Disease, Disparities, and Development: Evidence from Chagas Disease Control in Brazil*

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Abstract

Neglected tropical diseases (NTDs) primarily afflict the poorest people in developing economies and often lead to chronic health issues. Combating them could thus reduce inequality and the intergenerational transmission of poverty as well as burdens on healthcare systems in these countries. We show that these novel benefits of disease control indeed arose from Brazil's initial campaign to eliminate Chagas disease (1984-89), an NTD that occurs almost entirely among poor, non-white, and rural Latin Americans and can cause long-run heart problems. Exploiting the pre-treatment presence of its main vector, we find that having a childhood free of exposure to this NTD raised non-white adults' incomes by more than twice as much as for white adults (7.7 vs 3.4 percent), and non-literacy among their children declined substantively (0.8 p.p., or 10 percent). We also estimate that, coinciding with the expected reduction in chronic Chagas disease symptoms, public spending on circulatory disease hospital care declined by 13.5 percent (0.014 percent of 2019 GDP), yielding by itself an internal rate of return of 24.9 percent. These results suggest that NTD control can reduce (racial) disparities in one of the world's most unequal regions while improving the public and fiscal health of developing countries.

Keywords: Tropical Disease, Racial Inequality, Intergenerational Mobility, Health Spending
JEL Classification: H51, I14, I15, J62, O15

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1. Introduction

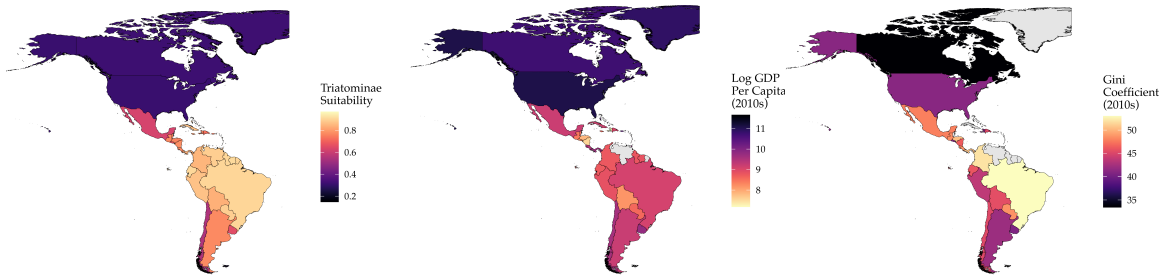
Latin America is one of the most unequal regions in the world (Chancel et al., 2021). The World Inequality Database calculates that, in Latin America, the top 10% captures 54% of the national income, with inequality being highest in Chile, Mexico, and Brazil (De Rosa, Flores and Morgan, 2020). Along with drastic income inequality, Latin America is also characterized by stark racial inequalities, with estimates suggesting that the average monthly income of white individuals is at least two times higher than that of individuals with dark skin shades (Woo-Mora, 2022). These high and interconnected income and racial inequalities have renewed calls to understand their underlying causes and to explore policies that might reduce these inequities in the region (World Economic Forum, 2016; Eslava and Caicedo, 2023).

An understudied but potentially important driver of income inequality in the region is Chagas disease (Galeano, 2005; Briceño-León and Méndez Galván, 2007; Franco-Paredes et al., 2007; Houweling et al., 2016). Chagas disease, also known as American trypanosomiasis, is a parasitic neglected tropical disease that is only found in the Americas. It affects both children and adults. Both groups can experience the acute phase's symptoms that last for weeks; then, around a decade or more later, a substantial share of those infected enter the chronic phase and develop cardiovascular problems, which affect the ability to work and are responsible for a substantial share of health problems in Latin America (Bocchi et al., 2009; World Health Organization, 2010).

Chagas disease is often described as a "neglected disease of poor, rural and forgotten populations" (Coura and Viñas, 2010; Houweling et al., 2016). For these reasons, it has been called "Latin America's Silent Killer" (Galeano, 2005). Poorer individuals are much more likely to contract the disease because the vector – triatomine bugs – lives in cracks in poor-quality housing. Additionally, lack of access to adequate health education, health care, and environmental management strengthen the link between poverty and Chagas disease (Hotez et al., 2013). Despite over 6 million individuals being affected by Chagas disease in the region, little is known about how Chagas disease exacerbates income and racial disparities, and whether its elimination might reduce inequalities and increase development.

The maps of the Americas in Figure 1 suggest that Chagas Disease is indeed associated with

Figure 1: Chagas Disease Vector Suitability, Income, and Inequality in the Americas



(a) Chagas Disease Vector Suitability (b) Log GDP per Capita (c) Gini Coefficient

Notes: The left panel shows a map of Chagas Disease vector suitability from Eberhard et al. (2020). The center panel shows log GDP per capita from the World Bank Development Indicators. The right panel shows the most recently reported Gini coefficient (scaled by 100) between 2010 and 2019 from the World Bank Development Indicators. The unit of observation is a country in the Americas.

lower incomes and greater inequality. In the left panel, we show the average value of the Eberhard et al. (2020) index measuring modern-day suitability for vectors of Chagas Disease (which we discuss in Section 2) for each country in the Western Hemisphere. This index appears to be correlated negatively with income (measured as log GDP per capita in the center panel) and positively with inequality (measured as the Gini coefficient in the right panel). Furthermore, following Alsan (2015) in the examination of a continent-specific disease, we regress these outcomes for all countries in the world on their suitability index values and the interaction between this index and an indicator for being in the Americas (as well as rich set of geographic controls) and present the results in Table 1.[1] The results in Table 1 suggest that, outside of the Western Hemisphere, Chagas Disease vector suitability has no significant correlation with development or disparities—but within the Americas, vector suitability is robustly associated with lower average incomes and greater inequality in the cross-section. These results provide suggestive evidence that combating Chagas disease could potentially help improve incomes and reduce income inequality in the region.

In spite of its presence only in the Americas, Chagas Disease is similar to many other Neglected Tropical Diseases (NTDs) in that it has both short and long run effects. As we describe in Section 2, in the short run, adults are unable to work due to its acute phase. In the long run,

[1] Alsan (2015) studies African trypanosomiasis, or sleeping sickness. Although Chagas Disease is also a trypanosomiasis, the only substantial similarity between the two diseases is that they are caused by parasites of the genus *Trypanosoma*.

Table 1: Chagas Disease, Income, and Inequality

	Dependent Variable:			
	log(GDP per capita)		*Gini Coefficient*	
	(1)	(2)	(3)	(4)
Chagas Suitability × **1**(Americas)	−0.02**	−0.03**	0.24***	0.25***
	(0.01)	(0.01)	(0.07)	(0.07)
Chagas Suitability	−0.00	−0.00	0.05*	0.04
	(0.01)	(0.01)	(0.03)	(0.03)
Continent FEs	Y	Y	Y	Y
Geography Controls	Y	Y	Y	Y
Disease Controls	N	Y	N	Y
Outcome Mean	8.91	8.91	37.16	37.16
Outcome SD	1.48	1.48	7.51	7.51
Adjusted R2	0.524	0.525	0.588	0.596
Observations	207	207	155	155

Notes: The unit of observation is a country. Robust standard errors in parentheses. In columns (1) and (2), the outcome variable is the log of the average GDP (in $1,000s) per capita in 2019 from the World Bank Development Indicators. In columns (3) and (4), the outcome variable is the most recently reported Gini coefficient (scaled by 100) between 2010 and 2019 from the World Bank Development Indicators. *Chagas Suitability* is a 0 to 100 measure of the ecological suitability for Chagas vectors from Eberhard et al. (2020). $\mathbb{1}$*(Americas)* is an indicator variable equal to one if a country is in North or South America. *Geography Controls* includes centroid longitude, centroid latitude, average rainfall, average temperature, elevation, area, and agricultural suitability. *Disease Controls* includes malaria suitability and tsetse fly suitability. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

its chronic phase can also affect adult incomes and employment as well as hospitalizations and spending in Brazil's government-run health care system, which is the largest in the world and consumes 4 percent of GDP. Additionally, as poorer individuals are more likely to contract this and other NTDs—in the case of Chagas Disease, the vector lives in cracks in the roof and wall, which are more common in houses made of earthen materials—and given the close link between poverty and race in Brazil, the disease may also exacerbate the country's wide racial disparities.

In the remainder of this paper, we provide within-country quasi-experimental evidence of the impacts of Chagas Disease on inequality and development. We focus on Brazil, which in Figure 1 has extremely high levels of vector suitability and income inequality. Importantly, as we discuss in Section 3, it also conducted a large Chagas Disease control campaign between 1984 and 1989, which was made possible by a technological breakthrough and a 1975-83 nationwide

entomological survey measuring the primary vector's presence at the municipal level.

As we describe in Section 4, these data allow us to compare cohorts from municipalities that were never infested with the vector and those that had it prior to insecticide spraying but were rid of it by 1989. The idea behind our approach is that individuals in municipalities where the vector was eliminated experienced a greater reduction in exposure to Chagas Disease than those where the vector was never found. Thus, we combine cohort variation in the timing of the control campaign with cross-municipality variation in vector presence to examine whether cohorts who grew up in municipalities that were infested with the vector had better long-run outcomes after the control program than those in municipalities that were never infested with the vector.

We formalize this empirical strategy in Section 5 and examine the long-run labor market results for adults treated as children using data from the 2010 census of Brazil and a difference-in-differences framework. We find positive and large increases in incomes for both white and non-white Brazilians. However, we find substantially larger effects for the latter (7.7-percent increase in incomes versus 3.4 percent), suggesting that the program may have helped reduced racial disparities in long run. We also find it that the control program decreased the interquartile range of incomes for treated cohorts by 3.3 percent. Additionally, children of treated parents have higher rates of literacy, suggesting that the control campaign has positive inter-generational impacts. We examine educational attainment as a channel for the income results in Section 5.4 but do not find evidence that it can explain a large share of the effect. Instead, results suggest that the health benefits of reducing acute Chagas disease – increasing employment in modern sectors in the short-run – and chronic Chagas Disease – increasing hours worked and reducing reliance on government support in the long-run –may play an important role in this reduction of racial disparities.

If reducing the occurrence of the disease's chronic phase contributed to the increase in incomes, it may also have resulted in substantial public finance effects through its impact on Brazil's government-run health care system. In Section 7, we examine hospitalizations, person-days spent in the hospital, and spending on hospital care covered by this system by modifying our differences-in-differences strategy to also compare the above outcomes due to circulatory system diseases against those due to all other causes (i.e., a triple-difference framework).

We find approximately 17-percent greater reductions in hospitalizations and person-days spent in the hospital due to circulatory diseases—which account for around 10 percent of these outcomes covered by Brazil's public health care system. Importantly, these declines started 10 years after vector elimination, which is about the point when the chronic phase symptoms of Chagas Disease would have begun to appear. These decreases appear to have led to a 14-percent greater decrease in spending on hospital care due to cardiovascular causes beginning at the same time, though our estimate is somewhat imprecise. Nonetheless, it is an economically significant magnitude given that circulatory diseases account for 20 percent of public hospital care spending.

We conduct a simple cost-benefit analysis comparing the costs of the vector spraying program to the public finance benefits of averted hospital care. The analysis in Section 8 finds that the internal rate of return for the disease control campaign was 24.9%. While the analysis abstracts from other potential government benefits (namely increases in income tax revenues) and therefore understates the economic benefits, the cost-benefit analysis provides strong evidence for the positive economic returns of the large-scale Chagas disease control campaign in the long-run.

Taken together, these results imply that controlling Chagas Disease had important benefits for a developing economy both in the short run (structural transformation) and in addition to individuals' labor market returns in the long run (public health care spending). They also suggest that, in a multi-racial country in which race and poverty are closely linked, combating diseases that primarily affect the poorest citizens can contribute to reductions in disparities between these groups. As such, we provide novel and important evidence of the benefits of controlling neglected tropical diseases in the developing world that should impact donors' and policymakers' decisions to do so.

Our results intersect with several strands of literature. First, our study contributes to the growing literature in economics that explores the ramifications of neglected tropical diseases on economic development (e.g., Miguel and Kremer, 2004; Bleakley, 2007, 2010; Alsan, 2015; Hamory et al., 2021). Neglected Tropical Diseases (NTDs) epitomize a class of health challenges that disproportionately afflict impoverished populations in low and middle-income countries. These diseases often have both acute and chronic effects, necessitating a long-run perspective to study their impacts. We highlight that eradicating NTDs can have benefits via reductions in

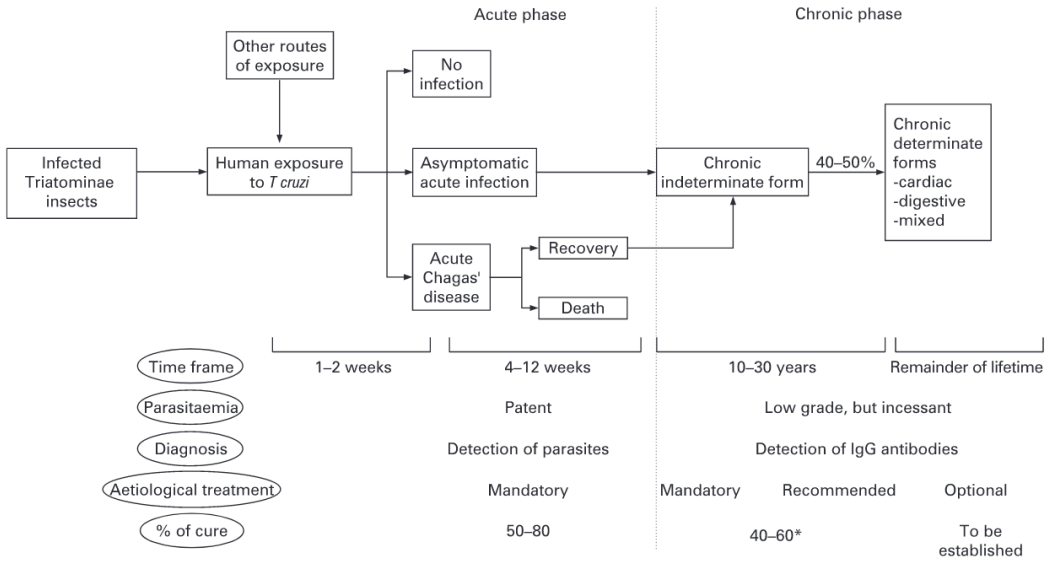
racial inequality and public health care spending, further augmenting the economic arguments for investing in eradication.

Second, by examining a disease that has mainly afflicted Latin America and has been called "a continent's scourge" (Delaporte, 2012), our results contribute to our understanding of the comparative development of Latin America. A growing literature has sought to understand Latin America's specific development path, characterized by disappointing growth and pronounced inequality (see Eslava and Caicedo, 2023, for a review). By spotlighting Chagas disease, a malady deeply entrenched in the socio-economic fabric of Latin America, we offer a novel perspective on the region's unique development trajectory. We first highlight that Chagas disease is negatively correlated with development in the cross-section today (Table 1).[2] Furthermore, we provide quasi-experimental evidence on how eliminating Chagas disease can have important long-run development benefits. Our results imply that further reducing Chagas disease could help improve development and reduce inequality in the region.

Finally, our study contributes to a large literature on health and inequality (e.g., Farmer, 2001; Deaton, 2003; O'Donnell, Van Doorslaer and Van Ourti, 2015; Bütikofer and Salvanes, 2020). Chagas Disease, as an NTD, encapsulates the vicious cycle of poverty and disease, where impoverished living conditions foster disease transmission, and the ensuing health ailments further entrench poverty. Our investigation into the economic and racial disparities stemming from Chagas Disease in Brazil highlights how NTDs that have both acute and chronic effects can impede economic development in the long-run. Moreover, the relevance of our study extends beyond Latin America as Chagas disease has been increasingly detected in non-endemic regions including the United States via climate change and migration (Hernández, 2021; Irish et al., 2022), underscoring the global pertinence of understanding and addressing this NTD.

[2] Additionally, Figure A2 shows that Chagas disease had pre-colonial development impacts as well: societies in Latin America in the Standard Cross-Cultural Sample that were recorded to have trypanosomes had lower levels of pre-colonial centralization.

Figure 2: Phases of Chagas Disease



Notes: Diagram taken from Rassi et al. (2009, p. 527).

2. Overview of Chagas Disease

2.1. Causal Agent and Vectors

The parasite *Trypanosoma cruzi* causes Chagas Disease. Around 90 percent of those infected contracted it from infected blood-sucking triatomine bugs.[3] Triatomine bugs live in cracks in roofs and walls and infect humans when they emerge at night to take blood meals, often from sleeping humans.[4] In Brazil, the most important vector species is *Triatoma infestans*, responsible for 80 percent of all transmission (Schofield and Dias, 1999).[5] Appendix Figure A1 presents an example image of *Triatoma infestans*. *T. infestans* became domesticated and spread via rural settlements after Brazilians began clearing forests for agriculture and ranching in the south and southeast of the country in the late nineteenth century (Schofield, 1988).

[3] The other 10 percent of transmission occurs through blood transfusions and the placenta.

[4] Triatoma bugs are also known by the following names: *Kissing bugs* (in English), *barbeiros* (in Brazil), *vinchucas* (in Argentina, Bolivia, Uruguay, and Chile), *chipos* (in Venezuela), or *chinches* (in Central America), among other names.

[5] The other main Brazilian vector is *T. brasiliensis*, which accounts for 10 percent of all transmission. In Central and northern South America, the main vectors are *Rhodnius prolixus* and *T. dimidiata*.

2.2. *Phases*

There are two phases of Chagas Disease. Figure 2 shows the progression of the disease from exposure through the rest of the patient's life. The acute stage begins after 1 to 2 weeks of incubation, lasts 4 to 12 weeks, and has non-specific symptoms like malaise and fever (Rassi et al., 2009). Children become more seriously ill in this phase than young adults, with as many as 10 percent of them dying from it, but more than 40 percent of those infected progress through this stage mostly symptom-free (Khan, 2011).

Individuals then enter the chronic phase. For 10 to 30 years, they experience no symptoms and approximately 50% of them will remain in a chronic indeterminate stage in which they never develop any lesions (Rassi, Rassi and Little, 2000). However, the other portion progresses to the chronic determinate phase. Most commonly, this group develops cardiac complications such as the heart muscle becoming degraded and being replaced by fibrous tissue. Such cardiomyopathy is the cause of most of the morbidity and mortality from Chagas Disease (Nunes et al., 2018).

2.3. *Economic Consequences of Chagas Disease*

Both stages have features that might affect economic development. Experiencing the acute phase in childhood should reduce lifetime income by affecting the knowledge component of adult human capital. Because of their symptoms, children would spend 1 to 3 months less able to focus in class or absent from school. In that case, they may fall behind their peers and repeat grades or decide to drop out entirely.[6] It is also possible for an adult to experience the acute phase's symptoms if they become infected, in which case they may struggle to work.

Entering the chronic determinate state of Chagas Disease in adulthood would have substantial negative impacts on developing countries as well. For individuals, years of shortness of breath, fatigue, and dizziness resulting from a heart with difficulty pumping blood should lead to reduced output at work, absenteeism, and even an inability to remain employed.

[6] In contrast to other parasitic infections (e.g., helminthiases), the medical literature on acute Chagas Disease does not mention sequelae like anemia and stunting that affect childhood development. But in a letter to the editor of *The Lancet*, Schofield (1981) estimated that individuals in the average house with *T. infestans* lose 2 to 3 ml of blood per day to the bugs, which might lead to anemia in children with inadequate diets or other parasitic infections. Subsequent studies did not investigate this claim, however.

Therefore, Chagas Disease should also have broader consequences for developing countries. A universal public health insurance program like Brazil's *Sistema Único de Saúde* (SUS, which consumes around 4 percent of GDP) would have to spend more heavily on hospitalizations and doctor's visits for those suffering from acute and chronic symptoms. As the disease is most likely to be transmitted to humans living in houses with cracks in the walls and roof, it also disproportionately affects those who were already poor. In the context of a strong correlation between race and poverty, the disease may exacerbate gaps between white and non-white Brazilians.

3. Brazil's Vector Control Program

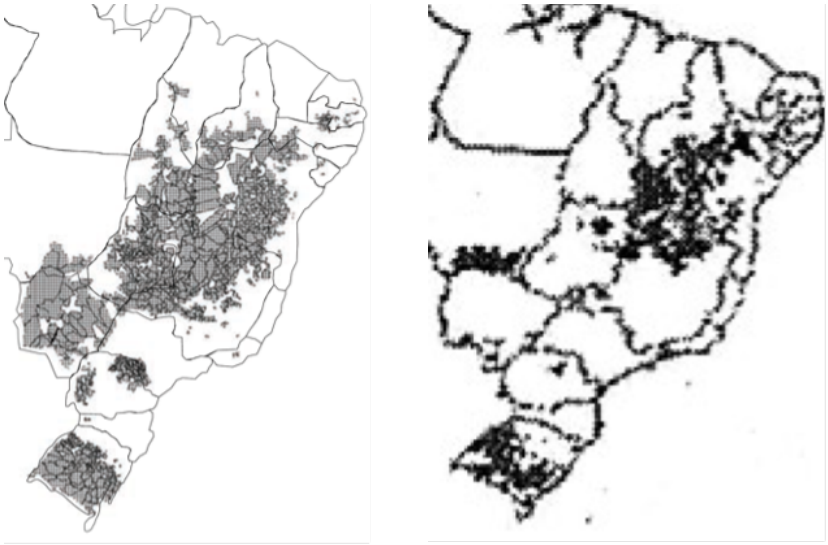
In this section, we provide an overview of Brazil's vector control program, which generates the quasi-experimental variation in exposure to Chagas Disease that we use to test the above hypotheses. To do so, we largely summarize Section 4 of Schofield and Dias (1999).

3.1. Breakthrough of Pyrethroid Insecticides

The post-World War II campaigns against malaria used organochlorine insecticides like DDT that were ineffective against Chagas Disease vectors. Several trials found that γ-benzene hexachloride (BHC) was effective if sprayed on the walls and roofs of triatomine-infested houses in high doses. In the 1960s, São Paulo's vector control superintendency began a program using BHC to effectively eliminate *T. infestans* from the state. However, São Paulo was the only state with the resources to implement such an intensive program.

The second generation of pyrethroid insecticides became available in the 1970s and studies by the end of the decade showed their effectiveness against triatomine bugs. Importantly, they were effective when sprayed less frequently and at low doses, which made them more cost-effective than BHC in spite of a higher price per kilogram. They were also easy to apply and did not have unpleasant odors.

Figure 3: Progress of Vector Control Campaign, 1975-89



(a) *T. infestans*, 1975-83 **(b)** *T. infestans*, 1989

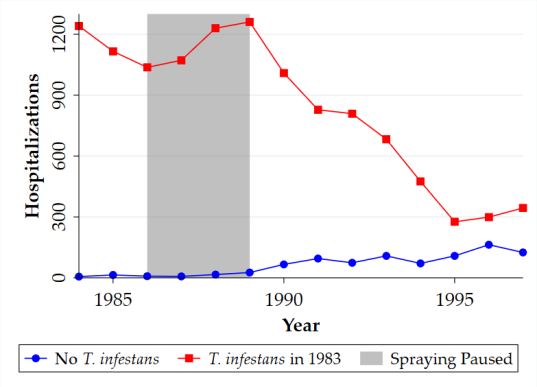
Notes: Figures taken from Silveira (2011) and Coura and Dias (2009) show municipalities with *T. infestans* in each period.

3.2. National Surveys and (an Interrupted) Vector Control Campaign

In 1975, the Brazilian government started a national campaign against Chagas Disease. It began because of, among other factors, the "development of suitable vector control methods, ... and continuous campaigning by scientists including demonstrations that vector control was feasible" (Dias, 1987, p. 338). The first stage consisted of serological and entomological surveys through the early 1980s. They found a national rural *T. cruzi* prevalence rate of 4.2 percent—including 8.8 percent rural prevalence in the heavily-infested states of Minas Gerais and Rio Grande do Sul—and vectors present in 36 percent of Brazil's territory (Dias, 1987). Figure 3a shows a map of municipalities that had *T. infestans* in dwellings from 1975 to 1983.

After the surveys concluded, thousands of sprayers visited millions of homes across the endemic region. However, the program came to a halt in 1986 due to the arrival of the *Aedes aegypti* mosquito in coastal areas and resulting outbreaks of dengue fever. Due to political pressure, the public health campaign superintendency diverted 40 percent of its personnel to dengue control, leading to a reemergence of triatomine in recently-sprayed municipalities (Dias, 1987; Schofield and Dias, 1999). But vector control resumed in 1989, and the reduction in *T. infestans* in that year

Figure 4: Hospitalizations for Acute Chagas Disease, 1984-97



Notes: Graph shows the evolution of hospitalizations for acute Chagas Disease (ICD-9 code 086) in states without (blue circles) and with (red squares) *T. infestans* in 1983. Data are from DATASUS. The shaded years (1986-89) denote the interruption in vector control.

compared to 1975-83 is clear in Figure 3b.

To show the health impacts of (the interruption in) spraying, Figure 4 presents a graph of 1984-97 hospital admissions due to (acute) Chagas Disease.[7] They declined at the beginning of the campaign through 1986 and then increased with the interruption of control efforts lasting until 1989. Subsequently, admissions declined again as the program resumed, and by 2006, the Pan American Health Organization certified Brazil as having interrupted transmission of Chagas Disease through *T. infestans* in every state.[8]

4. Data and Treatment Definition

4.1. Data

Given the discussion of the potential effects of Chagas Disease in Section 2.3, we focus on both the short- and long-run economic impacts of the health improvement induced by control of triatomine. To study the former, we use microdata from the National Household Sample Survey (PNAD) for 11 of the 18 years from 1982 to 1999.[9] These surveys contain data on respondents' state of residence, household characteristics and sociodemographic information, and schooling

[7] Because of the non-specific symptoms of the acute phase, these numbers are almost certainly an undercount. Nonetheless, they are helpful in verifying the effects of the vector control campaign.

[8] https://www.paho.org/english/AD/DPC/CD/dch-incosur-xv.htm

[9] The missing years are 1983, 1984, 1985, 1988, 1991, 1994, and 1996.

and labor market outcomes. While the geographic resolution of these data are coarse and several years immediately around the start of spraying are missing, their near-annual frequency allows us to examine how quickly outcomes changed in states of residence after spraying began.

We also use IPUMS microdata from the 1980 and 2010 censuses of Brazil (Minnesota Population Center, 2020) to examine the long-run effects of Chagas Disease vector control. These 25- and 10-percent samples include information on a respondent's consistent 1980-2010 municipality of residence and consistent 1960-2010 state of birth, as well as an indicator for whether a respondent was born in their municipality of residence. We use the 1980 data to construct our measure of exposure to the treatment when only a respondent's state of residence or birth is known, as we describe in the next section. The census also contains more detailed household, sociodemographic, schooling, and labor market data than the PNAD datasets, which we exploit in the 2010 data.

4.2. Treatment Definition

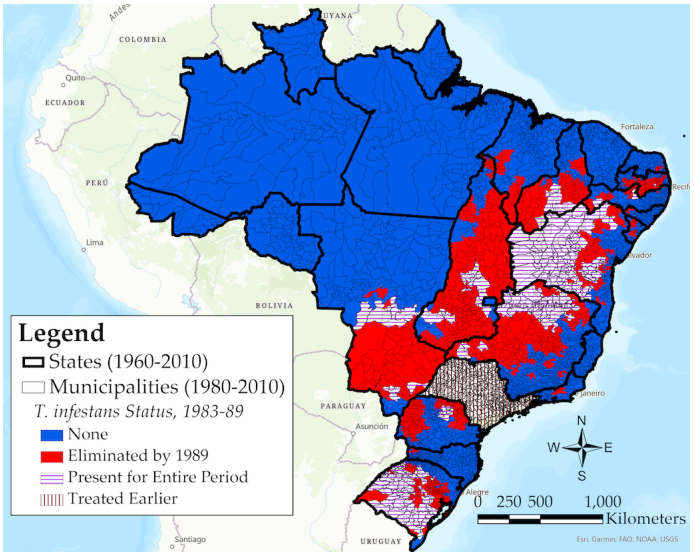
We define control municipalities as those that were never treated with spraying—i.e., they did not have *T. infestans* in 1975-83 and did not require vector control to achieve this status.[10] Our treatment group consists of those with *T. infestans* present in 1975-83 but eliminated by 1989 as a result of the spraying that began in 1984.[11] We use these years as endpoints because it ensures that our 2010 census sample has birth cohorts that were had spent their entire childhoods living without *T. infestans*. Because we cannot determine in which year a municipality became free of the vector, we assume that all were treated in 1984 to be conservative.

Figure 5 shows control municipalities in blue, treatment ones in red, and those we exclude in white with either horizontal purple lines (not yet treated) or vertical brown ones (treated earlier). Table 2 shows summary statistics for demographic, labor market, and human capital variables among all individuals in these groups in 1980, 4 years before the start of spraying. Municipalities in our treatment group were more white and had slightly less schooling than those in our control

[10] The latter condition leads us to exclude the state of São Paulo, as its prior vector control campaign means it is not part of the never-treated group but rather an earlier-treated one (Goodman-Bacon, 2021).

[11] We also exclude not-yet-treated municipalities that had triatomine bugs in 1975-83 and 1989. Note that this group also includes municipalities where spraying began but the 3-year pause allowed the vector to return by 1989.

Figure 5: Municipalities by 1983-89 *T. infestans* Status



Notes: Map shows control municipalities (never had *T. infestans*) in blue and treatment municipalities (*T. infestans* eliminated by 1989) in red. Municipalities that still had *T. infestans* in 1989 (white with horizontal purple lines) or were treated prior to this period (white with vertical brown lines) are omitted from our sample. Data are from Silveira (2011) and Coura and Dias (2009) (see Figures 3a and 3b). The underlying shapefile of consistent 1980-2010 municipalities and 1960-2010 states is from IPUMS.

Table 2: Summary Statistics, 1980

	Municipalities by 1983-89 *T. infestans* Status			
	None	Eliminated	Present	Treated Earlier
Panel A. Demography				
Age	23.748	23.717	23.722	25.054
	(34.251)	(31.166)	(33.434)	(27.835)
Female	0.506	0.501	0.500	0.501
White	0.457	0.587	0.541	0.749
Brown	0.477	0.355	0.396	0.185
Black	0.065	0.056	0.059	0.046
Asian	0.002	0.003	0.004	0.020
Panel B. Labor Markets				
Log Monthly Income	4.258	4.261	4.002	5.374
	(4.361)	(4.343)	(4.283)	(4.532)
Employed (if in LF)	0.975	0.980	0.978	0.983
Panel C. Human Capital				
Years of Schooling	4.490	4.305	4.047	5.210
	(4.774)	(4.643)	(4.675)	(4.523)
Observations	15,328,696	4,228,428	1,914,177	5,686,026
Municipalities	1,156	402	173	309

Notes: Table lists means for variables of interest in the 1980 census and standard deviations in parentheses under the means for continuous variables. Categories correspond to Figure 5. Data are from IPUMS.

group, though incomes were equal.

5. Effects on Individuals: Long-run and Next Generation

In this section, we examine the long-run effects of triatomine control on adults who were children around the time that spraying began. Our results show that spraying raised incomes more among adults who were more exposed to it in childhood. These results are markedly different by race: incomes and employment rates rose substantially more for non-white adults. We also show that the inter-quartile range in incomes for treated cohorts decreased. The implication is that vector control decreased inequality and increased the speed of racial convergence in Brazil in the long run.

5.1. Empirical Strategy

We compare outcomes for adults in the 2010 census across municipalities of birth with varying levels of pre-treatment *T. infestans* presence. Our baseline estimating equation is the dynamic difference-in-differences model:

$$y_{i,m,c} = \alpha_m + \gamma_c + \sum_{k \neq 1965} \tau_k \cdot \mathbf{1}[\mathbb{P}(Treat_m) > 0] \cdot \mathbf{1}[c = k] + \mathbf{X}_i\beta + \epsilon_{i,m,c} \tag{1}$$

$$\text{for } k \in \{1960, \ldots, 1979\}$$

where $y_{i,m,c}$ is an outcome of interest for individual i born in municipality m from birth-year cohort c. α_m and γ_c are municipality and cohort fixed effects, $\mathbb{P}[Treat_m]$ is the probability in the 1980 census that an individual in a state of that sex and race resides in a treatment municipality, $\mathbb{1}[c = k]$ indicates whether an observation is from the given year k, $\mathbf{X}_i$ is a vector of individual-level covariates (age, age squared, and fixed effects for female sex and Asian, Black, and Brown racial categories), and $\epsilon_{i,s,c}$ is the idiosyncratic error term. We cluster standard errors by municipality of birth. We estimate equation (1) using the two-way fixed effects estimator proposed by Borusyak, Jaravel and Spiess (2022).

Note that we use 1965 as the omitted cohort, as these adults were 18 years old in 1983. The oldest group we include is the 1960 birth cohort (age 50 in 2010) for two reasons. At the

time of this census, workers could retire with social security benefits after having paid into the system for 30 to 35 years. Additionally, DDT spraying began in the late 1950s, so we want to avoid comparing cohorts with different levels of exposure to that treatment. The youngest group we include is the 1979 cohort (age 31 in 2010) so that our sample contains prime-age adults with varying exposure to Chagas Disease in childhood and who had already made substantial progress along their lifetime earnings trajectories.

Our assumption is that the 1960 to 1965 cohorts were too old to have experienced the benefits of Chagas Disease control during their childhood years. Given that Chagas eradication can reduce the acute phase, it is important to note that these benefits could have accrued to both them *as well as their parents* from averting the acute phase—the latter may have implied more available resources as a child. On the other hand, these too-old cohorts still may have benefited from not contracting Chagas Disease in young adulthood, thus avoiding its acute phase in the short run and its chronic determinate phase in the long run. The implication is that null results using this strategy could arise from either the absence of long-run effects from controlling this disease during childhood or the greater importance of its chronic phase for adult outcomes.[12]

While our estimates are at the level of municipality of birth m because of the greater geographic detail in the census data, the 2010 census only reports municipality of birth if individuals reside in their municipality of birth; otherwise, it reports an individual's state of birth. Nearly two-thirds of the sample live in their municipality of birth, so for these adults $\mathbb{P}[Treat]_m$ is either zero (55 percent of the sample) or one (11 percent). For those living away from their birth municipality, we assign them the probability in the 1980 census that an individual of their sex and race who was living in their state of birth was in a treatment municipality.

The coefficients of interest are the τ_k, which measure the difference in an outcome for a given birth cohort as the probability of having been born in a treatment municipality goes from 0 to 1, relative to the size of that difference for the 1965 cohort that was aged 18 in the year prior to the start of the control campaign in 1983. Because the mean value for $\mathbb{P}(Treat_m)$ is approximately

[12] It also means our long-run results have a somewhat different interpretation from those for childhood exposure to malaria (e.g., Bleakley, 2010; Lucas, 2010; Cutler et al., 2010), which primarily affects children. However, it is similar to the results on childhood exposure to tuberculosis, which also affects both children and adults (e.g., Bütikofer and Salvanes, 2020).

0.24, we frame our results as moving the municipality of birth from 25% to 0% of population with vector exposure and discuss the implied magnitude of the estimate in the text.

Along with equation (1), we also estimate the static difference-in-difference model

$$y_{i,m,c} = \alpha_m + \gamma_t + \tau \cdot \left(\mathbf{1}[\mathbb{P}(Treat_m) > 0] \cdot \mathbb{1}[c > 1965]\right) + \mathbf{X}_i\beta + \epsilon_{i,c,m} \tag{2}$$

which is identical to the previous equation except that the probability of being born in a treatment municipality is interacted with a single variable $\mathbb{1}[c > 1965]$, an indicator for whether an individual was born after 1965. By pooling the post-treatment cohorts, the estimate of τ is likely to be more precise, though it comes at the cost of imposing a single value across all cohorts in this period. We view these strategies as complementary because they both have an important drawback but each helps to address the concern regarding the other.

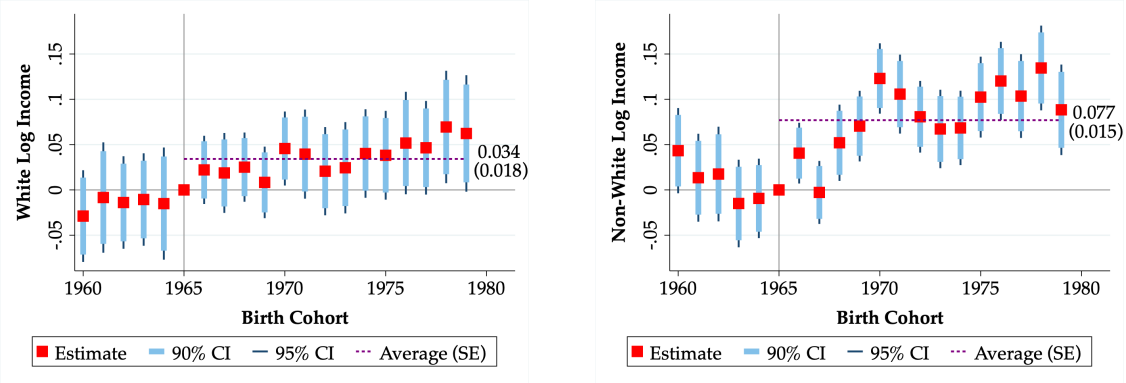
To examine the potential for Chagas disease control to reduce racial inequality in the long run, we estimate equations (1) and (2) separately for white and non-white individuals. Rather than imposing additional identification assumptions and conducting statistical tests for another difference in trends across racial groups (i.e., a triple-differences framework) given the potential for imprecision, we simply compare the respective estimates' magnitudes patterns and discuss implications.

5.2. *Income and Inequality Results*

Our first outcome of interest is (the natural log of) monthly income for the 1960 to 1979 birth cohorts in 2010. Figure 6 shows that while incomes for the adults who turned 18 before the start of spraying evolved in parallel for both white (6a) and non-white Brazilians (6b), those who were children during triatomine control experienced slightly greater increases the more they were exposed to treatment. The static estimate implied for moving from the 25% to 0% of the population being exposed to the triatome vector imply that incomes increased 3.4% for white adults and 7.7% for non-white adults treated in childhood. These results by race suggest that, in the long-run, the Chagas Disease control program helped reduce racial disparities.

Given that the results in Figure 6 suggest that the Chagas control campaign helped reduce racial inequities, we next examine how the campaign impacted income inequality. For each

Figure 6: Long-Run Effects on Incomes
Differential Effects by Race



(a) White: Log Monthly Income **(b)** Non-White: Log Monthly Income

Notes: Graphs plot dynamic (red squares) and static (dotted purple line) estimates for the respective outcomes and racial groups with 90-percent (light blue) and 95-percent (dark blue) confidence intervals for dynamic estimates. Static estimate magnitudes are next to the dotted purple lines with standard errors below them in parentheses. Data are from the IPUMS sample of the 2010 census. Regressions control for fixed effects for municipality, year, female sex, and racial category (Asian, Black, and Brown) as well as age and age squared. Standard errors are clustered by the municipality of birth. Regressions use the following number of observations: (a) 1,964,673 and (b) 1,907,724. For pre-treatment cohorts, means of the respective outcomes were: (a) 6.58 and (b) 5.86.

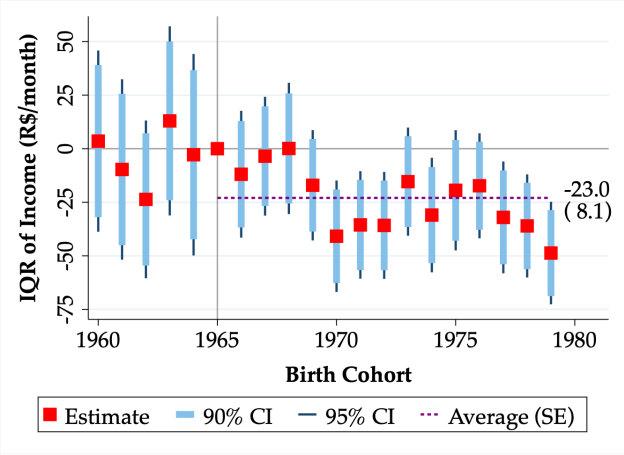
municipality of birth, we calculate the inter-quartile range of earned income for each cohort, and examine how the campaign impacted the inter-quartile range in Figure 7. We find that the Chagas control campaign reduced the inter-quartile range of earned in come by R$ 23/month (3.3%) for adults born in treatment municipalities. These results provide additional evidence that the Chagas control campaign helped reduce income disparities.

5.3. *Inter-Generational Impacts*

The 2010 census data also allows us to study the inter-generational impacts of the control campaign on the children of exposed cohorts of parents. For children living in the same household as their parents, we are able to link parents' exposure to the campaign to children and their outcomes. We examine literacy and schooling outcomes for children aged between 6 and 18.[13]

[13] We limit the sample to exclude children over 18 due to potential selection effects via lower probability of them living with their parents after 18. We select 6 years of age as the lower age bound since age 6 is when primary schooling begins in Brazil.

Figure 7: Long-Run Effects on Income Inequality



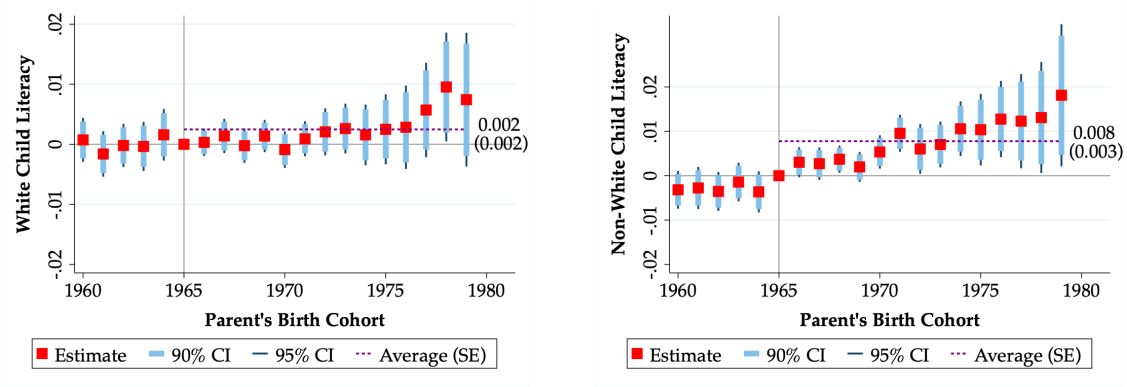
Notes: Graphs plot dynamic (red squares) and static (dotted purple line) two-way fixed effects estimates for the inter-quartile range of earned income with 90-percent (light blue) and 95-percent (dark blue) confidence intervals for dynamic estimates. Static estimate magnitudes are next to the dotted purple lines with standard errors below them in parentheses. Data are from the IPUMS sample of the 2010 census. Observations are municipalities of birth. Regression uses 31,633 observations. Average effect is displayed next to dotted purple line with standard errors clustered by municipality of birth in parentheses. For pre-treatment cohorts, the average IQR of income was R$ 699.7 per month.

We present the results on children's literacy in Figure 8, for children of white (8a) and non-white parents (8b). We find that the control program is associated with an increase of 0.8 p.p. for children of non-white parents who were treated in childhood. Relative to the low average illiteracy rate for non-white children of 6.5%, the estimated effects corresponds to approximately a 9% decline in illiteracy relative to the mean. These results provide some evidence that a portion of the benefits of the Chagas control program is transferred from parents to their children, highlighting the positive inter-generational impacts of combating neglected tropical diseases.

5.4. Mechanisms Behind Income and Inequality Results

A natural question is what explains the long-run income and inequality results. Much of the literature on the positive impacts of neglected tropical disease treatment has focused on its impacts via increased school attainment (e.g., Miguel and Kremer, 2004; Bleakley, 2007, 2010). Thus, we first examine whether years of schooling in the long-run can account for the observed impacts. However, like many NTDs, Chagas disease is a complex disease with both acute and chronic health impacts. This suggests that eliminating Chagas disease transmission could affect human

Figure 8: Long-Run Inter-Generational Effects on Children
Differential Effects by Race



(a) White: Child Literacy **(b)** Non-White: Child Literacy

Notes: Graphs plot dynamic (red squares) and static (dotted purple line) two-way fixed effects estimates for the respective outcomes and racial groups with 90-percent (light blue) and 95-percent (dark blue) confidence intervals for dynamic estimates. Static estimate magnitudes are next to the dotted purple lines with standard errors below them in parentheses. Data are from the IPUMS sample of the 2010 census. Regressions control for fixed effects for parent's municipality of birth, birth year, female sex, age, and racial category (Asian, Black, and Brown) as well children's female sex, number of siblings, and child age fixed effects. Standard errors are clustered by the municipality of birth. Regressions use 2,045,779 child-parent observations in (a) and 3,118,342 in (b). For pre-treatment cohorts, the mean rate of literacy was 97.2 percent for whites and 93.5 percent for non-whites. Children are limited to be between 6 and 18 years old.

capital beyond just years of schooling.[14] In particular, eliminating Chagas can have short-run benefits on health and household resources via reductions in the acute-phase of Chagas disease. Furthermore, it might have subsequent long-run benefits to health via reducing the chronic phase. For these reasons, we examine whether the control campaign is associated with increases structural transformation in treated municipalities. We then turn to specifically examining the long-run health benefits (in Section 7).

5.4.1. Mechanisms: Schooling

We examine the role of schooling in the explanation for our labor market results. Using the 2010 census data and the same identification strategy as in 5.2, we examine whether the control campaign led to large enough increases in schooling to explain the income and inequality re-

[14] See also Mora-Garcia (2018) and Bütikofer and Salvanes (2020) for evidence of how NTD control affects income via channels outside just schooling.

sults. Figure 9 presents the estimates and shows the absence of pre-treatment differential trends followed by increases in post-treatment cohorts' completed years for white (9a) and non-white adults (9b). Interestingly, Figures 9a and 9b show a larger increase in absolute terms for white cohorts (implied static estimate of 0.11 years) than non-white ones (0.08 years), but relative to the means for pre-treatment cohorts, the non-white effect is slightly larger (1.5 percent for white cohorts vs 1.6 percent for non-white ones). This absence of substantive differences across white and non-white adults suggests there may not have been any differential effects by racial group in terms of schooling attainment, making it an unlikely explanation for the reduction in racial disparities.

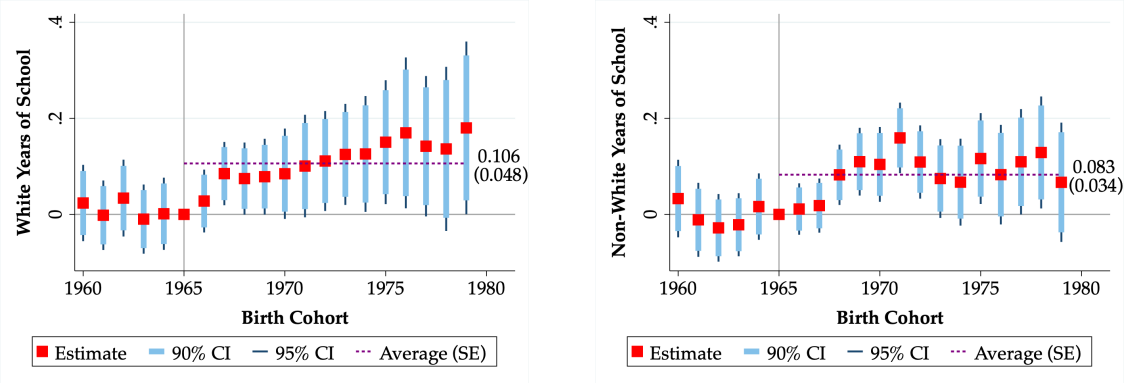
To further understand the importance of the schooling channel for the white vs. non-white income result, we make a back-of-the-envelope calculation of the upper bound of its contribution. Using the Mincerian return to schooling in Brazil of 15.7 percent reported in Psacharopoulos and Patrinos (2018)—which almost certainly overstates the causal effect of schooling on income—the additional schooling for non-white cohorts induced by vector control can account for at most 1.84 p.p. (one-fourth) of the 7.7-percent increase in their incomes. It suggests that avoiding chronic Chagas Disease in adulthood may play an important role in explaining differentially larger income increases for non-white Brazilians.

5.4.2. Mechanisms: Long-Run Benefits Related to Health

Reductions in chronic Chagas, which often occurs ten (or more) years after infection, could be particularly meaningful for long-run incomes since "Chagas disease can cause young adults to develop heart conditions, so that they fill hospital beds instead of the labour force" (World Health Organization, 2010). Therefore, the health benefits from reductions in chronic Chagas could help explain the long-run income patterns.

We explore whether better health outcomes due to reductions in Chagas conditions can help explain part of the income results in Section 5. The 2010 census occurred over 20 years since the end of the control program, meaning that reductions in chronic Chagas disease would have manifested by the time of the census. Because the 2010 census does not include questions directly on health, we use proxies for better health. (We directly examine long-run health benefits in

Figure 9: Long-Run Effects on Educational Attainment
Differential Long-Run Effects by Race



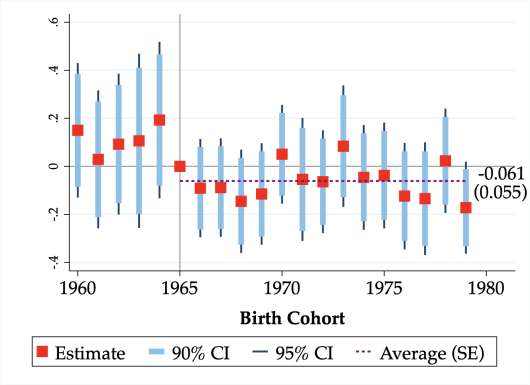
(a) White: Years of Schooling **(b)** Non-White: Years of Schooling

Notes: Graphs plot dynamic (red squares) and static (dotted purple line) two-way fixed effects estimates for the respective outcomes and racial groups with 90-percent (light blue) and 95-percent (dark blue) confidence intervals for dynamic estimates. Static estimate magnitudes are next to the dotted purple lines with standard errors below them in parentheses. Data are from the IPUMS sample of the 2010 census. Regressions control for fixed effects for state, year, female sex, and racial category (Asian, Black, and Brown) as well as age and age squared. Standard errors are clustered by the 24 consistent 1960-2010 states. Regressions use the following number of observations: (a) 2,568,352 and (b) 2,730,649. For pre-treatment cohorts, means of the respective outcomes were: (a) 6.91 years and (b) 5.10 years.

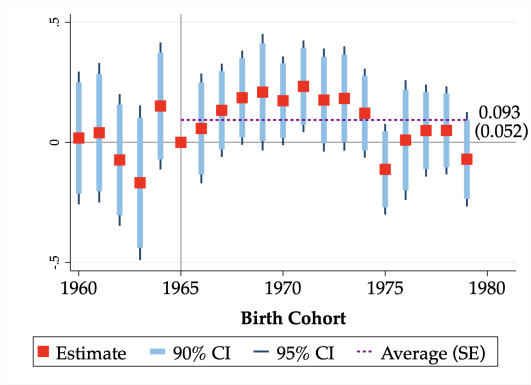
Section 7.)

Our first outcome of interest is hours worked. If treated cohorts are healthier in 2010 due to reductions in chronic Chagas, then we might expect to see them work more and receive fewer government benefits. Using the same data and identification strategy as in 5, we examine whether the control campaign led to increases in hours worked. Figure 10 presents the estimates and shows the absence of pre-treatment differential trends followed by increases in hours worked for white (10a) and non-white treated cohorts (10b). These results suggest that treated cohorts are able to work more, potentially due to better health. Furthermore, Figure 10 also presents the estimates for whether individuals are receiving a government pension for white (10a) and non-white (10b) cohorts. The results suggest that treated non-white cohorts are also less likely to receive government support. These results provide suggestive evidence that reductions in chronic Chagas could play an important role in the long-run economic benefits and reductions in inequities from controlling Chagas disease.

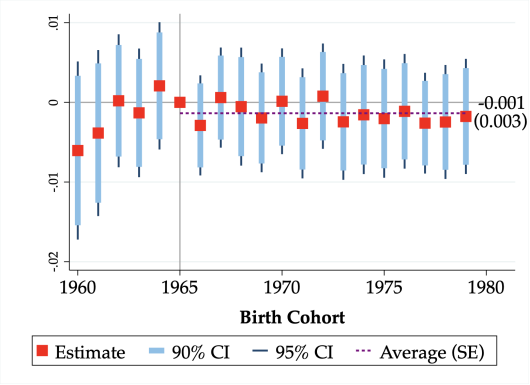
Figure 10: Long-Run Effects on Work
Differential Effects by Race



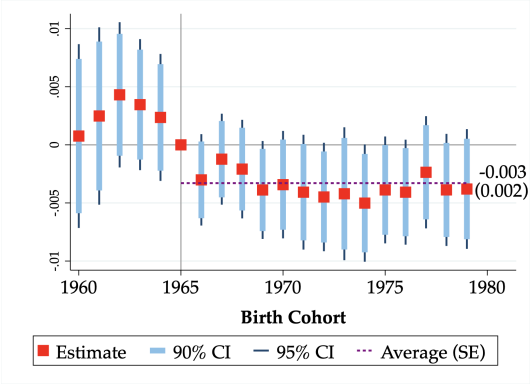
(a) White: Usual Hours Worked



(b) Non-White: Usual Hours Worked



(c) White: Receives Pension



(d) Non-White: Receives Pension

Notes: Graphs plot dynamic (red squares) and static (dotted purple line) two-way fixed effects estimates for the respective outcomes and racial groups with 90-percent (light blue) and 95-percent (dark blue) confidence intervals for dynamic estimates. Static estimate magnitudes are next to the dotted purple lines with standard errors below them in parentheses. Data are from the IPUMS sample of the 2010 census. Regressions control for fixed effects for municipality, year, female sex, and racial category (Asian, Black, and Brown) as well as age and age squared. Standard errors are clustered by the municipality of birth. Regressions use the following number of observations: (a) 1,964,673 and (b) 1,907,724. For pre-treatment cohorts, means of the respective outcomes were: (a) 6.58 and (b) 5.86.

6. Effects on Places: Short-run and Long-run Structural Transformation

We examine how reducing exposure to Chagas Disease affected municipalities in both the short- and long-run. We compare treatment and control municipalities across decadal population census waves to assess how reductions in acute Chagas Disease (in the short-run) and chronic Chagas Disease (in the long-run) affected structural transformation. First, we examine how reductions

in acute Chagas Disease affected outcomes shortly after the eradication campaign, as the 1 to 3 months of symptoms could interfere with both of these outcomes. Second, we explore whether the effects accumulate over time, especially as chronic symptoms were reduced.

Our results show that the eradication campaign led to short-run benefits. Treated municipalities experienced slightly more structural transformation: decreases in the share of the labor force employed in agriculture and in rural areas, and increases in the share of the labor force employed in manufacturing and in incomes. Thus, reduction in acute Chagas Disease seems to have led to immediate short-run benefits. Furthermore, these benefits tend to grow more pronounced over time, especially in census waves 10 years after the end of the eradication campaign. These results suggest that reducing chronic Chagas in the long-run augmented the positive short-benefits.

6.0.1. Empirical Strategy

Our evidence comes from comparing treated and non-treated municipalities across population census waves (1970, 1980, 1991, 2000, and 2010), motivated by the idea that vector control induced greater improvements in health where there was more exposure to Chagas Disease prior to spraying. Our estimating equation is the dynamic difference-in-differences model:

$$y_{m,c} = \alpha_m + \gamma_c + \sum_{k \neq 1980} \tau_k \cdot \mathbf{1}[\mathbb{P}(Treat_m) > 0] \cdot \mathbf{1}[c = k] + \gamma_{s(m)y} + \mathbf{X}_m \beta + \epsilon_{m,c} \tag{3}$$

$$\text{for } k \in \{1970, 1980, 1991, 2000, 2010\}$$

where $y_{m,c}$ is an outcome of interest in municipality m in census wave c. α_m and γ_c are municipality and census-wave fixed effects, $\mathbf{1}[\mathbb{P}(Treat_m) > 0]$ is an indicator function equal to 1 if municipality m was part of the control campaign and 0 otherwise, $\mathbb{1}[c = k]$ indicates whether an observation is from the given census k, $\mathbf{X}_m$ is a vector of municipality-level covariates measured prior to the control campaign (share of population that is female, Asian, Black, and Brown), $\gamma_{s(m)y}$ are state-by-census fixed effects, and $\epsilon_{m,c}$ is the idiosyncratic error term. We cluster standard errors by municipality. As with equation (1), we estimate equation (6) using the two-way fixed effects estimator proposed by Borusyak, Jaravel and Spiess (2022).

The coefficients of interest are the τ_k, which measure the difference in an outcome in a given

census as the probability of being a treated municipality goes from 0 to 1, relative to the size of that difference in outcomes in 1980. For $k \geq 1991$, τ_k will allow us to assess whether reductions in exposure to acute (and eventually chronic) Chagas disease led to meaningful economic benefits. Conversely, $k < 1980$, we expect τ_k to be of economically and statistically insignificant magnitudes if our identifying assumption of parallel trends is valid.

Along with equation (6), we also estimate the static two-way fixed effects model

$$y_{m,c} = \alpha_m + \gamma_c + \tau \cdot \left(\mathbf{1}[\mathbb{P}(Treat_m) > 0] \cdot \mathbb{1}[k > 1980]\right) + \gamma_{s(m)y} + \mathbf{X}_m\beta + \epsilon_{m,c} \tag{4}$$

which is identical to the previous equation except that the probability being a treated municipality is interacted with a single variable $\mathbb{1}[k > 1980]$, an indicator for whether the decadal census was conducted after 1980 (i.e., after the control campaign ended in 1989).

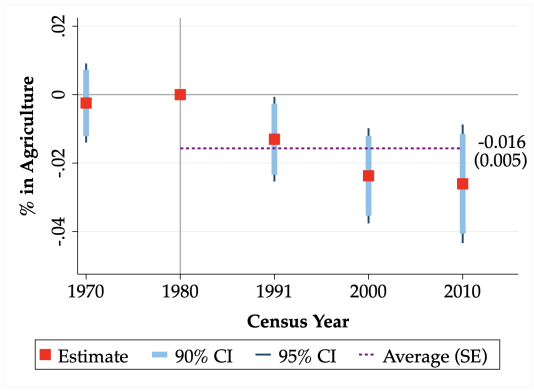
6.0.2. Structural Transformation Results

We first examine the effects of Chagas Disease control on employment across different sectors. Figure 11 plots the estimates from equations (6) and (7) for the share of the labor force employed in agriculture (11a), manufacturing (11b), and services (11c). Figure 11 suggests that individuals in treatment municipalities were less likely to work in the agricultural sector after spraying began, and became more likely to work in manufacturing and services. The patterns in the dynamic estimates suggest that these effects grow over time. Figure 11 also plots the share of the population in rural areas (11d). The results suggest that the control campaign reduced the share of individuals in a municipality that resided in rural areas, and that these effects become more pronounced over time.

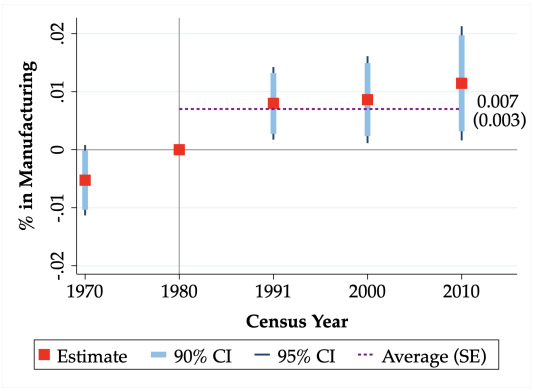
We also examine the effects of the control campaign on years of schooling and income, to validate our previous results that found little increases in school attainment despite increases in incomes. Figure 12 plots the estimates for the average years of schooling (12a) and log monthly income (12b). The estimates suggest that the control campaign did not meaningfully impact schooling in neither the short- nor long-run. However, the control campaign was associated with increases in incomes.[15]

[15] Despite large income effects in each census wave post-campaign, we avoid inferring much from the dynamic pattern of the income effects because of changes in how income was measured across different

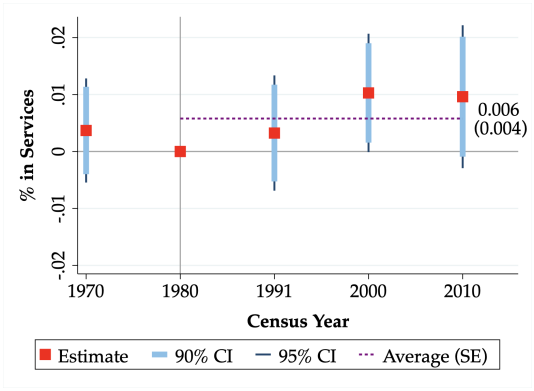
Figure 11: Short- and Long-Run Effects on Structural Transformation Outcomes



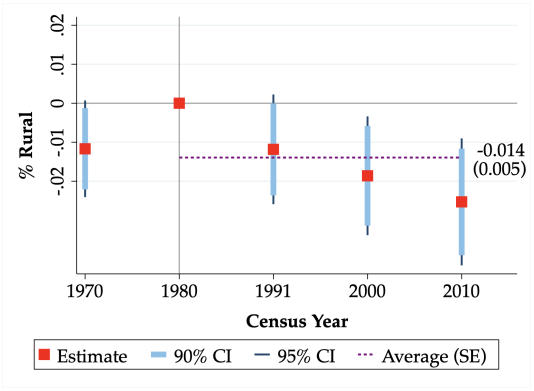
(a) % in Agriculture



(b) % in Manufacturing



(c) % in Services



(d) % Rural

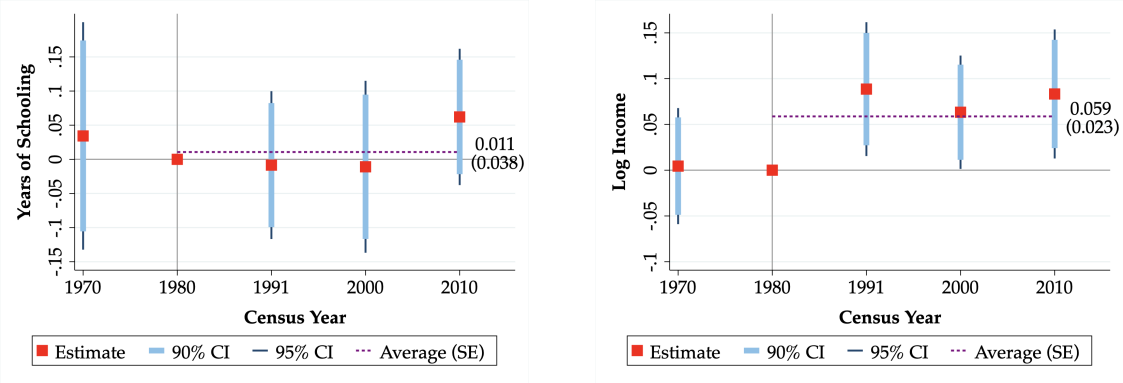
Notes: Graphs plot dynamic (red squares) and static (dotted purple line) two-way fixed effects estimates for the respective outcomes with 90-percent (light blue) and 95-percent (dark blue) confidence intervals. Static estimate magnitudes are next to the dotted purple lines with standard errors in brackets. Data are from the 1970, 1980, 1991, 2000, and 2010 Brazil population censuses. Regressions control for fixed effects for municipality, census wave, and state-by-census wave, and control for the share of the population in 1980 that is female, Asian, Black, Brown, Indigenous, and White interacted with census-wave fixed effects. Standard errors are clustered at the municipality level. Regression uses 1,750 municipalities each census wave for a total of 8,750 observations. For pre-treatment municipalities, the average % of the labor force in agriculture was 56.8%, the % in manufacturing was 9.2%, the % in services was 32.8%, and the % rural was 55.0%.

Take together, the results suggest that reductions in acute Chagas Disease in the short-run led to important benefits: individuals in treated municipalities were more able to take part in structural transformation.[16] These benefits tended to increase over time as well, especially in time periods when the benefits to reduced chronic Chagas disease are likely to become important.

survey waves.

[16] Appendix Section Appendix B examines a related set of outcomes using household survey data (PNAD) from Brazil (which are conducted yearly but are aggregated to the state level) and similarly shows short-run benefits from the Chagas disease control.

Figure 12: Short- and Long-Run Effects on Schooling and Incomes



(a) Years of Schooling **(b)** Log Income

Notes: Graphs plot dynamic (red squares) and static (dotted purple line) two-way fixed effects estimates for the respective outcomes with 90-percent (light blue) and 95-percent (dark blue) confidence intervals. Static estimate magnitudes are next to the dotted purple lines with standard errors in brackets. Data are from the 1970, 1980, 1991, 2000, and 2010 Brazil population censuses. Regressions control for fixed effects for municipality, census wave, and state-by-census wave, and control for the share of the population in 1980 that is female, Asian, Black, Brown, Indigenous, and White interacted with census-wave fixed effects. Standard errors are clustered at the municipality level. Regression uses 1,750 municipalities each census wave for a total of 8,750 observations. For pre-treatment municipalities, the average years of schooling was 3.1 and the average log income was 4.4.

These improvements in health in both the short- and long-run likely played an important role in the income results in Section 5. In the next section, we more carefully examine the long-run health benefits of reducing chronic Chagas disease.

7. Effects on Public Health Care System

If the morbidity from chronic Chagas Disease was severe enough to keep adults from being productive or even working, reducing it through triatomine control may have impacted more than just individuals' labor market outcomes. As Brazil has the world's largest government-run health care system (the *Sistema Único de Saúde*, or SUS) consuming about 4 percent of its GDP, improvements in adults' cardiovascular health could have had important effects on its public finances. According to SUS data, circulatory system diseases caused one-tenth of the hospitalizations that it paid for since 2010 (over 850,000 per year), which accounted for one-fifth of its spending on hospital care in this period (averaging nearly 1.5 billion 2019 Brazilian *reais* annually, or around 0.1 percent of GDP).

Therefore, in this section we examine the long-run effects of triatomine control on circulatory system-related hospitalizations covered by SUS and the resulting spending. Using a triple-differences strategy comparing circulatory and non-circulatory system-related causes, we show that hospitalizations and spending resulting from the former decreased more in states more exposed to treatment. The implication is that controlling Chagas Disease transmission has yielded substantial benefits for the public health care system and public finances in Brazil.

7.1. Data and Empirical Strategy

Our outcomes of interest are (the natural logs of) hospitalizations, person-days spent in the hospital, spending on hospital care, and deaths in each state by cause from 1984 to 2019. These data are from the SUS's Hospital Information System (SIH/SUS), and we deflate the last of them so that figures are in constant (log) 2019 Brazilian *reais* (BRL). Given that chronic Chagas Disease manifests primarily as cardiovascular problems, we focus on all diseases of the circulatory system.[17] We combine these data with each state's 1980 share of its population living in treatment municipalities to create a state-level treatment measure.

However, there is an additional dimension to our empirical approach that was not necessary in previous sections. The SUS is a heavily decentralized system with transfers of responsibilities and funds to state and municipalities (Castro et al., 2018). Therefore, there likely are confounders varying across both state and year (e.g., public health priorities, non-hospital care spending) in violation of the common trends assumption.

To address this complication, we use a triple-differences strategy with all other disease categories as the additional control group. Our assumption is that they are subject to the same state-specific, time-varying factors as circulatory diseases. If it is the case, the triple-difference approach is a valid strategy when the one in previous sections rejects the absence of differential pre-treatment trends for each disease category (Olden and Møen, 2022). The specification we use to estimate dynamic effects is

$$y_{s,t,d} = \alpha_{s,t} + \gamma_{t,d} + \delta_{s,d} + \sum_{k \neq 1999} \tau_k \cdot \left(\mathbf{1}[\mathbb{P}(Treat)_s > 0] \cdot \mathbb{1}[t = k] \cdot \mathbb{1}[d = circ]\right) + \epsilon_{s,t,d}, \tag{5}$$

[17] For 1984 to 1997, we use ICD-9 codes 390-459, and for 1998 onwards, we use ICD-10 codes I00-I99.

where $y_{s,t,d}$ is state s's log outcome in year t due to disease category d, $\alpha_{s,t}$ are state-year fixed effects, $\gamma_{t,d}$ are year-disease category fixed effects, $\delta_{s,d}$ are state-disease category fixed effects, and $\mathbb{1}[d = circ]$ indicates whether the category is circulatory diseases.

This strategy compares first compares the differences in log outcomes due to circulatory diseases in a given year as the probability of a state's population living in a treatment municipality goes from 0 to 1, relative to the size of that difference in 1999 (see below for why 1999 is the natural reference year for chronic Chagas). Then it compares this double-difference estimate to the analogous one for non-circulatory diseases. Because the mean value of this probability is approximately 25 p.p., we frame our results as moving from 25% to 0% of population with vector exposure. For inference, we compute wild cluster bootstrap confidence sets after clustering standard errors by the "small" number of states.

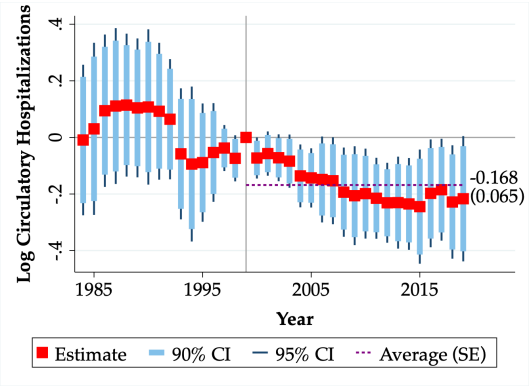
We set 1999 as the reference year because it is 10 years after all treatment municipalities became *T. infestans*-free, and it takes at least 10 years for chronic Chagas Disease to manifest (see Section 2). Our hypothesis is that differences within states between circulatory and non-circulatory outcomes should have begun to emerge at or around that point, yielding larger decreases in the former. To account for heterogenous and potentially increasing treatment effects, we estimate equation (5) using the two-way fixed effects estimator proposed by Borusyak, Jaravel and Spiess (2022). We also measure this decrease in a static context using a version of equation (5) analogous to equation (7).

7.2. Circulatory Disease Hospitalizations and Hospital Stay Length

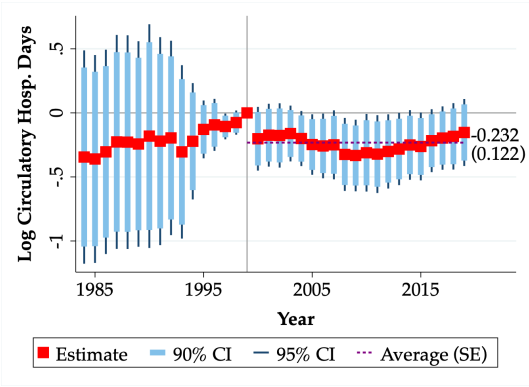
Consistent with our hypothesis, Figure 13a shows that the double-difference estimates for log hospitalizations evolved in parallel across disease categories prior to 1999 and then subsequently diverged.[18] In the late 2000s and beyond, the dynamic coefficients implied additional decreases in hospitalizations of at least 8 percent, each of which was significant at the 5-percent level. The economic significance of the implied static coefficient (16.8 percent) as well as its precision suggests that controlling Chagas Disease transmission had a substantial impact on Brazilian

[18] In results not shown, we verify that the two categories' double-difference estimates for this and the other two outcomes did not have parallel pre-treatment trends prior to 1999. The implication is that there were indeed state-specific, time-varying confounders necessitating the triple-differences strategy.

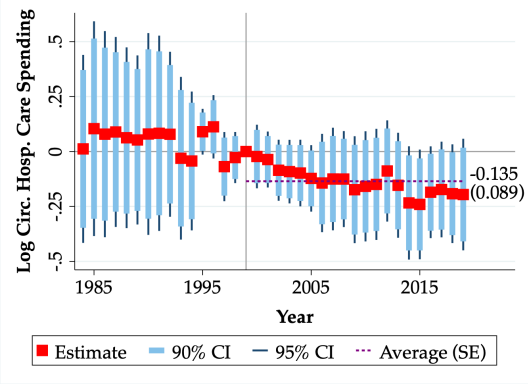
Figure 13: Long-Run Effects on Circulatory Disease Hospital Care



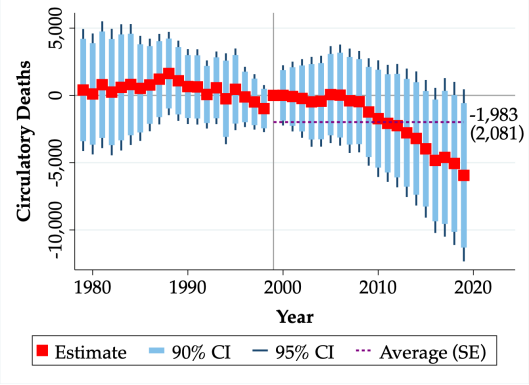
(a) Log Circulatory Disease Hospitalizations



(b) Log Circulatory Disease Hospital Days



(c) Log Circulatory Disease Spending



(d) Circulatory Disease Deaths

Notes: Graphs plot dynamic (red squares) and static (dotted purple line) triple-difference estimates for the respective outcomes with 90-percent (light blue) and 95-percent (dark blue) confidence sets for dynamic estimates. Static estimate magnitudes are next to the dotted purple lines with 90-percent wild cluster bootstrap confidence sets below them in parentheses. Data are from DATASUS. Regressions control for state-year, year-disease category, and state-disease category fixed effects. Standard errors are clustered by the 24 consistent 1960-2010 states. Regressions use 1,512 state-year-disease category observations. For pre-treatment years, mean log hospitalizations was 9.94 for circulatory diseases and 12.4 for non-circulatory diseases, mean log person-days in the hospital was 11.9 for circulatory diseases and 14.2 for non-circulatory diseases, mean log spending was 12.2 for circulatory diseases and 18.1 for non-circulatory diseases, and mean deaths by cause was 6,921 for circulatory diseases and 19,707 for non-circulatory diseases.

health care.

The patterns in log person-days spent in the hospital are highly similar: after pre-1999 trends evolved for the most part in parallel in Figure 13b, time spent in the hospital due to circulatory diseases decreased more. By the late 2000s, these differences stabilized at an implied magnitude of around 20 percent and achieved 5-percent significance, as did the static coefficient (implied estimate of 23.2 percent). While the latter may be overstated given the positive but noisy coeffi-

cients at the start of the sample, these results nonetheless suggest that Brazilians relying on the SUS for health care spent less time in hospitals due to circulatory problems right when we expect this decline to have occurred.

7.3. Circulatory Disease Hospital Spending and Deaths

Because these declines should have translated into a drop in SUS outlays on hospital care, we expect to find a similar pattern in the spending results. Figure 13d is consistent with this prediction but there is more noise in these estimates. Following pre-1999 coefficients of small magnitudes, the dynamic estimates become consistently more negative and achieve 5-percent significance in the 2010s, although those for last few years of the sample are less precise. But the implied static estimate of an additional 14.0-percent decrease in hospital spending (just outside of 10-percent significance) suggests that SUS has benefited—and continues to benefit—from substantial savings on circulatory disease hospital care spending resulting from triatomine control that occurred more than 3 decades ago.

Given that chronic Chagas disease—that manifests at least 10 years after exposure—can translate into mortality via cardiac problems approximately 10 years following the start of chronic Chagas Disease (see Section 2), we explore whether we observe a drop in circulatory deaths in the long-run for treatment states following vector eradication. Figure 13d is consistent with this prediction: estimates between 2000 and 2009 remain similarly stable and of small magnitudes as the pre-1999 coefficients; however, post-2009, dynamic estimates become consistently more negative and become statistically significant towards the end of our sample period starting approximately in 2017. These results suggests that the triatomine control program has begun to lead to substantial improvements in mortality more than 3 decades after the end of the program.

8. Cost-Benefit Analysis

Given the substantial public health benefits of the Chagas disease control project documented in Section 7, we provide a simple cost-benefit analysis to understand the economic viability of such public health interventions aimed at controlling neglected tropical diseases. We conduct the

analysis from the government's perspective, focusing on the costs associated with the spraying campaign and the benefits derived from averted hospital care.

On the cost side, the cost of the spraying campaign reported in Dias (1986) for spending on Chagas Disease control in 1985 translates to R$ 10 million in 2019 values. We consider this cost as an annual expenditure for the years 1984-89.

On the benefit side, we focuses on the savings from averted hospital care from Section 7. The Sistema Único de Saúde (SUS), Brazil's public health care system, spends approximately 0.1% of GDP on cardiac hospital care per year. Given that Chagas disease is a significant cause of cardiac complications, the reduction in Chagas disease prevalence due to the spraying campaign was estimated to lead to a 14% reduction in cardiac hospital care. This reduction would save 0.014% of GDP per year. Given that Brazil's GDP in 2019 was approximately R$ 1.9 trillion, this translates to annual savings of R$ 260 million in 2019 values.

The cost-benefit analysis thus reveals a highly favorable internal rate of return (IRR) of 24.9% just from averted hospital care. This calculation considers the costs of R$ 10 million per year for 1984-89 and the benefits of R$ 260 million per year for 2000-19. Importantly, the analysis abstracts from other potential government benefits, such as increased formalization and income tax revenues, likely understating the potential economic benefits of reducing Chagas disease.

For comparison, a study by Hamory et al. (2021) on the long-run impacts of deworming interventions in Kenya calculated a 37% IRR for 20-year earnings and consumption gains over the direct costs of deworming and indirect costs of additional teachers in Kenya, suggesting that the IRR for the insecticide spraying campaign in Brazil is in a comparable range. Thus, the cost-benefit analysis provides strong evidence for the economic viability of the large-scale Chagas disease control campaign.

9. Conclusion

Our understanding of the role of disease in explaining differences in economic development between and within countries has mostly been limited to its effects on childhood human capital (usually measured as schooling), which subsequently affects adult incomes for those treated

as children. While such impacts are very important for development in the long run, it takes decades to realize their full returns and they are by no means the only long-run economic gains from disease control programs in developing countries. As a result of discounting these benefits and considering those only in this domain, cost-benefit analyses of these campaigns may fail to justify them to policymakers and development practitioners.

However, this paper has shown there were important short-run benefits to Brazil's campaign to control the main vector of Chagas Disease, which has both acute and chronic phases, and important long-run benefits beyond individuals' labor market returns. We found that shortly after spraying began, employment rates increased for *older adults already in the labor force*, which likely resulted in quickly-improved living standards for them and their families. In the long run, vector control raised adult incomes for non-white Brazilians treated as children, potentially helping to increase the speed of racial convergence in a country with wide disparities in this dimension. We also found small and imprecisely estimated effects on school attendance and comparable eventual educational attainment impacts for both white and non-white adults, suggesting that reducing the cardiovascular morbidity arising from Chagas Disease's chronic phase may be more important in explaining the income result.

Because circulatory diseases result in a substantial share of hospitalizations (10 percent) and spending (20 percent) covered by Brazil's publicly-run health care system, which consumes around 4 percent of GDP, this paper also showed that these outcomes decreased substantially more for circulatory causes than non-circulatory ones in states more exposed to vector control beginning around the time we expected such a difference to arise. A simple cost-benefit analysis that considers just averted cardiovascular hospital care finds a 24.9% internal rate of return to the Chagas disease control campaign. We interpret these results as evidence for Chagas Disease control having a significant impact on Brazil's public finances by improving adult health in the long run, which is another important impact not previously examined in the literature.

Taken together, these results present a more complete picture of the economic consequences of Chagas Disease control for developing countries. Whether they generalize beyond this unique malady—which almost exclusively afflicts the Americas, can affect both children and adults in its acute stage, and can cause long-run cardiovascular problems—is an open question we leave to

future research. Nonetheless, we believe that this paper has identified novel areas through which health can impact economic development, helping to strengthen justifications for controlling transmission of this neglected tropical disease affecting an estimated 6 million people throughout the Western Hemisphere.

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Online Appendix for:

Disease, Disparities, and Development: Evidence from Chagas Disease in Brazil

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February 15, 2024

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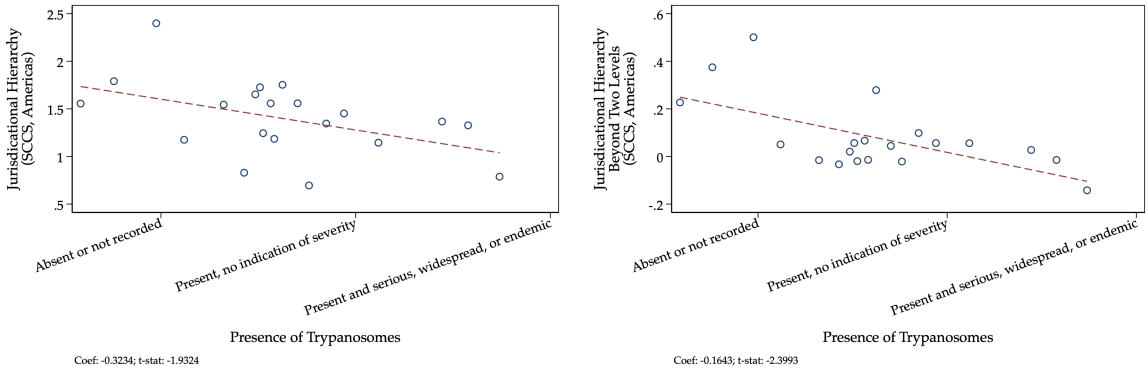
Appendix A. Additional Figures

Figure A1: Example of Triatoma Infestans ("Kissing Bug")



Notes: Figure presents a picture of Triatoma Infestans, the main vector in Brazil prior to the 1984-1989 control campaign. Triatoma bugs are also known by the following names: Kissing bugs (in English), *vinchucas* (in Argentina, Bolivia, Chile, Ecuador, and Uruguay), *chinches* (in Central America), *barbeiros* (in Brazil), *chipos* (in Venezuela), and *pitos* (in Colombia), among other names.

Figure A2: Pre-Colonial Presence of Trypanosomes and Jurisdictional Hierarchy



a. Jurisdictional Hierarchy b. Centralization

Notes: The figure presents binscatters between the whether a pre-colonial society had trypanosomes present and the levels of jurisdictional hierarchy beyond the local community (a.) and an indicator variable equal to one if the levels of jurisdictional hierarchy is above two, and zero otherwise (b.). The unit of observation is a society in the Standard Cross-Cultural Survey (SCCS) (Murdock and White, 1969) in the Americas. All plots include controls for latitude, longitude, average rainfall, average temperature, elevation, agricultural suitability, and malaria ecology. The bottom-left of each figure presents the estimated bivariate coefficient and t-statistic using robust standard errors.

Appendix B. Short-Run Benefits: PNAD Survey Data Results

We examine whether reducing exposure to acute Chagas Disease affected a child's ability to attend school (ages 8 to 18) and an adult's ability to be employed (ages 35 to 50), as the 1 to 3 months of symptoms could interfere with both of these outcomes using household survey data from Brazil (PNAD). Our results show that while spraying had positive but imprecise effects on school attendance, it noticeably increased adult employment within a few years, implying that policymakers did not need to wait decades for there to be economically meaningful returns to reducing Chagas Disease transmission. When examining results by racial group, we estimate that attendance and employment increased more (and more precisely) for non-white Brazilians, which would have helped to speed convergence in the country's racial disparities.

B.1. Empirical Strategy

Our evidence comes from comparing individuals of interest in each year of the PNAD (1982-2015) across states with varying levels of pre-treatment *T. infestans* presence, motivated by the idea that vector control induced greater improvements in health where there was more exposure to Chagas Disease prior to spraying. While our long-run analysis compares individuals across municipalities, municipality of birth is not reported in the PNAD surveys, so we instead comparing individuals across states. Our estimating equation is the dynamic difference-in-differences model:

$$y_{i,s,t} = \alpha_s + \gamma_t + \sum_{k \neq 1986} \tau_k \cdot \mathbf{1}[\mathbb{P}(Treat)_s > 0] \cdot \mathbf{1}[t = k] + \mathbf{X}_i\beta + \epsilon_{i,s,t} \tag{6}$$

where $y_{i,s,t}$ is an outcome of interest for individual i living in state s in year t, α_s and γ_t are state and year fixed effects, $\mathbb{P}[Treat]_s$ is the probability in the 1980 census that an individual in a state of that sex and race resides in a treatment municipality, $\mathbb{1}[t = k]$ indicates whether an observation is from the given year k, $\mathbf{X}_i$ is a vector of individual-level covariates (age, age squared, and fixed effects for female sex and Asian, Black, and Brown racial categories), and $\epsilon_{i,s,t}$ is the idiosyncratic error term. Because we cluster standard errors by the "small" number of states in our sample (24), we follow Cameron, Gelbach and Miller (2008) and use the wild cluster bootstrap to generate confidence sets. We estimate equation (6) using the two-way fixed effects estimator proposed by Borusyak, Jaravel and Spiess (2022).

The coefficients of interest are the τ_k, which measure the difference in an outcome in a given year as the probability of residing in a treatment municipality goes from 0 to 1, relative to the size of that difference in outcomes in 1986. Because nearly one-third of observations were from states with no treatment municipalities and the average probability in residing in a treatment municipality is approximately 0.25, the magnitude of these estimates overstates any policy-relevant parameter. Therefore, when discussing the results, we frame them in the context of moving the population with vector exposure

in a state from 25% to 0% —i.e., this shift would have an impact of just under one-quarter of the estimated magnitudes.

Note that equation (6) makes 1986 the reference year despite that the final entirely pre-treatment year was actually 1983. We do this for two reasons. First, there are no PNAD data available for 1983-1985. Thus, we chose to make 1986 the reference year because it was only 2 years after spraying began and spraying paused in that year, making it only "lightly" treated. Importantly, this choice biases estimates toward zero if the sign of the 1982 coefficient is the opposite of the treatment effect's, assuming that the size of the difference in outcomes in hypothetical 1983 data would have been between those in 1982 and 1986. Second, estimating an effect for 1982 also permits a qualified assessment of pre-treatment trends, as a hypothetical 1983 coefficient should not have been "too different" from the 1982 estimate. Economically and statistically insignificant magnitudes for the pre-treatment year suggest that outcomes evolved in parallel prior to vector control, and significant estimates in years after spraying began indicate that outcomes diverged as a result of the decline in acute Chagas Disease.

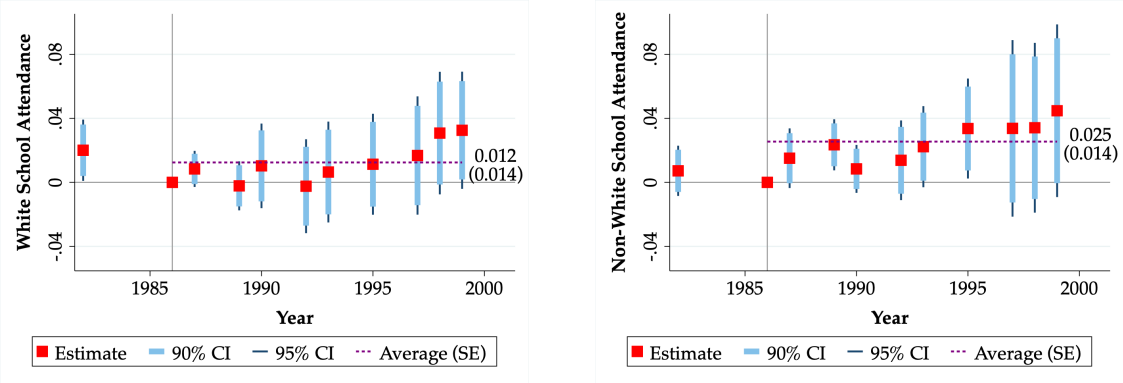
Along with equation (6), we also estimate the static two-way fixed effects model

$$y_{i,s,t} = \alpha_s + \gamma_t + \tau \cdot \left(\mathbf{1}[\mathbb{P}(Treat)_s > 0] \cdot \mathbb{1}[t > 1986]\right) + \mathbf{X}_i\beta + \epsilon_{i,s,t}, \tag{7}$$

which is identical to the previous equation except that the probability of residing in a treatment municipality is interacted with a single variable $\mathbb{1}[t > 1986]$, an indicator for whether a year is after 1986.

As with the dynamic estimates, the static estimate will be biased toward zero if the 1982 coefficient and the treatment effect have opposite signs. However, this bias will be less severe than in the dynamic estimates in this case because the static coefficient is estimated against the average of the difference in outcomes in 1982 and 1986. The result is that the static coefficient will lie closer to the largest dynamic estimates (in absolute magnitude) instead of falling in the middle of the range.

Figure B3: Short-Run Effects on School Attendance
Differential Short-Run Effects by Race



(a) White: Attending School, Ages 8-18 **(b)** Non-White: Attending School, Ages 8-18

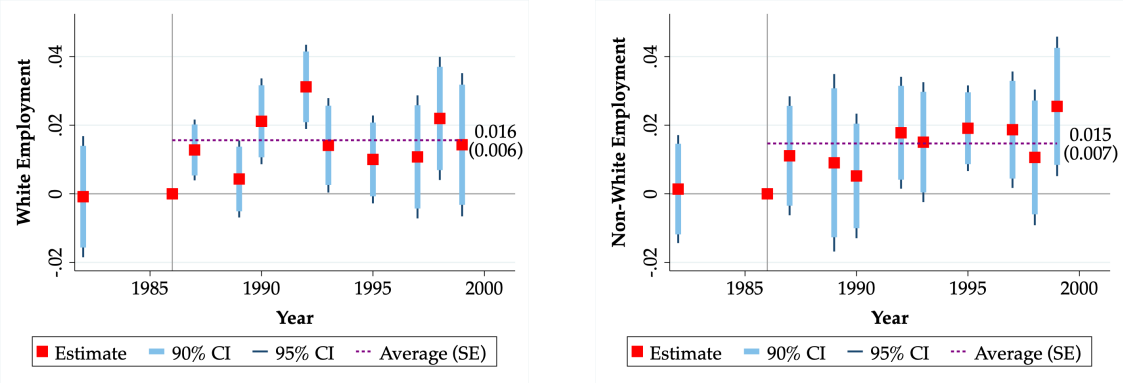
Notes: Graphs plot dynamic (red squares) and static (dotted purple line) two-way fixed effects estimates for the respective outcomes and racial groups with 90-percent (light blue) and 95-percent (dark blue) wild cluster bootstrap confidence sets for dynamic estimates. Static estimate magnitudes are next to the dotted purple lines with 90-percent wild cluster bootstrap confidence sets below them in brackets. Data are from the 1982-1999 PNAD surveys. Regressions control for fixed effects for state, year, female sex, and racial category (Asian, Black, and Brown) as well as age and age squared. Standard errors are clustered by the 24 consistent 1960-2010 states. Regressions use 389,414 observations for white and 446,017 observations for non-white children. In the pre-treatment year, 77.7 percent of white and 74.1 percent of non-white children attended school.

To examine the potential for health improvements to reduce racial inequality in the short run, we estimate equations (6) and (7) separately for white and non-white individuals.

B.2. Reducing Racial Disparities in the Short Run: School Attendance

We first examine the effects of Chagas Disease on school attendance in the years around the start of vector control and examine evidence for differential effects by race, which is closely linked with poverty in Brazil. Figure B3 plots the estimates from equations (6) and (7) for school attendance for white (B3a) and non-white (B3b) children ages 8 to 18. Figure B3 suggests that both white and non-white children were more likely to attend school after spraying began, but that the increase for non-white children was larger and less noisy. The patterns in the dynamic estimates suggest a positive but imprecise impact of reducing acute Chagas Disease exposure on attendance for white children, but a larger

Figure B4: Short-Run Effects on Adult Employment
Differential Short-Run Effects by Race



(a) White: Employed, Ages 35-50 **(b)** Non-White: Employed, Ages 35-50

Notes: Graphs plot dynamic (red squares) and static (dotted purple line) two-way fixed effects estimates for the respective outcomes and racial groups with 90-percent (light blue) and 95-percent (dark blue) wild cluster bootstrap confidence sets for dynamic estimates. Static estimate magnitudes are next to the dotted purple lines with 90-percent wild cluster bootstrap confidence sets below them in brackets. Data are from the 1982-1999 PNAD surveys. Regressions control for fixed effects for state, year, female sex, and racial category (Asian, Black, and Brown) as well as age and age squared. Standard errors are clustered by the 24 consistent 1960-2010 states. Regressions use 648,336 observations for white and 550,144 observations for non-white adults. In the pre-treatment year, 67.2 percent of white and 66.9 percent of non-white adults were employed.

and more sustained positive impact for non-white children.

The static strategy recovers a coefficient of similar magnitudes to the later years' dynamic estimates for non-white Brazilians but with greater precision. The implied effect of moving from a 0 to one-quarter probability of living in a treatment municipality is 2.5 p.p. (3.5 percent of the pre-treatment mean). For white children, the static estimate is imprecise and suggests that the effect of moving from a 0 to one-quarter probability of living in a treatment municipality is 1.2 p.p. (1.6 percent of the pre-treatment mean). Thus, both the dynamic and static estimates suggest that the reduction in acute Chagas disease increase school attendance for children, especially for non-white children.

B.3. Reducing Racial Disparities in the Short Run: Adult Employment

Next, we study the short-run effects on employment (having worked in the past week) for adults ages 35 to 50 and plot the dynamic and static estimates in Figure B4 for

white (B4a) and non-white Brazilians (B4b). For both groups, there is little evidence of differential trends prior to the start of spraying, but in contrast to the attendance results, the divergence in employment trends occurs more rapidly and is more precisely estimated. By 1990, the implied dynamic estimates remain around 1.5 p.p. (2.1 percent) for both group, which are very similar in magnitude to the implied static estimates. This result is particularly important because it shows that controlling Chagas Disease has relatively rapid effects on *adults already in the labor force*. While increasing the human capital of children makes large contributions to economic development, doing so yields benefits that are only realized after they enter the labor force a decade later and over decades of their working lives. Instead, triatomine control appears to have induced some unemployed adults to (re-)enter employment, which could have quickly improved standards of living for them and their families.

Figure B4 also show that employment increased for both groups but somewhat more for non-white adults: the implied white static estimate was 1.5 p.p. (2.1 percent) while the implied non-white one was 1.6 p.p. (2.2 percent). Importantly, the identical differences in outcomes for both racial groups in 1982 and 1986 are consistent with the post-treatment coefficients being accurately estimated. These results thus suggest that Chagas Disease control may have had relatively immediate impacts on the speed of racial convergence in living standards by increasing employment more among non-white Brazilian adults. It also could have set the stage for greater convergence in the future if non-white children's human capital increased more as well.