B.3 discusses the estimates for the prevalence of such diseases during the period analyzed in this paper and concludes that, although present in Brazil, their numbers were in a much smaller scale compared to malaria incidence and are not likely to contaminate the estimates below.

[18] If parents are able to systematically choose the month of birth of their offspring in response to seasonal malaria burden, my empirical strategy would likely be biased towards my estimates. In appendix B.7, I find no statistical association between individuals' month of birth and underlying socioeconomic characteristics, suggesting that timing of birth is not endogenous.

[19] Ideally, the measure of risk of malaria transmission would rely on average monthly malaria cases during the period around the eradication era. However, due to data limitations, monthly malaria cases can only be observed for the period 2007–2017, as detailed above.

is captured by the average number of reported cases between 2007 and 2017 at month k and state s as a ratio of the state-level mean number of cases throughout the given period.[20]

The importance of scaling the relative intensity monthly measure with state-level yearly cases during the timing of exposure is to capture the overall trends in malaria levels over the years in different areas. Since overall variations in malaria incidence across states might change over time, due to, for instance, state-level policy measures or unobservable factors (such as population characteristics), scaling the relative monthly incidence might improve precision of the constructed exposure variable by controlling for prevailing trends. An important drawback of such inclusion, however, is the possibility of introducing measurement error to the estimation, since the data does not allow for a precise disaggregated information regarding exact timing of in utero exposure.[21]

The implicit assumption carried out throughout the analysis, then, is that relative risk across month-state cells is constant across time. The implication of this assumption is that the eradication efforts was homogeneous across the country and 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.[22] The constructed measure to represent in utero exposure of individuals born in month m, state s, and year y is given by

$$\text{Exposure}_{smy} = \sum_{k=m-9}^{m-1} \text{REL}_{sk} \times \text{SCALE}_{sy},$$

where REL_{sk} denotes the average risk of transmission in month k and state s and SCALE_{sy} is the average number of reported cases between 2007 and 2017 at month k and state s, as described above.

[20]Specifically, I construct Exposure_{sky} as the ratio of average monthly-level number of reported cases to the mean number of cases between 2007 and 2017, at the state level, multiplied by the ratio of the state-level total number of cases in year y, per 1,000 inhabitants. For the years before 1959, for which overall malaria burden is unavailable, I consider the observed incidence in the year 1953, from Griffing et al. (2015).

[21]I thank an anonymous referee for pointing this out. Facing this trade-off, I decided to include the Scale component as an additional variation to the data. Given the high frequency of the data required to estimate in utero effects of exposure to malaria, the additional variation might help to improve the precision of the estimated coefficients. Moreover, estimation of the model without the scale effect (not shown) does not qualitatively change the results. Results are available upon request.

[22]The analysis essentially assumes that if the observed likelihood of being infected with malaria in state s at month m is twice as much as the one observed in state s' and month m' in 2007, it is also twice as likely to be infected with malaria in state s at month m relative to state s' and month m' in the years around the eradication era. Appendix C discusses the validity of this assumption in a great deal of details, including potential biases in the estimated results if the underlying assumption fails to hold.

I consider individuals born during the roll-out of the campaign to estimate the effects of early life exposure to malaria on socioeconomic conditions. The baseline equation to be estimated is given by

$$Outcome_{ismy} = \beta Exposure_{smy} + \alpha_c + \gamma_s + X'_{ismy}\phi + \varepsilon_{ismy}, \quad (2)$$

where $Exposure_{smy}$ is defined as the risk of transmission at a given month of birth m and state of birth s. The terms α_c and γ_s capture a cohort-specific fixed effects and the state of birth fixed effects, respectively, whereas X_{ismy} is a matrix of control variables. I estimate equation 2 for two consecutive birth cohorts: 1959 and 1960 (treatment and placebo cohorts, respectively, henceforth). Individuals born in 1959, in general, were largely exposed to malaria transmission while in utero, while postnatal exposure was relatively shortened.[23] 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 2 for the treatment birth cohort, while the treatment effect of early life exposure on individuals members of the placebo birth cohort is expected to be very small (if any). The large estimated decline in the malaria burden between these two adjacent years is of extreme relevance to compare outcomes of cohorts whose in utero exposure to malaria was dramatically different.[24] The identifying assumption of the research design, thus, is that any unobservable factors that affect human capital accumulation vary continuously over time (at least during the time frame considered in this paper).

An important concern is that 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 had some exposure while in utero. 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 are considered not

[23] According to the WHO, DDT spraying total coverage started in August, 1959.

[24] 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 1960. Assuming a linear path in the yearly reduction along this period, malaria rates would have been around 14.40 cases per 1,000 inhabitants per year in 1959; this corresponds to 22.85 times the intensity of exposure in the following year. However, considering the time of personnel training, and bureaucratic delays in the initial stage of the eradication, the decline in the malaria burden from 1959 to 1960 is likely to be larger.

exposed. 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 at a monthly level. Second, the results are robust to restricting the date of birth to specific month brackets around the years of 1959 and 1960.[25] Additionally, in table 2 and in appendix B.6, I construct broader definitions of birth cohorts to ensure that treatment birth cohorts were indeed exposed. The results are qualitatively similar across different birth cohorts.

According to 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 light of this potential heterogeneous treatment effects during different periods of early life, I employ the same strategy as defined above to identify 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

$$\text{Exposure}^{T}_{ms} = \sum_{i=m-k}^{m-n} \text{REL}_{si} \times \text{SCALE}_{y},$$

where the subscript T denotes the trimester of exposure, $k = 9, 6, 3$ and $n = 7, 4, 1$ whenever $T = 1, 2, 3$, respectively. The trimester exposure analysis, then, can be represented by

$$\text{Outcome}_{ismy} = \beta_1 \text{Exposure}^{1}_{msy} + \beta_2 \text{Exposure}^{2}_{msy} + \beta_3 \text{Exposure}^{3}_{msy} + \alpha_c + \gamma_s + X'_{imsy}\phi + \varepsilon_{ims}, \tag{3}$$

where the variable definitions are as above.

The empirical strategy described above has some limitations and challenges that are worth mentioning. First, general equilibrium effects might 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 groups. The net general equilibrium effect in this situation is uncertain.

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

Second, 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. In appendix B.1, I show that mortality selection is not a first-order issue in my main analysis.

Another important concern is that the constructed measure of malaria exposure is contaminated with measurement error, should the assumption regarding the dynamics of malaria transmission across locations fail to hold. Appendices B.4 and B.5 provide Instrumental Variables estimates and appendix B7 tests for several different proxies for exposure, corroborating the main findings of this paper and alleviating concerns related to the specific construction of the exposure measure in this paper introducing measurement error to the estimation.

Finally, the estimated coefficients might overestimate the independent impact of malaria if exposure to DDT has long-run negative consequences to educational attainment.[26] Given the empirical limitation in controlling for such effect, my estimated results can be interpreted as an upper bound for the effects of exposure to malaria. However, to rule out the possibility that the results are entirely driven by the effects of the DDT, I use a proxy for in utero exposure to malaria based on the intrinsic climatic factors that determine the survival and proliferation of both vector and parasite and estimate its effects on educational attainment of cohorts born before the time in which DDT was first used in Brazil to fight malaria, in 1945.[27] The results corroborate the main findings of this paper, suggesting that in utero exposure to malaria indeed has a negative effect on education that is independent of DDT exposure.[28]

4 Main Results

To estimate the effects of in utero malaria exposure on adult outcomes across treatment and placebo groups (1959 and 1960 birth cohorts respectively), it is necessary that individuals are similar along different socioeconomic dimensions to ensure comparability across both groups. Panels A and B of table 1 describes the summary statistics for the main variables analyzed in this paper for the 1959 and 1960 birth cohorts separately. In general, both samples are similar

[26] Medical studies have documented a positive association between DDT exposure and cognitive development among infants and children in different contexts (Eskenazi et al., 2006; Ribas-Fito et al., 2006; Jusko et al., 2006; Eskenazi et al., 2008; Al-Saleh et al., 2012; Jusko et al., 2012).

[27] Lack of data on reported cases at earlier dates precludes the use of the main exposure variable. See appendix B.8 for a detailed description of the construction of this alternative measure of exposure.

[28] Results are available upon request.

in all dimensions, suggesting balance across individuals on different group cohorts, on average. Additionally, panel C displays some summary statistics of the constructed exposure variables. Consistent with the overall trends, individuals born in 1959 were relatively more exposed to malaria while in utero than the 1960 birth cohort.

The first set of results below addresses estimates of 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. Then, I consider the potential long-term consequences of in utero exposure to malaria on a range of different socioeconomic conditions as well as potential differential treatment effects across the income distribution.

4.1 Average In Utero Effects

If early life exposure to malaria affects long-term outcomes, the baseline estimation in equation 2 should display stronger effects on the treatment cohort. Since the sample is restricted to only two consecutive birth cohorts, any differences in long-term outcomes can plausibly 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.[29] Column (1) displays the coefficient of the simple regression, without controls. The results suggest no discernible relationship between in-utero malaria exposure and educational attainment. However, in column (2), after controlling for sociodemographic and economic characteristics, the results suggest a statistically significant effect or the treatment birth cohort sample. Individuals in utero in months with higher risk of exposure to malaria, on average, have less years of education than less-exposed individuals during the same period.[30] Column (3) performs the previous estimation including state-of-birth fixed effects, which essentially captures the effect of monthly-level differences in malaria exposure by shutting down the between-states variation mechanism. The size of the coefficient is somewhat smaller but still economically meaningful and statistically significant, corroborating the hypothe-

[29]Standard errors are clustered at the state-month level in all regressions.

[30]The full set of controls include individual-level demographic characteristics (gender, age, race), as well as socioeconomic confounding factors that might be correlated with the treatment variable (sector of activity, migration and rural status). Following Bleakley (2010b), I include sector of activity as a control to capture sectoral shocks from import substitution policies in place during the analyzed period. Migration status is added to control for baseline differences in educational attainment between migrants and non-migrants. Finally, rural status controls for educational gaps across rural and urban residents.

sis that within-state seasonal exposure to malaria is an important component of the main overall effect.

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.[31] The results in table 2 indicate a negative effect of in utero exposure to malaria on years of education and primary completion on treated cohorts (born in 1959). The point estimate for the effect of in utero exposure on educational attainment is -17.52 (column 3). The mean value of the in utero risk is 0.0436, with a standard deviation of 0.0029. An increase in malaria risk in the magnitude of one standard deviation would induce an effect of 0.0029 times the estimated coefficient. Therefore, a one standard deviation increase in exposure, as measured by the risk of in utero malaria infection, leads to, on average, 0.0516 fewer years of education. Carrying out the same computations above for the primary degree coefficient (-1.634), a one standard deviation increase of malaria risk would generate an estimated reduction of 0.48 percentage points on the probability of completing primary education. The results in Panel A further indicate statistically significant treatment effect of in utero exposure to malaria on secondary education completion, although smaller in magnitude and less significant. The small effect size on higher education seems to suggest that early life malaria exposure affects different socioeconomic groups of the population in an heterogeneous manner, given that black (including *pardos*) *and* poor population in Brazil are disproportionately less represented in college enrollment rates.[32] Finally, 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.

[31] Although the independent variable of the analysis is expressed in terms of number of cases, it does not carry a direct interpretation of the magnitude of the estimated coefficients, since I construct a *relative* measure of malaria intensity based on available incidence data.

[32] For example, in my sample, 13.6 percent of whites have completed tertiary education, whereas this share is only 5.3 percent for non-whites. College degree is also unequal across the income distribution: among the bottom 10%, only 2.5% of the individuals in the sample have college degree, whereas this share is about 16.9% among the top 10%. I return to the issue of the heterogeneity of the results in section 4.4.3.

4.2 Exploring Heterogeneous Effects According to Different Trimesters of In Utero Early Life

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 2 for exposure in each trimester of pregnancy for both birth cohort groups (1959 and 1960).

The results indicate a negative and statistically significant long-run effect of exposure during the first three months of pregnancy on human capital accumulation in the pre-eradication birth cohort, as observed in Panel A. In general, higher in utero exposure to malaria during first trimester leads to less years of education and lower likelihood of primary as well as secondary completion. The point estimates are qualitatively similar to ones detailed in table 2, albeit somewhat larger magnitudes. In fact, these results seem to corroborate theoretical predictions in which 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.[33]

Exposure to malaria in the second trimester of pregnancy seems to also have negative impact on years of education, albeit a smaller treatment effect when compared to exposure during the first trimester.[34] These 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. The results also do not indicate any treatment effect of exposure during the second trimester on the other outcomes, as shown in columns (4) through (12) of table 3. 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 in Panel B indicate no exposure effects on educational outcomes, except for secondary education, during the first trimester, as shown in column (5) of table 3.

[33] Fetuses with symmetric growth restrictions are more likely to develop permanent neurological sequalae (Umbers et al., 2011).

[34] However, the results are only statistically significant at the 10% level for the specification with state-of-birth fixed effects.

4.3 Discussion and Extension of the Main Results

The main results of this paper suggest a small albeit significant effect of early life exposure to malaria on educational attainment. The results are similar in magnitude to other studies that estimate the impact of early life health insults on human capital in different contexts. For example, Barreca (2010) finds that an increase of 10 deaths per 100,000 inhabitants is associated with a reduction of about 0.4 years of completed education and a 5 percent lower probability of secondary education completion. Moreover, employing differences-in-differences strategies to estimate the gains in education after eradication programs, Cutler et al. (2010) and Lucas (2010) also find modest effects: 2.5 to 5.6 percentage points and 0.1 years, respectively. Although exposure is measured somewhat differently, my results suggest a reduction of 1.75 years of education following an increase in exposure by 10 cases per 100,000 inhabitants (or, alternatively, a 0.14 years reduction after a 10 percentage points increase in exposure).[35]

Among studies that address in utero exposure to other health environmental shocks, Almond (2006) and Lin and Liu (2014), for instance, find significantly lower educational attainment of individuals who were in utero during the 1918 Flu Pandemic (between 0.04 and 0.4 less years of education). Field et al. (2009) find an increased 0.5 years of education of children who were in utero during a iodine supplementation program in Tanzania. The magnitudes of these results suggest comparable effects with the ones in this paper.

Other Socioeconomic Outcomes

Given the sizable negative effects of in utero exposure to malaria on educational attainment, I test whether these effects are also observed in other socioeconomic outcomes. Particularly, in table 4, I provide the estimates for both average (Panel A) and trimester effects (Panel B) of in utero exposure to malaria on literacy, personal income, and whether the individual works in the agricultural sector for the two consecutive birth cohorts (1959 and 1960). First, there does not seem to be any significant effects of timing of in utero exposure on either literacy or personal

[35] However, it is worth mentioning that the constructed exposure measure in this paper does not directly translate into number of cases, since I use information on reported cases at different periods to assess relative differences in exposure across locations.

income on either cohort (columns 1 through 4).[36] Another important socioeconomic outcome that might have been affected by early life health shocks is the sector of activity in which individuals work.[37] Columns (5) and (6) address the question of whether early life exposure to malaria during the Eradication era might have played a role in this process. As the results show, the point estimates for both birth cohorts are positive, suggesting that early life exposure to malaria is associated with a higher probability of working in the low productivity agriculture sector. As expected, the results are only statistically significant and larger in magnitude for the pre-eradication group.[38]

Heterogeneity of the Effects

Across the Income Distribution: Given the main effects of in utero exposure on educational attainment reported in tables 2 and 3, a natural subsequent question is whether these results are observed across all the income distribution. To answer this question, I re-estimate the model for different quartiles, as shown in table 5.

In general, in utero malaria exposure reduces years of education on first and third quartiles, as observed in figure 4a and table 5. Primary education completion is affected by in utero exposure only for the individuals at or below the second quartile, whereas the effects on secondary and tertiary degrees are observed on the third and fourth quartiles of the income distribution, respectively, as seen in figure 4b and table 5. These results are intuitive since lower income population groups generally do not have access to higher degree education, and only exposed individuals at the top of the income distribution are less likely to complete either secondary or tertiary education. Importantly, the observed treatment effect of early life exposure to malaria on primary completion only on lower-income groups can shed light on the potential exacerbation of inequality by the differential impacts from exposure to early life insults.

[36] 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 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.

[37] Sector of activity might provide additional (and a less noisy) information about the socioeconomic status of the individuals in the sample.

[38] Sawyer (1993) shows that economic mobility and the distribution of the economic activity might depend on the incidence of malaria. Specifically, malaria-ridden areas might render difficult the establishment of permanent settlements for agriculture production, attracting only temporary labor for other activities.

Across Socioeconomic Groups: Another important dimension of heterogeneity of the effects is related to the socioeconomic and demographic characteristics of the population. In this section, I estimate the long-run effects of in utero exposure to malaria across race and gender, documented in table 6.

While higher in utero exposure to malaria for whites is associated with lower probability of completing both primary and secondary degrees, the effects are somewhat smaller and not significant among non-whites on the 1959 birth cohort. There are two competing effects that might determine the long-run consequences of in utero exposure to environmental factors, such as malaria incidence: a (negative) *scarring* effect on the survivors' educational attainment, and a (positive) selection effect, which happens when only the healthiest individuals survive.[39] Selection effects are a possible factor mitigating the *scarring* negative effects observed on non-white individuals, usually the demographic group facing the most challenging early life circumstances (Marteleto, 2012; Wood et al., 2010; Barros et al., 2001; Gradín, 2009).[40]

The results in table 6 further indicate net *scarring* effects of in utero exposure to malaria on both men and women. In general, the effects are somewhat stronger for primary completion on women, again suggesting that a selection mechanism might be at play given that male fetuses are usually more vulnerable to environmental shocks than female ones. It is worth noting further that I find little to no effect of in utero exposure to malaria for the 1960 birth cohort, further corroborating the hypothesis that this particular comparison group has indeed received a placebo treatment.

Despite the evidence for potential selection effects discussed above, it is important to note that it is likely to play a limited role in my estimates, as opposed to Rawlings (2016)'s, in which selection dominates the net effect on the estimates for men, for example. Indeed, Kudamatsu et al. (2012) finds that selection might be a more important contributing factor to the overall effects in epidemic locations relative to endemic ones (Rawlings, 2016).

[39] Rawlings (2016), for example, found consistent heterogeneity of the effects of early life exposure to a malaria epidemic outbreak in two states in Brazil across gender and race, with the results suggesting important selection effects, specifically among the non-white women group.

[40] In Brazil, race can be used as a *proxy* for socioeconomic status (SES), given the sizable correlation between them (Rawlings, 2016; Harris et al., 1993; Barros et al., 2001).

5 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 strategy, since unobserved socioeconomic differences across different groups of people might otherwise 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 disproportionately affect highly exposed pregnant women 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 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 to identify the extent of the in utero exposure.

The results found on this paper suggest that initial health plays an important role in adult human capital accumulation. 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 about 0.2 years more of education, and be 2 percentage points more likely to be primary graduate 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. Moreover, the poor population face stronger effects, given the lack of sufficient and adequate resources to protect against malaria exposure or even treatment upon infections. Therefore, policies devoted to improve socioeconomic conditions in afflicted areas would likely be effective and achieve long-run desired outcomes should they target low-income and pregnant women, the vulnerable socioeconomic groups – especially those in early stages of gestation. The results in this paper suggest potential future relative gains in socioeconomic conditions for these particular groups.

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, as well as its impact on life expectancy and adult mortality. 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. Unfortunately, comprehensive official micro-level data on test scores are only available starting from 1995, after the malaria eradication efforts. 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.

Figures

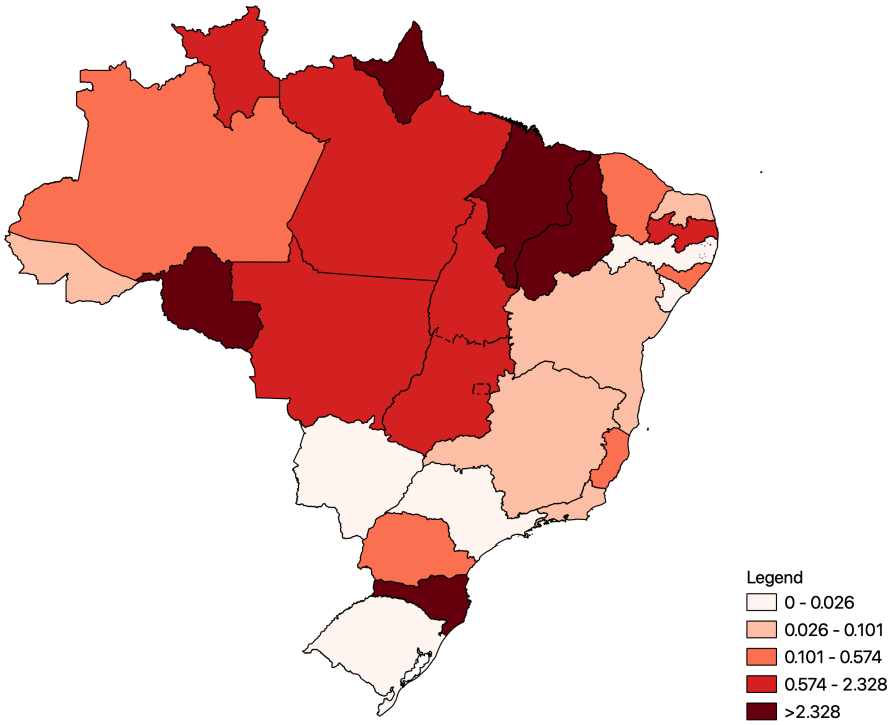


Figure 1: State-Level Average Malaria Cases – 1959. Source: DATASUS – Ministry of Health. The figure represents state-level average reported number of cases per 1,000 inhabitants at in 1959 (the first period of available state-level malaria incidence). The figure depicts the state-level variation in malaria incidence that identifies the heterogeneity in the scale of the risk of malaria infections for the pre-eradication birth cohort.

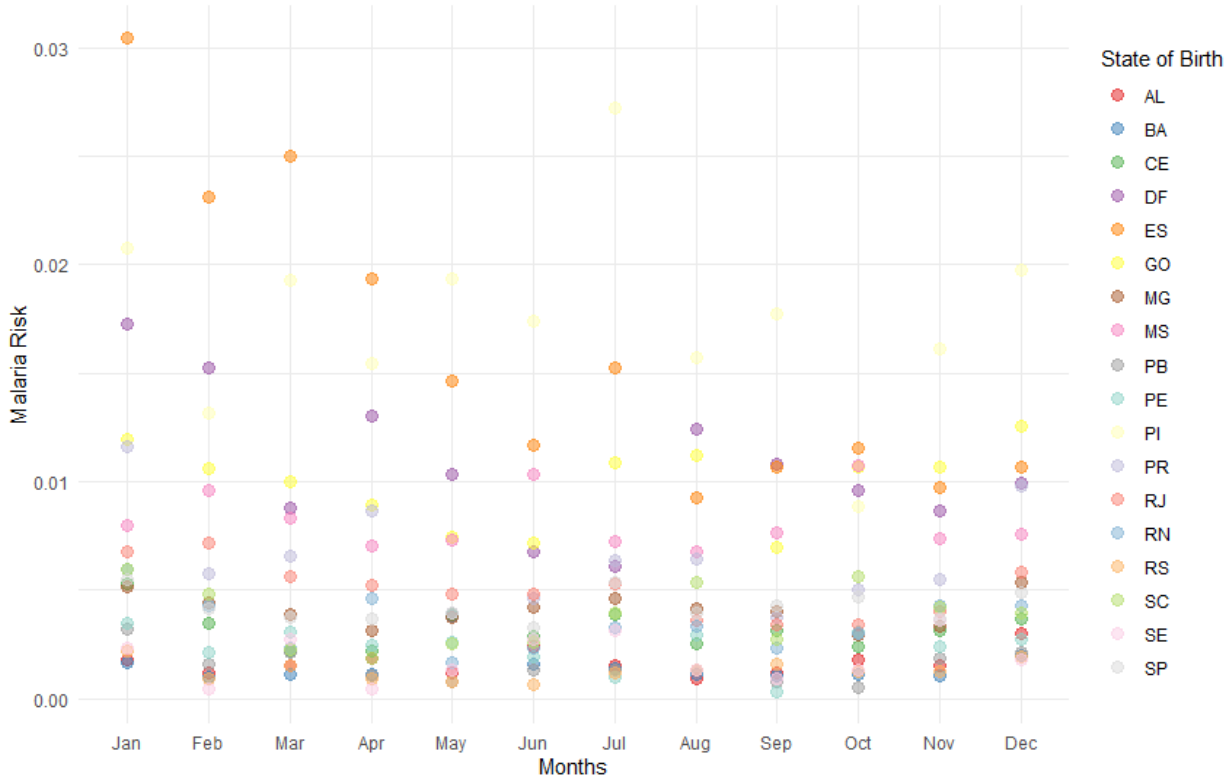


Figure 2: State-Level Monthly Average Malaria Cases per 1,000 inhabitants (2007–2017). Source: DATASUS – Ministry of Health. The figure represents state-level average reported number of cases per 1,000 inhabitants at the monthly level for the period 2007–2017. The figure depicts the malaria incidence variation both at the state- and month-level that identifies the source of heterogeneity in exposure across individuals in the sample.

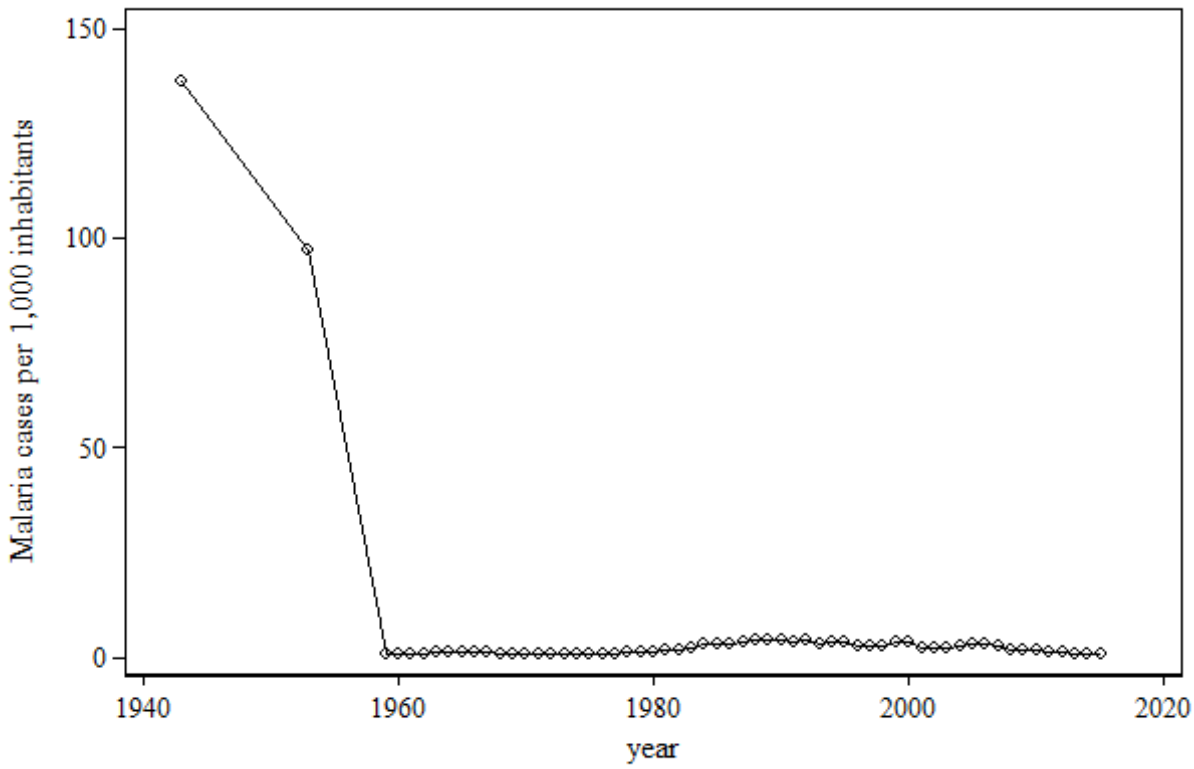
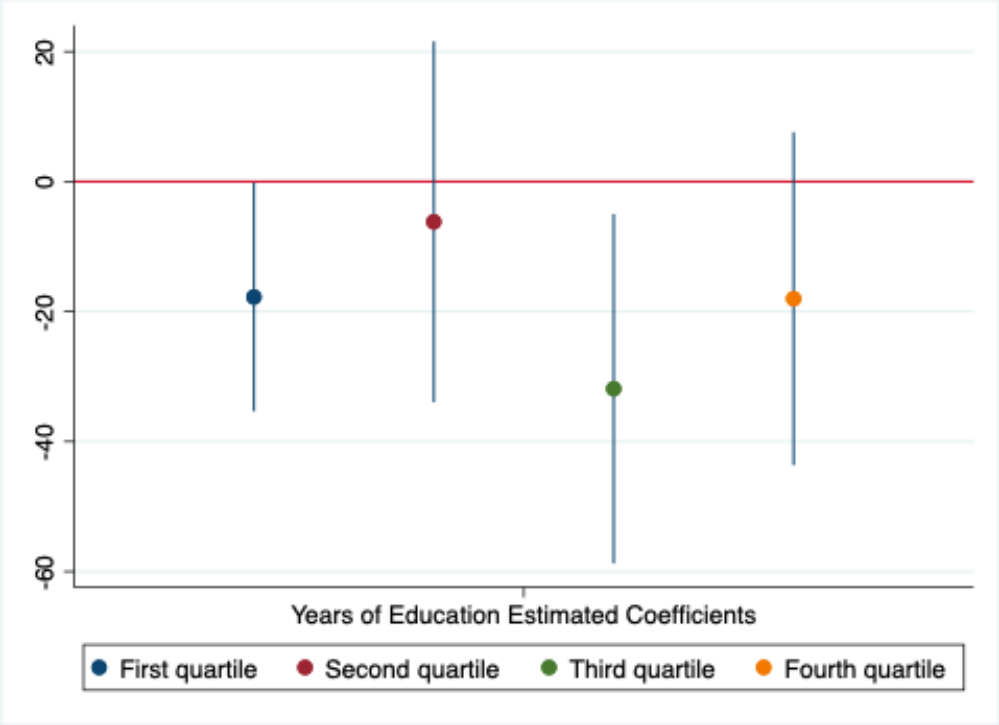
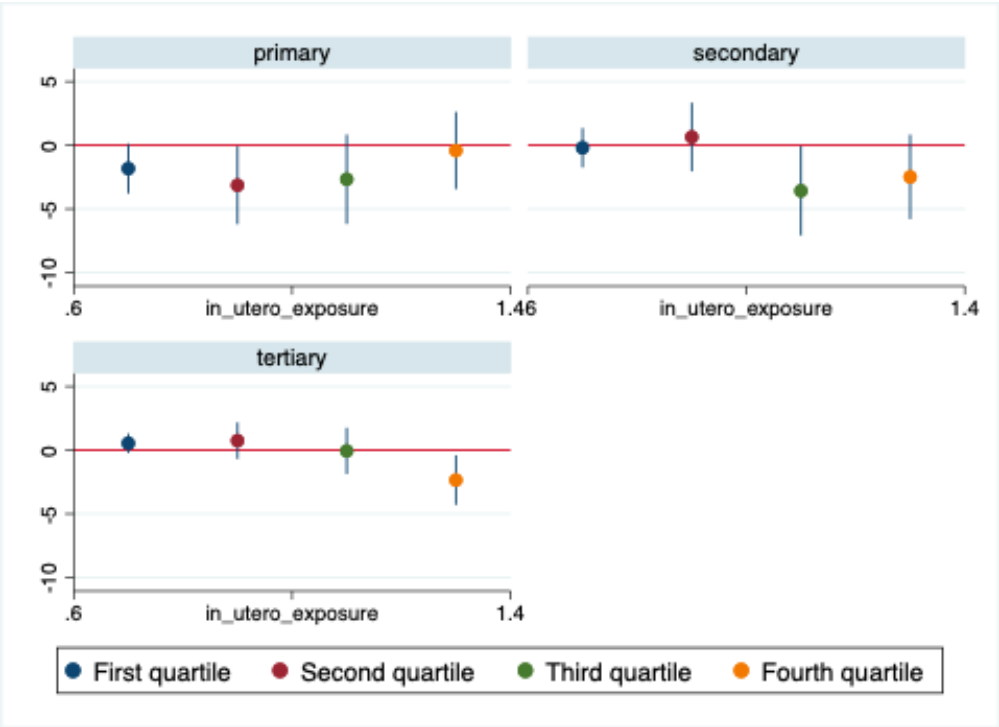


Figure 3: Reduction in Malaria Cases per 1,000 inhabitants after the Eradication Campaign. Source: DATASUS – Ministry of Health and Griffing et al. (2015). The figure represents the time series of the average reported number of cases per 1,000 inhabitants at the yearly level for the whole country during the period between 1959–2017. The figure adds two data points for the years of 1943 and 1953 based on estimates from Griffing et al. (2015). The figure depicts the large scale and rapid reduction of malaria incidence after the malaria eradication campaign started in 1957.



(a) Effects of In utero Exposure to Malaria on Years of Education across the Income Distribution



(b) Effects of In utero Exposure to Malaria on Degree Completion across the Income Distribution

Figure 4: The Effects of In Utero Exposure to Malaria Across the Income Distribution. The figure depicts OLS estimation coefficients and associated 95% confidence intervals for the effects of average in utero exposure, expressed in terms of state-level monthly average cases, on human capital accumulation across different quartiles of the income distribution for the pre-eradication birth cohort. All regressions include full set of controls, state fixed effects and survey year fixed effects.

Tables

Table 1: Summary Statistics

	1959 birth cohort		1960 birth cohort	
	Mean	S.D.	Mean	S.D.
Panel A. Outcome variables				
Years of Education	6.84	4.57	6.85	4.57
Primary	0.59	0.49	0.55	0.50
Secondary	0.32	0.47	0.31	0.46
Tertiary	0.10	0.30	0.10	0.30
Panel B. Demographic Characteristics				
White	0.55	0.50	0.54	0.50
Indigenous	0.00	0.05	0.00	0.05
Black	0.07	0.26	0.07	0.26
Parda	0.37	0.48	0.38	0.49
Mover	0.23	0.42	0.22	0.42
Rural	0.15	0.36	0.15	0.36
Age	44.57	6.84	43.64	6.90
Panel C. Exposure Measures				
Avg In utero Exposure	0.0436	0.0028	0.0388	0.0026
First Trimester Exposure	0.0145	0.0028	0.0129	0.0025
Second Trimester Exposure	0.0146	0.0028	0.0129	0.0025
Third Trimester Exposure	0.0145	0.0028	0.0129	0.0025
Number of Observations	91,414		102,126	
Number of States	18			

Notes. Means and standard deviations are weighted using sample weights. Sample includes individuals 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, the proportion of individuals living in rural areas, and age. Panel C reports average and standard deviation of the constructed exposure measures.

Table 2: Early Life Exposure to Malaria OLS Results – Baseline Exposure

	Years of Education			Primary			Secondary			Tertiary		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
Panel A. Pre-Eradication Birth Cohort (1959)												
In utero Exposure	−21.95	−30.47**	−17.52***	−1.915	−3.168*	−1.634**	−0.577	−1.545	−1.323*	−0.012	0.121	−0.390
	(32.93)	(16.84)	(6.079)	(3.128)	(1.877)	(0.669)	(2.280)	(1.454)	(0.768)	(1.227)	(0.882)	(0.420)
R-squared	0.000	0.271	0.289	0.000	0.139	0.158	0.000	0.155	0.167	0.000	0.096	0.105
Observations	81,772	57,907	57,907	82,050	58,119	58,119	82,050	58,119	58,119	82,050	58,119	58,119
Panel B. Post-Eradication Birth Cohort (1960)												
In utero Exposure	−19.43	−17.44	3.526	−1.360	−2.601	−1.024	−0.860	−1.417	−0.482	−0.293	0.00891	0.248
	(37.40)	(19.92)	(5.685)	3.489)	(2.298)	(0.661)	(2.554)	(1.494)	(0.696)	(1.302)	(0.870)	(0.530)
R-squared	0.000	0.239	0.294	0.000	0.128	0.168	0.000	0.127	0.169	0.000	0.059	0.103
Observations	90,789	64,744	64,744	91,098	64,971	64,971	91,098	64,971	64,971	91,098	64,971	64,971
Full Set of Controls		✓	✓		✓	✓		✓	✓		✓	✓
State Fixed Effects			✓			✓			✓			✓
Survey Year Fixed Effects		✓	✓		✓	✓		✓	✓		✓	✓

Notes. OLS estimates with robust standard errors clustered at the state-month 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 in utero exposure, expressed in terms of state-level monthly average cases. Legal Amazon is excluded. Individual controls include gender, age, race, sector of activity, migration and rural status. All regressions include survey year fixed effects. *** p<0.01, ** p<0.05, * p<0.1

Table 3: Early Life Exposure to Malaria – Exposure by Trimester

	Years of Education			Primary			Secondary			Tertiary		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
Panel A. Pre-Eradication Birth Cohort (1959)												
First Trimester Exposure	−33.57	−38.16**	−26.25***	−3.013	−4.035*	−2.746***	−2.281	−2.525	−2.225**	−0.559	−0.712	−0.935*
	(41.85)	(19.19)	(7.643)	(4.045)	(2.190)	(0.779)	(2.975)	(1.761)	(0.889)	(1.541)	(1.001)	(0.509)
Second Trimester Exposure	−28.50	−28.89	−14.13*	−2.452	−3.022	−1.229	−0.576	−1.051	−0.752	−0.331	−0.135	−0.346
	(39.83)	(20.94)	(7.692)	(3.696)	(2.226)	(0.849)	(2.900)	(1.877)	(0.922)	(1.420)	(1.115)	(0.518)
Third Trimester Exposure	−2.553	−20.48	−8.164	−0.184	−1.990	−0.396	1.079	−0.730	−0.710	0.913	0.859	0.464
	(43.18)	(21.67)	(7.674)	(4.223)	(2.480)	(0.864)	(3.055)	(1.985)	(0.953)	(1.653)	(1.243)	(0.514)
R-squared	0.000	0.271	0.289	0.000	0.139	0.158	0.000	0.155	0.167	0.000	0.096	0.105
Observations	81,772	57,907	57,907	82,050	58,119	58,119	82,050	58,119	58,119	82,050	58,119	58,119
Panel B. Post-Eradication Birth Cohort (1960)												
First Trimester Exposure	−12.53	−14.00	5.958	−1.114	−2.598	−1.062	−1.229	−1.677	−0.755	0.236	0.277	0.517
	(49.24)	(23.91)	(6.971)	(4.643)	(2.733)	(0.802)	(3.427)	(1.877)	(0.873)	(1.774)	(1.102)	(0.611)
Second Trimester Exposure	−28.05	−23.09	−1.116	−1.999	−2.759	−1.128	−1.460	−1.693	−0.647	−0.799	−0.541	−0.255
	(43.99)	(22.69)	(7.053)	(3.971)	(2.536)	(0.772)	(3.042)	(1.768)	(0.823)	(1.447)	(0.967)	(0.562)
Third Trimester Exposure	−15.59	−13.97	7.029	−0.811	−2.345	−0.781	0.249	−0.520	0.287	−0.189	0.463	0.619
	(48.88)	(26.47)	(7.732)	(4.649)	(2.959)	(0.942)	(3.311)	(2.007)	(0.834)	(1.743)	(1.258)	(0.697)
R-squared	0.000	0.271	0.294	0.000	0.147	0.168	0.000	0.157	0.169	0.000	0.095	0.103
Observations	90,789	64,744	64,744	91,098	64,971	64,971	91,098	64,971	64,971	91,098	64,971	64,971
Full Set of Controls		✓	✓		✓	✓		✓	✓		✓	✓
State Fixed Effects			✓			✓			✓			✓
Survey Year Fixed Effects		✓	✓		✓	✓		✓	✓		✓	✓

Notes. OLS estimates with robust standard errors clustered at the state-month 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 exposure on each of the three trimesters while in utero, expressed in terms of state-level monthly average cases. Legal Amazon is excluded. Individual controls include gender, age, race, sector of activity, migration and rural status. All regressions include survey year fixed effects. *** p<0.01, ** p<0.05, * p<0.1

Table 4: Early Life Exposure to Malaria on Socioeconomic Outcomes

	Literacy		Log(Income)		Agriculture	
	(1)	(2)	(3)	(4)	(5)	(6)
	(1959)	(1960)	(1959)	(1960)	(1959)	(1960)
	Panel A. Average In Utero Exposure					
Average Treatment Effect	0.249	0.308	0.845	−1.095	0.797**	0.208
	(0.460)	(0.610)	(1.637)	(1.483)	(0.378)	(0.355)
R-squared	0.163	0.162	0.274	0.282	0.363	0.360
Observations	58,116	64,969	51,716	57,573	82,040	91,084
	Panel B. Trimester Exposure					
First Trimester Exposure	0.614	0.561	−0.00911	−1.199	1.030**	0.536
	(0.518)	(0.742)	(1.920)	(1.782)	(0.462)	(0.402)
Second Trimester Exposure	−0.226	0.00821	0.753	−0.346	0.642	−0.0779
	(0.577)	(0.646)	(1.929)	(1.938)	(0.437)	(0.471)
Third Trimester Exposure	0.392	0.363	2.471	−2.201	0.647	0.0925
	(0.533)	(0.761)	(1.971)	(2.087)	(0.451)	(0.471)
R-squared	0.163	0.162	0.274	0.282	0.363	0.360
Observations	58,116	64,969	51,716	57,573	82,040	91,084
Full Set of Controls	✓	✓	✓	✓	✓	✓
State Fixed Effects	✓	✓	✓	✓	✓	✓
Survey Year Fixed Effects	✓	✓	✓	✓	✓	✓

Notes. OLS estimates with robust standard errors clustered at the state-month 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 in utero exposure, expressed in terms of state-level monthly average cases in Panel A. In Panel B, treatment is identified by the average exposure on each of the three trimesters while in utero, expressed in terms of state-level monthly average cases. Legal Amazon is excluded. Individual controls include gender, age, race, sector of activity, migration and rural status. All regressions include state fixed effects and survey year fixed effects. *** p<0.01, ** p<0.05, * p<0.1

Table 5: Early Life Exposure to Malaria by Income Groups – Baseline Exposure

	Years of Education		Primary		Secondary		Tertiary	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	(1959)	(1960)	(1959)	(1960)	(1959)	(1960)	(1959)	(1960)
In utero Exposure								
Bottom 25%	−17.76**	1.025	−1.830*	0.666	−0.196	−0.830	0.543	0.0107
	(8.930)	(10.13)	(0.995)	(1.237)	(0.786)	(0.813)	(0.387)	(0.413)
R-squared	0.212	0.211	0.122	0.115	0.067	0.061	0.022	0.020
Observations	16,426	18,645	16,480	18,710	16,480	18,710	16,480	18,710
25%–50%	−6.193	−7.760	−3.141**	−1.503	0.646	−0.928	0.746	0.206
	(14.09)	(14.06)	(1.551)	(1.945)	(1.368)	(1.843)	(0.728)	(0.457)
R-squared	0.170	0.182	0.090	0.101	0.101	0.097	0.024	0.019
Observations	11,081	12,870	11,132	12,924	11,132	12,924	11,132	12,924
50%–75%	−31.88**	27.04**	−2.670	−0.545	−3.579**	0.628	−0.0580	1.994**
	(13.64)	(13.20)	(1.782)	(1.707)	(1.780)	(1.759)	(0.921)	(0.910)
R-squared	0.235	0.240	0.093	0.103	0.155	0.155	0.093	0.095
Observations	12,140	13,597	12,207	13,664	12,207	13,664	12,207	13,664
75%–100%	−18.03	9.277	−0.414	−1.932	−2.485	0.401	−2.349**	−0.156
	(13.00)	(12.88)	(1.540)	(1.252)	(1.684)	(1.464)	(0.989)	(1.501)
R-squared	0.319	0.322	0.113	0.131	0.183	0.188	0.207	0.205
Observations	18,260	19,632	18,300	19,673	18,300	19,673	18,300	19,673
Full Set of Controls	✓	✓	✓	✓	✓	✓	✓	✓
State Fixed Effects	✓	✓	✓	✓	✓	✓	✓	✓
Survey Year Fixed Effects	✓	✓	✓	✓	✓	✓	✓	✓

Notes. OLS estimates with robust standard errors clustered at the state-month 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 in utero exposure, expressed in terms of state-level monthly average cases. Legal Amazon is excluded. Individual controls include gender, age, race, sector of activity, migration and rural status. All regressions include state fixed effects and survey year fixed effects. *** p<0.01, ** p<0.05, * p<0.1

Table 6: Early Life Exposure to Malaria by Socioeconomic Characteristics

	Years of Education				Primary				Secondary				Tertiary			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
	(1959)	(1959)	(1960)	(1960)	(1959)	(1959)	(1960)	(1960)	(1959)	(1959)	(1960)	(1960)	(1959)	(1959)	(1960)	(1960)
In utero Exposure																
Race																
White	−30.95***		0.397		−2.703***		−1.750		−2.998***		−0.939		−0.826		0.210	
	(9.763)		(10.99)		(0.997)		(1.106)		(1.132)		(1.210)		(0.742)		(0.813)	
Non-white		−5.369		5.062		−0.723		−0.381		0.215		−0.231		0.00295		0.188
		(9.333)		(9.451)		(0.980)		(1.012)		(1.131)		(1.143)		(0.590)		(0.649)
R-squared	0.233	0.243	0.241	0.251	0.105	0.145	0.126	0.148	0.153	0.114	0.154	0.123	0.103	0.050	0.105	0.049
Observations	31,835	26,072	34,929	29,815	31,944	26,175	35,046	29,925	31,944	26,175	35,046	29,925	31,944	26,175	35,046	29,925
Gender																
Female	−16.65*		5.219		−2.752***		−0.340		−1.322		−0.739		−0.325		0.245	
	(10.02)		(9.977)		(1.029)		(1.024)		(1.103)		(1.115)		(0.754)		(0.890)	
Male		−16.35**		1.490		−0.542		−1.583*		−1.141		−0.305		−0.391		0.331
		(7.497)		(8.019)		(1.033)		(0.916)		(0.857)		(0.906)		(0.595)		(0.587)
R-squared	0.268	0.314	0.261	0.326	0.146	0.170	0.145	0.184	0.165	0.175	0.163	0.178	0.107	0.102	0.104	0.103
Observations	25,282	32,625	28,266	36,478	25,388	32,731	28,390	36,581	25,388	32,731	28,390	36,581	25,388	32,731	28,390	36,581
Full Set of Controls	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
State Fixed Effects	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
Survey Year Fixed Effects	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓

Notes. OLS estimates with robust standard errors clustered at the state-month 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 in utero exposure, expressed in terms of state-level monthly average cases. Legal Amazon is excluded. Individual controls include gender, age, race, sector of activity, migration and rural status. All regressions include survey year fixed effects. *** p<0.01, ** p<0.05, * p<0.1

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Appendix A. Data sources and Details

Malaria Variables

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[41]. The malaria risk variable is number of reported cases per 1,000 inhabitants for a given state and month.[42]

Individual-level outcomes and controls

Education

The education variables used throughout the analysis are number of years of education which corresponds to the number of completed years in school, as well as indicator variables for degree completion, which are constructed based on the survey question referring to the last completed degree. 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 observations whose hourly income is higher than 90 percentile. In the analysis, I use the natural log of income. The primary data source is the Brazilian National Household Sample Survey (PNAD).

Health

The health variable is given by the self-reported general health status. Respondents categorize their perceived general health by reporting a number from 1 (excellent) to 5 (poor). 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 the self-reported individuals' race.
Mover: dummy variable indicating whether the individual resides in the state of birth.
Age: constructed variable, based on self-reported individuals' date of birth.
Sector of activity: Sector of main activity in the previous week. I define the following groups as separate sectors of activity: agriculture, manufacturing, construction, other industries, services, transport and communications, public administration, and other activities.
The primary data source is the Brazilian National Household Sample Survey (PNAD).

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

[41] http://www.ipeadata.gov.br/Default.aspx Accessed on 10/17/2018.
[42] Population data is given at an yearly basis. I use the same constant population for each month in a given year.

– Global Climate Data (v.2), available at http://worldclim.org/version2. The climatic data set 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. Sensitivity Analysis

In this section, I discuss several possible alternative mechanisms that might affect the main results of this paper. First, 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. Second, I discuss the potential issue of age heaping in my data, followed by the role of other vector-borne diseases in explaining the results in my analysis. The subsequent section deals with potential omitted variable bias and measurement error in my main specification. Specifically, I construct a set of instruments based on the intrinsic relationship between malaria transmission and climatic factors, namely temperature and precipitation. The plausibility of the exclusion restriction assumption of the Instrumental Variables estimation is discussed in the following subsection. Next, it is possible that the observed effects are not robust to different choices of cohorts (both high versus low in utero exposure). I address this issue by testing different timings of birth in defining exposure across cohorts. Then, I address the potential endogeneity associated with timing of birth, followed by a final subsection testing for alternative measures of exposure.

B.1 Mortality Selection

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 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.[43] I address the issue more formally by testing whether more exposed women at childbearing age during pre-eradication era (i.e. born prior to 1914) had higher likelihood of miscarriages or stillbirths.[44] In particular, I use number of deceased children while in utero as the dependent variable, and I use a state-level malaria ecology-based index (henceforth MSI) , which captures the stability and strength of malaria transmission, as the main independent variable.[45] Figure B1 depicts the distribution of the MSI in Brazil.

[43] *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.

[44] Introducing younger cohorts only corroborate the main results of this section.

[45] Specifically, 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. The resulting index is defined as

$$\sum_{m=1}^{12} a_{i,m}^2 p_{i,m}^E / -\ln(p_{i,m}),$$

where m = month, i = identity of the dominant vector, a = proportion of biting people (0-1), and p = daily survival rate, and E = Extrinsic incubation period in days.

The estimation uses the following set of controls: race, migration status, whether the respondent lives in a rural area, sector of employment, age, year of birth, and number of children.[46] All estimates also include birth cohort fixed effects, state of birth fixed effects.

The results are shown in table B1. Column 1 tests whether differential exposure to malaria prior to the eradication era is associated with differences in the number of miscarriages and/or stillbirths.[47] Columns 2 and 3 break down the analysis for boys and girls, whereas column 4 displays the results of a Linear Probability Model (LPM) where the dependent variable is an indicator of whether the respondent has ever had either a miscarriage or stillbirth. The results indicate no relationship between malaria burden and mortality differences among women who experienced greater or lesser levels of exposure to the disease.

An important limitation of the above analysis is that miscarriages are likely to be considerably underreported, since most of them happen within the first weeks of gestation, when the mothers might not even be aware of the pregnancy. Column 5 addresses this issue by reporting the effects of exposure to malaria on the sex ratio of births among childbearing aged women prior and post eradication. Specifically, I run a Differences-in-Differences (DID) model, where the (continuous) treatment variable is the malaria stability index interacted with the Post dummy, indicating whether the individual has spent her childbearing period after the eradication period, as defined below:

$$Sex\,ratio_{ismy} = \beta MSI_s \times Post + \gamma Post + \eta MSI_s + \alpha_c + X'_{isy} \times Post\phi + \varepsilon_{isy}, \tag{4}$$

where $Sex\,ratio_{isy}$ is defined as the number of surviving boys relative to the number of surviving girls for each woman i aged 45 or more, born in state s and year y; Post is a dummy variable indicating whether the individual was born after 1960; α_c is a cohort-specific fixed effects term; X_{isy} is the vector of sociodemographic controls used on the previous estimations, including a term capturing state-specific time trends to partially control for inherent differential pre-intervention trends across different states.

If mortality selection is an issue, the data should display a relatively larger imbalance towards a higher sex ratio (boys relative to girls) for individuals born post-eradication era. This expected result is based on the widely observed fact that female infants are more likely to survive to negative shocks during in utero life (Waldron, 1983; Astolfi and Zonta, 1999; Kraemer, 2000; Zaren et al., 2000; Sakamoto et al., 2001; Barker, 2004; Catalano et al., 2005, 2006). The results in column 5 show no significant change in the sex composition of births after the eradication relative to births prior to the eradication period, although the sign of the estimated coefficient suggests a relative increase in the sex ratio in favor of male children being born after the improvement in the early life health environment.

Besides the fact that the prevailing malaria parasite in Brazil is mildly lethal, the evidence collected in this section does not seem to indicate that the main results of this paper are driven by selection due to mortality of the weakest fetuses.

B.2 Age Heaping

In self-reported survey data, such as the PNAD, individuals might misreport their age, approximating it to the nearest round number. This might result in a potential source bias if the misreporting is systematically correlated with individual characteristics, such as socioeconomic status.

[46] It is important to control for the number of children, since, according to Regan et al. (1989), prior live births affect the likelihood of miscarriages.

[47] Stillbirth is defined as an infant death after survival of the first 28 weeks of in utero life. Death prior to this date is considered miscarriage.

To detect such anomaly in the data, figure B2 reports the distribution of the reported age structure of the sample by different quartiles of the income distribution. As observed, age heaping does not seem to be an important issue driving the results above. Nevertheless, I re-estimate the models above, excluding individuals with ages ending in either 0 or 5. The results do not contradict the previous findings.[48]

B.3 Other Vector-Borne Diseases

A possible confounding factor that might inflate the estimates of the long-term effects of early-life exposure to malaria is the role played by the eradication campaign in reducing the transmission of other vector-borne diseases such as yellow fever and dengue. Both diseases are transmitted by the same vector, the *Aedes aegypti*, which was eradicated in Brazil by 1955.[49]

Although the individuals in our sample might have been exposed to yellow fever and dengue during childhood, it is not likely that this effect is substantial enough to contaminate the main estimates. The national efforts in an eradication campaign of the *Aedes aegypti* had started more than ten years before the malaria eradication campaign, in 1942 (Catão, 2011).[50]. Moreover, during the time of its highest incidence rates, dengue was not an important public health problem. In fact, endemicity rates were lower in Brazil than in other countries of the American continent (Catão, 2011).

Additionally, Benchimol (1994) reports a relatively small number of cases of yellow fever between 1957 and 1959 (37 cases in three different states) in Brazil. Subsequent outbreak episodes in different regions has led to spikes in yellow fever cases, reaching 167 potential cases in the South in 1966. For comparison, the smallest number of malaria cases, after the success of the DDT spraying and antimalarial distribution was around 36,900 cases, in 1961 (Ferreira and Castro, 2016).[51]

B.4 Instrumental Variables Approach

One potential concern in the analysis is that the constructed exposure variable may suffer from measurement error or omitted variable bias, 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 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.[52] If this under-reporting is associated with regional characteristics, such as average educational levels, my results would suffer from attenuation bias. In my main specification, this concern is alleviated by the fact that I make use of within-state variations in exposure to malaria by leveraging on the seasonal differences in the reported cases due to the combination of high temperatures and rainfall.[53] Nevertheless, I discuss the sensitivity of my results to such potential biases by instrumenting the risk of malaria transmission with

[48] Results are available upon request.

[49] Brazil was officially recognized as having eradicated the *Aedes aegypti* in 1958. However, the mosquito was reintroduced in the country around late 1960s and early 1970s (Teixeira and Barreto, 1996).

[50] However, the vector was reintroduced in the Brazilian territories in the following decades for consecutive times (Catão, 2011).

[51] Bleakley (2010b) reports similar figures for Colombia: 22 reported cases of yellow fever and 167 leishmaniasis cases during 1962. The number of malaria cases for the same year was 21,245.

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

[53] I compare, therefore, individuals born in the same state and year, controlling for potential state-level unobserved factors, in different months.

specific climatic factors that affect the distribution and survival of both the mosquito and parasite of malaria.[54]

I leverage on the biological and climatic aspects that determine the malaria vector survival and proliferation likelihood and identify short-term exposure to malaria as a function of monthly climatic variations, which capture the intrinsic relationship between vector and parasite development with temperature and precipitation both across space and seasons of the year. 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.[55] The first stage estimating equation can, thus, be described as

$$\text{Exposure}_{smy} = \alpha + \text{Temp}_{smy}\psi_1 + \text{Prec}_{smy}\psi_2 + \epsilon_{smy}, \tag{5}$$

where Temp_{smy} and Prec_{smy} represent quarterly average temperature and precipitation (and squared terms) faced by individuals born in state s, month m, and year y.[56] The second stage of the estimation is analogous to equation 2.

Table B2 describes the two-stage least squares (2SLS) results of the IV estimation. Column (1) shows the IV results for years of education of the 1959 birth cohort, whereas column (2) displays the result for the 1960 birth cohort sample. I also report the first stage F test statistics from all regressions, indicating a strong first stage of the 2SLS estimation.[57] The 2SLS estimation produces more precisely estimated treatment effects for average in utero exposure on years of education on the more exposed birth cohort. The estimated coefficients are larger in magnitude than the OLS estimates and statistically significant at usual confidence levels. The point estimate for the 1959 sample is six times greater in magnitude than for the the 1960 sample, which is not statistically significant. The remaining columns in table B2 show the IV estimates for the effects of in utero exposure to malaria on the likelihood of graduating primary through tertiary degrees. The 2SLS results find statistically significant average treatment effects, at a least 1% significance level, for primary completion from the 1959 birth cohort, whereas the results for the 1960 birth cohort, as expected, are smaller in magnitude and not statistically significant.

Table B3 reports the IV estimates according to the different trimester of in utero exposure to malaria. Compared to the OLS estimates, the IV results suggests larger treatment effects from in utero exposure during different gestational periods. Across all estimates for the 1959 cohort, the effects seem to be stronger during first and last trimesters (although the results are more precisely estimated for the first).

In general lines, the first three months of in utero life seem to be important contributors to brain development and associated long-term negative consequences of fetal growth and cognition. A one standard deviation in the in utero exposure measure during the first trimester reduces education by 0.43 years (6% of the mean) and is associated with a reduction in the probability of primary completion of about 4.5 percentage points, as well as a decline in the likelihood of secondary and tertiary education by 2.78 and 0.89 percentage points, respectively. The 1960 birth

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

[55] See Section 2.2.1 for further discussion on the relationship between short-term malaria outbreaks and climatic variation.

[56] Although I am not able to directly assess the validity of the exclusion restriction assumption, it is worthwhile noting that the first stage above captures the *joint* effect of the quarterly temperature, rainfall, and squarred terms on exposure to malaria. The first stage of the IV estimate, thus, relies on the *simultaneous* importance of both variations. See section B.5 below for further discussion on the plausibility of the exclusion restriction in my analysis.

[57] All statistics are greater than the commonly used value of 10 as a rule of thumb, suggested by Staiger and Stock (1997).

cohort does not seem to exhibit statistically significant effects from in utero exposure to malaria over different trimesters of gestational life, as suggested by columns (2), (4), (6), and (8) in table B3.[58]

Collectively, the results of this section further underscores the long-term effects of in utero exposure to malaria on human capital accumulation. The findings suggest 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.

B.5 Climatic Confounders

Since malaria transmission is intrinsically correlated with climatic and geographic factors, it is possible that the measure of malaria intensity is correlated with other mechanisms that might directly affect adult mortality irrespective of malaria incidence. For example, temperature variability might affect in utero health through the development of respiratory diseases or transmission of maternal insults from mental health or food insecurity for instance (Molina and Saldarriaga, 2017). Another possible channel arises from the effects of temperatures that favor agricultural productivity, which might affect nutrition and agricultural income (Barreca, 2010). Sugarcane, corn, and soy constitute the main agricultural crops in Brazil, with sugar and corn alone accounting for around 24.5 percent of the total agricultural production between 1965 and 2010.[59] Sugarcane ideal temperatures range from 22°C to 30°C, with growing season between January and March, whereas ideal temperatures for corn yields are from 25°C to 30°C and can be grown between the months of August and May.[60]

To address the concern about the climatic confounding mechanisms raised above, I construct two additional sets of instruments: one that measures the proportion of the individual's in utero life in which monthly average temperatures are at least one standard deviation above yearly average temperature to capture the adverse health effects from extreme weather variations.[61] Next, I introduce temperature ideal instruments for both sugarcane and corn yields, as well as soy, as the proportion of the "growing season", in which monthly level average temperatures are between 22°C and 30°C, during sugar cane growing season, between 25°C and 30°C during corn growing season, and between 28°C and 32°C during soy growing season.

Table B6 shows the estimated results for the alternative set of instruments described above.[62] As one can observe, the effects are not statistically significant at any usual confidence levels for any of the outcomes. Importantly, the first stage F statistics for the joint significance of the excluded instruments are extremely small, suggesting a weak correlation between the constructed measure of exposure to malaria and climatic factors that are ideal to agricultural productivity. Indeed, the important factors explaining in utero exposure to malaria is the *joint* contribution of the specific biological and geographic factors that determine the strength and stability of malaria

[58] Additionally, first stage F statistics are sufficiently high to meet the rule of thumb requirement for a strong first stage estimate, as suggested by the table.

[59] Data from Ministry of Agriculture and IBGE.

[60] See http://www.agencia.cnptia.embrapa.br/ (in Portuguese) for information on ideal temperatures for the crop yields and USDA Agricultural Weather Assessment (https://www.usda.gov/oce/weather/pubs/Other/MWCACP/samerica.htm for information on their corresponding growing seasons. Accessed on 03/30/2019.

[61] See Deschenes and Moretti (2009) and Barreca (2012) for discussions on the effects of extreme weather shocks in the United States.

[62] Estimates are for the cohorts born prior to 1960 (between 1953 and 1959) to make sure that malaria was in fact prevalent.

transmission across different environments, captured by the state-level short-term seasonal variations in temperature *and* precipitation.

B.6 Exploring Alternative Timing of Exposure

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, one might 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. I might, therefore, be misclassifying individuals according to the degree of exposure. If that is the case, my results above would be capturing a different source of negative shock on early life exposure. Data limitations preclude an exact construction of exposed versus non-exposed cohorts, 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 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 limited 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 group does not alter the main results of no treatment effects.[63]

To ensure that the effects observed for the 1959 birth cohort (and for cohorts around that period) are indeed due to malaria exposure, I add older cohorts to the pre-eradication sample, 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 the joint effect of in utero exposure to malaria on those individuals born between 1956 and 1963. This particular choice of years covers the period in which malaria was endemic countrywide to being nearly eradicated in most parts of the country.[64] The estimation results, displayed in table B4, show both OLS and 2SLS estimates for the 1956–1959 and 1960–1963 samples. The estimates show statistically significant treatment effects of exposure to malaria on years of education and primary completion for the different choices of birth groups, especially the IV results, suggesting that the main results are not likely to be driven by the specific choice of the birth year groups.[65] Moreover, table B4 displays the IV estimation results for a broader set of cohorts. Panel A expands the treatment birth cohort to include individuals born between 1953 and 1959, whereas panel B provides estimation results for the whole sample, including individuals born between 1953 and 1963.[66]

[63] Results are available upon request.

[64] The choice of cohorts in my analysis imposes an important trade-off: while introducing older cohorts ensures a significant difference of overall malaria exposure across treatment and control groups, the estimation might render less precise results and of difficult interpretation, as longer horizon comparisons might introduce biases from compositional effects because broader birth year ranges would likely be contaminated by changes in the socioeconomic environment. For example, in 1964, Brazil underwent a series of political events that led to a military coup d'tat, which affected society in ways that would likely be correlated with the outcome variables in the analysis.

[65] However, the results in this section should be taken with a grain of salt, given the potential biases introduced by the larger cohorts.

[66] OLS estimates are not included, since the non-instrumented effects are not precisely estimated for the larger sample.

B.7 Endogenous Timing of Birth

The results of previous sections might also suffer from bias toward my estimates if households are able to systematically and endogenously choose the timing of gestation based on the seasonal risks of malaria infections.[67] Although data limitations do not allow me to directly test whether the particular timing of birth is correlated with household characteristics (I do not observe mother's information, for example), I analyze whether individuals' sociodemographic characteristics are correlated with their own timing of birth.[68] I find no statistically significant correlation between most of the individuals' characteristics and their month of birth, controlling for state-specific linear trends.[69] These findings, thus, provide no sufficiently robust evidence to support the claim that timing of birth is endogenously determined according to individual-level socioeconomic and demographic characteristics.

B.8 Alternative Measures of Exposure

Throughout the analysis in this paper, I construct a measure of in utero exposure to malaria based on reported number of cases at the state-level. Due to data limitations, monthly exposure can only be estimated according to the underlying assumption that the seasonal variation in malaria transmission is stable over time. In this section, I consider alternative measures of malaria exposure to validate the analysis carried out so far.

B.8.1 $\text{MSI} \times \text{Post}$

The first alternative exposure measure tested below is based on the state-level average Malaria Stability Index (MSI) interacted with a dummy variable indicating whether the individual was born after the overall reduction in malaria incidence, in the early 1960s. The main relevant feature of this measure is that it does not suffer from biases arising from differences in institutional factors, such as health coverage and reporting systems, across locations, since it is constructed according to ecologic factors, mitigating concerns related to endogenous state-level responses to malaria risk.[70] However, given the lack of variability at the temporal dimension, the estimations below do not allow for testing the effects of in utero exposure on different trimesters of gestation. The estimating equation is

$$\text{Education}_{ismy} = \beta \text{MSI}_s \times \text{Post} + \alpha_c + X'_{isy} \times \text{Post}\phi + \varepsilon_{isy}, \tag{6}$$

where Education_{ismy} is defined as either the number of completed years of education or whether the individual completed primary, secondary, and tertiary education; Post is a dummy variable indicating whether the individual was born after 1960; α_c is a cohort-specific fixed effects term; γ_s is state-of-birth fixed effects; X_{isy} is the vector of sociodemographic controls used in the previous estimations.

Table B7 shows the estimates of the above equation. All specifications include full set of controls as well as state of birth fixed effects and birth year fixed effects. The coefficients of columns 1 and 2 show that individuals born in relatively higher malarious states after the overall reduction

[67] Castilla and Sawyer (1993) points out to the role played by socioeconomic status on the behavioral responses to malaria risk.

[68] I essentially use individual level characteristics as *proxies* for parental background. This assumption is based on the fact that intergenerational transmission of wealth is extremely relevant and salient in the Brazilian context (Dunn, 2007; Ferreira and Veloso, 2006).

[69] Results are available upon request.

[70] See footnote 45 for a description of the constructed MSI.

have more years of education and are more likely to complete primary degree relative to individuals born in these areas prior to such reduction. Surprisingly, the sign of the coefficients become negative for the secondary and tertiary education completion. However, the effects, although statistically significant, are hardly economically meaningful. For comparison, I calculate the estimated effect of a change from the fifth percentile of the malaria risk distribution (0.41004) to the ninety-fifth (5.49863) on the probability of degree completion as follows. The primary education effect size of malaria exposure, measured by the MSI, on the cohort born in the post period, relative to the individuals born in the pre-period, is 0.0224, as column 2 in table B7 displays. Therefore, moving from the fifth to the ninety-fifth percentile of the MSI distribution makes the post cohort 11 percentage points $((5.49863 - 0.41004) \times 0.0224)$ more likely to complete primary education relative to the pre-period cohort. Performing the same computation for the coefficients in columns 3 and 4 results in an effect of -1.16 percentage points for the likelihood of completing secondary education and -1.66 percentage points for the likelihood of completing tertiary education. The magnitude of these somewhat puzzling negative coefficients on secondary and tertiary completion rates are, thus considerably smaller than the positive coefficient on primary degree.[71]

B.8.2 Malaria Suitable Months

As a second alternative proxy for malaria exposure, 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 of vector and parasite development with temperature and precipitation. I identify months in which malaria transmissions are likely the highest throughout the year according to climatic variations that satisfy the following conditions:[72]

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.

Specifically, I construct an exposure measure as follows: first I construct dummy variables for each in utero month indicating whether each of the above conditions is satisfied. Thus, for each gestational month, the constructed exposure measure is given by the sum

$$\sum_{i=1}^{3} I_k^i$$

where $I_k^i = 1$ if condition i is satisfied in month k. Next, I calculate a trimester-level exposure variable as the sum of the dummy variables for each month in a given trimester.[73]

[71] Recall that the estimated effects are according to a large variation in malaria risk (fifth to the ninety-fifth percentile). This corresponds to comparing individuals being born after and before the large reduction in malaria in the states of Rio Grande do Norte, in the Northeast (or Acre in the highly malarious Amazon region), and Rio Grande do Sul, in the South. The small magnitude of the estimated effects on secondary and tertiary degrees suggests a negligible marginal effect of exposure to malaria on these educational outcomes.

[72] Cervellati et al. (2018) 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.

[73] This specification is agnostic in terms of the relative importance of each condition listed above, assigning equal weights for each. For example, consider an individual whose first trimester of gestation happened during months in

The main advantage of such proxy for exposure to malaria, besides the high frequency of the data allowing to test for the potential heterogeneity in the effects at the trimester level, is that the necessary climatic conditions determining the parasite and mosquito survival and proliferation is specific enough to make sure the constructed measure is not simply capturing general climatic effects over the early life environment.

Table B8 displays the results of equation 3 using the aforementioned proxy for trimester exposure for a larger sample, including individuals born between 1926 and 1964. A similar picture emerges as the results from tables 3 and B3: the effects of in utero exposure to malaria on educational attainment seem to be stronger during the first trimester, although the third trimester also seems to be relevant to some of the outcomes. As expected, results are not statistically nor economically significant for the cohort born post eradication.[74]

B.8.3 Relative Exposure Only

As discussed in section 3, the constructed measure of in utero exposure might suffer from measurement error if the SCALE component does not capture overall state-level trends in malaria risk. Since the most important source of variation of the data is the seasonality in malaria distribution across space, I construct a similar measure of exposure solely based on the relative exposure with data from 2007 to 2017. The results are largely similar in magnitude and statistical significance as the main results above.[75]

B.8.4 Using Amazon as a Counterfactual

As a final check, I make use of the fact that the Eradication Campaign was only successful in reducing malaria transmission in the regions outside Legal Amazon to employ a simple differences-in-differences (DID) estimation in which individuals born in the Amazon basin constitute a plausible comparison group.[76] The aim of the analysis is to gouge the extent to which improvements in the early-life health environment through malaria declining rates improved educational attainment. To assess the plausibility of the comparison, figure B3 plots average education for individuals born in both treatment and control groups, corroborating the validity of the parallel trends assumption.

Table B9 displays the results of the DID. In general lines, individuals born in states outside Legal Amazon experienced larger gains in educational attainment relative to individuals born in the Legal Amazon region following the overall decline in malaria rates outside the Amazon basin. As cohorts born in areas outside Legal Amazon experienced larger improvements in early life health environment compared to cohorts born within this area, as expected, average educational attainment improved faster in treated areas.[77]

which all conditions were satisfied. Then, their first trimester exposure is constructed as the sum $I_1^1 + I_1^2 + I_1^3 + I_2^1 + I_2^2 + I_2^3 + I_3^1 + I_3^2 + I_3^3 = 9$. By the same token, an individual who faced none of the conditions above during their first trimester of exposure will have an average first trimester exposure of 0. The exposure measure, therefore, ranges from 0 to 9.

[74] Except for the years of education and tertiary degree results during the first trimester, which are significant at the 10 percent level.

[75] Results are available upon request.

[76] I thank an anonymous referee for pointing this out.

[77] The results indicate a negative treatment effect for primary completion, though.

B.9 Appendix B Figures

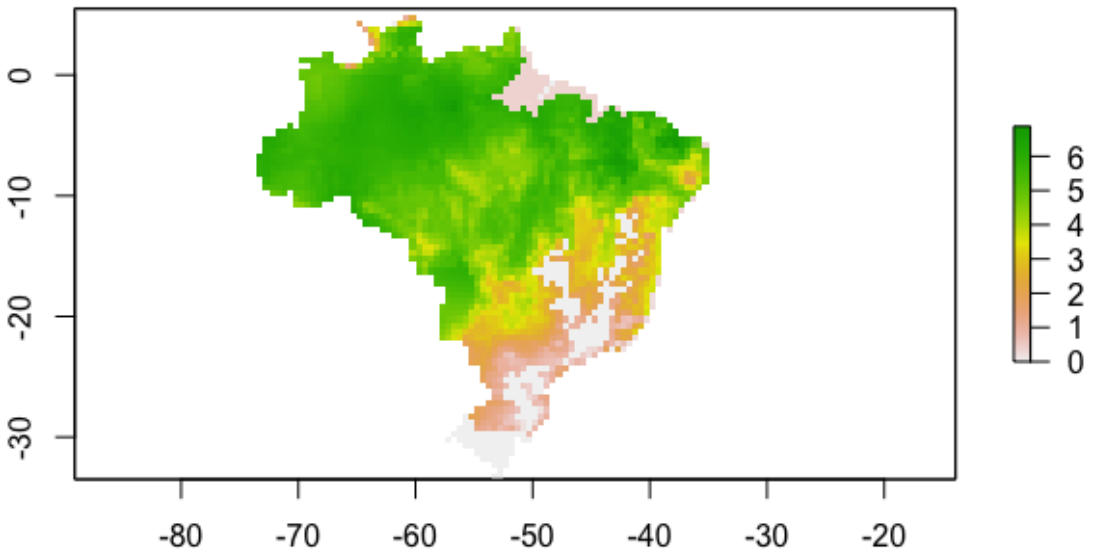


Figure B1: Malaria Stability Index – Brazil. Source: Kiszewski et al. (2004). The figure captures the geographical distribution of the dominant *Anopheles* mosquito based on information about climatic, biologic, and geographic conditions that contribute to the survival and spread of vector and parasite, on a 0.5 degrees grid level.

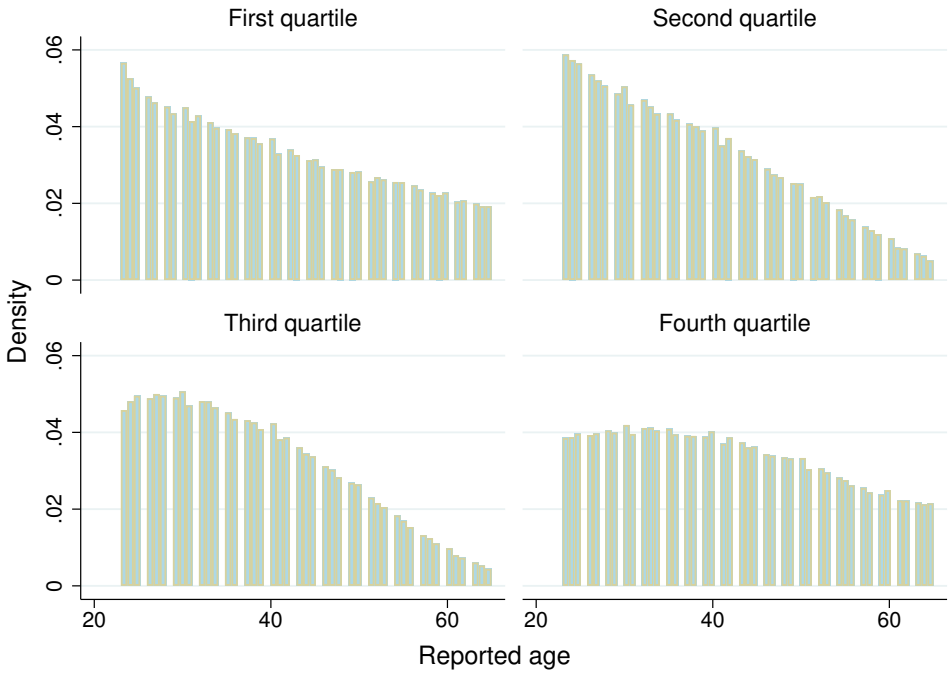


Figure B2: Reported age structure according to income quartiles. Brazil, 1993–2015. Source: PNAD (1993–2015). The figure depicts the distribution of reported age across the different quartiles of the income distribution. The sample includes individuals aged 23-65 years old in years 1959 and 1960.

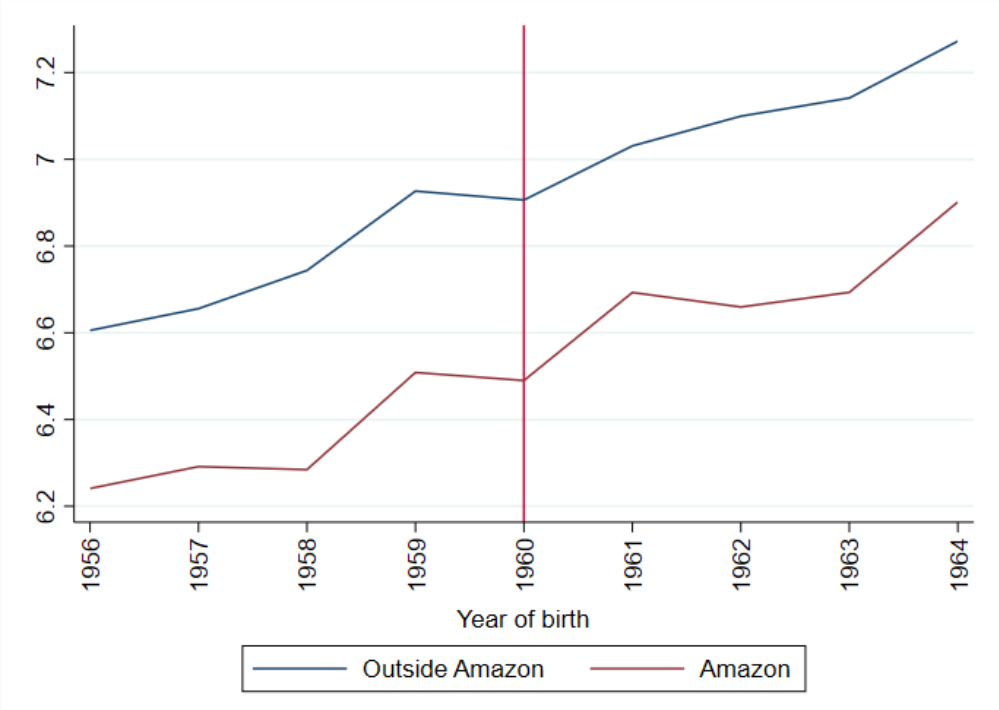


Figure B3: Average educational attainment across treatment and control groups between 1956 and 1964. Treatment group consists of individuals born in states outside Legal Amazon. Control group includes individuals born in states within Legal Amazon.

B.10 Appendix B Tables

Table B1: Mortality Selection Estimates

	Miscarriages/ Stillbirths (1)	Miscarriages/ Stillbirths (Boys) (2)	Miscarriages/Stillbirths Stillbirths (Girls) (3)	Ever Had Miscarriage/ Stillbirth (4)	Sex Ratio (Pre-Post) (5)
Malaria Index	0.0298	0.0160	0.0138	0.0139	-0.0749
	(0.337)	(0.340)	(0.427)	(0.335)	(0.197)
Observations	685	685	685	685	55,284
R-squared	0.060	0.075	0.034	0.089	0.109
Full set of controls	✓	✓	✓	✓	✓
Birth Cohort Fixed Effects	✓	✓	✓	✓	✓

OLS estimates with robust standard errors clustered at the state level in parentheses. Sample weights are used. The sample of regressions 1-4 includes females who completed their childbearing period prior to the Eradication Campaign, in 1959. Treatment is identified by the average risk of exposure, expressed in terms of state-level average malaria stability index, as described in Kiszewski et al. (2004). For regression 5, 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 race, whether the individual lives in a rural area, whether the individual has migrated , sector of activity, and total number of children. *** p<0.01, ** p<0.05, * p<0.1

Table B2: Early Life Exposure to Malaria IV Results – Baseline Exposure

	Years of Education		Primary		Secondary		Tertiary	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	(1959)	(1960)	(1959)	(1960)	(1959)	(1960)	(1959)	(1960)
In utero Exposure								
Treatment Effect	−36.81**	−6.049	−3.517***	−1.342	−1.641	−1.322	−1.280	−0.725
	(14.94)	(11.60)	(1.286)	(1.442)	(1.591)	(1.401)	(0.926)	(0.922)
First stage F-statistics	11.92	11.48	11.91	11.48	11.91	11.48	11.91	11.48
Observations	57,907	64,744	58,119	64,971	58,119	64,971	458,119	64,971
Full Set of Controls	✓	✓	✓	✓	✓	✓	✓	✓
State Fixed Effects	✓	✓	✓	✓	✓	✓	✓	✓
Survey Year Fixed Effects	✓	✓	✓	✓	✓	✓	✓	✓

Notes. 2SLS estimates with robust standard errors clustered at the state-month 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 in utero exposure, expressed in terms of state-level monthly average cases. Legal Amazon is excluded. Individual controls include gender, age, race, sector of activity, migration and rural status. All regressions include state fixed effects and survey year fixed effects. *** p<0.01, ** p<0.05, * p<0.1

Table B3: Early Life Exposure to Malaria IV Results – Exposure by trimester

	Years of Education		Primary		Secondary		Tertiary	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	(1959)	(1960)	(1959)	(1960)	(1959)	(1960)	(1959)	(1960)
In utero Exposure								
First Trimester	−68.11***	−7.208	−6.741***	−1.232	−4.800**	−2.086	−2.265**	−0.780
	(18.95)	(16.01)	(1.658)	(1.884)	(2.295)	(1.940)	(1.151)	(1.226)
Second Trimester	−23.58	−6.042	−1.929	1.224	−0.231	−0.890	−0.666	−0.733
	(16.58)	(11.79)	(1.508)	(1.492)	(1.710)	(1.366)	(0.999)	(0.949)
	(0.0270)	(0.129)	(0.0380)	(0.143)	(0.109)	(0.307)	(0.363)	(0.235)
Third Trimester	−48.72**	−8.376	−3.610*	−0.605	−2.463	−0.989	−0.668	−0.871
	(21.34)	(17.74)	(1.905)	(2.254)	(2.449)	(2.042)	(1.239)	(1.469)
First stage F-statistics	20.46	19.90	20.46	19.90	20.46	19.90	20.46	19.90
Observations	57,907	64,744	58,119	64,971	58,119	64,971	58,119	64,971
Full Set of Controls	✓	✓	✓	✓	✓	✓	✓	✓
State Fixed Effects	✓	✓	✓	✓	✓	✓	✓	✓
Survey Year Fixed Effects	✓	✓	✓	✓	✓	✓	✓	✓

Notes. 2SLS estimates with robust standard errors clustered at the state-month 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 exposure on each of the three trimesters while in utero, expressed in terms of state-level monthly average cases. Legal Amazon is excluded. Individual controls include gender, age, race, sector of activity, migration and rural status. All regressions include state fixed effects and survey year fixed effects. *** p<0.01, ** p<0.05, * p<0.1

Table B4: Early Life Exposure to Malaria Results – Alternative cohorts.

	Years of Education		Primary		Secondary		Tertiary	
	Panel A. (1956 – 1959) Cohorts							
	(1) OLS	(2) 2SLS	(3) OLS	(4) 2SLS	(5) OLS	(6) 2SLS	(7) OLS	(8) 2SLS
In utero Exposure								
Treatment Effect	−0.0124**	−0.612***	0.000740	−0.115***	−0.000599	−0.00877	−0.000670**	−0.00388
	(0.00504)	(0.237)	(0.000517)	(0.0321)	(0.000564)	(0.0195)	(0.000327)	(0.00750)
First stage F-statistics		11.66		11.64		11.64		11.64
Observations	319,581	186,313	320,550	187,007	320,550	187,007	320,550	187,007
Full Set of Controls	✓	✓	✓	✓	✓	✓	✓	✓
State Fixed Effects	✓	✓	✓	✓	✓	✓	✓	✓
Survey Year Fixed Effects	✓	✓	✓	✓	✓	✓	✓	✓
		Panel B. (1960 – 1963) Cohorts						
	(9) OLS	(10) 2SLS	(11) OLS	(12) 2SLS	(13) OLS	(14) 2SLS	(15) OLS	(16) 2SLS
In utero Exposure								
Treatment Effect	−1.157	−36.23	−0.0494	−4.619**	0.0552	−0.552	−0.114**	−0.368
	(0.771)	(19.35)	(0.0847)	(2.259)	(0.0735)	(1.774)	(0.0546)	(0.672)
First stage F-statistics		10.68		10.69		10.69		10.69
Observations	363,216	220,811	364,618	221,790	364,618	221,790	364,618	221,790
Full Set of Controls	✓	✓	✓	✓	✓	✓	✓	✓
State Fixed Effects	✓	✓	✓	✓	✓	✓	✓	✓
Survey Year Fixed Effects	✓	✓	✓	✓	✓	✓	✓	✓

Notes. OLS and 2SLS estimates with robust standard errors clustered at the state-month level. Sample weights are used. The sample includes individuals aged 23-65 years old in years between 1953 and 1964. Treatment is identified by the average in utero exposure, expressed in terms of state-level monthly average cases. Legal Amazon is excluded. All regressions include state fixed effects and survey year fixed effects. *** p<0.01, ** p<0.05, * p<0.1

Table B5: Early Life Exposure to Malaria IV Results – Alternative cohorts.

	Years of Education	Primary	Secondary	Tertiary
	Panel A. 1953 – 1959 as alternative pre-eradication cohort			
	(1)	(2)	(3)	(4)
In utero Exposure				
Treatment Effect	−0.470**	−0.0926***	−0.00489	−0.000504
	(0.200)	(0.0269)	(0.0177)	(0.00594)
First stage F-statistics	11.35	11.29	11.29	11.29
Observations	306,413	307,445	307,445	307,445
	Panel B. 1953 – 1963			
	(5)	(6)	(7)	(8)
In utero Exposure				
Treatment Effect	−0.809**	−0.132***	−0.00891	−0.00275
	(0.340)	(0.0412)	(0.0301)	(0.0104)
First stage F-statistics	11.35	11.24	11.24	11.24
Observations	527,224	529,235	529,235	529,235
Full Set of Controls	✓	✓	✓	✓
State Fixed Effects	✓	✓	✓	✓
Survey Year Fixed Effects	✓	✓	✓	✓

Notes. 2SLS estimates with robust standard errors clustered at the state-month level. Sample weights are used. The sample includes individuals aged 23-65 years old in years between 1953 and 1964. Treatment is identified by the average in utero exposure, expressed in terms of state-level monthly average cases. Legal Amazon is excluded. Individual controls include gender, age, race, sector of activity, migration and rural status. All regressions include state fixed effects and survey year fixed effects. *** p<0.01, ** p<0.05, * p<0.1

Table B6: Early Life Exposure to Malaria IV Results – Alternative set of Instruments.

	Years of Education	Primary	Secondary	Tertiary
	Panel A. Extreme weather variability			
Treatment Effect	−1.601	0.101	0.0130	−0.111
	(7.300)	(0.429)	(0.164)	(0.506)
First stage F-statistics	0.0513	0.0523	0.0523	0.0523
Observations	366,820	367,924	367,924	367,924
	Panel B. Agriculture ideal temperatures			
Treatment Effect	−0.0103	−0.0235	0.00318	0.00791
	(0.169)	(0.0178)	(0.0182)	(0.0117)
First stage F-statistics	3.679	3.672	3.672	3.672
Observations	366,820	367,924	367,924	367,924
Full Set of Controls	✓	✓	✓	✓
State Fixed Effects	✓	✓	✓	✓
Survey Year Fixed Effects	✓	✓	✓	✓

Notes. 2SLS estimates with robust standard errors clustered at the state-month level. Sample weights are used. The sample includes individuals aged 23-65 years old between years 1953 and 1959. Treatment is identified by the average in utero exposure, expressed in terms of state-level monthly average cases. Legal Amazon is excluded. Individual controls include gender, age, race, sector of activity, migration and rural status. All regressions include state fixed effects and survey year fixed effects. *** p<0.01, ** p<0.05, * p<0.1

Table B7: Interaction MSI and Post as Alternative Exposure Measure

	Years of Education (1)	Primary (2)	Secondary (3)	Tertiary (4)
Malaria Index×Post	0.0224**	0.0207***	−0.00306***	−0.00327***
	(0.00977)	(0.000890)	(0.000972)	(0.000556)
Observations	1,984,205	1,994,561	1,994,561	1,994,561
R-squared	0.328	0.176	0.169	0.108
Full set of controls	✓	✓	✓	✓
Birth Cohort Fixed Effects	✓	✓	✓	✓
State Fixed Effects	✓	✓	✓	✓

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 after 1956. Treatment is identified by the average risk of exposure, expressed in terms of state-level average malaria stability index, as described in Kiszewski et al. (2004), interacted with a Post dummy. Legal Amazon is excluded. Individual controls include race, whether the individual lives in a rural area, whether the individual has migrated , sector of activity, and total number of children. *** p<0.01, ** p<0.05, * p<0.1

Table B8: Climatic Variables as Alternative Exposure Measure

	Years of Education		Primary		Secondary		Tertiary	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	(Before 1959)	(After 1960)	(Before 1959)	(After 1960)	(Before 1959)	(After 1960)	(Before 1959)	(After 1960)
In utero Exposure								
First Trimester	−0.0244**	−0.0169*	−0.00188**	−0.000624	−0.00206**	−0.00135	−0.00112*	−0.00111*
	(0.0103)	(0.00962)	(0.000893)	(0.00108)	(0.000899)	(0.00101)	(0.000570)	(0.000596)
Second Trimester	0.00061	−0.00213	−7.73e − 05	−0.000499	−0.000163	−0.000350	−0.000217	−0.000216
	(0.00327)	(0.00266)	(0.000281)	(0.000319)	(0.000255)	(0.000287)	(0.000163)	(0.000185)
Third Trimester	−0.0213**	−0.0131	−0.00175**	0.00113	−0.00142*	−0.000634	−0.000731	−0.000791
	(0.00980)	(0.00990)	(0.000871	(0.00107)	(0.000846)	(0.000977)	(0.000540)	(0.000609)
Observations	1,372,555	517,038	1,375,302	519,229	1,375,302	519,229	1,375,302	519,229
R-squared	0.216	0.193	0.159	0.127	0.109	0.104	0.045	0.055
Full Set of Controls	✓	✓	✓	✓	✓	✓	✓	✓
State Fixed Effects	✓	✓	✓	✓	✓	✓	✓	✓
Survey Year Fixed Effects	✓	✓	✓	✓	✓	✓	✓	✓

Notes. OLS estimates with robust standard errors clustered at the state-month level in parentheses. Sample weights are used. The sample includes individuals aged 23-65 years old in the full sample. Treatment is identified by the average exposure on each of the three trimesters while in utero, expressed in terms of state-level monthly conditions that determine the risk of malaria infections. Legal Amazon is excluded. Individual controls include gender, age, race, migration and rural status. All regressions include state fixed effects and survey year fixed effects. *** p<0.01, ** p<0.05, * p<0.1

Table B9: Differences-in-Differences Approach

	Years of Education (1)	Primary (2)	Secondary (3)	Tertiary (4)
Treatment×Post	0.122***	−0.0784***	0.0119**	0.0121***
	(0.0382)	(0.00468)	(0.00504)	(0.00211)
R-squared	0.361	0.181	0.174	0.104
Observations	2,583,895	2,595,345	2,595,345	2,595,345
Full set of controls	✓	✓	✓	✓
Survey Year Fixed Effects	✓	✓	✓	✓
State Fixed Effects	✓	✓	✓	✓

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 after 1956. Treatment group is identified by individuals born outside Legal Amazon region. Post is a dummy indicating whether individuals were born after 1959. Individual controls include gender, age, race, sector of activity, migration and rural status. *** p<0.01, ** p<0.05, * p<0.1

Appendix C. The Stability of Malaria Transmission

Throughout the analysis above, I assume that the relative risk of malaria transmission is constant (or at least does not vary much). This assumption is necessary for identifying relative exposure levels across states and months of births, given the absence of incidence data for the period around the eradication era. In this section, I discuss the plausibility of the stability malaria transmission assumption as well as its shortcomings. If malaria relative transmission does not change over time, we should observe similar patterns over the time period in which monthly data is available. Figure C1 shows the monthly reported number of cases per capita across the period 2007-2017 in Brazil.

In general, January is the month in which malaria transmission rates are the largest. This high incidence coincides with the peak of the summer season, in which temperatures are well above average. Moreover, a second peak in transmission can be identified during the month of June, which coincides with the peak of the raining season in most regions of Brazil. The remaining months follow a similar pattern, with a relative decline both after January and June, and picking up as the year approaches December. The general trends are observed across different years, suggesting a potential stability in relative malaria transmission rates for different periods.

A potential threat to the plausibility of the assumption of constant relative risk of malaria transmission is the heterogeneous effects of climatic variation over time due to either climate change (Martens et al., 1995; Bhattacharya et al., 2006; Caminade et al., 2014; Bauch et al., 2015) or deforestation (Yasuoka and Levins, 2007; Olson et al., 2010; Hahn et al., 2014; Santos and Almeida, 2018; MacDonald and Mordecai, 2019).[78] Indeed, the second half of the Nineteenth century has experienced alarming consequences of climate change and deforestation has been an important contributing environmental problem faced in Brazil.[79]

To address the issues raised above, I collect state-level climatic data for two distinct periods (1961–1969 and 2007–2017) and test for potential differential monthly variation in both precipitation and temperature.[80] Table C1 details the results of the analysis. Columns 1 and 2 display the results of a regression with precipitation as the dependent variable and month fixed effects as main independent variable for the initial and final periods. The results point out to the importance of the seasonal component in rainfall variations over time. Importantly, column 3 shows the difference in the coefficients and their lack of significance suggest no discernible differences in the effects of months on rainfall across states. Columns 4 and 5 show the estimation results for temperature for both samples, with the differences in the coefficients in column 6. As one can notice, the differences are not statistically significant for most months (except March, April, and July, which are significant at least at the 5% level). Finally, columns 7 and 8 pool both samples together and show estimates for the effects of months interacted with a dummy variable indicating whether a given observation is from the final period (2007–2017). The results for both precipitation and temperature indicate no statistically significant difference in monthly climatic variation across initial and final periods.[81] Therefore, although climate change and deforestation might pose a challenge to the validity of the constant relative malaria risk assumption, the results in ta-

[78] Deforestation has been shown to directly impact early-life health conditions through the smoke from wood burning. See, for example, Carrillo et al. (2019) and Sant'Anna and Rocha (2020) for the Brazilian case.

[79] Although deforestation in Brazil has increased over the years, its incidence is mostly concentrated in the Amazon basin, which is not part of the main sample, thus alleviating some of the concern related to the invalidation of the assumption of constant relative risk of malaria transmission.

[80] The data used in this analysis is from the Worldclim data (Fick and Hijmans, 2017). For each month-year 21-km^2 grid data, I overlay a Brazilian map with state boundaries and calculate average state-level precipitation and minimum temperature for the grids within each state for the period between 1961-1969 and 2007-2017.

[81] Results are robust to the inclusion of month and year fixed effects.

ble C1 provide further assurance that the monthly climatic variations are not sufficiently affected by them.

Two additional factors might invalidate the assumption of constant relative malaria transmission: heterogeneous evolution of public health system efficiency across states and in individual-level prophylactic (and remedial) measures adopted by across both locations and time. If the evolution of malaria treatment and prevention did not evolve homogeneously across regions, the relative risk of transmission might have changed over time. Figure C2 addresses this potential issue by describing regional evolution of two important health sector components. Panel C2a displays how the number of public health facilities have evolved between 2005 and 2017, whereas panel C2b shows the decline in infant mortality between 1979 and 2017, across the four regions (North excluded) in Brazil.

Panel C2a exhibits a clear positive and parallel trend across different regions, which does not refute the hypothesis that the overall reduction in malaria transmission rates have happened in a proportional fashion. Provision of resources to control malaria is a task attributed to the central government, which transfers funds to the local authorities, who in their turn, use them on measures to prevent and treat malaria. It is relevant to notice an important characteristic of the Brazilian public sector: the distribution of the public resources transfers from the central government to states (and municipalities) is determined by the constitution, as to ensure equal regional development across different regions. Therefore, it is expected that local health institutions to have evolved in a similar manner. The observed regional trends, along with the institutional arrangements in Brazilian public sector spendings, then, alleviate the concern of heterogeneous efficiency of the health sector in controlling malaria over time.

Prophylactic measures – such as the use of bednets, mosquito repellents, avoidance of exposure to risky environments – and treatment measures – including intake of antimalarial drugs – might have evolved differently across different locations in Brazil since the time of the eradication era. Since one could expect such measures to correlate with improvements in the health status of the country as a whole, panel C2b uses regional infant mortality rates to proxy for these changes. The figure points out to a similar pattern as observed in the above panel: infant mortality seems to have been steadily declining uniformly across regions in Brazil. Taken together, the evidences provided in this section provide suggestive evidence in favor of the assumption that relative malaria transmission rates across locations are stable over time, reassuring the robust treatment effects estimated above.

Despite the compelling plausibility of the assumption of stable relative malaria transmission over time, it is worth mentioning the implications for my results if this assumption is violated. First, suppose that wealthier regions were able to control malaria transmission rates disproportionately more efficiently than poorer ones. That is, although the constitutional fund enforces equitable allocation of resources, their utilization might have been disproportionately more efficient in more developed regions. In this case, the malaria intensity measure used in the above analysis might in fact be underestimating the malaria burden in wealthier regions, rendering biased OLS estimates in favor of my results.[82] Suppose now that poorer regions were able to catch up with wealthier ones with respect to the use of the public resources. In this situation, the relative measure of malaria transmission would underestimate the prevailing malaria burden in poorer regions around the eradication era. The estimates above would, then, constitute a lower bound for the true treatment effect of in-utero exposure to malaria.

[82]The Instrumental Variables approach might alleviate some of the potential measurement error bias, although the assumption of classical measurement error is likely to be violated in this situation.

Appendix C Figures

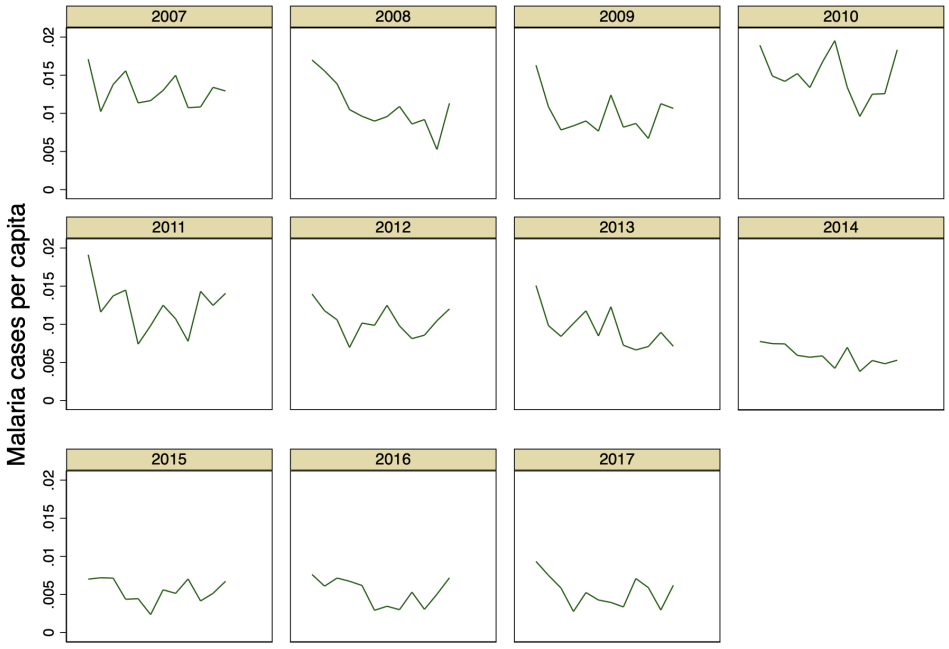
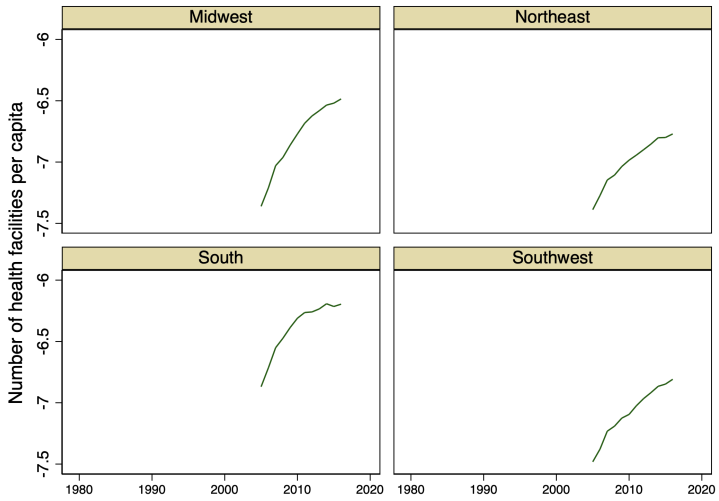
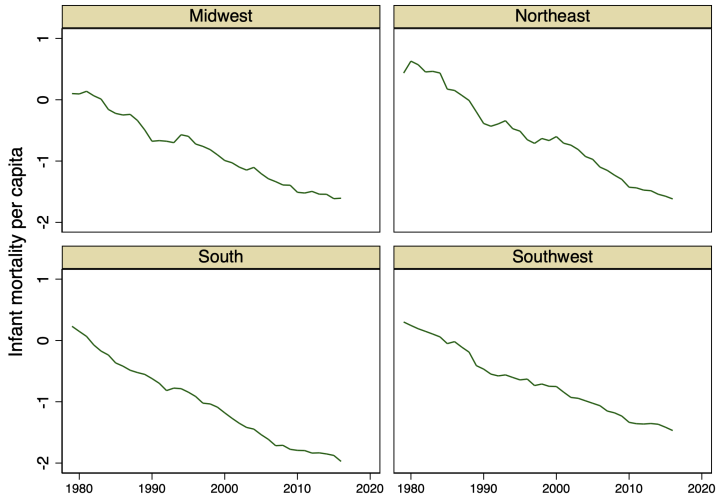


Figure C1: Monthly Malaria Cases per 1,000 inhabitants (2007–2017) – Brazil. Source: DATASUS – Ministry of Health. The figure represents country-wide average reported number of cases per 1,000 inhabitants at the monthly level for the period 2007–2017. The figure depicts the relative stability of malaria incidence variation over different seasons.



(a) Log of Number of Health Facilities by Regions



(b) Log of Infant Mortality by Regions

Figure C2: Source: DATASUS – Ministry of Health. Panel (a) shows the Log of Number of Health Facilities by Regions in Brazil over the period 2005–2016. Panel (b) shows the Log of Infant Mortality by Regions in Brazil over the period 1979-2017.

Appendix C Table

Table C1: Precipitation and Temperature Stability Analysis

	Precip. 1960-1969	Precip. 2007-2017	Diff (p-value)	Temp. 1960–1969	Temp. 2007–2017	Diff (p-value)	Precip. (Month×Post)	Temp. (Month×Post)
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Jan	Excluded						9.664	−0.589
							(48.87)	(0.507)
Feb	−3.197	−4.152	0.955	−0.0661	0.0300	−0.0961	8.434	−0.480
	(20.81)	(23.25)	(0.9080)	(0.478)	(0.397)	(0.3887)	(49.77)	(0.507)
Mar	−0.643	4.895	−5.538	−0.378***	−0.187	−0.191	14.93	−0.385
	(20.79)	(22.64)	(0.5033)	(0.527)	(0.470)	(0.0245)	(48.83)	(0.504)
Apr	−34.51*	−26.64**	−7.87	−1.241***	−0.923	−0.318	17.26	−0.258
	(20.67)	(21.48)	(0.3220)	(0.662)	(0.589)	(0.0012)	(49.62)	(0.505)
May	−71.69***	−63.06***	−8.63	−2.562***	−2.422***	−4.984	18.02	−0.435
	(23.74)	(23.39)	(0.2540)	(0.826)	(0.780)	0.2074	(49.56)	(0.503)
Jun	−103.1***	−102.4***	−0.70	−3.882***	−3.674***	−0.208	10.15	−0.367
	(22.30)	(21.98)	(0.9172)	(0.872)	(0.836)	0.0759	(49.50)	(0.499)
Jul	−122.1***	−112.4***	−9.70	−4.441***	−4.126***	−0.315	19.04	−0.260
	(20.57)	(21.30)	(0.1474)	(0.883)	(0.844)	0.0113	(49.45)	(0.514)
Aug	−138.7***	−129.5***	−9.20	−3.438***	−3.407***	−0.031	18.62	−0.546
	(18.09)	(19.75)	(0.1402)	(0.809)	(0.759)	0.8024	(49.43)	(0.504)
Sep	−129.1***	−123.6***	−5.50	−1.817***	−1.723***	−0.094	14.89	−0.481
	(18.00)	(19.24)	(0.3765)	(0.694)	(0.658)	0.3776	(49.46)	(0.504)
Oct	−89.39***	−81.92***	−7.47	−1.088***	−0.864	−0.224	16.86	−0.352
	(19.69)	(20.78)	(0.2501)	(0.639)	(0.577)	0.0501	(49.37)	(0.505)
Nov	−64.21***	−55.90***	−8.31	−0.483*	−0.462	−0.021	17.70	−0.555
	(20.37)	(22.46)	(0.2236)	(0.556)	(0.534)	0.8079	(49.26)	(0.500)
Dec	−24.37	−20.76	−3.61	−0.395	−0.195	−0.20	13.01	−0.375
	(22.10)	(23.63)	(0.6126)	(0.527)	(0.455)	0.0601	(49.65)	(0.505)

Notes. OLS estimates with robust standard errors clustered at the state-month level. The sample includes state-level precipitation and temperature data between years 1961–1969 and between 2007–2017. The last two columns include month and year fixed effects. *** p<0.01, ** p<0.05, * p<0.1