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 the likelihood that non-white adults reported non-zero incomes in the 2010 census by 1 to 1.5 percentage points (p.p., or 1 to 2 percent) as well as literacy among their daughters by 0.5 p.p. (0.5 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 (0.017 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.

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JEL Classification: H51, I14, I15, J62, O15

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Like hunger, it kills in silence.
Like hunger, it kills the silent. . . .
Chagas disease holds no allure for the pharmaceutical industry, for politicians or the press. It selects its victims from among the poor. It bites and, slowly, relentlessly, they waste away.
—Eduardo Galeano (Médicos Sin Fronteras, 2013)

1. Introduction

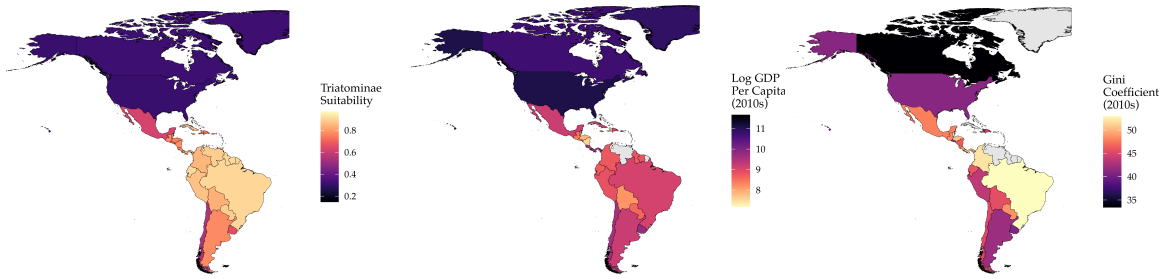
Latin America is one of the most unequal regions in the world (Chancel et al., 2021), with the top 10% capturing 54% of national incomes (De Rosa, Flores and Morgan, 2020). Alongside drastic income inequality, the region faces stark racial inequalities (Telles et al., 2023): white individuals earn at least twice as much as those with darker skin tones (Woo-Mora, 2022). These high and intertwined inequalities in the region have renewed calls to understand their underlying causes and explore policies to reduce them (World Economic Forum, 2016; Eslava and Valencia Caicedo, 2023).

An understudied but potentially important driver of income inequality in the region is Chagas disease (Briceño-León and Méndez Galván, 2007; Franco-Paredes et al., 2007). Chagas disease is often described as a "neglected disease of poor, rural, and forgotten populations" (Coura and Viñas, 2010; Houweling et al., 2016). It is a parasitic disease that is only found in the Americas. Poorer individuals are much more likely to contract the disease because the vector – triatomine bugs – lives in cracks in poor-quality housing.[1] Chagas disease 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).[2] Despite more than 6 million individuals being infected by Chagas disease and 75 million exposed to the disease in the region, little is known about how Chagas disease – "Latin America's Silent Killer" (Médicos Sin Fronteras, 2013) – exacerbates income and racial disparities, and whether its elimination might reduce inequalities and increase development.

[1] 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).

[2] The World Health Organization (2010) notes that "Chagas disease can cause young adults to develop heart conditions, so that they fill hospital beds instead of the [labor] force."

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.

Studying the economic consequences of Chagas Disease is challenging for several reasons. First, the long timeline of the disease – particularly its chronic phase, which can manifest decades after initial infection – requires a long-term perspective to capture the full impacts of the disease. Second, accurate measurement of Chagas exposure is often lacking, as it requires detailed assessment of vector presence or serological testing. Finally, the vector that transmits the disease, triatomine bugs, lives in regions with certain environmental characteristics, which may independently affect economic outcomes. The maps of the Americas in Figure 1 highlight that areas with greater vector suitabiltiy also tend to have lower incomes and higher inequality. However, omitted variable bias and reverse causality – where poverty exacerbates exposure to the disease through inadequate housing – further complicate efforts to rigorously assess Chagas disease's impact on development and inequality.

In this paper, we address these challenges and 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, 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. We exploit the timing of the campaign combined with long-run outcome data to examine the impacts of controlling Chagas disease on development and disparities.

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, its chronic phase can substantially 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. Furthermore, since poorer individuals are more likely to contract this (and other) NTD(s) and given the close link between poverty and race in Brazil, the disease may also exacerbate the country's wide racial disparities.

Features of the control campaign that we detail in Section 2.3 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. As we outline in Section 3, 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 4 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 (9-percent increase in incomes versus 3 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 4.3 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 4.6 but do not find evidence that it can explain a large share of the effect. Instead, the health benefits of reducing acute and chronic Chagas Disease may play an important role in this reduction of racial

disparities.

We complement the individual-level results by also examining how reducing exposure to Chagas Disease affected municipalities in both the short- and long-run in Section 5. 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 incomes and structural transformation. We find that treated municipalities experienced decreases in income inequality and the share of the labor force employed in agriculture, and increases in income and in the share of the labor force employed in manufacturing or service. These benefits tend to grow more pronounced over time, especially in census waves 10 years after the end of the eradication campaign when chronic Chagas first begins to manifest.

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 6, 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 7.1 finds that the internal rate of return for the disease control campaign was 24.9%. Although 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.

We then examine the broader implications of Chagas disease on inequality and development across Latin America in Section 7. First, we show 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. Second, to assess the potential economic gains of controlling Chagas Disease across Latin America, we extrapolate our Brazil-based findings using WHO data on exposure to Chagas disease. Our back-of-the-envelope estimates suggest that controlling Chagas Disease could increase GDP per capita by about 0.9%, with larger gains in countries where the disease remains widespread. Additionally, we estimate a modest reduction in income inequality, underscoring the broader benefits of disease control for economic development.

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 contribute to several strands of literature. First, by examining a disease that has primarily 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 Valencia Caicedo, 2023; Telles et al., 2023; Attanasio et al., 2024). 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 highlight that Chagas disease suitability is negatively correlated with development in the cross-section today in the Americas; this relationship does not

hold outside of the Americas (Table 2). Furthermore, we provide quasi-experimental evidence on how eliminating Chagas disease can have important long-run development benefits. Our results imply that policies that further reduce Chagas disease could help improve development and reduce inequality in the region.

Second, 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; Lucas, 2010; Cutler et al., 2010; Alsan, 2015; Mora-Garcia, 2018; Depetris-Chauvin and Weil, 2018; Dillon, Friedman and Serneels, 2021; Hamory et al., 2021) and to a larger literature on health and inequality (e.g., Farmer, 2001; Deaton, 2003; O'Donnell, Van Doorslaer and Van Ourti, 2015; Bütikofer and Salvanes, 2020). NTDs such as Chagas disease 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. The growing incidence of Chagas Disease in non-endemic regions, including the United States (Hernández, 2021; Irish et al., 2022), underscores the global importance of understanding and addressing this health challenge.

2. Chagas Disease and Its Control in Brazil

2.1. Vectors and Disease Progression

The parasite *Trypanosoma cruzi* causes Chagas disease, also known as American trypanosomiasis. Around 90 percent of those infected contracted it from blood-sucking triatomine bugs carrying the parasite.[3] These bugs live in cracks in roofs and walls and infect humans when they emerge at night to take blood meals. In Brazil, the most important vector species is *Triatoma infestans*, thought to have been responsible for 80 percent of infections (Schofield and Dias, 1999).[4] Appendix Figure A1 presents an image of this bug, which became domesticated and spread through

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

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

rural settlements in southern and southeastern Brazil in the late nineteenth century after forests were cleared for agriculture and ranching (Schofield, 1988). Shortly thereafter in a rural area of Minas Gerais state, the Brazilian physician Carlos Chagas identified the parasite and the disease it caused (Chagas, 1909).

Appendix Figure A2 shows the progression of Chagas disease from a patient's exposure to *T. cruzi* through the rest of their life. The acute stage begins after 1 to 2 weeks of incubation, lasts 1 to 3 months, and is characterized by nonspecific symptoms such as malaise and fever (Rassi et al., 2009). Children generally become more seriously ill than adults in this phase, though more than 40 percent of those infected are asymptomatic (Khan, 2011). The individual then enters the chronic stage of the disease. Around half of *T. cruzi* carriers have no subsequent symptoms, but for the others, after 10 to 30 years, the usual outcome is that the heart muscle becomes enlarged and fibrous (Rassi, Rassi and Little, 2000).[5] This cardiomyopathy, which makes it progressively more difficult for the heart to pump blood, causes most of the morbidity and mortality from Chagas disease (Nunes et al., 2018). Importantly, individuals can be reinfected with the parasite and experience acute stage symptoms multiple times, with more reinfections linked to increased likelihoods of experiencing symptomatic chronic phases of the disease (Olivo Freites et al., 2022).

2.2. *Association with Poverty and Race*

In announcing the discovery of *T. cruzi*, Chagas (1909) also noted how frequently its vector inhabited fissures in the unplastered walls of patients' homes. He was thus the first of many scholars to link the malady named after him to rural poverty, especially poor-quality housing (e.g., Coura and Viñas, 2010; Hotez et al., 2013; Houweling et al., 2016). In this sense, exposure to *T. cruzi* is a consequence of poverty, but due to the acute and chronic symptoms described above, Chagas disease can be one of its causes as well:

> Many people forgotten by [society] are ... the same people who encounter infection with *T. cruzi*. The vast majority with Chagas [disease] has the lowest incomes, poor sanitation and nutrition, the worst opportunities for education, low quality housing, and endure an inter-generational persistence of social inequalities. Poverty not only

[5] Dilation of the esophagus or colon (megasyndromes) can also occur, but cardiovascular involvement is more common. See Zingales et al. (2012) for a discussion of the respective chronic phase symptoms caused by the six lineages of *T. cruzi*.

> restricts patients' access to diagnosis and treatment of the disease, but it ... limit[s] an individual's ability to recover and return to work. In a vicious cycle, poor living conditions lead to increased incidence of Chagas disease[,] which cripples the population[,] leaving it unable to work, [earn income], and reach its full potential. Poverty and underdevelopment, therefore, persist. (Franco-Paredes et al., 2007, p. 4)

Given the close link in Latin America between poverty and darker skin tones (Telles et al., 2023), it is therefore not surprising that mixed-race ("Brown") Brazilians—who predominate in the rural interior of the country—comprised over 60 percent of hospitalizations for acute Chagas disease symptoms while only being around 40 percent of the population (Santos et al., 2020).[6]

2.3. Eradication of Vectorial Transmission in Brazil

In describing Brazil's campaign against Chagas disease, we largely summarize the account in Schofield and Dias (1999), as one of the authors was the director of the Chagas Disease Division of the Ministry of Health in this period. It began in the wake of the post-World War II campaigns against malaria, which used organochlorine insecticides like DDT that were found to be ineffective against vectors of Chagas disease. However, several trials showed that benzene hexachloride (BHC) was effective if sprayed on the walls and roofs of triatomine-infested houses in high doses. In the 1960s, the vector control superintendency of Brazil's richest state (Sao Paulo) began a program using BHC to effectively eliminate *T. infestans* from its territory, but no others had the resources to implement such an intensive program. Nonetheless, new pyrethroid insecticides became available in the 1970s, and studies by the end of the decade showed their efficacy against triatomine bugs. More important was the fact that they worked when sprayed less frequently and in lower doses than BHC, making them more cost-effective, with the additional virtues of being easy to apply and lacking unpleasant odors.

Due to this "development of suitable vector control methods ... and continuous campaigning by scientists[,] including demonstrations that vector control was feasible," the Brazilian government launched a national campaign against Chagas disease in 1975 (Dias, 1987, p. 338). The first stage consisted of serological and entomological surveys lasting until the early 1980s. They

[6] In contrast, 20 percent of those hospitalized were white (around 45 percent of the population) and 7 percent were Black (around 8 percent of the population). For the population's racial composition since 1991, see: https://g1.globo.com/economia/censo/noticia/2023/12/22/censo-2022-cor-ou-raca.ghtml.

found a national rural *T. cruzi* prevalence rate above 4 percent—including nearly 9 percent rural prevalence in the heavily-infested states of Minas Gerais and Rio Grande do Sul—and vectors present in just over one-third of Brazil's territory (Dias, 1987).[7] Notably, in some municipalities, estimated prevalence was above 30 percent.

After the surveys concluded, thousands of public health personnel visited millions of homes across the endemic region to conduct indoor residual spraying (IRS).[8] However, the program was paused in 1986 due to outbreaks of dengue fever in coastal (i.e., tourist) areas, leading public health officials to divert sprayers to the control of this mosquito-borne disease. Triatomine bugs thus reestablished themselves in some recently-sprayed municipalities (Dias, 1987; Schofield and Dias, 1999), but there was nonetheless a clear decline in the presence of *T. infestans* before Chagas disease vector control restarted in 1989 (see Appendix Figure A4 from Coura and Dias, 2009). The program continued into the 21st century, and by 2006, the Pan American Health Organization certified that Brazil had interrupted transmission of Chagas disease by *T. infestans*.[9]

3. Difference-in-Differences Framework

At the core of our study of the effects of eliminating Chagas disease transmission is a difference-in-differences strategy comparing outcomes across municipalities (of birth) and years (of birth). We now give a conceptual overview of this approach, but because our individual- and place-level specifications differ in important ways, we provide more detail on them in subsequent sections.

3.1. Treatment, Control, and Excluded Municipalities

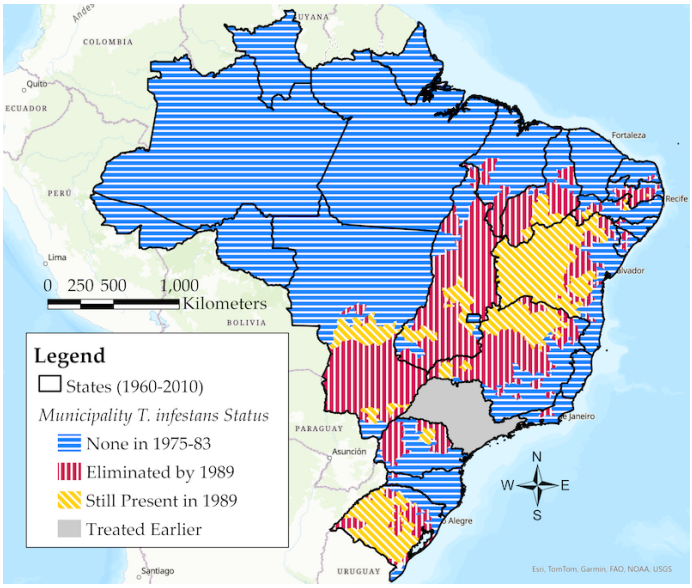
To determine which municipalities belong in our treatment and control groups, we combine the maps in Appendix Figure A4 showing the presence of *T. infestans* in the entomological surveys before (1975-83) and after IRS began but had been interrupted (1989). Figure 2 shows the resulting four categories of municipalities. Our control group consists of those in which the vector was not

[7] Appendix Figure A3 presents a map of rural *T. cruzi* seroprevalence across municipalities from Costa Passos and Silveira (2011). Appendix Figure A4 shows a map of municipalities that had *T. infestans* in dwellings from 1975 to 1983 (Silveira, 2011).

[8] See Appendix Figure A5 for images from Dias (1987) of insecticide being sprayed on rural homes with many cracks and spaces in their roofs and walls for *T. infestans* to colonize.

[9] https://www3.paho.org/english/ad/dpc/cd/dch-incosur-xv.htm

Figure 2: Brazilian Municipalities by *T. infestans* Status, 1975-89



Notes: Map shows municipalities with no *T. infestans* in 1975-83 (control group) with blue horizontal lines and those in which the vector was eliminated by 1989 (treatment group) with red vertical lines. Municipalities with *T. infestans* still present in 1989 (diagonal yellow lines) or that were treated prior to this period (solid gray) are excluded from our analysis. Data are from Silveira (2011) and Coura and Dias (2009) (see Appendix Figures A4a and A4b).

found prior to spraying, so we assume that they were never exposed to transmission of Chagas disease via *T. infestans*.[10] We compare them to treated municipalities in which the vector was present before IRS began but had been eliminated by 1989. We thus exclude from our sample those where the vector was observed again in that year (i.e., treated later) and the state of Sao Paulo (treated earlier).[11] By doing so, we examine the effects of eliminating vectorial transmission of Chagas disease over the longest time horizon and limit our comparisons to a single treatment time (Goodman-Bacon, 2021).

Table 1 presents summary statistics for these groups of municipalities using data from the IPUMS 25-percent sample of the 1980 census (Ruggles et al., 2024). Notably, the share of white Brazilians was lower in the control municipalities than in treatment ones (Panel A), likely because the former includes more rural territory in Brazil's interior. Nonetheless, in all cases, Panel B shows that the non-white population was around 3 times more likely to be living in housing

[10] This assumption is strong, but if it means that our control group contains municipalities where vectorial transmission was occurring or had recently occurred, our results would understate the true effects.

[11] Note that the first of these groups includes municipalities were the vector may have been temporarily eliminated but the interruption in spraying allowed it to return.

Table 1: Summary Statistics, 1980

	Municipalities by 1975-89 *T. infestans* Status			
	None (Control)	Eliminated (Treatment)	Still Present (Omitted)	Treated Earlier (Omitted)
Panel A. Demographics				
Age	24.01	24.34	24.23	25.95
	(19.12)	(19.16)	(19.45)	(18.93)
Female	0.507	0.502	0.502	0.501
Asian	0.002	0.003	0.003	0.019
Black	0.065	0.056	0.060	0.045
Brown	0.473	0.352	0.393	0.182
White	0.460	0.589	0.544	0.754
Panel B. Living in Housing Vulnerable to Infestation				
Non-White	0.341	0.353	0.463	0.056
White	0.112	0.116	0.173	0.026
Panel C. Income and Human Capital				
Monthly Income (in 2019 BRL)	241	228	193	427
	(882)	(889)	(941)	(1,327)
Years of Schooling: Adults 19+	3.70	3.43	2.98	4.68
	(3.86)	(3.62)	(3.43)	(3.90)
Literate: Children 10-18	0.768	0.802	0.752	0.955
Panel D. Within-Municipality Income Inequality				
Gini Coefficient	0.763	0.771	0.774	0.734
	(0.045)	(0.047)	(0.039)	(0.043)
Interquartile Range (as Share of Mean)	0.723	0.833	0.877	0.749
	(0.327)	(0.343)	(0.298)	(0.226)
p90-p10 Gap (as Share of Mean)	1.461	1.732	1.836	1.550
	(0.777)	(0.787)	(0.791)	(0.431)
p90-p50 Gap (as Share of Mean)	1.361	1.584	1.753	1.285
	(0.688)	(0.640)	(0.705)	(0.343)
Observations	16,431,739	4,554,538	2,057,166	6,181,009
Municipalities	1,156	402	173	309

Notes: Means for variables of interest are displayed, with standard deviations in parentheses for continuous measures. Variable definitions are given in the text and municipality groupings correspond to those in Figure 2. Data are from the IPUMS sample of the 1980 Brazilian census.

vulnerable to vector infestation—which we define as having a roof made of wood, straw, or scraps of material, walls made of uncoated lathe and plaster or straw, or a floor made of dirt—than the white population, consistent with racial disparities in exposure to Chagas disease transmission.[12] The means in Panel C imply a correlation between greater exposure to Chagas disease and worse economic development outcomes. It is also associated with greater within-municipality income

inequality (Panel D), particularly when measured as the gaps between various percentiles of their distributions as a fraction of their average incomes.

3.2. *Pre- and Post-Treatment Years (of Birth)*

We cannot observe when in the 1984-89 period vectorial transmission of Chagas disease was eliminated in a municipality, so we make the conservative assumption that it occurred uniformly throughout the treatment group in 1984.[13] When studying the impacts on municipalities and states, our final pre-treatment year is thus 1983 when using annual data and 1980 when comparing across decennial censuses. However, in our examination of the effects of additional childhood years free from exposure to Chagas disease (e.g., Bleakley, 2010; Cutler et al., 2010; Bütikofer and Salvanes, 2020), we define the end of childhood to be the last age at which school attendance rates were (just under) 50 percent; in the 1980 census, it was age 16. Therefore, our pre-treatment cohorts were born in 1967 or before (i.e., aged 17 or older when IRS began in 1984).

Underlying this cutoff is the assumption that pre-1968 birth cohorts were too old to have benefited from *T. infestans* control. But as noted in Section 2, both children and adults can experience symptoms of the acute stage of Chagas disease again when reinfected with *T. cruzi*, which also increases the chances of developing its chronic manifestations. Therefore, the interpretation of our individual-level results is closer to those for childhood exposure to tuberculosis (e.g., Bütikofer and Salvanes, 2020), which also affects all ages, than to diseases like malaria that primarily impact children (e.g., Bleakley, 2010; Lucas, 2010; Cutler et al., 2010).

For several reasons, we limit our attention to cohorts born between 1960 and 1979, who were aged 31 to 50 in 2010. First, in that year, workers could retire with social security benefits after having paid into the system for 30 to 35 years. In addition, DDT spraying began in the late 1950s, so we want to avoid comparing cohorts with different levels of exposure to that public health intervention. Lastly, the restriction at the other end of our age range ensures that our sample

[12] The fact that a much greater share of the population in municipalities where the vector was still present in 1989 were poorer and living in vulnerable housing suggests that it could have been more difficult to eliminate there. One implication is that the benefits of eliminating transmission estimated by comparing our treatment and control groups might understate the effects on the later-treated group. However, the consequences for the cost-benefit analysis are more ambiguous, as it was likely more expensive to eliminate *T. infestans* from these areas.

[13] As a result, our estimated effects for the first few post-treatment years are likely biased toward zero.

contains prime-age adults with varying exposure to Chagas disease transmission in childhood and who had already made substantial progress along their lifetime earnings trajectories.

4. Long-Run and Intergenerational Effects

In this section, we examine the impact of eliminating Chagas disease transmission on adults who were children around the time that *T. infestans* control began as well as its effects on their children. As predicted, our results show that it raised incomes substantially more for non-white than white Brazilians, and that measures of income inequality decreased more within treated cohorts born in treated municipalities. We also find that the children of non-white fathers experienced larger increases in their literacy rates than those with white fathers. The implication is that vector control decreased racial inequality in Brazil in the long run and across generations. Interestingly, when exploring mechanisms, we find that increases in adults' years of schooling are unlikely to explain most of the income results, suggesting that improved long-run health could be a more important channel.

4.1. Data

To study these impacts on post-treatment cohorts and their children, we use the IPUMS 10-percent sample of Brazil's 2010 census (Ruggles et al., 2024). It contains information on our outcomes of interest (monthly income, years of schooling, and literacy), demographic characteristics (age, racial category, and sex), and whether an individual was born in their municipality or state of residence. The last of these variables gives us the municipality of birth for just under three-fifths of the sample, but for the remainder, we can only observe their state of birth.

4.2. Assignment to Treatment and Control Groups

Therefore, we place individuals with known municipalities of birth into the treatment or control groups or exclude them from the sample, as discussed in the previous section. We then assign to those with unknown municipalities of birth the probability in the 1980 census that a 6- to 10-year-old child of their race, sex, and state of birth was living in a treatment municipality. Because we

know with certainty that just over 45 percent of the sample was born in a control municipality, we add to the treatment group anyone with a strictly positive value of the probability mentioned above, which is nearly identical to performing a median split.

This decision means that we compare anyone who could have been born in a treatment municipality against those we are certain were not. As such, we interpret our treatment effects as resulting from a reduction in the share of children potentially exposed to Chagas disease transmission from around 45 percent—the average of the probability values in the treatment group—to 0 percent.[14] We view this interpretation as both intuitive and policy relevant, as we discuss in Section 7. It also allows us to avoid making the much stronger assumptions required in a continuous-treatment difference-in-differences framework (Callaway, Goodman-Bacon and Sant'Anna, 2024).

4.3. Empirical Strategy

Our baseline estimating equation is thus the dynamic difference-in-differences specification

$$y_{i,m,c} = \alpha_m + \gamma_c + \sum_{k \neq 1967} \tau_k \cdot \mathbb{1}[\mathbb{P}_i(m \in \text{Treat}) > 0] \cdot \mathbb{1}[c = k] + \mathbf{X}_i\beta + \delta_{s(m)} \times \gamma_c + \epsilon_{i,m,c}, \quad (1)$$

where $y_{i,m,c}$ is an outcome of interest for individual i born in municipality m and of birth cohort c, α_m and γ_c are fixed effects for m and c, $\mathbb{1}[\mathbb{P}_i(m \in \text{Treat}) > 0]$ indicates whether the probability that i was born in a treatment municipality is strictly positive, $\mathbb{1}[c = k]$ indicates whether i was born in year k, $\mathbf{X}_i$ is a vector of individual-level covariates (fixed effects for female sex and Asian, Black, and Brown racial categories), $\delta_{s(m)}$ is a fixed effect for m's state interacted with γ_t to control for trends at this level, and $\epsilon_{i,m,c}$ is the idiosyncratic error term.[15]

The coefficients of interest are the τ_k, which measure the difference in an outcome for a given birth cohort as the share of children potentially exposed to Chagas disease transmission is reduced from about 45 to 0 percent, relative to the size of that difference for the 1967 cohort. Using OLS with standard errors clustered by municipality of birth, we estimate these coefficients sepa-

[14] Our use of the word "exposure" is in the intent-to-treat sense, as we cannot observe the quality of an individual's childhood home.

[15] We create a pseudo-municipality for all individuals born in a given state for whom we cannot observe more detailed information about their birthplace.

rately by broad racial category (non-white and white) and sex to examine the potential for Chagas disease control to reduce inequality along these dimensions without imposing the additional assumptions necessary for—or risking the greater imprecision in—a triple-differences framework. Instead, we simply discuss and compare patterns across groups, which is facilitated by estimating the average of the post-treatment τ_k, though it requires that we impose a single treatment effect across these cohorts. In addition, we test in ROBUSTNESS APPENDICES whether our results are robust to focusing only on the three-fifths of the sample with exact municipality of birth information or using state-of-birth measures for the entire sample, and we complement the individual-level results with cross-census municipality-level estimates in Section 5.

DISCUSS APPENDICES WITH NEW ESTIMATORS.

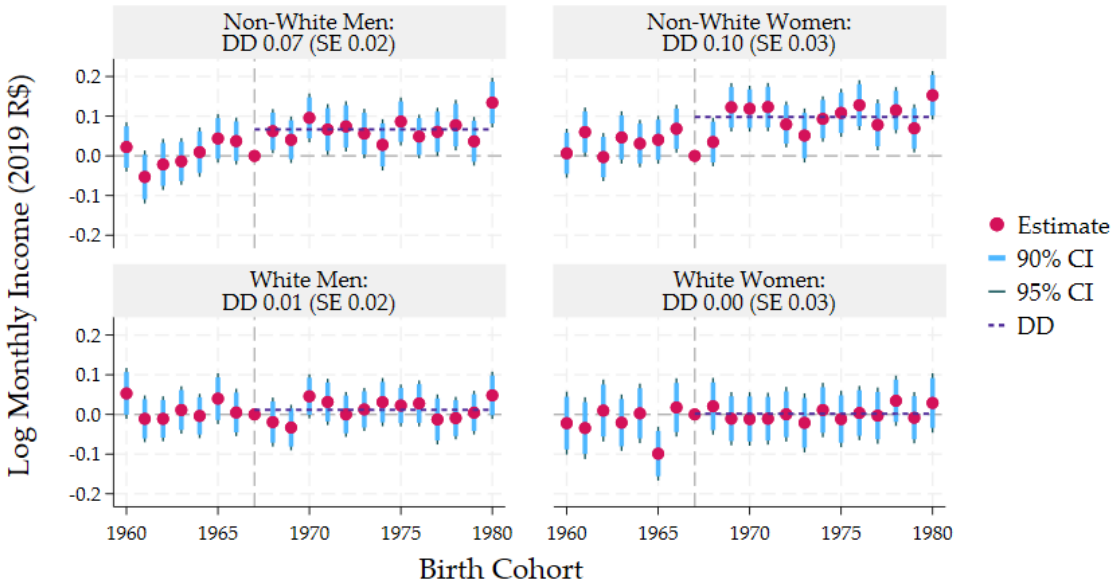
4.3.1. Addressing Threats to Identification

An important concern with this approach is the potential influence of concurrent policies or economic shocks that might have differentially affected post-treatment cohorts. For instance, if the roll-out of IRS against *T. infestans* coincided with other public health campaigns, our results would not be solely attributable to the elimination of vectorial *T. cruzi* transmission. Although historical accounts suggest little to no overlap with other health campaigns or targeted policies during this period, the specific progression of Chagas disease in humans provides a way to assess the relevance of this concern. Specifically, because its chronic phase often results in serious cardiovascular complications after a latency period of 10 or more years, significant improvements related to these conditions—but not any others—would thus not be expected until a decade or more after IRS began. As discussed in Section 6, our findings on health outcomes align with this timeline, mitigating concerns that concurrent economic shocks or government policies drive the effects discussed below.

4.4. Income and Inequality

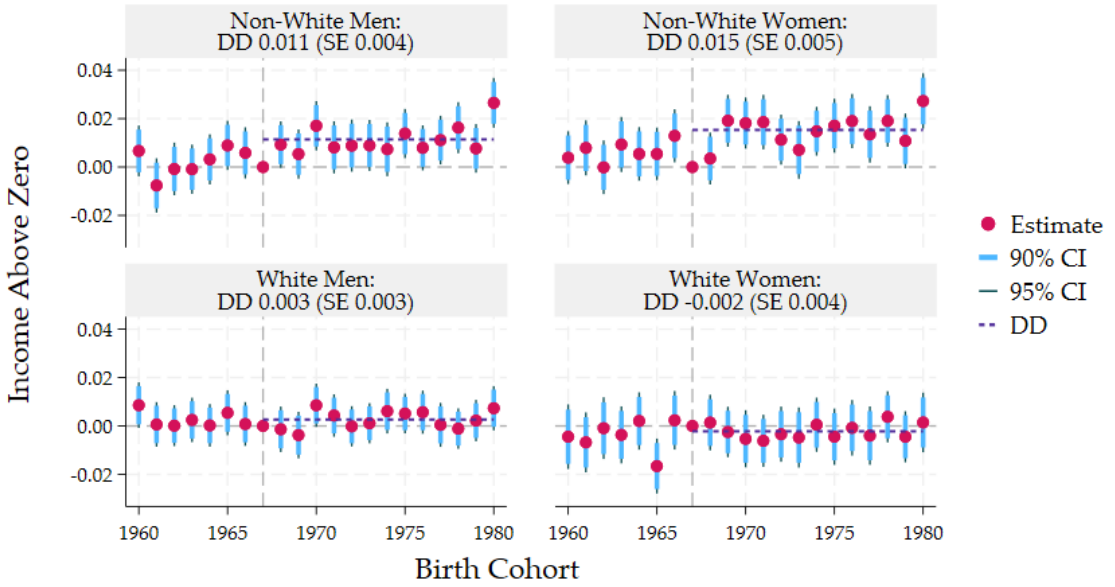
Our first outcome of interest is (the natural log of) monthly income as reported in the census for July 2010. Figure 3a shows that while incomes mostly evolved in parallel across treatment and control groups for cohorts that turned 16 before IRS began, the incomes of non-white men

Figure 3: Long-Run and Intergenerational Effects of Chagas Disease Vector Control



1.01-1.32 million observations, 1,582 clusters, pre-1968 birth cohort means (in 2019 R$): non-white men 468, non-white women 258, white men 904, white women 479

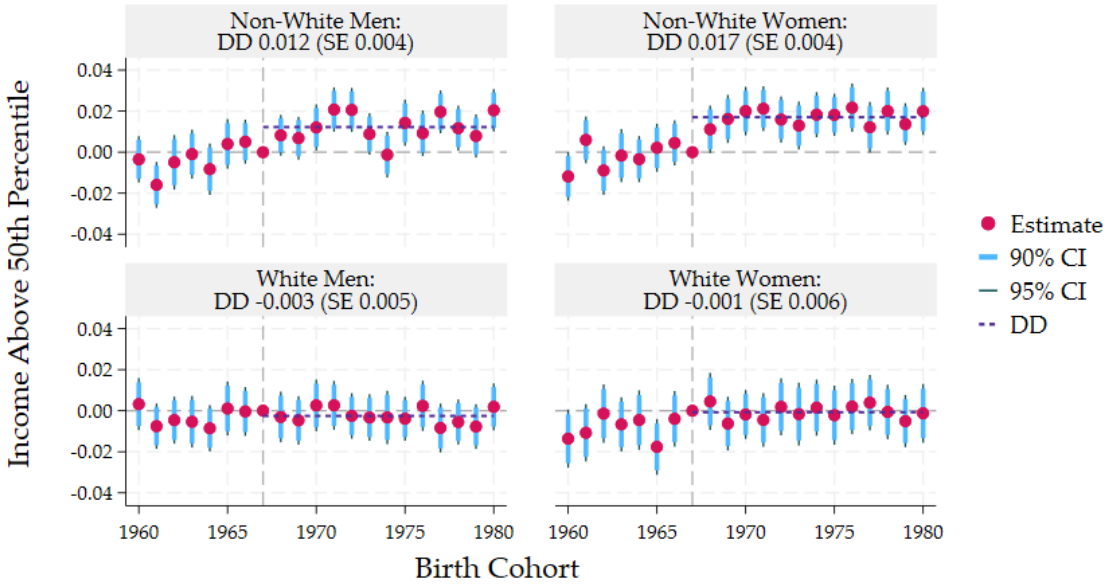
(a) Log Monthly Income



1.01-1.32 million observations, 1,582 clusters, pre-1968 birth cohort means: non-white men 0.807, non-white women 0.727, white men 0.888, white women 0.732

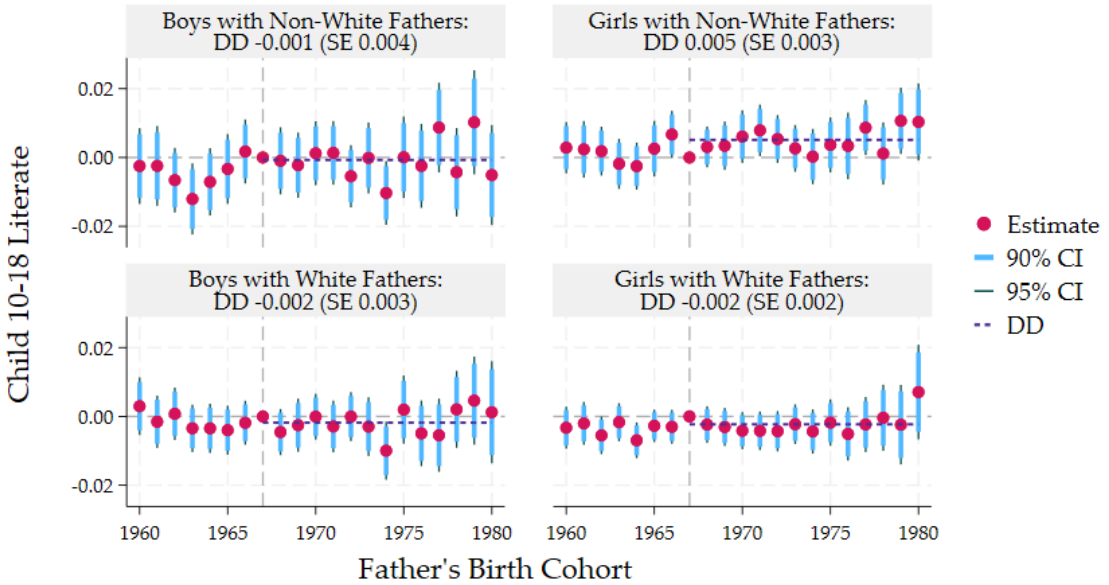
(b) Income Above Zero

Figure 3: Continued



1.01-1.32 million observations, 1,582 clusters, pre-1968 birth cohort means: non-white men 0.625, non-white women 0.408, white men 0.781, white women 0.548

(c) Income Above Median



0.25-0.44 million observations, 1,574 clusters, pre-1968 father's birth cohort means: boys with non-white fathers 0.951, girls with non-white fathers 0.977, boys with white fathers 0.980, girls with white fathers 0.989

(d) Literacy: Children Ages 10-18

Notes: Graphs show dynamic and post-treatment average (DD) estimates for the respective subgroups, with 90- and 95-percent confidence intervals for dynamic estimates. Data are from the IPUMS sample of the 2010 census. All regressions include fixed effects for (father's) birth cohort, birth municipality, and racial category. For children's literacy, regressions also control for own age and age squared. Standard errors are clustered by (father's) birth municipality.

and women who were exposed to treatment increased by 6 and 9 percent more than the incomes of their non-white peers from control municipalities. These estimates are also very precise. In contrast, there are no similar treatment-control divergences in the incomes of white men and women. As such, we view these results as consistent with our hypothesis that Chagas disease control reduced racial income disparities in Brazil.

Next, we turn to measures of income inequality at the municipality-cohort level. For each municipality of birth, we calculate various measures of inequality within each birth cohort: the Gini coefficient, the interquartile range (IQR), the gap between the 90th and 10th percentiles ("p90-p10 gap"), and the gap between the 90th percentile and the median ("p90-p50 gap"). Figure **??** shows that these indicators fell by more for cohorts exposed to treatment: the Gini coefficient by 0.8 percentage points (p.p., or 1.3 percent of the pre-treatment mean), the IQR by R$ 30/month (4.4 percent), the p90-p10 gap by R$ 69/month (4.4 percent), and the p90-p50 gap by R$ 68/month (5.8 percent). As each estimate is precise, especially for the percentile gaps, these results provide additional evidence that the Chagas disease control campaign helped mitigate Brazil's overall income inequality.

4.5. Next Generation's Literacy

To study the impacts on the children of men exposed to vector control during childhood, we attach men's birth municipality and cohort information to the children aged 6 to 18 living in their households in the 2010 census.[16] We then estimate a modified version of equation (1) that uses fathers' birth municipalities and cohorts, and we add children's age and age squared to the vector of controls. Given the children's ages, our outcome of interest is their literacy, which we view as a measure of their knowledge and thus their future labor market prospects. Notably, even through Brazil had made extensive progress toward universal child literacy between 1980 (see Table 1) and 2010, Figure 3d shows that it increased by 0.8 p.p. more for sons with non-white fathers who were children in treatment municipalities after IRS began than for sons of their non-white peers in the control group, and similarly by 0.9 p.p. more for their daughters. In addition

[16] We limit the sample to exclude those older than 18 due to selection into living with their parents after that age. We set 6 years old as the lower bound because it is when primary schooling begins in Brazil.

to being precisely estimated, these effects amount to large reductions in non-literacy: 8.5 percent of the mean for sons with non-white fathers in pre-treatment cohorts and 14.1 percent for their daughters. Once again, there are no comparable effects for the children of white fathers, which is in line with the income results above. Therefore, these results provide evidence that the some of the benefits of Chagas disease vector control were transferred from parents to their children, highlighting the positive intergenerational impacts of combating NTDs.

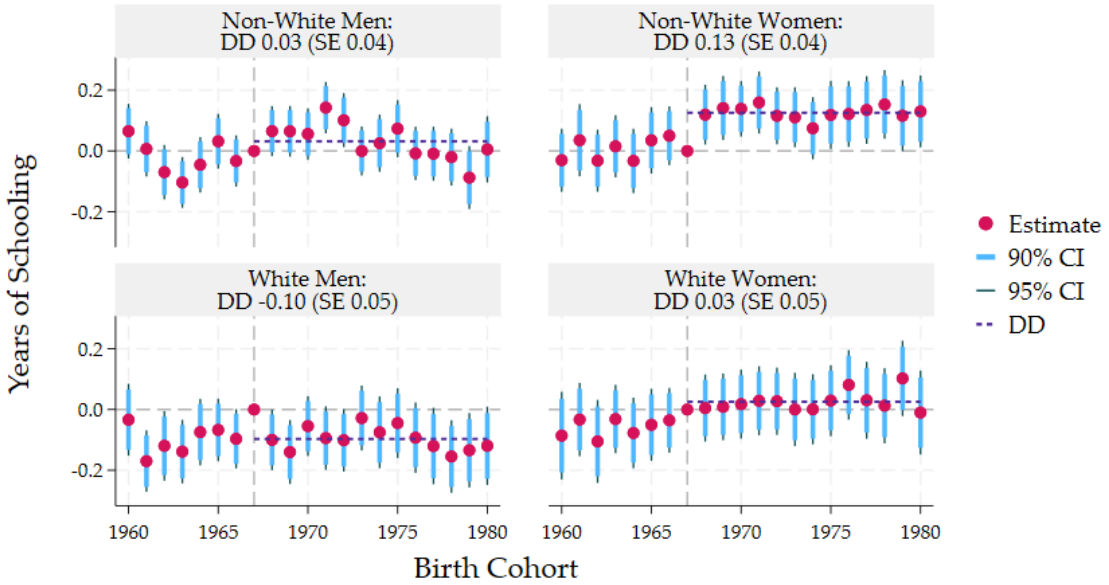
4.6. Mechanisms: Examining Years of Schooling and a Possible Role for Long-Run Health

Much of the literature on the effects of NTD control has focused on increased education as a primary mechanism (e.g., Miguel and Kremer, 2004; Bleakley, 2007, 2010). Thus, we use the 2010 census data and the same identification strategy as in Section 4.4 to study years of schooling among adults who were children around the time that Chagas disease vector control began. Figure 4a shows that the results are not particularly consistent with time in the classroom having a predominant role. Specifically, while there is a precisely estimated effect for non-white women (0.13 years, or 2.4 percent of the mean for pre-treatment cohorts), it is small and noisy for non-white men (0.04 years, or 0.8 percent) and non-white women (0.08 years, or 1.1 percent), though the latter appears to be a continuation of a trend among pre-treatment cohorts.

To support our interpretation, we perform a back-of-the-envelope calculation of the upper bound of years of schooling's contribution to the income results. Using the Mincerian return to schooling in Brazil of 15.7 percent (as reported in Psacharopoulos and Patrinos, 2018)—which almost certainly overstates the causal effect of schooling on income—the 0.13-year increase for non-white women can account for at most 2.1 p.p. (or less than one-fourth) of the 9-percent increase in their incomes. For non-white men, taking the imprecise 0.04-year estimate at face value generates at most a 0.6-p.p. increase in their incomes, which is just 7.1 percent of the observed effect. As such, the implication is that increased time in the classroom is unlikely to explain the long-run increases in incomes and reductions in racial inequities due to Chagas disease control.

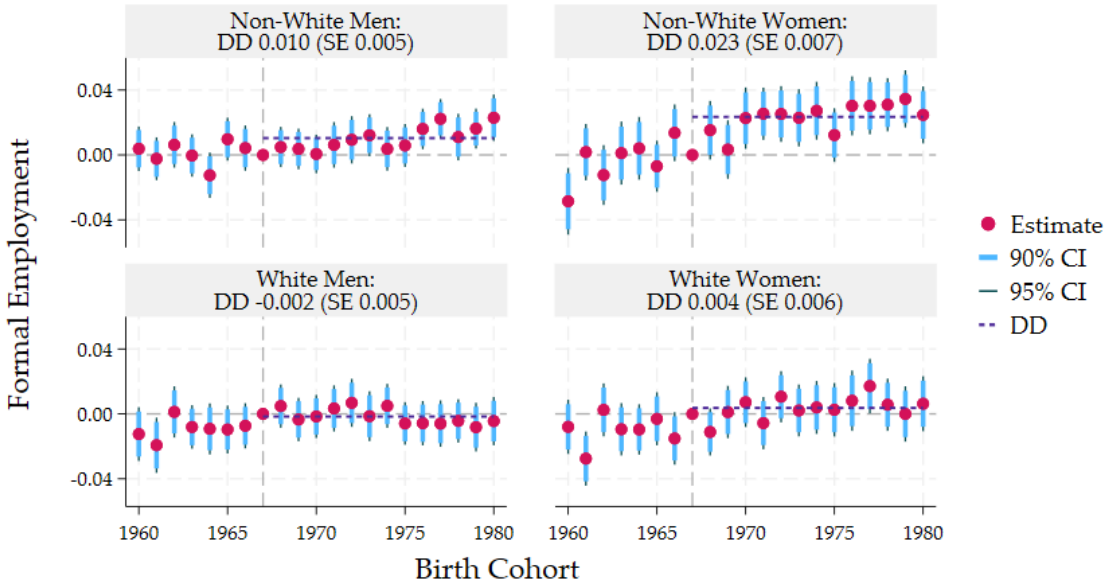
Instead, we view reductions in its chronic symptoms—which "can cause young adults to develop heart conditions, so that they fill hospital beds instead of the [labor] force" (World

Figure 4: Mechanisms Underlying Long-Run and Intergenerational Effects



1.01-1.32 million observations, 1,582 clusters, pre-1968 birth cohort means: non-white men 4.80, non-white women 5.41, white men 6.44, white women 6.96

(a) Years of Schooling



0.69-1.03 million observations, 1,582 clusters, pre-1968 birth cohort means: non-white men 0.449, non-white women 0.467, white men 0.491, white women 0.542

(b) Formal Employment

Notes: Graphs show dynamic and post-treatment average (DD) estimates for the respective subgroups, with 90- and 95-percent confidence intervals for dynamic estimates. Data are from the IPUMS sample of the 2010 census. All regressions include fixed effects for birth cohort, birth municipality, and racial category. Standard errors are clustered by birth municipality.

Health Organization, 2010)—as a more promising candidate. Because they manifest 10 or more years after infection and over two decades had passed between the start of IRS and the 2010 census, it is very much possible that these symptoms would have begun to manifest before 2010 in the absence of the 1984-89 vector control program. However, we cannot test this proposition directly in the census data, as they lack information on health status, so we use a place-based strategy to investigate long-run health effects in Section 6.

5. Effects on Municipalities across Censuses

In this section, we take a complementary approach to studying the long-run effects of Chagas disease vector control by comparing repeated cross-sections of individuals living in treatment and control municipalities. Consistent with our previous results, we find that it led to increased incomes and reduced inequality. Importantly, these benefits were evident in the first post-treatment census and they became more pronounced in subsequent years.

5.1. Data and Empirical Strategy

We use data from five Brazilian censuses: two 25-percent samples from pre-treatment years (1970 and 1980) and three 10-percent samples from post-treatment years (1991, 2000, and 2010), all of which are from IPUMS (Ruggles et al., 2024). Our outcomes of interest are the distributions of incomes (in 2019 R$) in consistent municipalities across census years.[17] Because the 1970 census does not contain information on race, we cannot assess parallel trends assumptions within subgroups, so we pool all observations within a municipality m and census year t to estimate

$$y_{m,t} = \alpha_m + \gamma_t + \sum_{k \neq 1980} \tau_k \cdot \mathbb{1}[m \in \text{Treat}] \cdot \mathbb{1}[t = k] + \mathbf{X}_{m,1980} \times \gamma_t + \delta_{s(m)} \times \gamma_t + \epsilon_{m,t} \tag{2}$$

where $\mathbb{1}[m \in \text{Treat}]$ indicates whether m is in the treatment group, $\mathbf{X}_{m,1980}$ is a vector of m's characteristics in 1980 (the female, Asian, Black, and Brown shares of the population), $\delta_{s(m)}$ is a fixed effect for m's state, and all other variables are analogous to those in equation (1). We also include the interactions of $\mathbf{X}_{m,1980}$ and $\delta_{s(m)}$ with γ_t to control for 1980 characteristics' time-

[17] We match 1970 municipalities to the IPUMS 1980-2010 municipalities containing their centroids.

varying influences and state-specific trends across census years.

The coefficients of interest are the τ_k, which measure the difference in an outcome in a given census year across the treatment and control groups, relative to the size of that difference in 1980. As these data are at the municipality level, we know with certainty which group an observation is in, changing the interpretation of our treatment definition from the one in the previous section. In particular, because we pool all observations in a municipality, we observe how treated municipalities evolve over time rather than how treatment effects change with additional years of childhood free from vectorial transmission of Chagas disease. Another important difference is that the composition of the sample changes across census years because our municipality-level measures are derived from repeated cross-sections.

With these differences in mind, we once again use OLS with standard errors clustered by municipality, but now we weight observations by their 1980 population to account for differences in municipality sizes. We also estimate the average of the post-treatment τ_k to minimize imprecision and generate a single policy-relevant estimate, though at the cost of imposing a single treatment effect across census years.

DISCUSS APPENDICES WITH NEW ESTIMATORS.

5.2. *Results*

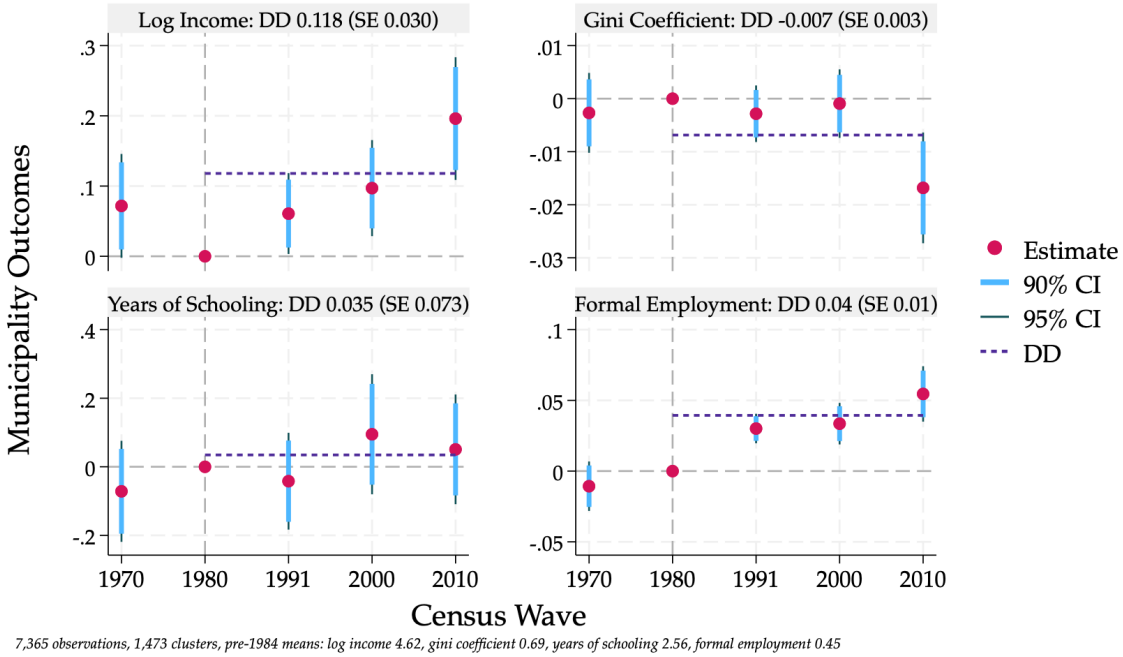
We first study the effects of IRS on (the natural log of) monthly income and its distribution within municipalities, with values converted into 2019 BRL. Figure 5 shows that

We also examine the effects of the control campaign on years of schooling.

Taken together, the results suggest that reductions in Chagas disease transmission led to important benefits: municipalities saw increases in incomes and decreases in income inequality.[18] These benefits tend to increase over time, especially in time periods when the benefits of reduced chronic Chagas disease are likely to become important. In the next section, we directly examine the long-run health benefits of reducing the incidence of these symptoms.

[18] 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 shows similar short-run benefits from Chagas disease control.

Figure 5: Effects of Chagas Disease Vector Control on Municipalities



Notes: Observations are municipality-years. Graphs show dynamic and post-treatment average (DD) estimates for the respective outcomes, with 90- and 95-confidence intervals for dynamic estimates. Data are from the IPUMS samples of the 1970, 1980, 1991, 2000, and 2010 censuses. All regressions include fixed effects for census year and municipality and the interactions of census year fixed effects with a vector of 1980 characteristics (shares of the population that were female, Asian, Black, and Brown) and state fixed effects. Standard errors are clustered by municipality.

6. Effects on State Public Health Care Systems

If cardiovascular morbidity from chronic Chagas disease was severe enough to reduce the productivity of adults exposed to its transmission as children, the costs it imposed on society could have extended beyond income and (racial) inequality. Specifically, as Brazil has the world's largest government-run health care system (the *Sistema Único de Saúde*, or SUS)—which consumes about 4 percent of GDP and 70 percent of the population depends on—improvements in adults' heart health could have had important effects on public finances.[19] 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 2019 R$ 1.5 billion annually, or around 0.1 percent of GDP).

[19] For more information on the SUS, see: https://agenciagov.ebc.com.br/noticias/202409/sistema-unico-de-saude-comemora-34-anos-de-democracia-e-cidadania.

Therefore, in this section we examine the long-run effects of Chagas disease vector control on cardiovascular-related hospital care covered by the SUS. 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 savings for the public health care system in Brazil. IRR, MVPF?

6.1. 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, each of which is by International Classification of Diseases (ICD) code. The first three of these measures are from the SUS's Hospital Information System (SIH/SUS), and we deflate the spending data so that figures are in 2019 BRL. The data on deaths are from the SUS's Mortality Information System (SIM). Given that Chagas disease is highly under-diagnosed and its chronic effects manifest primarily as cardiovascular problems 10 or more years after infection (see Section 2), we focus on all diseases of the circulatory system.[20] In addition, we set our omitted year to 1994, or a decade after IRS began.[21] However, as the SUS does not consistently provide municipality-level data for years prior to 1995, our focus in the main text is on state-level data from 1991 to 2019.[22]

In a similar vein as our approach in Section 4, our measure of each state's exposure to Chagas disease vector control is the share of its 1980 population living in treatment municipalities. However, because the SUS is a heavily decentralized system with transfers of responsibilities and funds to states and municipalities (Castro et al., 2018), there are likely confounders that vary across both state and year (e.g., public health priorities, non-hospital care spending) in violation of the difference-in-differences common trends assumption. To address this complication, we use a triple-differences strategy using all other non-circulatory system disease categories as the

[20] For 1991 to 1997 hospital care outcomes, we use ICD-9 Chapter 7 (codes 390-459), and for 1998 onwards, we use ICD-10 Chapter 9 (codes I00-I99). For deaths, the final year using ICD-9 was 1995.

[21] Once again, we assume that the elimination of vectorial Chagas disease transmission occurred immediately after IRS began. This conservative approach implies that our treatment effect estimates for the first several post-1994 years might be biased toward zero.

[22] In APPENDICES, we verify that the same divergence in trends occurred in the post-1994 municipality-level data, though we cannot test whether outcomes evolved in parallel before then.

additional control group, under the assumption that they are subject to the same state-specific, time-varying factors.[23] The specification that we estimate is thus

$$y_{s,t,d} = \alpha_{s,t} + \gamma_{t,d} + \delta_{s,d} + \sum_{k \neq 1994} \tau_k \cdot \mathbb{1}[\mathbb{P}_{s,1980}(m \in \text{Treat}) > 0] \cdot \mathbb{1}[t = k] \cdot \mathbb{1}[d = \text{Circ}] + \eta_{r(s)} \times \gamma_{t,d} + \epsilon_{s,t,d}, \tag{3}$$

where $y_{s,t,d}$ is state s's outcome in year t for disease category d, $\alpha_{s,t}$, $\gamma_{t,d}$, and $\delta_{s,d}$ are fixed effects for state-year, year-disease category, and state-disease category, $\mathbb{1}[\mathbb{P}_{s,1980}(m \in \text{Treat}) > 0]$ indicates whether s had any of its population living in treatment municipalities in 1980, $\mathbb{1}[d = \text{Circ}]$ indicates whether d is circulatory system diseases, $\eta_{r(s)}$ is a fixed effect for s's region, and all other variables are analogous to those in previous specifications.[24] As in equation (2), we include the interaction of $\eta_{r(s)}$ and $\gamma_{t,d}$ to control for region-disease category-specific trends.

This strategy first estimates the differences in log outcomes due to circulatory system diseases in a given year between states with any individuals living in treatment municipalities in 1980 and those that did not, relative to the size of that difference in 1994. Then we compare this double-difference to the analogous one for non-circulatory diseases in the same year. 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. To increase precision and policy relevance, we also estimate the average of the post-1994 τ_k. For inference in both cases, we compute wild cluster bootstrap confidence intervals after clustering standard errors by the 21 consistent states in the sample (Cameron, Gelbach and Miller, 2008).

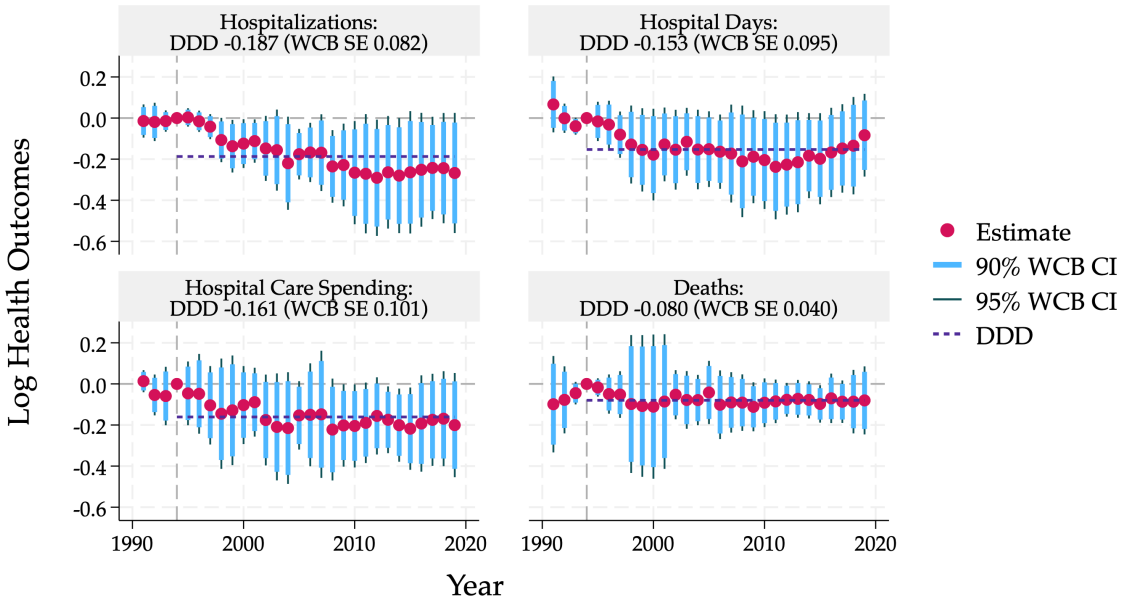
6.2. Circulatory Disease Hospital Care

Consistent with our hypothesis that Chagas disease control would improve cardiovascular outcomes, the top-left panel of Figure 6 shows that hospitalizations paid for by the SUS due to circulatory system disease decreased by 21 percent more than those due to all other causes. This effect is precisely estimated and emerges after outcomes evolved in parallel prior to 1995—indeed, the divergence began close to the year predicted in the medical literature. Similarly, in

[23] If it holds, the triple-difference approach is a valid strategy when difference-in-differences rejects the absence of differential pre-treatment trends for each disease group (Olden and Møen, 2022)

[24] Brazilian states are grouped into five regions: North, Northeast, Southeast, South, and Center-West.

Figure 6: Long-Run Effects on Circulatory Disease Outcomes



1,392 state-year-disease category observations, 24 clusters, pre-1995 means: 0.24 million hospitalizations, 1.54 million hospital days, 2019 R$ 307 million hospital care spending (1995 mean to avoid distortions from 1991-94 hyperinflation), 0.02 million deaths

Notes: Graphs show dynamic and post-treatment average (DDD) estimates for the respective outcomes, with 90- and 95-percent wild cluster bootstrap (WCB) confidence intervals for dynamic estimates. Data are from DATASUS. All regressions include fixed effects for state-year, year-disease category, and state-disease category and the interaction of region and year-disease category fixed effects. WCB standard errors are clustered by state.

the top-right panel, SUS-covered days spent in the hospital as a result of cardiovascular problems decreased by 17 percent more, and this estimate is also precise. Consequently, we also find a 13.7-percent greater decrease in the SUS's spending on circulatory disease hospital care relative to spending on care due to other causes. While the dynamic and post-treatment average effects in the bottom-left panel are noisier than those in the top panels, the patterns over time are effectively identical, which we consider further evidence of the spending effect. Therefore, these results suggest that eliminating Chagas disease transmission yielded significant benefits for public health and public finances in Brazil, which we quantify below.

6.3. Circulatory Disease Deaths

However, the impact on deaths by cause is less clear in the bottom-right panel. Although the post-treatment average effect is precisely estimated, there is a much larger pre-treatment difference

in trends across circulatory and non-circulatory diseases than in the other panels, and there are some extremely wide confidence intervals in the late 1990s and early 2000s. Nonetheless, we view the absence of strong evidence of an effect on deaths until the mid- to late 2000s to be reassuring. Given the uncertainty in the medical literature about when chronic symptoms manifest and the lack of a timeline for how long after that point deaths can occur (see Section 2), it seems likely that there should be a substantive gap between the two events.

7. Cost-Benefit Analyses and Extrapolation

7.1. Cost-Benefit Analysis

Given the substantial public health benefits of the Chagas disease control project documented in Section 6, 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 6. 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.

7.2. Implications for Chagas Disease Control across Latin America

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

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 2.[25] The results in Table 2 suggest that, within the Americas, vector suitability is robustly associated with lower average incomes and greater inequality in the cross-section. Importantly, outside of the Americas, the suitability of the Chagas disease vector does not have a significant correlation with development or disparities, suggesting that the relationship between the suitability of the Chagas disease vector and income and disparities is not due to the direct effects of ecological factors that increase the suitability of the vector. Instead, the results suggest that the presence of the vector and associated Chagas Disease in the Americas is significantly associated with lower development and higher disparities. These results provide suggestive evidence that combating Chagas disease could potentially help improve incomes and

[25] 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 2: 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. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

reduce income inequality in the region.[26]

To assess the broader implications of controlling Chagas Disease beyond Brazil, we extrapolate the estimated effects on income and inequality to other Latin American countries using World Health Organization (WHO) data on population exposure to the disease (World Health Organization, 2015). Appendix Figure A8 provides a map with the estimated share of the population that is exposed to Chagas disease across countries; the World Health Organization (2015) estimates that approximately 13% of the population of Latin America is still exposed to Chagas disease.

[26] Additionally, Figure A7 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, suggesting that the presence of the disease may have hindered the formation of more complex and centralized societies. This historical impact underscores the long-standing influence of Chagas Disease on development in the region.

Following Bleakley (2007), by multiplying the percentage of the population in each country that is exposed to Chagas disease by our estimated income and inequality effects from Section 5, we can provide back-of-the-envelope calculations for the impact that controlling Chagas disease might have on the GDP per capita and Gini coefficients of other Latin American countries where Chagas is still present.[27]

Appendix Table A1 presents these extrapolated impacts. The analysis suggests that, across Latin America, GDP per capita could be approximately 0.9% higher if Chagas Disease were controlled, with significant variation across countries depending on the share of their population exposed to the disease. Furthermore, the Gini coefficient would be, on average, 0.08 percentage points lower, reflecting a modest reduction in inequality. Although this change may seem small, it is worth noting that the Gini coefficients in Latin America are already much higher than those in other regions, such as Europe, where the average Gini gap between Latin America and Europe is about 8 percentage points. Thus, even small reductions in inequality could represent meaningful progress. In general, the estimates underscore the potential for public health interventions targeting Chagas disease to yield substantial economic benefits throughout

8. 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.

[27] While this extrapolation provides valuable insights, several caveats should be considered. Similar to the analysis in Bleakley (2007), potential general equilibrium effects, such as changes in labor market dynamics, could influence the magnitude of these estimates, either amplifying (e.g., via human capital spillovers) or dampening the impact (e.g., from higher labor supply). These factors imply that general equilibrium effects could be either larger or smaller than the extrapolated estimates.

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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November 1, 2024

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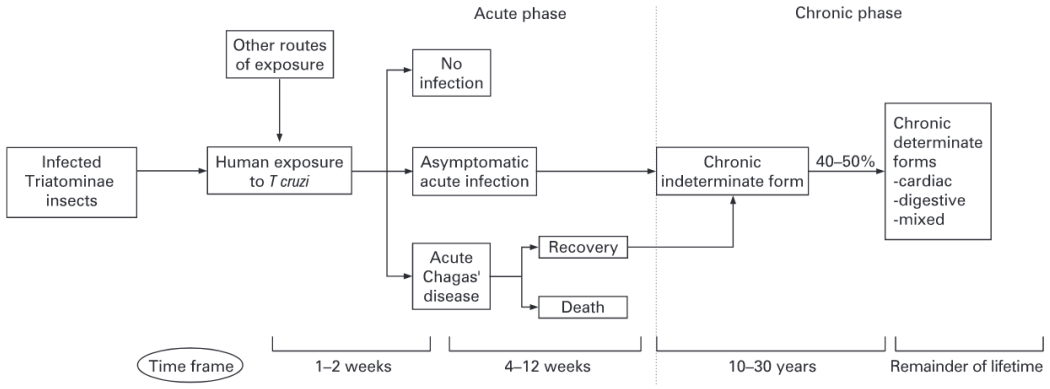
Appendix A. Additional Figures

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: Phases of Chagas Disease



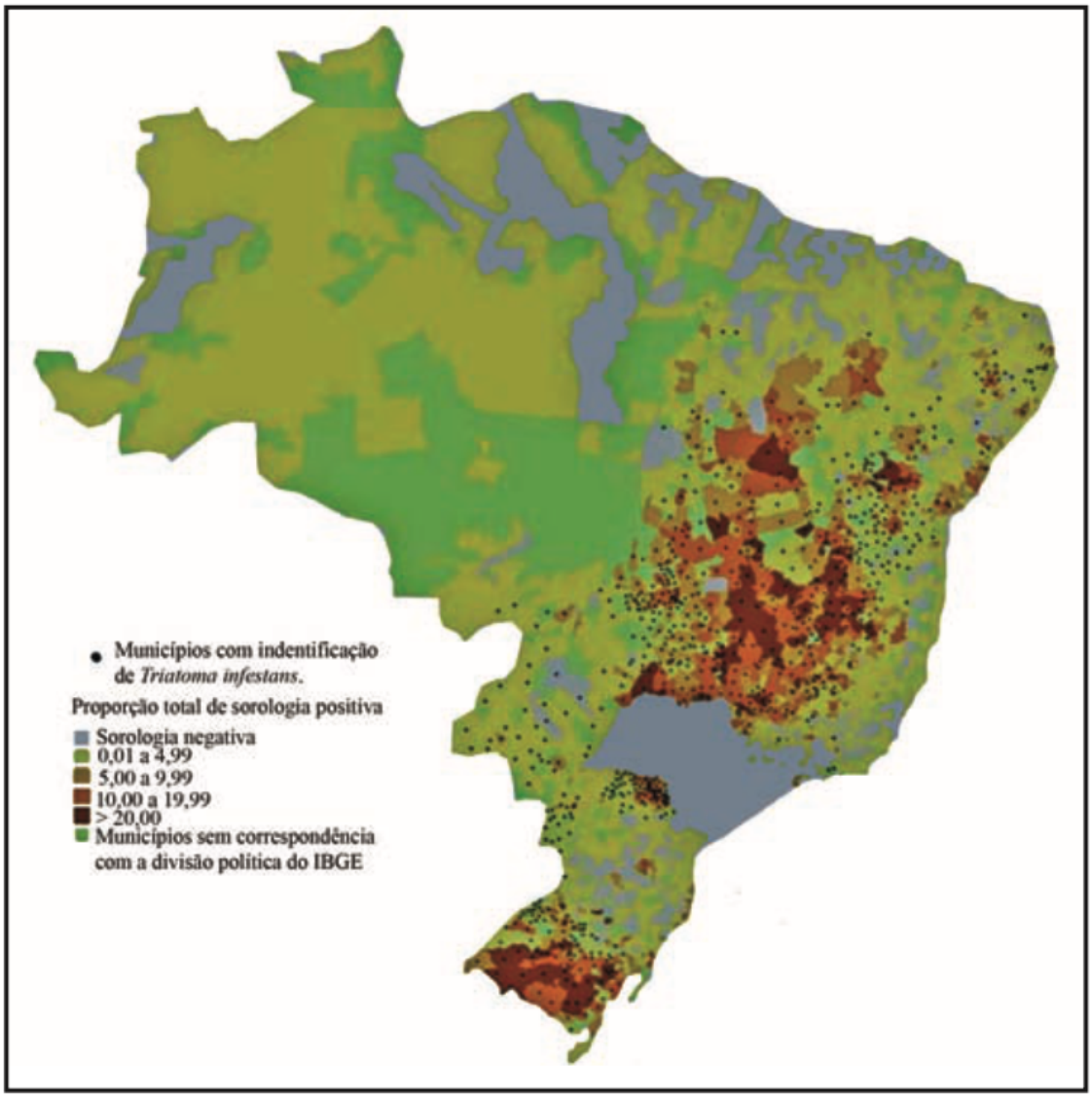
Notes: Diagram taken from Rassi et al. (2009, p. 527).

Table A1: Extrapolation: Impact of Controlling Chagas Disease on Latin American Development and Inequality

Country	*Estimated % of Population Exposed to Chagas Disease*	*GDP Per Capita*	*Extrapolated Δ in GDP Per Capita From Controlling Chagas Disease*	*Gini Coefficient*	*Extrapolated Δ in Gini Coefficient From Controlling Chagas Disease*
(1)	(2)	(3)	(4)	(5)	(6)
Argentina	0.05	10076	49.19	42.90	-0.03
Belize	0.22	4983	100.03	–	–
Bolivia	0.06	3552	18.85	41.60	-0.03
Brazil	0.13	8876	106.68	53.50	-0.10
Chile	0.00	14699	0.00	44.40	-0.00
Colombia	0.11	6419	60.71	51.30	-0.07
Costa Rica	0.05	12762	59.35	48.20	-0.03
Ecuador	0.29	6223	162.39	45.70	-0.18
El Salvador	0.15	4168	54.98	38.80	-0.08
Guatemala	0.10	4639	43.13	48.30	-0.07
Guyana	0.25	6610	149.41	–	–
Honduras	0.15	2574	33.96	48.20	-0.09
Mexico	0.21	9950	186.92	46.70	-0.13
Nicaragua	0.11	1924	19.87	46.20	-0.07
Panama	0.13	15774	186.22	49.80	-0.09
Paraguay	0.20	5384	95.23	45.70	-0.12
Peru	0.04	7023	28.18	41.60	-0.02
Suriname	0.25	6854	154.93	–	–
Uruguay	0.00	17688	0.00	39.70	-0.00
Average	0.13	7904	79.47	45.79	-0.07
Average (>0 Exposure)	0.15	6929	88.82	46.32	-0.08

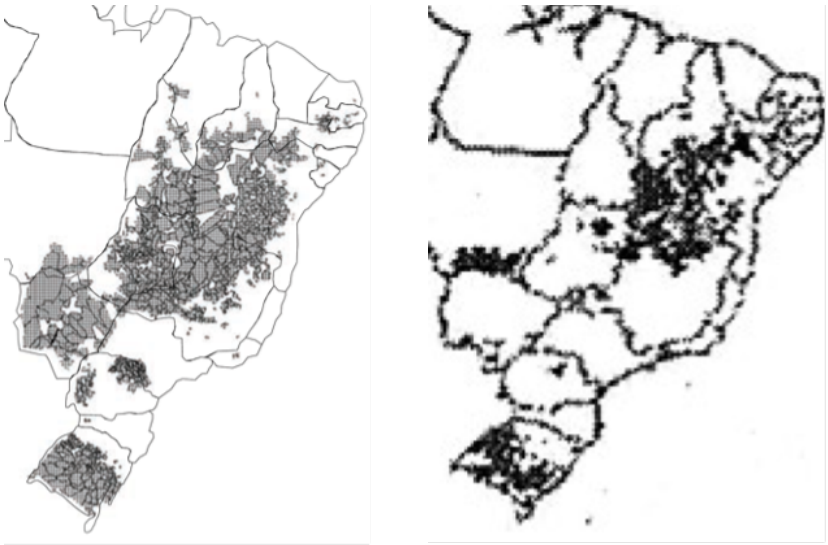
Notes: The unit of observation is a country. *Estimated % of Population Exposed to Chagas Disease* is the share of the population reported to be exposed to Chagas disease vectors according to the World Health Organization (2015). *GDP Per Capita* is GDP per capita in 2019 (in $1,000s) from the World Bank Development Indicators. *Extrapolated Δ in GDP PC From Controlling Chagas Disease* multiplies the GDP per capita by the % of population that is exposed to Chagas and the estimated % change in incomes from Figure 5. *Gini Coefficient* is the most recently reported Gini coefficient (scaled by 100) between 2010 and 2019 from the World Bank Development Indicators. *Extrapolated Δ in Gini Coefficient From Controlling Chagas Disease* multiplies the Gini Coefficient by the % of population that is exposed to Chagas and the estimated % change in income gini from Figure 5.

Figure A3: Rural *T. cruzi* Seroprevalence



Notes: Figure reproduces a map of *T. cruzi* Seroprevalence prior to the Chagas control campaign across municipalities from Costa Passos and Silveira (2011).

Figure A4: 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.

Figure A5: Picture of Insecticide Spraying Campaign

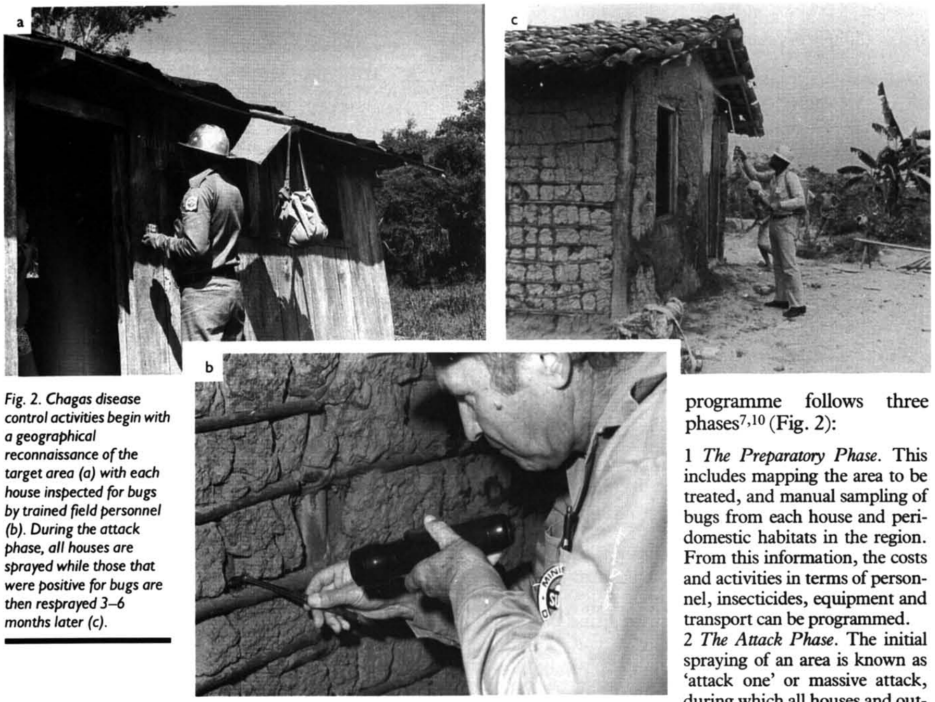


Fig. 2. Chagas disease control activities begin with a geographical reconnaissance of the target area (a) with each house inspected for bugs by trained field personnel (b). During the attack phase, all houses are sprayed while those that were positive for bugs are then resprayed 3–6 months later (c).

programme follows three phases[7,10] (Fig. 2):

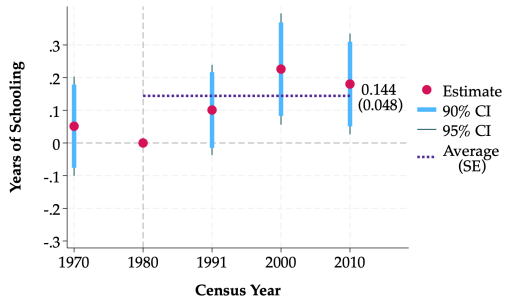
1 *The Preparatory Phase*. This includes mapping the area to be treated, and manual sampling of bugs from each house and peridomestic habitats in the region. From this information, the costs and activities in terms of personnel, insecticides, equipment and transport can be programmed.

2 *The Attack Phase*. The initial spraying of an area is known as 'attack one' or massive attack, during which all houses and outbuildings are sprayed regardless of whether or not they were found to be infested. A second selective spraying is then carried out 3–6 months later only in houses known to have been infested.

– development of suitable vector control methods, both in trials against Chagas disease itself, and from experience with malaria control;

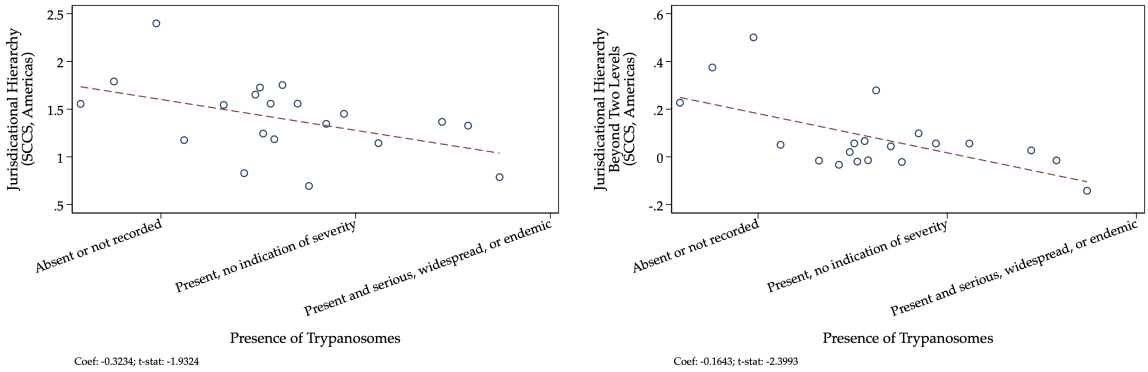
Notes: Images and text on the Chagas disease control activites reproduced from Dias (1987).

Figure A6: Effects on Years of Schooling Across Census Waves



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.

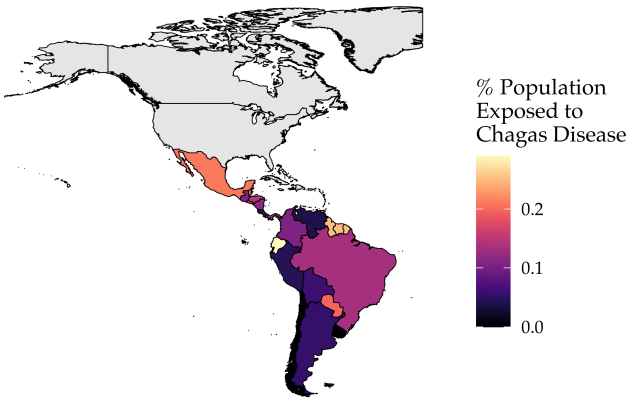
Figure A7: Pre-Colonial Presence of Trypanosomes and Jurisdictional Hierarchy



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.

Figure A8: Estimated % of Population Exposed to Chagas Disease



Notes: Figure presents a map of the estimated percent of the population in each country in Latin America that is exposed to Chagas from the World Health Organization (2015). Countries without information on Chagas disease exposure are in light grey.

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). We use microdata for 11 of the 18 years from 1982 to 1999.[28] These surveys contain data on respondents' state of residence, household characteristics and sociodemographic information, and schooling 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.

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

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

the PNAD surveys, so we instead comparing individuals across states. Our estimating equation is the dynamic difference-in-differences model:

$$y_{i,s,t} = \alpha_s + \gamma_t + \sum_{k \neq 1986} \tau_k \cdot \mathbb{1}[\mathbb{P}(Treat)_s > 0] \cdot \mathbb{1}[t = k] + \mathbf{X}_i\beta + \epsilon_{i,s,t} \tag{4}$$

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 (4) 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 (4) 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 (4), we also estimate the static two-way fixed effects model

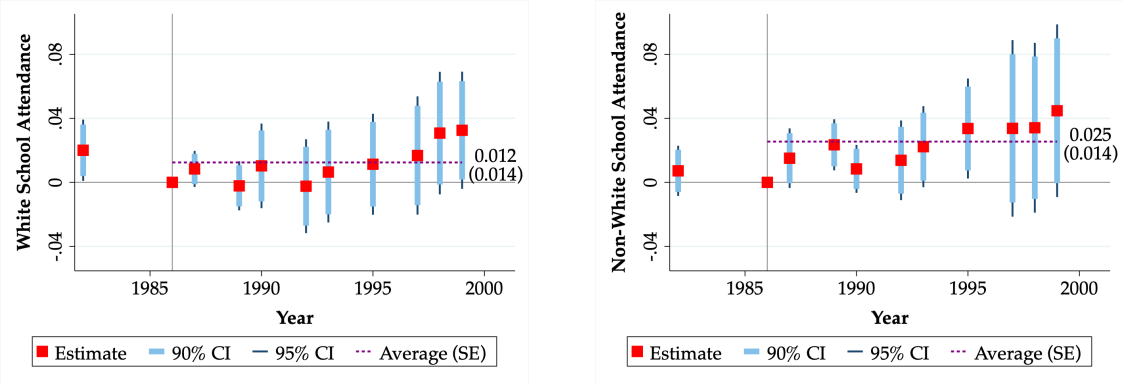
$$y_{i,s,t} = \alpha_s + \gamma_t + \tau \cdot \left(\mathbb{1}[\mathbb{P}(Treat)_s > 0] \cdot \mathbb{1}[t > 1986]\right) + \mathbf{X}_i\beta + \epsilon_{i,s,t}, \tag{5}$$

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.

To examine the potential for health improvements to reduce racial inequality in the short run, we estimate equations (4) and (5) separately for white and non-white individuals.

Figure B9: 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

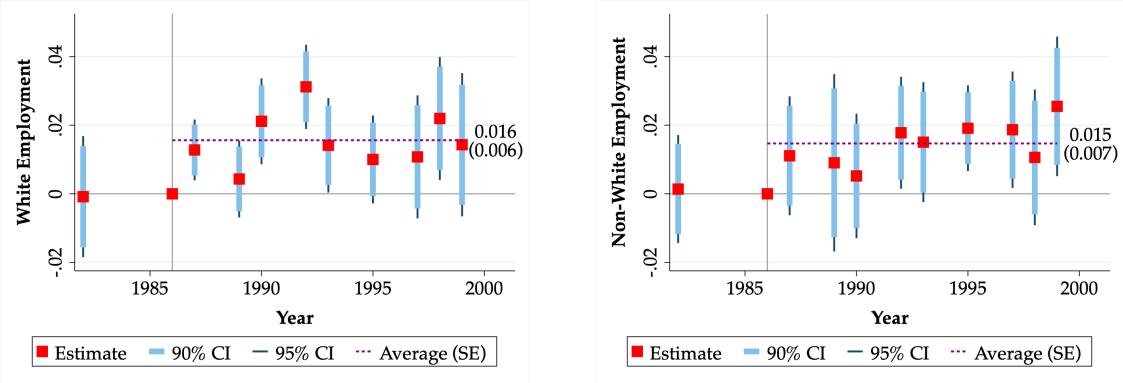
Notes: Graphs plot dynamic (red squares) and static (dotted purple line) two-way fixed effects estimates for the respective outcomes and racial groups with 90-percent (light blue) and 95-percent (dark blue) wild cluster bootstrap confidence sets for dynamic estimates. Static estimate magnitudes are next 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.

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 B9 plots the estimates from equations (4) and (5) for school attendance for white (B9a) and non-white (B9b) children ages 8 to 18. Figure B9 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 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

Figure B10: 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.

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 B10 for white (B10a) and non-white Brazilians (B10b). 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 B10 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.