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

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Abstract

We show that Brazil's 1984-89 efforts to eliminate the vectorial transmission of Chagas Disease – a neglected tropical disease with a weeks-long acute phase and often a chronic cardiovascular phase at least 10 years later – had short- and long-run effects in domains that are important for economic development but have not yet been included in cost-benefit analyses of disease control. Using a difference-in-differences strategy, we make comparisons before and after the vector control campaign across states and municipalities with varying levels of pre-treatment vector prevalence. We find that adults' employment rates rose shortly after spraying began and cohorts treated as children had higher incomes as adults, and the latter effect was much larger for non-white Brazilians. Using a triple-differences strategy, we also show that Brazil's government-run health care system – which consumes 4 percent of GDP – spent substantially less on hospitalizations due to circulatory diseases than other causes. These results imply that the benefits of disease control can be larger than previously assumed, and that combating neglected tropical diseases can help to speed convergence in societies with large racial disparities.

Keywords: Disease, Employment, Racial Disparities, Health Care Spending
JEL Classification: H51, I14, I15, O15

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1. Introduction

In the last two decades, the impact of disease on economic development has received much scholarly attention. While some studies argue for limited or even positive macroeconomic effects of disease in the modern era (e.g., Young, 2005; Acemoglu and Johnson, 2007; Ashraf, Lester and Weil, 2009), the large majority find substantial negative effects on a more micro level, with a focus on (anticipated) labor market returns (e.g., Miguel and Kremer, 2004; Bleakley, 2007). Importantly, they arise by affecting the children's human capital, whether through their ability to learn and attend school (Bleakley, 2010*a*) or expectations about the time horizon over which they will use it (e.g., Jayachandran and Lleras-Muney, 2009).

While this channel makes substantial contributions to long-run economic development by raising the adult incomes of those treated as children, there are two major downsides to relying solely on it to justify health interventions.[1] First, it takes nearly two decades for the first cohorts exposed to treatment for their entire childhoods to begin earning labor market returns from their increased human capital. As most of these returns are realized decades into their earnings trajectories, they are heavily discounted in cost-benefit analyses conducted when deciding whether to undertake an intervention. In addition, while schooling and labor income are easily measured, it is highly unlikely that they are the only development-relevant domains that health improvements affect. The result is that the benefit side of the ledger can be substantially understated.

This underestimation of the benefits of health interventions can be especially critical for neglected tropical diseases—those afflicting the poorest populations and causing them substantial morbidity—as donors may focus on quickly reducing mortality and developing-country governments may prioritize more immediate or certain payoffs.[2] Therefore, researchers can make substantial contributions to improving the lives and living standards of the world's poorest people by demonstrating that disease control has impacts in the short run as well as in the long run

[1] The main exception to the primary focus on childhood human capital has been the literature on HIV (e.g., Tompsett, 2020) because it primarily affects sexually-active adults, but it is still an important component of research on the virus's economic impacts (e.g., Baranov and Kohler, 2018).

[2] In the case of soil-transmitted helminths, see Taylor-Robinson et al. (2019) for a systematic review that is cautious about the evidence for the benefits of mass deworming.

in areas relevant for economic development beyond individuals' labor market returns.[3]

We take a step in this direction by examining the effects of Brazil's efforts to eliminate the transmission of Chagas Disease (also known as American trypanosomiasis). As we discuss in Section 2, this parasitic neglected tropical disease almost exclusively afflicts the Americas south of the Rio Grande, and it affects both children and adults. Specifically, both groups can experience the acute phase's symptoms that last for weeks. And 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), as the disease affects around 6 million people in the region.

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

We test these hypotheses by exploiting the geographic distribution of the main Chagas Disease vector prior to the control campaign. In Section 3, we describe the breakthrough that allowed for such a program, the 1975-83 nationwide entomological survey measuring the vector's presence at the municipal level, and the progress of insecticide spraying between 1984 and 1989. As we describe in Section 4, these data allow us to compare municipalities that were never infested with the vector and those that had it prior to spraying but were rid of it by 1989.

The idea behind our approach is that municipalities where the vector was eliminated experienced a greater reduction in exposure to Chagas Disease than those where the vector was never found. We formalize this empirical strategy in Section 5 and use nationwide survey data from that period and a difference-in-differences framework to examine the short-run effects of the vector control campaign. As hypothesized, we find relatively rapid effects on employment rates for adults: moving from the 25th percentile of the probability of living in a treatment municipality

[3] To the extent that these benefits are public goods, they strengthen the case for collective action.

to the 75th percentile results in a 1.3-percentage point (p.p., 1.9 percent) greater increase in the likelihood of being employed in post-spraying years. These effects are larger for non-white (1.9 p.p., or 2.6 percent) than white Brazilians (1.1 p.p., or 1.5 percent), suggesting that it raised the speed of racial convergence in the short run.

In Section 6, we turn to the long-run labor market results for adults treated as children using data from the 2010 census of Brazil and the same empirical strategy. When pooling racial groups, we find positive but imprecise increases in incomes. However, when we separate the sample into white and non-white adults, we find substantially larger effects for the latter (1.5-percent increase in incomes versus 0.2 percent). We examine educational attainment as a channel for these results but do not find evidence that it can explain a large share of the effect, suggesting that the effects of chronic Chagas Disease may play an important role in this reduction of racial disparities.

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

We find approximately 10-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 6-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.

Taken together, these results imply that controlling Chagas Disease had important benefits for a developing economy both in the short run (adult employment) 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.

2. Overview of Chagas Disease

2.1. *Causal Agent and Vectors*

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

2.2. *Phases*

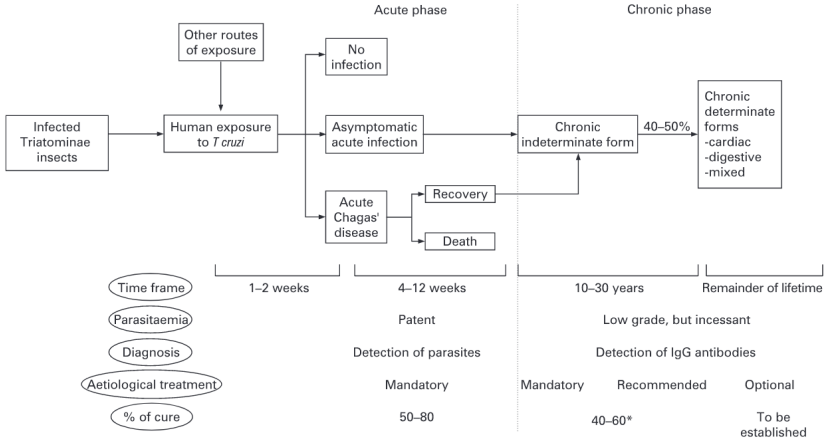
There are two phases of Chagas Disease. Figure 1 shows the progression of the disease from exposure through the rest of the patient's life. The acute stage begins after 1 to 2 weeks of incubation, lasts 4 to 12 weeks, and has non-specific symptoms like malaise and fever (Rassi et al., 2009). Children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

Individuals then enter the chronic phase. For 10 to 30 years, they experience no symptoms and the majority will remain in a chronic indeterminate stage in which they never develop any lesions (Rassi, Rassi and Little, 2000). However, the other portion progresses to the chronic determinate phase. Most commonly, this group develops cardiac complications such as the heart

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

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

Figure 1: Phases of Chagas Disease



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

muscle becoming degraded and being replaced by fibrous tissue. Such cardiomyopathy is the cause of most of the morbidity and mortality from Chagas Disease (Nunes et al., 2018).

2.3. Economic Consequences of Chagas Disease

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

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

Therefore, Chagas Disease should also have broader consequences for developing countries.

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

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

3. Brazil's Vector Control Program

Much of this section summarizes Section 4 of Schofield and Dias (1999).

3.1. Breakthrough of Pyrethroid Insecticides

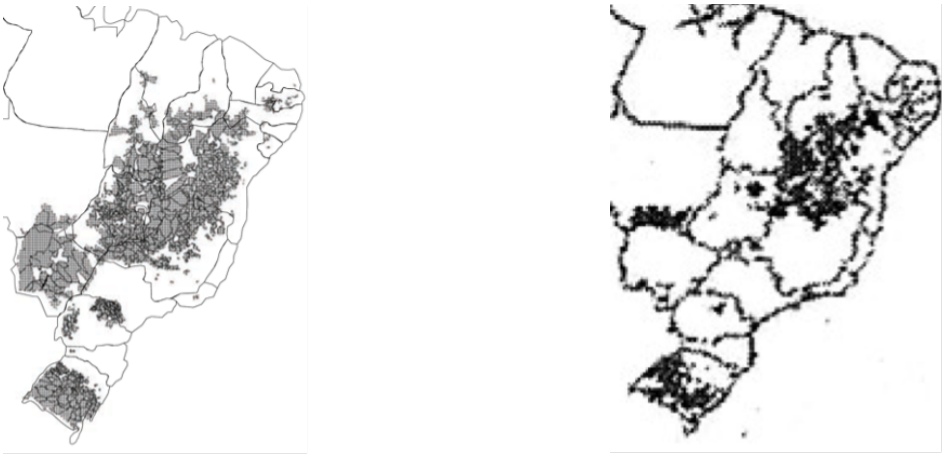
The post-World War II campaigns against malaria used organochlorine insecticides like DDT that were ineffective against Chagas Disease vectors. Several trials found that γ-benzene hexachloride (BHC) was effective if sprayed on the walls and roofs of triatomine-infested houses in high doses. In the 1960s,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

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

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

In 1975, the Brazilian government started a national campaign against Chagas Disease. It began because of, among other factors, the "development of suitable vector control methods, ... and continuous campaigning by scientists including demonstrations that vector control was feasible" (Dias, 1987, p. 338). The first stage consisted of serological and entomological surveys through

Figure 2: 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.

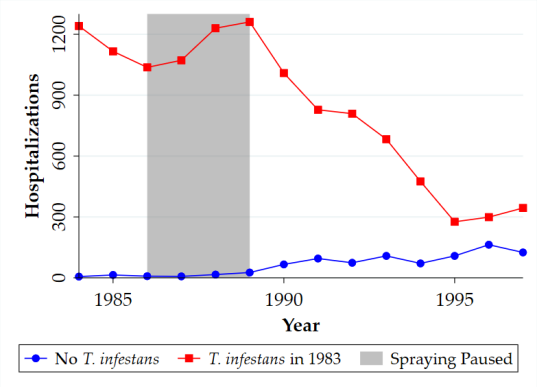
the early 1980s. They found a national rural *T. cruzi* prevalence rate of 4.2 percent—including 8.8 percent rural prevalence in the heavily-infested states of Minas Gerais and Rio Grande do Sul—and vectors present in 36 percent of Brazil's territory (Dias, 1987). Figure 2a shows a map of municipalities that had *T. infestans* in dwellings from 1975 to 1983.

After the surveys concluded, thousands of sprayers visited millions of homes across the endemic region. However, the program came to a halt in 1986 due to the arrival of the *Aedes aegypti* mosquito in coastal areas and resulting outbreaks of dengue fever. Due to political pressure, the public health campaign superintendency diverted 40 percent of its personnel to dengue control, leading to a reemergence of triatomine in recently-sprayed municipalities (Dias, 1987; Schofield and Dias, 1999). But vector control resumed in 1989, and the reduction in *T. infestans* in that year compared to 1975-83 is clear in Figure 2b.

To show the health impacts of (the interruption in) spraying, Figure 3 presents a graph of 1984-97 hospital admissions due to (acute) Chagas Disease.[7] They declined at the beginning of the campaign through 1986 and then increased with the interruption of control efforts lasting until 1989. Subsequently, admissions declined again as the program resumed, and by 2006, the Pan American Health Organization certified Brazil as having interrupted transmission of Chagas

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

Figure 3: Hospitalizations for Acute Chagas Disease, 1984-97



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

Disease through *T. infestans* in every state.

4. Data and Treatment Definition

4.1. Data

Given the discussion of the potential effects of Chagas Disease in Section 2.3, we focus on both the short- and long-run economic impacts of the health improvement induced by control of triatomine. To study the former, we use microdata from the National Household Sample Survey (PNAD) for 11 of the 18 years from 1982 to 1999.[8] 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.

We also use IPUMS microdata from the 1980 and 2010 censuses of Brazil (Minnesota Population Center, 2020) to examine the long-run effects of Chagas Disease vector control. These 25- and 10-percent samples include information on a respondent's consistent 1980-2010 municipality of residence and consistent 1960-2010 state of birth, as well as an indicator for whether a respondent was born in their municipality of residence. We use the 1980 data to construct our

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

measure of exposure to the treatment when only a respondent's state of residence or birth is known, as we describe in the next section. The census also contains more detailed household, sociodemographic, schooling, and labor market data than the PNAD datasets, which we exploit in the 2010 data.

4.2. Treatment Definition

We define control municipalities as those that were never treated with spraying—i.e., they did not have *T. infestans* in 1975-83 and did not require vector control to achieve this status.[9] Our treatment group consists of those with *T. infestans* present in 1975-83 but eliminated by 1989 as a result of the spraying that began in 1984.[10] We use these years as endpoints because it ensures that our 2010 census sample has birth cohorts that were had spent their entire childhoods living without *T. infestans*. Because we cannot determine in which year a municipality became free of the vector, we assume that all were treated in 1984 to be conservative.

Figure 4 shows control municipalities in blue, treatment ones in red, and those we exclude in white with either horizontal purple lines (not yet treated) or vertical brown ones (treated earlier). Table 1 shows summary statistics for demographic, labor market, and human capital variables among all individuals in these groups in 1980, 4 years before the start of spraying. Municipalities in our treatment group were more white and had slightly less schooling than those in our control group, though incomes were equal.

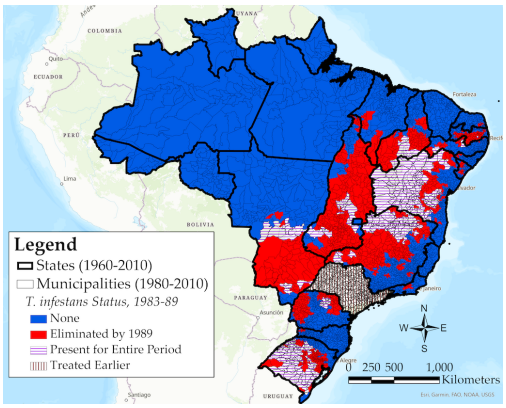
5. Short-Run Effects on Schooling and Labor Market Outcomes

We first 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. 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

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

[10] We also exclude not-yet-treated municipalities that had triatomine bugs in 1975-83 and 1989. Note that this group also includes municipalities where the vector to returned during the 1986-89 pause in spraying.

Figure 4: Municipalities by 1983-89 *T. infestans* Status



Notes: Map shows control municipalities (never had *T. infestans*) in blue and treatment municipalities (*T. infestans* eliminated by 1989) in red. Municipalities that still had *T. infestans* in 1989 (white with horizontal purple lines) or were treated prior to this period (white with vertical brown lines) are omitted from our sample. Data are from Silveira (2011) and Coura and Dias (2009) (see Figures 2a and 2b). The underlying shapefile of consistent 1980-2010 municipalities and 1960-2010 states is from Minnesota Population Center (2020).

Table 1: Summary Statistics, 1980

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

Notes: Table lists means for variables of interest in the 1980 census and standard deviations in parentheses under the means for continuous variables. Categories correspond to Figure 4. Data are from Minnesota Population Center (2020).

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.

5.1. 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. Our baseline estimating equation is the dynamic two-way fixed effects model

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

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.

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.[11] 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.

[11] The final entirely pre-treatment year was actually 1983, but there are no PNAD data available for that year. 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.

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.

Because nearly one-third of observations were from states with no treatment municipalities and the maximum probability of living in one was 0.740, the magnitude of these estimates overstates any policy-relevant parameter. Therefore, when discussing the results, we frame them in the context of moving from the 25th percentile of this probability to the 75th, which is an increase from 0 to 24.6 percentage points (p.p.)—i.e., this shift would have an impact of just under one-quarter of the estimated magnitudes.

Along with equation (1), we also estimate the static two-way fixed effects model

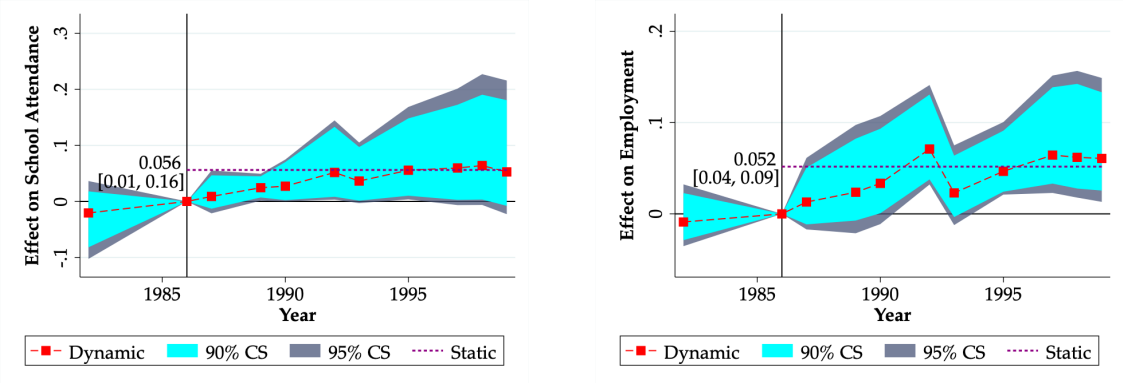
$$y_{i,s,t} = \alpha_s + \gamma_t + \tau \cdot \left(\mathbb{P}[Treat]_s \cdot \mathbb{1}[t > 1986]\right) + \mathbf{X}_i\beta + \epsilon_{i,s,t}, \tag{2}$$

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. By pooling the post-treatment years, the estimate of τ is likely to be more precise, though it comes at the cost of imposing a single value across all years in this period. We view these strategies as complementary because they both have an important drawback but each helps to address the concern regarding the other.

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.

Finally, to examine the potential for health improvements to reduce racial inequality in the short run, we estimate equations (1) and (2) separately for white and non-white individuals. Rather than conducting statistical tests for another difference in trends across racial groups (i.e.,

Figure 5: Short-Run Effects on School Attendance and Adult Employment



(a) Attending School, Ages 8-18 **(b)** Employed, Ages 35-50

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) 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 836,331 observations for children and 1,198,480 observations for adults. In pre-treatment years, 74.7 percent of children attended school and 67.0 percent of adults were employed.

a triple-differences framework) given the potential for imprecision, we simply compare the respective estimates' magnitudes patterns and discuss implications.

5.2. *School Attendance*

We first examine the effects of Chagas Disease on school attendance in the years around the start of vector control. Figure 5a plots the estimates from equations (1) and (2) for school attendance among children ages 8 to 18. The patterns in the dynamic estimates suggest a positive but imprecise impact of reducing acute Chagas Disease exposure on attendance. However, it is important to note that they likely underestimate the true magnitudes given the opposite signs of the 1982 coefficient and the treatment effect.

The static strategy recovers a coefficient of similar magnitude to the later years' dynamic estimates but with greater precision. The implied effect of moving from a 0 to one-quarter probability of living in a treatment municipality is 1.4 p.p. (1.9 percent of the pre-treatment mean) and it is significant at 10 percent. This estimate is likely closer to the true value than the dynamic estimates because it is calculated against the average of the 1982 and 1986 differences.

5.3. *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 5b. 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 1.3 p.p. (1.9 percent), which equals the magnitude of the implied static estimate.

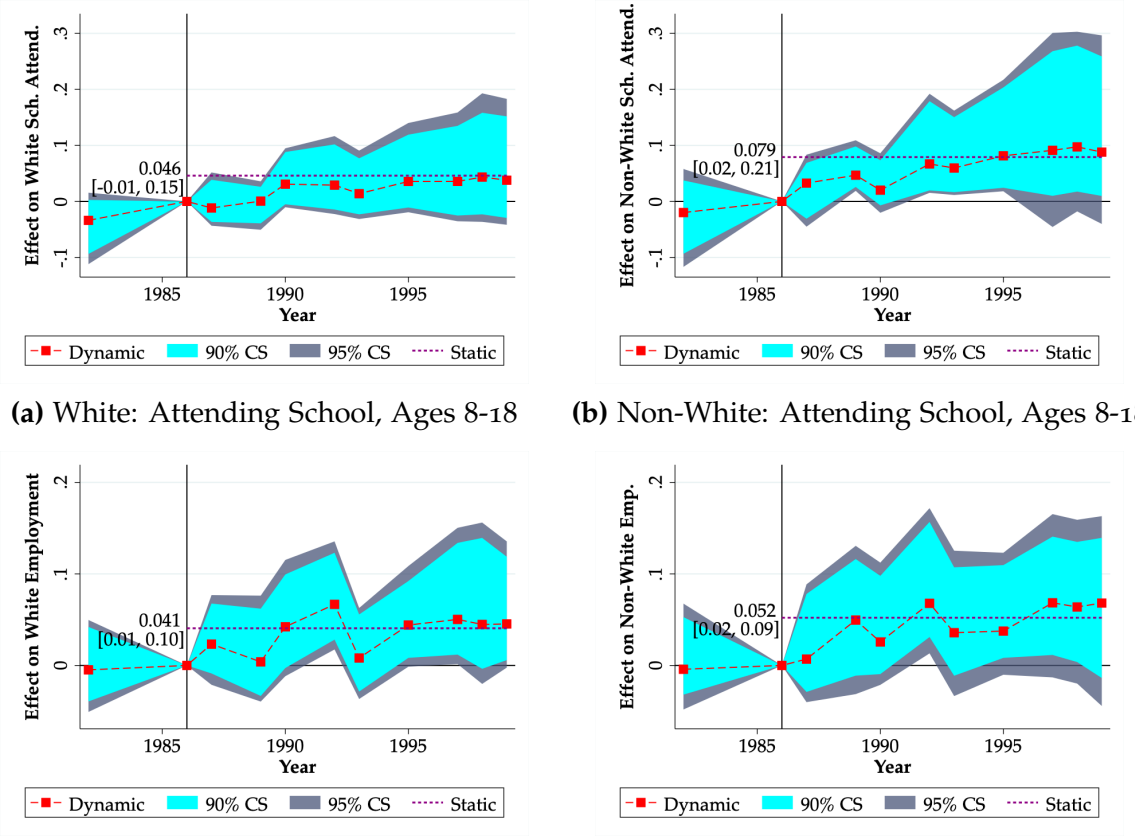
This result is particularly important because it shows that controlling Chagas Disease has relatively rapid effects on *adults already in the labor force*. While increasing the human capital of children makes large contributions to economic development, doing so yields benefits that are only realized 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.

5.4. *Reducing Racial Disparities in the Short Run*

Finally, we examine evidence for differential effects by race, which is closely linked with poverty in Brazil. Figures 6a and 6b suggest 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. In particular, the implied static magnitude for the former group was 1.1 p.p. (1.5 percent) whereas for the latter it was 1.9 p.p. (2.6 percent) and more precisely estimated. However, because the 1982 estimate for white children was more negative than for non-white children, the apparent racial difference may simply be an artifact of greater bias toward zero in the white coefficients.

Figures 6c and 6d also show that employment increased for both groups but somewhat more for non-white adults: the implied white static estimate was 1.0 p.p. (1.5 percent) while the implied non-white one was 1.3 p.p. (1.9 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 rel-

Figure 6: Differential Short-Run Effects by Race



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

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

(c) White: Employed, Ages 35-50

(d) 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 389,414 observations for white and 446,017 observations for non-white children, and 648,336 observations for white and 550,144 observations for non-white adults. In the pre-treatment year, 77.7 percent of white and 74.1 percent of non-white children attended school, and 67.2 percent of white and 66.9 percent of non-white adults were employed.

atively 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.

6. Long-Run Effects on Labor Market Outcomes

In this section, we examine the long-run effects of triatomine control on adults who were children around the time that spraying began. Our results show that spraying raised incomes and employment rates more among adults who were more exposed to it in childhood. In examining channels, we find small and imprecise increases in years of schooling, suggesting that this effect does not (primarily) arise through increased educational attainment—a more likely explanation is the direct effect of avoiding the phases of Chagas Disease morbidity. However, these results were markedly different by race: incomes and employment rates rose substantially more for non-white adults despite equivalent (and imprecise) increases in years of schooling. The implication is that vector control increased the speed of racial convergence in Brazil in the long run.

6.1. Empirical Strategy

Similar to our approach in the previous section, we compare adults of interest in the 2010 census across municipalities of birth with varying levels of pre-treatment *T. infestans* presence. For dynamic effects, we modify equation (1) to be

$$y_{i,m,c} = \alpha_m + \gamma_c + \sum_{k \neq 1965} \tau_k \cdot \left(\mathbb{P}[Treat]_m \cdot \mathbb{1}[c = k] \right) + \mathbf{X}_i \beta + \epsilon_{i,m,c}, \quad \text{for } k \in \{1960, \ldots, 1979\} \tag{3}$$

where the differences are in the subscripts and the omitted period.[12] First, our variables are now at the level of municipality of birth m because of the greater geographic detail in the census data. Nearly two-thirds of the sample live in their municipality of birth, so for these adults $\mathbb{P}[Treat]_m$ is either 0 (55 percent of the sample) or 1 (11 percent). For those living away from their birth municipality, we assign them the probability in the 1980 census that an individual of their sex and race who was living in their state of birth was in a treatment municipality.

Our focus also shifts to birth cohort c. We use 1965 as the omitted cohort, as these adults were 18 years old in 1983. The oldest group we include is the 1960 birth cohort (age 50 in 2010) for 2 reasons. At the time of this census, workers could retire with social security benefits after

[12] We make analogous changes to equation (2) to estimate the static effect.

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

Our assumption is that the 1960 to 1965 cohorts were too old to have experienced the benefits of Chagas Disease control during their childhood years. Given the results in the previous section, it is important to note that these benefits could have come from both them *as well as their parents* averting the acute phase—the latter may have implied more available resources as a child. On the other hand, these too-old cohorts still may have benefitted from not contracting Chagas Disease in young adulthood, thus avoiding its acute phase in the short run and its chronic determinate phase in the long run. The implication is that null results using this strategy could arise from either the absence of long-run effects from controlling this disease during childhood or the greater importance of its chronic phase for adult outcomes.[13]

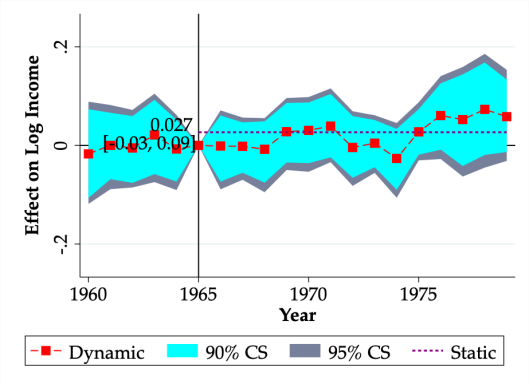
The comparisons we make in this section are very similar to those made previously. The τ_k measure the difference in an outcome for a given birth cohort as the probability of having been born in a treatment municipality goes from 0 to 1, relative to the size of that difference for the 1983 cohort. Because the median value is 0 while the 75th percentile remained just under a quarter, we frame our results as an increase in this probability from 0 to 24.7 p.p. and discuss the implied magnitude of the estimate in the text. For inference, we continue to calculate wild cluster bootstrap confidence sets because we cluster standard errors at the state level.

6.2. Labor Market Outcomes

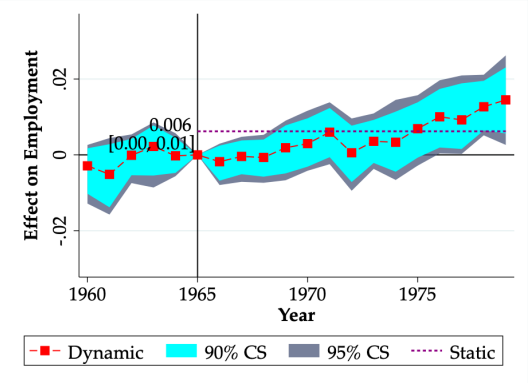
Our first outcome of interest is (the natural log of) monthly income for the 1960 to 1979 birth cohorts in 2010. Figure 7a shows that while incomes for the adults who turned 18 before the start of spraying evolved in parallel, those who were children during triatomine control experienced slightly greater but imprecise increases the more they were exposed to treatment. The static

[13] It also means our long-run results have a somewhat different interpretation from those for childhood exposure to malaria (e.g., Bleakley, 2010*b*; Lucas, 2010; Cutler et al., 2010), which primarily affects children.

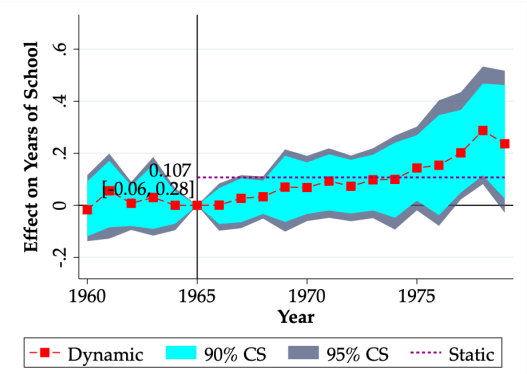
Figure 7: Long-Run Effects on Labor Market Outcomes and Educational Attainment



(a) Log Monthly Income



(b) Employed



(c) Years of Schooling

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) 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 IPUMS sample of the 2010 census (Minnesota Population Center, 2020). Regressions control for state and year fixed effects, female sex, and racial category (Asian, Black, Brown, and Indigenous). Standard errors are clustered by the 24 consistent 1960-2010 states. Regressions use 3,872,397 observations for (a), 4,075,538 for (b), and 5,299,001 for (c). For pre-treatment cohorts, mean log monthly income was 6.242, 91.2 percent of those in the labor force were employed, and mean years of schooling was 6.02.

estimate implied for moving from the median to the 75th-percentile probability of having been born in a treatment municipality is of incomes increasing by 0.7 percent more for treated cohorts.

We then turn to studying the employment status of members of these birth cohorts who were in the labor force. After verifying in Appendix A1 that vector control did not differentially affect labor force participation, Figure 7b shows no evidence of differential trends prior to treatment but divergent employment rates after spraying started. The implied static estimate is of an additional 0.1-p.p. increase in employment for treated cohorts. Whether this estimate is of a large relative

magnitude depends on how this binary variable is framed—it is 0.2 percent of the employment rate of adults in the labor force from the 1960-65 birth cohorts, which is alternatively 1.7 percent of this group's unemployment rate. Either way, these results provide some evidence that greater exposure to Chagas Disease control in childhood leads to better adult labor market outcomes.

6.3. Assessing Channels: Schooling

Next, we examine the role of schooling in the explanation for our labor market results, especially considering the small and imprecise attendance results in Section 5.2. Figure 7c shows the absence of pre-treatment differential trends followed by imprecise increases in post-treatment cohorts' completed years. The implied static estimate is of an additional 0.03-year (0.4-percent) increase in years of schooling for cohorts exposed to vector control as children, though it is noisy.

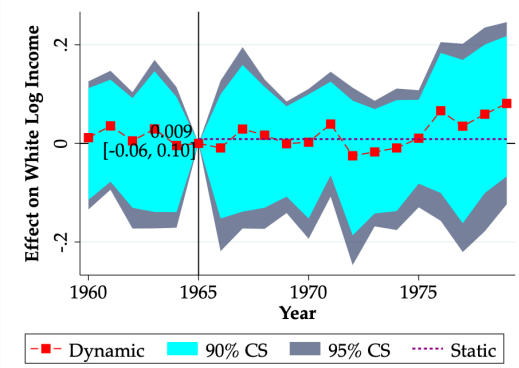
6.4. Reducing Racial Disparities in the Long Run

The imprecision in these results suggests there may be important heterogeneity by race masked by pooling white and non-white Brazilians. Therefore, we study the long-run contributions of Chagas Disease control to reducing racial disparities by examining these outcomes separately for the two groups. Figures 8a and 8b support our hypothesis of differential effects: while there were small, if any, increases in white incomes for post-treatment cohorts (implied static estimate of 0.2 percent), non-white incomes rose substantially more (implied static estimate of 1.5 percent). In addition, this estimate and the dynamic ones were precise, and the latter imply that non-white cohorts with only a few years of treatment exposure had substantively larger income increases.

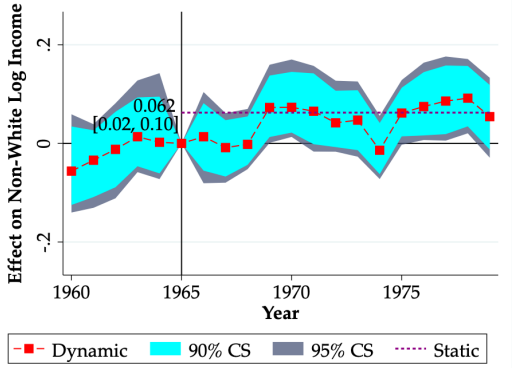
There are also somewhat different effects on employment in adulthood. After verifying that vector control did not have differential outcomes on either racial group's labor force participation in Appendix A2, we show increases in the implied static estimates of 0.1 p.p. for white rates and for 0.2 p.p for non-white ones in Figures 8c and 8d. Depending on the framing, these magnitudes are 0.1 percent and 0.3 percent of the respective pre-treatment cohorts' employment rates or 1.5 percent and 2.0 percent of their respective unemployment rates.

The results for years of schooling are more ambiguous. Figures 8e and 8f show a larger but imprecise increase in absolute terms for white cohorts (implied static estimate of 0.04 years) than

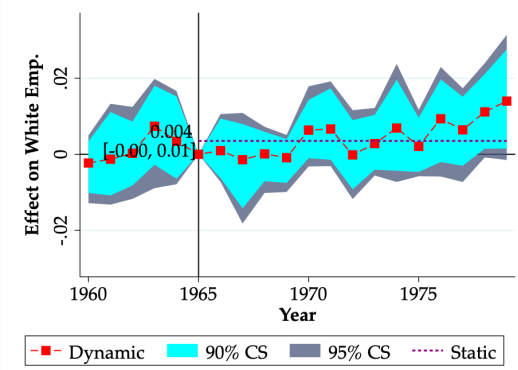
Figure 8: Differential Long-Run Effects by Race



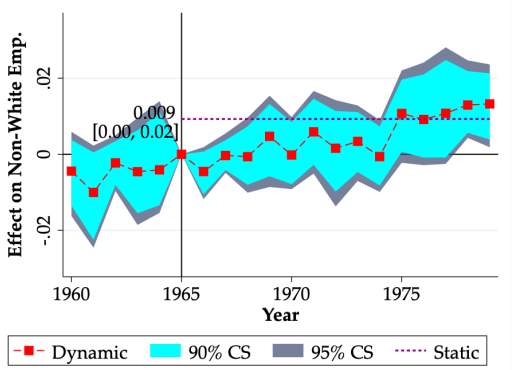
(a) White: Log Monthly Income



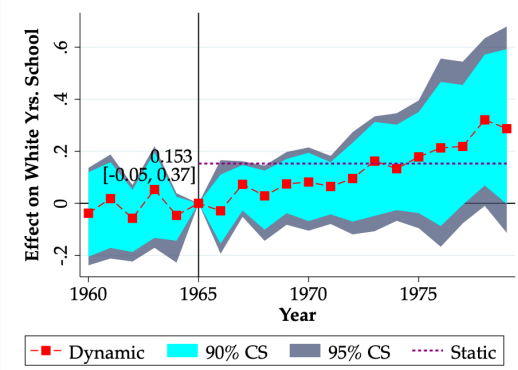
(b) Non-White: Log Monthly Income



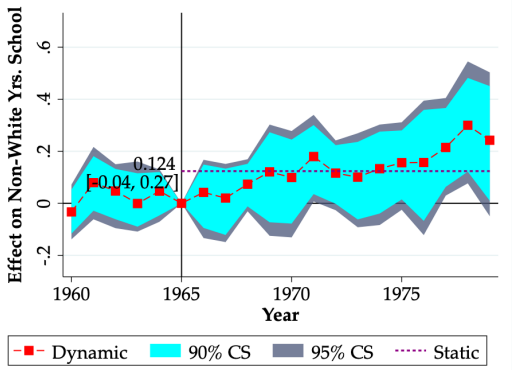
(c) White: Employed



(d) Non-White: Employed



(e) White: Years of Schooling



(f) Non-White: Years of Schooling

Notes: Graphs plot dynamic (red squares) and static (dotted purple line) two-way fixed effects estimates for the respective outcomes and racial groups with 90-percent (light blue) and 95-percent (dark blue) 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 IPUMS sample of the 2010 census (Minnesota Population Center, 2020). Regressions control for fixed effects for state, year, female sex, and racial category (Asian, Black, and Brown) as well as age and age squared. Standard errors are clustered by the 24 consistent 1960-2010 states. Regressions use the following number of observations: (a) 1,964,673, (b) 1,907,724, (c) 2,044,592, (d) 2,030,946, (e) 2,568,352, (f) 2,730,649. For pre-treatment cohorts, means of the respective outcomes were: (a) 6.58, (b) 5.86, (c) 93.6 percent, (d) 88.5 percent, (e) 6.91 years, (f) 5.10 years.

non-white ones (0.03 years), but relative to the means for pre-treatment cohorts, the non-white effect is slightly larger (0.5 percent for white cohorts vs 0.6 percent for non-white ones). This absence of substantive differences is consistent with the interpretation of the estimates in Section 5.2: namely, after adjusting for the larger downward bias in the white attendance coefficients, there may not have been any differential effects by racial group.

6.5. Assessing Channels for Reducing Racial Disparities

Finally, to understand the importance of the schooling channel for the non-white income result, we make a back-of-the-envelope calculation of the upper bound of its contribution. Using the Mincerian return to schooling in Brazil of 15.7 percent reported in Psacharopoulos and Patrinos (2018)—which almost certainly overstates the causal effect of schooling on income—the additional schooling for non-white cohorts induced by vector control can account for at most 0.5 p.p. (one-third) of the 1.5-percent increase in their incomes. It suggests that avoiding the cardiovascular problems arising from chronic Chagas Disease in adulthood played an important role in explaining differentially larger income increases for non-white Brazilians.

7. Long-Run Effects on Public Health Care and Spending

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

Therefore, in this section we examine the long-run effects of triatomine control on circulatory system-related hospitalizations covered by SUS and the resulting spending. Using a triple-

differences strategy comparing circulatory and non-circulatory system-related causes, we show that hospitalizations and spending resulting from the former decreased more in states more exposed to treatment. The implication is that controlling Chagas Disease transmission has yielded substantial benefits for the public health care system and public finances in Brazil.

7.1. Data and Empirical Strategy

Our outcomes of interest are (the natural logs of) hospitalizations, person-days spent in the hospital, and spending on hospital care in each state by cause from 1984 to 2019. These data are from the SUS's Hospital Information System (SIH/SUS), and we deflate the last of them so that figures are in constant (log) 2019 Brazilian *reais* (BRL). Given that chronic Chagas Disease manifests primarily as cardiovascular problems, we focus on all diseases of the circulatory system.[14] We combine these data with each state's 1980 share of its population living in treatment municipalities to create a state-level treatment measure as in Section 5.

However, there is an additional dimension to our empirical approach that was not necessary in previous sections. The SUS is a heavily decentralized system with transfers of responsibilities and funds to state and municipalities (Castro et al., 2018). Therefore, there likely are confounders varying across both state and year (e.g., public health priorities, non-hospital care spending) in violation of the common trends assumption. In Appendix A3, we verify that the difference-in-differences estimates for these outcomes show that they did not evolve in parallel prior to the omitted year.

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

$$y_{s,t,d} = \alpha_{s,t} + \gamma_{t,d} + \delta_{s,d} + \sum_{k \neq 1999} \tau_k \cdot \left(\mathbb{P}[Treat]_s \cdot \mathbb{1}[t = k] \cdot \mathbb{1}[d = circ] \right) + \epsilon_{s,t,d}, \tag{4}$$

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

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

This strategy compares first compares the differences in log outcomes due to circulatory diseases in a given year as the probability of a state's population living in a treatment municipality goes from 0 to 1, relative to the size of that difference in 1999 (see below). Then it compares this double-difference estimate to the analogous one for non-circulatory diseases. Because the 25th percentile of this probability was 0 and the 75th percentile was 26.3 p.p., we frame our results as moving between these two values. For inference, we compute wild cluster bootstrap confidence sets after clustering standard errors by the "small" number of states.

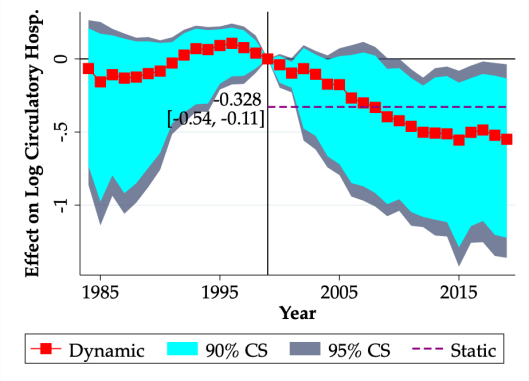
We set 1999 as the reference year because it is 10 years after all treatment municipalities became *T. infestans*-free, and it takes at least 10 years for chronic Chagas Disease to manifest (see Section 2). Our hypothesis is that differences within states between circulatory and non-circulatory outcomes should have begun to emerge at or around that point, yielding larger decreases in the former. We also measure this decrease in a static context using a version of equation (4) analogous to equation (2).

7.2. Circulatory Disease Hospitalizations and Hospital Stay Length

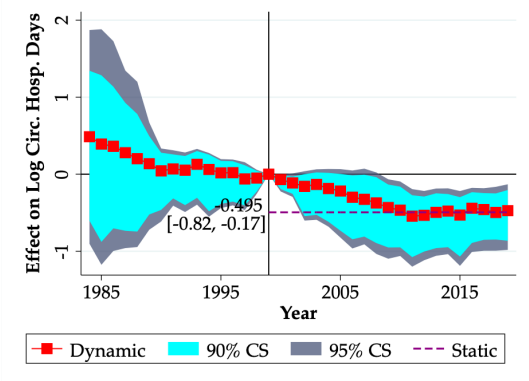
Consistent with our hypothesis, Figure 9a shows that the double-difference estimates for log hospitalizations evolved in parallel across disease categories prior to 1999 and then subsequently diverged. In the late 2000s and beyond, the dynamic coefficients implied additional decreases in hospitalizations of at least 8 percent, each of which was significant at the 5-percent level. The economic significance of the implied static coefficient (8.6 percent) as well as its precision suggests that controlling Chagas Disease transmission had a substantial impact on Brazilian health care.

The patterns in log person-days spent in the hospital are highly similar: after pre-1999 trends evolved for the most part in parallel in Figure 9b, time spent in the hospital due to circulatory diseases decreased more. By the late 2000s, these differences stabilized at an implied magnitude of around 13 percent and achieved 5-percent significance, as did the static coefficient (implied estimate of 13.0 percent). While the latter may be overstated given the positive but noisy coefficients

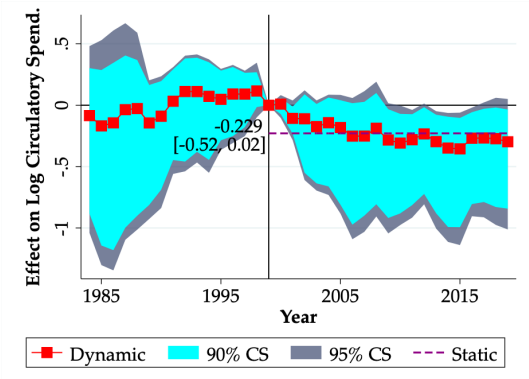
Figure 9: Long-Run Effects on Circulatory Disease Hospital Care



(a) Log Circulatory Disease Hospitalizations



(b) Log Circulatory Disease Hospital Days



(c) Log Circulatory Disease Spending

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

at the start of the sample, these results nonetheless suggest that Brazilians using SUS-provided health care spent less time in hospitals due to circulatory problems right when we expected.

7.3. Circulatory Disease Hospital Spending

Because these declines should have translated into a drop in SUS outlays on hospital care, we expect to find a similar pattern in the spending results. Figure 9c is consistent with this prediction

but there is more noise in these estimates. Following pre-1999 coefficients of small magnitudes, the dynamic estimates become consistently more negative and achieve 5-percent significance in the 2010s, although those for last few years of the sample are less precise. But the implied static estimate of an additional 6.0-percent decrease in hospital spending (just outside of 10-percent significance) suggests that SUS has benefited—and continues to benefit—from substantial savings on circulatory disease hospital care spending resulting from triatomine control that occurred more than 3 decades ago.

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.

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 eventual educational attainment 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. 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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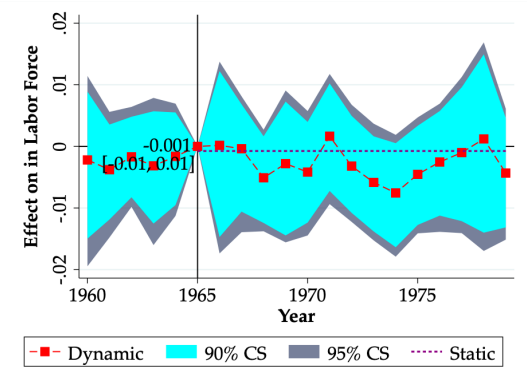
Appendix A. Additional Results

A1. Long-Run Effects on Labor Force Participation

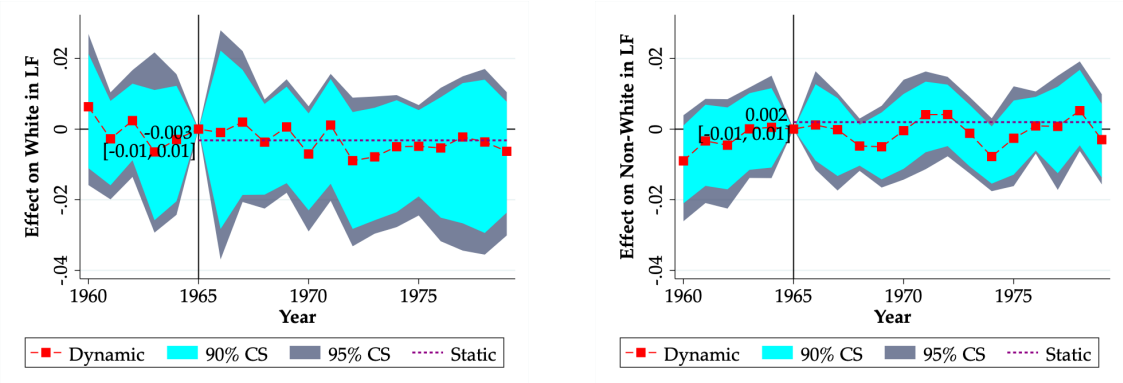
Figure A1: Long-Run Effects on Labor Force Participation [17]



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) 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 IPUMS sample of the 2010 census (Minnesota Population Center, 2020). Regressions control for state and year fixed effects, female sex, and racial category (Asian, Black, Brown, and Indigenous). Standard errors are clustered by the 24 consistent 1960-2010 states. Regression uses 4,455,390 observations. For pre-treatment cohorts, the labor force participation rate was 73.3 percent.

A2. Long-Run Effects on Labor Force Participation by Race

Figure A2: Long-Run Effects on Labor Force Participation by Race [19]

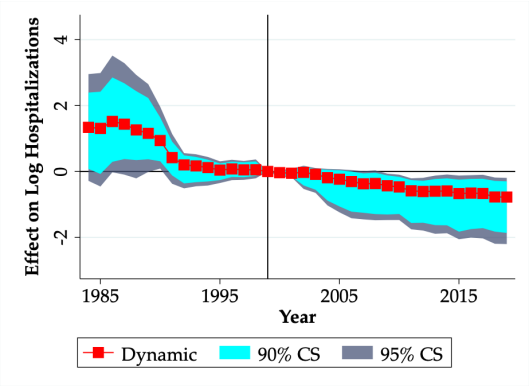


(a) White **(b)** Non-White

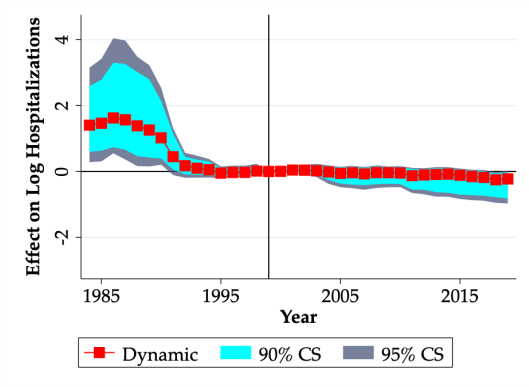
Notes: Graphs plot dynamic (red squares) and static (dotted purple line) two-way fixed effects estimates for the respective outcomes with 90-percent (light blue) and 95-percent (dark blue) wild cluster bootstrap confidence sets for dynamic estimates. Static estimate magnitudes are next to the dotted purple lines with 90-percent wild cluster bootstrap confidence sets below them in brackets. Data are from the IPUMS sample of the 2010 census (Minnesota Population Center, 2020). Regressions control for state and year fixed effects, female sex, and racial category (Asian, Black, Brown, and Indigenous). Standard errors are clustered by the 24 consistent 1960-2010 states. Regressions use 1,983,837 observations for (a) and 2,471,553 observations in (b). For pre-treatment cohorts, labor force participation rates were 76.3 for white adults and 70.6 for non-white adults.

A3. Difference-in-Differences Estimates for Hospital Care Outcomes

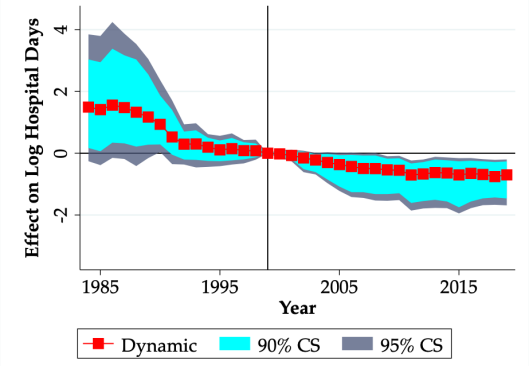
Figure A3: Difference-in-Differences by Disease Category: Hospital Care Outcomes [22]



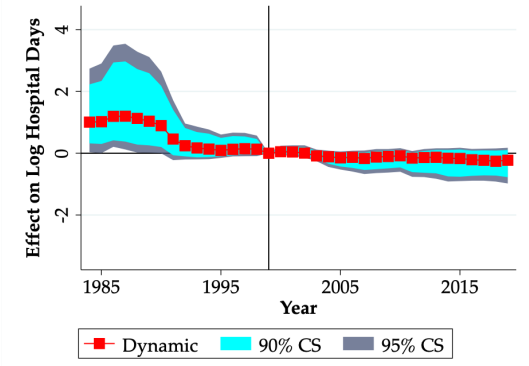
(a) Circulatory Disease: Log Hospitalizations



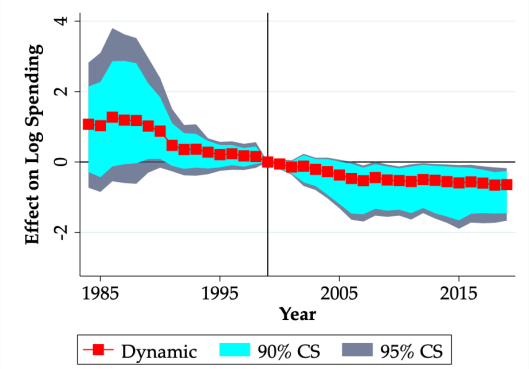
(b) Non-Circ. Disease: Log Hospitalizations



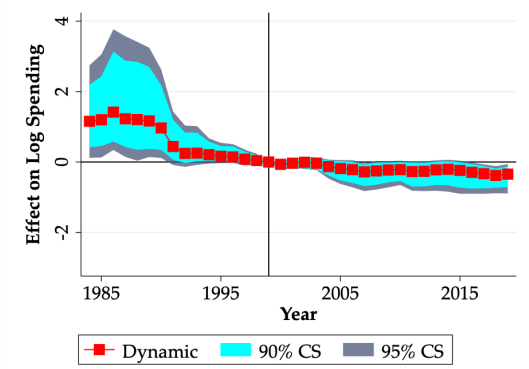
(c) Circulatory Disease: Log Hospital Days



(d) Non-Circ. Disease: Log Hospital Days



(e) Circulatory Disease: Log Spending



(f) Non-Circ. Disease: Log Spending

Notes: Graphs plot dynamic (red squares) and static (dotted purple line) difference-in-difference estimates for the respective outcomes with 90-percent (light blue) and 95-percent (dark blue) wild cluster bootstrap confidence sets. Data are from DATASUS. Regressions control for state and year fixed effects. Standard errors are clustered by the 24 consistent 1960-2010 states. Regressions use 756 state-year observations. See the notes for Figure 9 for pre-treatment means for the respective outcomes and disease categories.