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

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Abstract

In Latin America—the world's most unequal region—non-white rural populations disproportionately suffer from Chagas disease, a neglected tropical disease (NTD) that causes weeks of acute symptoms and can lead to chronic heart problems decades later. We demonstrate that Brazil's post-1983 campaign to eliminate the transmission of this disease significantly reduced (racial) income inequality, the intergenerational transmission of low human capital, and burdens on the world's largest government-run health care system. Exploiting the pre-treatment presence of Chagas disease's main vector, we find that controlling this NTD increased municipalities' GDP per capita by 11.1% and reduced their Gini coefficients by 1.1% in the long run. Furthermore, averting childhood exposure to Chagas disease increased the share of non-white adults with above-median incomes by 1.4 percentage points (p.p., or 2.8%) and their children's literacy rates by 0.4 p.p. (0.5%). Coinciding with the expected reduction in chronic heart problems, we also find that public spending on circulatory disease hospital care declined by 16%, contributing to a 24% internal rate of return and an infinite marginal value of public funds. These results suggest that NTD control can improve the economic and fiscal health of developing countries while mitigating (racial) disparities and intergenerational cycles of poverty.

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[A] tragedy that makes no sound, patients that [cannot] pay, [an] illness that will not sell. 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)

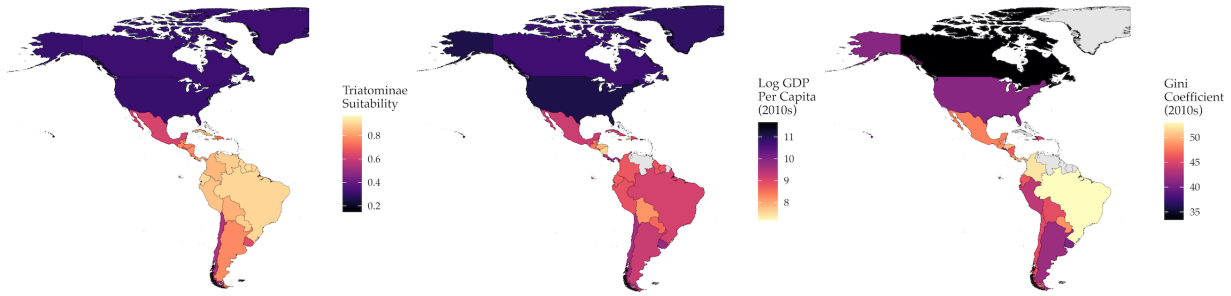
1. Introduction

Latin America is one of the most unequal regions in the world, with the richest 10% capturing 54% of national incomes (Chancel et al., 2021; De Rosa, Flores and Morgan, 2020). Race is an important element of these inequalities, as white individuals earn at least twice as much as those with darker skin tones (Telles et al., 2023; Woo-Mora, 2024). A potential contributor to this middle-income region's disparities and underdevelopment is Chagas disease, which afflicts 8 million predominantly non-white people in Latin America while putting an additional 75 million at risk of infection (Briceño-León and Méndez Galván, 2007; Franco-Paredes et al., 2007; Santos et al., 2020). Caused by a parasite found only in the Western Hemisphere, it is a "neglected disease of poor, rural, and forgotten populations" because its vectors—triatomine bugs—much more easily infest housing made of substandard materials (Coura and Viñas, 2010; Houweling et al., 2016).[1] In both children and adults, Chagas disease can lead to weeks of non-specific acute symptoms (e.g., fever, fatigue, and headaches) that may reoccur with reinfection. More importantly, between 10 and 30 years later, a substantial share of those (re)infected, including "young adults[,] develop heart conditions, so that they fill hospital beds instead of the [labor] force" (World Health Organization, 2010, p. iv). As such, this disease may be both a cause and a consequence of poverty, potentially contributing to the intergenerational transmission of low socioeconomic status.

In this paper, we study whether controlling what Delaporte (2012) described as "a continent's scourge" can contribute to its equitable growth, using a difference-in-differences approach exploiting the 1984 start of Brazil's campaign to eliminate vectorial transmission of Chagas disease. Data from across the Western Hemisphere suggest it could have had a substantive impact in this regard: the map of a proxy for transmission of the illness (the Eberhard et al., 2020, modern suitability index for its vectors) in Figure 1a is similar to the maps of GDP per capita (Figure

[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).

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



(a) 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 and the right panel shows the most recently reported Gini coefficient (scaled by 100) between 2010 and 2019, both from the World Bank.

1b) and Gini coefficients (Figure 1c) today.[2]

Despite its presence only in the Americas, Chagas disease shares an important trait with many other neglected tropical diseases (NTDs) that afflict the poorest billion people in the world: most of their burdens arise from the chronic health problems that they cause (Hotez, 2011). In the case of Chagas disease, the decades between initial infection and the manifestation of chronic symptoms make its effects very challenging to study without taking a long-run perspective. We can therefore gain important insights into the impacts of controlling NTDs on inequality and development by exploiting decades-old quasi-experimental variation in the exposure to Chagas disease. Specifically, we study the consequences of indoor residual spraying (IRS) against the main Chagas disease vector that began in 1984 in Brazil—a country that Figure 1 shows has high vector suitability, a middle income, and very high inequality—which was made possible by the invention of a new insecticide and a 1975-83 nationwide entomological survey measuring the vector's presence at the municipal level.

Our basic difference-in-differences strategy combines cross-municipality and cross-year variation in exposure to the main Chagas disease vector. For the former, our focus is on comparing

[2] In Appendix A1, we formally test these relationships and confirm that the ecological suitability for Chagas disease is strongly associated with lower GDP per capita and greater income inequality within the Western Hemisphere. This mirrors the continent-specific relationship documented by Alsan (2015) for African trypanosomiasis (sleeping sickness). Chagas disease is known as American trypanosomiasis, but only because it is also caused by parasites of the genus *Trypanosoma*. There is no overlap in these species, their vectors, or the symptoms they cause.

municipalities never infested by the vector against those in which the vector was present before IRS began. Following recent developments in the difference-in-differences literature (Goodman-Bacon, 2021), we exclude municipalities in the state of Sao Paulo, which was wealthy enough to have conducted its own vector control in the 1960s with a more expensive insecticide. With respect to time variation, we lack information on when each municipality was treated after the program started in 1984, so we make the conservative assumption that it occurred uniformly in 1984. As such, we do not exploit a staggered rollout of treatment for identification.[3]

We first study the long-run impacts of eliminating Chagas disease transmission on income and inequality across municipalities, comparing our treatment and control groups using data on GDP per capita and from population censuses between 1970 and 2010. We find that after 1984, treatment municipalities experienced larger increases in their output per person (11.1%) and greater decreases in their income Gini coefficients (1.1%). Importantly, these effects arose in the first post-treatment years and grew more pronounced over time, suggesting that reducing chronic Chagas disease augmented the benefits of averting its acute symptoms. When examining mechanisms, we find that these changes likely arose from a greater share of treatment municipalities' populations earning incomes above the nationwide median in each census sample: 1.0 percentage points (p.p., or 2.1%) among adults aged 20 to 29 and 0.4 p.p. (0.8%) among those 30 to 50 years old. Our results are consistent with increases in completed schooling among younger adults (0.14 years, or 3.6%) contributing to their income gains but not to those of older adults. Instead, to paraphrase the World Health Organization (2010) quote above, we find suggestive evidence that over time, older individuals were filling the labor force instead of hospital beds, in line with long-run reductions in chronic symptoms of Chagas disease.

We complement the municipality-level results by examining the long-run impacts on those who were children when Chagas disease transmission was eliminated. Using data from the 2010 census, we find clear evidence of reductions in racial inequality: the share of non-white Brazilians from treatment municipalities earning above the median income in the census sample rose 1.4 p.p. (2.8%), but there was no change among white adults. There was also an identical

[3] Such an approach has recently been shown to have important defects (see, e.g., Callaway and Sant'Anna, 2021; de Chaisemartin and D'Haultfœuille, 2020; Sun and Abraham, 2021).

increase in the non-white share earning strictly positive incomes (1.4 p.p., or 1.8%), implying that treatment benefited the poorest non-white Brazilians.[4] We also show that the literacy of children of non-white men from treatment municipalities increased by 0.4 p.p. (0.5%), suggesting that Chagas disease control could contribute to breaking intergenerational cycles of (racial) poverty. Consistent with our municipality-level results, we find that these results cannot be attributed solely to increases in completed schooling. Rather, non-white labor force participation increased by 0.9 p.p. (1.3%), and a mediation analysis suggests that this manifestation of reduced chronic Chagas disease symptoms contributed more to the increase in incomes than the result of averted acute stages during childhood (i.e., more schooling).

If the reduction in chronic morbidity due to Chagas disease contributed to the observed increase in non-white incomes, it may also have resulted in substantial savings for Brazil's universal health care system (known as SUS), as circulatory diseases account for 10% of the hospitalizations it pays for and 20% of its spending on hospital care. We therefore examine hospitalizations covered by SUS and its spending on hospital care 10 years after IRS began (given the lag between infection and the manifestation of chronic symptoms). To do so, we add a third layer to our differences-in-differences strategy by also comparing outcomes due to heart-related causes against all others. We show that from 1995 to 2019, there were greater reductions in hospitalizations (19%) and spending on hospital care (16%) resulting from circulatory system problems compared to all other causes. We do not find clear evidence of a greater decline in deaths, which we view as something of a placebo test because mortality should only occur many years after the development of substantial morbidity from chronic Chagas disease.

We conduct two simple cost-benefit analyses comparing the costs of IRS against the main Chagas disease vector against the increases in income and decreases in hospital care spending. The first is calculating the internal rate of return (IRR), or the discount rate required for the net present value to be exactly zero. Excluding any benefits that we do not directly measure—most importantly, the willingness to pay for better health—the IRR is 23.9%. A key lesson from this

[4] We use these income indicators rather than a log transformation following the recommendations of Chen and Roth (2024). First, the problems they identify are especially important when the extensive margin effect is large, as is the case in our results. In addition, these indicators are more informative about the income distribution than an average effect.

exercise is that including the hospital care savings increases the IRR by 2.3 p.p. (around 10%), implying that returns to controlling the other NTDs that cause chronic health problems may also be understated. Next, we calculate the marginal value of public funds (MVPF), or the benefits divided by the net cost to the government. Because programs that pay for themselves have infinite MVPFs, we estimate that Brazil's intervention belongs in this category because the hospital care savings discounted at 5% outweigh the costs of IRS.[5] As such, another important lesson is that if developing countries are considering interventions to improve the health of their poorest citizens but do not expect to collect taxes from them (e.g., due to high informality or incomes below a minimum level), they can still recoup their investments in controlling NTDs that cause chronic health problems because doing so will relieve future health care burdens.

Lastly, we conduct an extrapolation exercise to shed light on the potential impacts of eliminating Chagas disease transmission on disparities and underdevelopment across Latin America. We multiply each country's GDP per capita and Gini coefficient by the share of its population exposed to transmission (World Health Organization, 2015) and the percent effects on these outcomes for Brazilian municipalities. Our calculations suggest that on average, this intervention could increase Latin American incomes per person by 1.5% and reduce Gini coefficients by 0.2%. Notably, greater benefits would accrue to the countries with higher shares exposed to Chagas disease, which are precisely those that are more underdeveloped and unequal today.

Our results therefore contribute to several literatures. First, our study adds to our understanding of the comparative development of Latin America. A growing body of work has sought to understand the region's disappointing growth and pronounced inequality (see Eslava and Valencia Caicedo, 2023; Telles et al., 2023; Attanasio et al., 2024). By studying Chagas disease, a malady deeply entrenched in the socioeconomic fabric of Latin America, we offer a novel perspective on the region's unique development trajectory.[6] We also provide quasi-experimental evidence that eliminating Chagas disease transmission can have important long-run benefits such

[5] It would take a discount rate of 9.1% for there to be a net cost to the government, which is well above the standard 5% rate for developing countries (Haacker, Hallett and Atun, 2020).

[6] In Appendix A2, we provide evidence that Chagas disease has deep historical roots in the region's development trajectory. Specifically, a greater recorded presence of the parasite in precolonial Latin America is associated with lower levels of centralization in the Standard Cross-Cultural Survey (Murdock and White, 1969).

as higher rates of inclusive growth. Nonetheless, our results also have relevance beyond Latin America: as climate change and increased migration bring the parasite and its vectors into non-endemic areas in the United States and Europe (Eberhard et al., 2020; Hernández, 2021; Irish et al., 2022), it becomes ever more important to understand the consequences of this increasingly globalized health challenge.

Second, we contribute to a growing body of research in economics on tropical disease and long-run development (e.g., Miguel and Kremer, 2004; Bleakley, 2007, 2010; Cutler et al., 2010; Lucas, 2010; Alsan, 2015; Mora-Garcia, 2018; Depetris-Chauvin and Weil, 2018; Dillon, Friedman and Serneels, 2021; Hamory et al., 2021). In particular, NTDs epitomize a class of health challenges that disproportionately afflict the poorest people in low- and middle-income countries. Because they often have both acute and chronic effects, a long-run perspective is necessary when studying the full impacts of controlling them. We highlight that doing so can have lasting and novel benefits such as reductions in (racial) inequality and public health care spending, which can inform how policymakers allocate scare resources dedicated to promoting development.

Finally, our study adds to our understanding of the effects of health on inequality, and vice versa, and on inter-generational cycles of poverty (e.g., Farmer, 2001; Deaton, 2003; O'Donnell, Van Doorslaer and Van Ourti, 2015; Alsan and Wanamaker, 2018). In this respect, our paper is most closely related to Bütikofer and Salvanes (2020), who find that Norway's control of tuberculosis (TB)—another disease that primarily afflicts those at society's margins—beginning in 1948 led to larger increases in adult incomes for children from formerly high-TB municipalities and greater intergenerational mobility. We complement this work by showing that similar benefits obtain when controlling an NTD in a developing country, and that averting its chronic health impacts results in substantial cost savings, compounding the effects of reducing its acute symptoms. Moreover, our results imply disease control can be a race-neutral policy that nonetheless reduces racial income inequality in a country with a long history of skin color-based disparities, similar to minimum wage policies (e.g., Derenoncourt and Montialoux, 2021; Derenoncourt et al., 2021). As such, it can be a simple, relatively inexpensive, and politically palatable tool to help mitigate mutually reinforcing health and socioeconomic gaps between white and non-white individuals.

The rest of the paper is organized as follows. Section 2 provides background on Chagas

disease, detailing its transmission, progression, and close association with poverty and racial disparities in Latin America. It also describes Brazil's vector control campaign and its implementation timeline. Section 3 outlines the difference-in-differences framework used to estimate the effects of Chagas disease control. Section 4 presents the impacts of vector control on municipalities' GDP per capita and inequality. Section 5 examines individual-level outcomes, exploring the long-term benefits for children exposed to the campaign and their subsequent intergenerational effects. Section 6 examines the health spending implications of reduced chronic morbidity. Finally, Section 7 discusses the broader implications of the findings, including cost-benefit analyses and extrapolations of impacts for the rest of Latin America. We conclude with Section 8.

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% of those infected contracted it from blood-sucking triatomine bugs carrying the parasite.[7] 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% of infections (Schofield and Dias, 1999).[8] Appendix B1 presents an image of this bug, which became domesticated and spread through rural settlements in southern and southeastern Brazil in the late nineteenth century after the clearing of forests for farming and ranching (Schofield, 1988). Shortly thereafter, the physician Carlos Chagas identified the parasite and the disease it caused (Chagas, 1909).

Appendix B2 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% of those infected are asymptomatic (Khan, 2011). The individual then enters the chronic

[7] The other 10% of transmission occurs through blood transfusions and the placenta.

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

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).[9] 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) noted how frequently its vector inhabited fissures in the unplastered walls of patients' homes. He was thus the first of many to link exposure to the parasite with 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 its symptoms, Chagas disease can be a cause 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 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% of hospitalizations for acute Chagas disease symptoms while only being about 40% of the population (Santos et al., 2020). In contrast, 20% of those hospitalized were white (around 45% of the population) and 7% were Black (about 8%).

[9] 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*.

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 other state 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] demonstrations that vector control was feasible," the Brazilian government launched a national effort against Chagas disease in 1975 (Dias, 1987, p. 338). The first stage consisted of serological and entomological surveys lasting until the early 1980s. They found a national rural *T. cruzi* prevalence rate above 4%—including nearly 9% 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).[10] Notably, in some municipalities, estimated prevalence was above 30%.

After the surveys concluded, thousands of public health personnel began visiting millions of homes across the endemic region in 1984 to conduct indoor residual spraying (IRS) against *T. infestans*.[11] However, outbreaks of dengue fever in coastal (i.e., tourist) areas in 1986 resulted in a 3-year diversion of public health personnel to the control of this mosquito-borne disease.[12]

[10] Appendix B3 shows municipalities' *T. cruzi* seroprevalence (Costa Passos and Silveira, 2011).

[11] See Appendix B4 for images from Dias (1987) of insecticide being sprayed on rural homes with many cracks and spaces in their roofs and walls amenable to colonization.

[12] Triatomine bugs thus reestablished themselves in some recently-sprayed municipalities (Dias, 1987; Schofield and Dias, 1999), though the map in Coura and Dias (2009) shows a clear decline in the presence of *T. infestans* by 1989. However, we could not find any record of which municipalities had not yet had

IRS restarted in 1989 and 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*.

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 municipality- and individual-level specifications differ in important ways, we provide more detail in subsequent sections.

3.1. Control, Treatment, and Excluded Municipalities

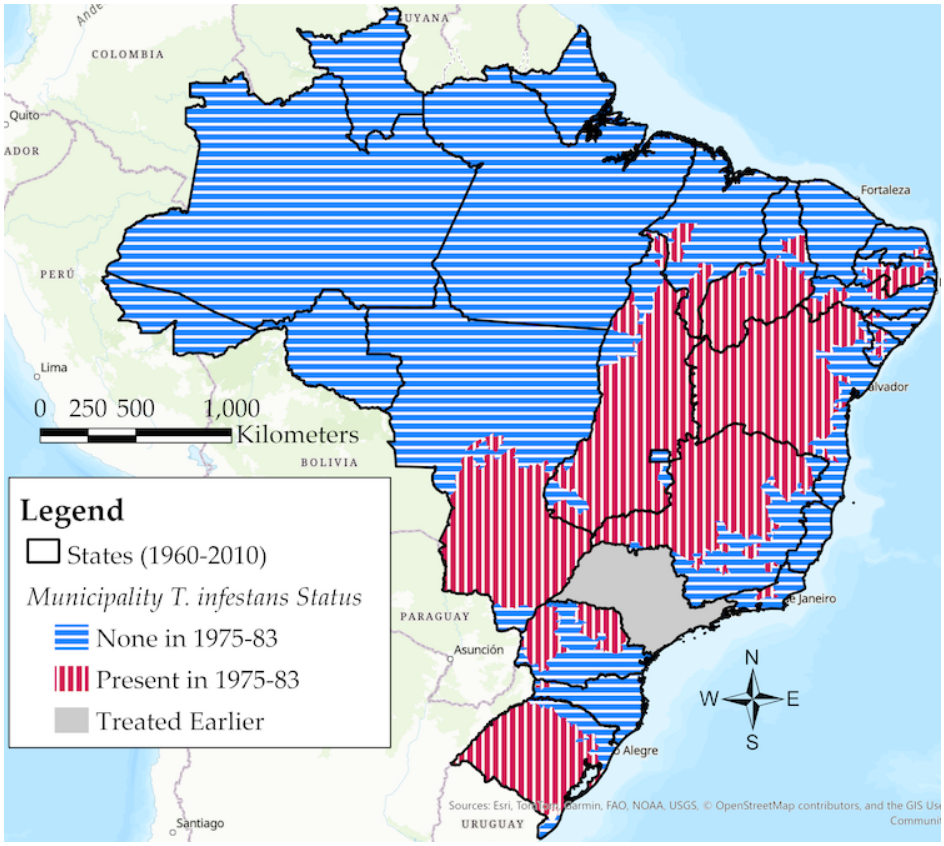
We digitized the map of Brazilian municipalities in which *T. infestans* were present in the 1975-83 entomological surveys (Silveira, 2011). Figure 2 shows these data. Our control group consists of those in which the vector was not found prior to spraying, so we assume that they were never exposed to transmission of Chagas disease via *T. infestans*.[13] Our treatment group consists of all municipalities in which the vector was present before IRS began. We cannot observe whether the vector was successfully—or only temporarily—eliminated from a municipality in 1984-86 (i.e., treatment had begun), or if it had not yet received IRS, though we do know that it was eventually eliminated in every case. Therefore, we include all of these municipalities in our treatment group to be conservative. Lastly, we exclude the state of Sao Paulo because it was treated much earlier, so we avoid treatment heterogeneity problems in our difference-in-differences estimation (Goodman-Bacon, 2021).

Table 1 presents summary statistics for these groups using individual-level data from the IPUMS 25% 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 vulnera-

IRS and which ones were treated before 1986 but had the bugs return during the pause in spraying.

[13] 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.

Figure 2: Brazilian Municipalities by *T. infestans* Status, 1975-83



Notes: Map shows municipalities with no *T. infestans* in 1975-83 (control group) with blue horizontal lines and those in which the vector was present in that period (treatment group) with red vertical lines. Municipalities in the state of Sao Paulo (solid gray) were treated earlier and are thus excluded from our sample. Data are from Silveira (2011).

ble 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. The means by racial group in Panels C and D imply links between exposure to Chagas disease and somewhat worse labor market and human capital outcomes, especially for non-white adults. There is also somewhat greater within-municipality inequality (as measured by the average municipality-level Gini coefficient) in the treatment group evident in Panel E.

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

We cannot observe in which year IRS began in a municipality, so we assume that it occurred uniformly throughout the treatment group in 1984. The graph of hospitalizations for acute

Table 1: Summary Statistics, 1980

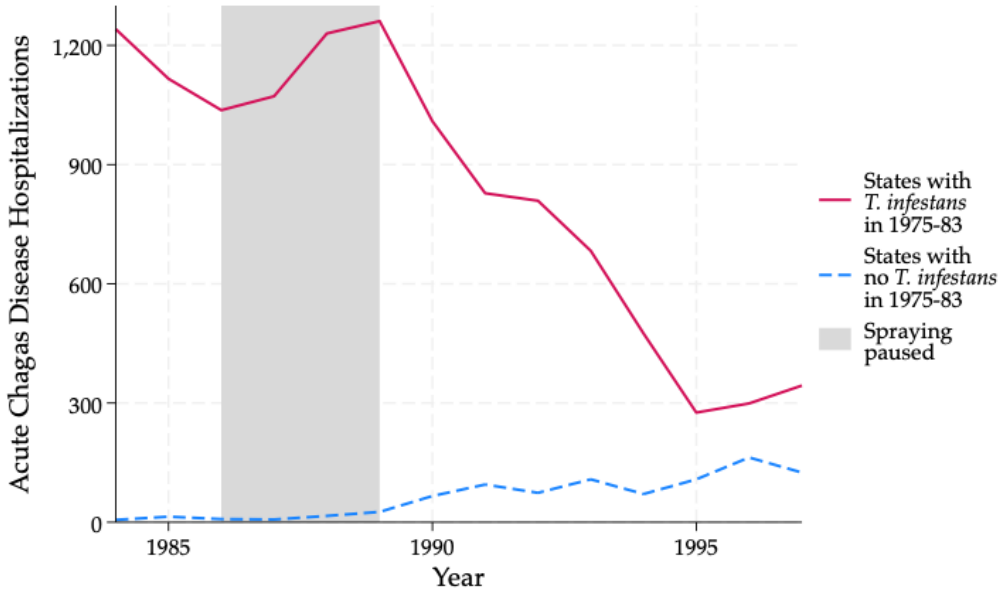
	Municipalities by 1975-83 *T. infestans* Status		
	None (Control)	Present (Treatment)	Treated Earlier (Excluded)
Panel A. Demographics			
Age	24.01	24.31	25.95
	(19.12)	(19.25)	(18.93)
Female	0.507	0.502	0.501
Asian	0.002	0.003	0.019
Black	0.065	0.057	0.045
Brown	0.473	0.365	0.182
White	0.460	0.575	0.754
Panel B. Living in Housing Vulnerable to Infestation			
Non-White	0.341	0.390	0.056
White	0.112	0.133	0.026
Panel C. Labor Market Outcomes			
Income Above Median: Non-White	0.455	0.444	0.607
Income Above Median: White	0.498	0.492	0.583
Labor Force Participant: Non-White	0.634	0.614	0.711
Labor Force Participant: White	0.626	0.629	0.660
Panel D. Human Capital			
Years of Schooling: Non-White Ages 19+	2.68	2.02	3.58
	(3.31)	(2.87)	(3.33)
Years of Schooling: White Ages 19+	4.78	4.13	5.02
	(4.10)	(3.73)	(4.00)
Literate: Non-White Ages 9-18	0.658	0.641	0.916
Literate: White Ages 9-18	0.867	0.872	0.962
Panel E. Income Inequality within Municipalities			
Gini Coefficient	76.30	76.94	72.31
	(4.02)	(4.28)	(3.17)
Observations	16,431,739	6,611,704	6,181,009
Municipalities	1,156	575	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.

Chagas disease in Figure 3 suggests that this assumption is conservative, as IRS appeared to reduce this measure of *T. cruzi* transmission but it did not immediately fall to zero.[14] When studying the impacts on municipalities and states, our final pre-treatment year is thus 1983

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

Figure 3: Hospitalizations Due to Acute Chagas Disease, 1984-97



Notes: Graph shows hospitalizations due to acute Chagas disease from 1984 to 1997 in states with any treatment municipality (solid red line) and those with only municipalities in the control group (dashed blue line). See Figure 2 for a map of these municipalities. Gray shading indicates years in which indoor residual spraying against *T. infestans* was paused. Data are from DATASUS.

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%; 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).

3.3. Assessing Threats to Identification

An important concern with our difference-in-differences approach is the potential influence of concurrent policies or economic shocks that might have differentially affected post-treatment municipalities or 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 Sections 4 and 6, our findings on municipality outcomes and health outcomes align with this timeline, mitigating concerns that other factors drove the effects discussed below.

4. Effects on Municipalities

We now study the long-run effects of Chagas disease vector control by comparing municipality-level outcomes across our treatment and control groups. We show that it led to increased GDP per capita and reduced income inequality, and that these effects became more pronounced 10 or more years after IRS began, suggesting that reductions in chronic Chagas disease augmented the positive short-run benefits of preventing the disease's acute stage. The evidence on mechanisms suggests a small positive effect within the first decade on young adults' educational attainment (consistent with reductions in children experiencing acute symptoms) and an increase around 25 years later in older adults' labor force participation (consistent with declines in chronic symptoms in the long run).

4.1. Data and Empirical Strategy

Our first main outcome is municipalities' GDP per capita (in constant 2019 R$), which we calculated in 8 years between 1970 and 2010 using data from Ipeadata.[15] In addition, we collapsed individual-level data for adults aged 20 to 50 into municipality-level means across five Brazilian censuses: two 25% samples from pre-treatment years (1970 and 1980) and three 10% samples from post-treatment years (1991, 2000, and 2010), all of which are from IPUMS (Ruggles et al., 2024).[16] Doing so allows us to calculate our other main outcome, which is a municipality's income Gini coefficient in each year. We also examine three others that can shed light on mechanisms:

[15] These years are 1970, 1975, 1980, 1985, 1996, 2000, 2007, and 2010. Ipeadata did not report municipality population estimates for 1975 and 1985, so we interpolated them using average annual growth rates for 1970-80 and 1980-91.

[16] We match 1970 municipalities to consistent IPUMS 1980-2010 municipalities containing their centroids.

the share earning incomes above the national median within a census year, the average years of schooling, and the share in the labor force.[17] For our analysis of these variables, we divide the data into ages 20 to 29 and 30 to 50 (almost exactly the median split) to test for differential impacts, which could arise from reductions in different symptoms of the illness.[18]

To study the effects of Chagas disease control on these outcomes in municipality m in year t, we estimate

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

where $y_{m,t}$ is an outcome of interest for m in t, α_m and γ_t are fixed effects for m and t, $\mathbb{1}[m \in \text{Treat}]$ indicates whether m is in the treatment group, $\mathbb{1}[t = k]$ is an indicator for whether t was a given year k, $\mathbf{X}_{m,1980}$ is a vector of m's characteristics in 1980 (the female, Asian, Black, and Brown shares of its population, and its changes in potential soy and maize yields when switching from traditional to genetically engineered seeds from Bustos, Caprettini and Ponticelli, 2016), $\delta_{s(m)}$ is a fixed effect for m's state, and $\epsilon_{m,t}$ is the idiosyncratic error term. We interact $\mathbf{X}_{m,1980}$ and $\delta_{s(m)}$ with γ_t to control for these characteristics' time-varying influences as well as state-specific trends.

The coefficients of interest are the τ_k, which measure the difference in an outcome in a given year across the treatment and control groups, relative to the size of that difference in 1980. In other words, they capture how treated municipalities evolved over time relative to those in the control group before and after the start of IRS. We estimate equation (1) using either Poisson regression (GDP per capita and Gini coefficients) or OLS (years of schooling and labor force participation rates) with standard errors clustered by municipality, and we weight observations by their 1980 population to account for differences in municipality sizes.[19] We also compute 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 constant treatment effect across 30 years.

[17] We use the share of above-median incomes to avoid log transformations and because it is more informative about the distributional impacts of treatment than an average effect (Chen and Roth, 2024).

[18] The 1970 census does not contain information on race, so we cannot assess municipal-level parallel trends assumptions by racial group. In Section 5, we incorporate race into our analysis by taking a cohort-based approach using individual-level data from 2010, and we also focus on adults aged 30 to 50.

[19] We use Poisson regression to recover effects as percentages of means in the control group, which are more intuitively applied in our cross-country extrapolations in Section 7.3.

4.2. GDP per Capita and Inequality

We first use Poisson regression to examine trends in output per person between 1970 and 2010. Figure 4a shows that GDP per capita increased by 11.1% more in treatment municipalities in the three decades after 1980, and this effect is precisely estimated. Importantly, the impact of Chagas disease control on GDP per capita increased over time, consistent with reductions in chronic symptoms compounding the positive effects of averting the acute phase. Next, we continue using Poisson regression to study income inequality within municipality-years. Figure 4b shows that Gini coefficients decreased by a precisely estimated 1.1% more in the treatment group. Though more difficult to assess given the smaller number of time periods, there is a qualitatively similar pattern of the effect sizes growing across the post-treatment decades. Taken together, these results provide evidence that eliminating Chagas disease transmission led to important improvements in standards of living across Brazilian municipalities and reductions in inequality within them.

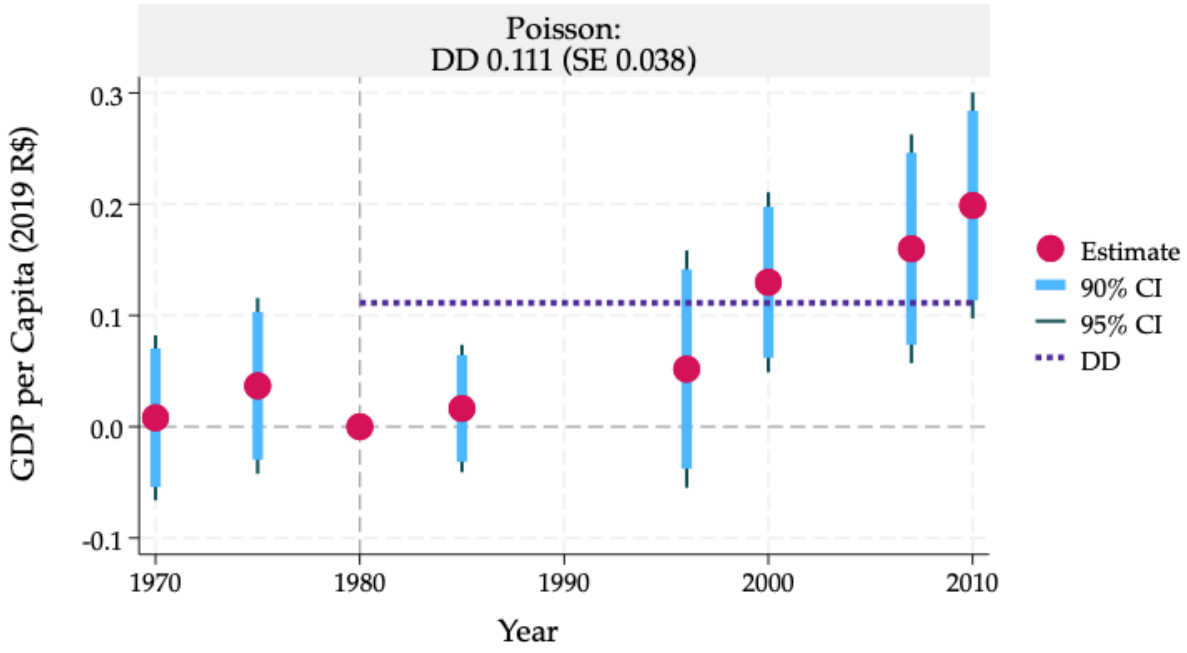
For robustness to the issues raised in the new difference-in-differences literature, we also present results using new estimators in Appendices C1 and C2 (Arkhangelsky et al., 2021; Callaway and Sant'Anna, 2021; de Chaisemartin and D'Haultfœuille, 2020; Sun and Abraham, 2021).[20] The patterns in these estimates are very similar to those above: treated municipalities experienced substantial increases in output per capita and decreases in inequality, though the magnitudes for the former are smaller.

4.3. Disentangling Effects of Acute and Chronic Symptoms

We then turn to the effects of IRS on the share of adults with incomes above the nationwide median in each census sample. The left panel of Figure 4c shows that for adults aged 20 to 29, it increased 1.0 p.p. (2.1%) more in treatment municipalities over this period. Notably, this average estimate is precise and the dynamic effects again grew over time. Because it is unlikely that 20- to 29-year-olds experienced chronic Chagas disease symptoms before 1984—and especially because any reduction in chronic symptoms should not have occurred before 1994 (see Section 2)—the implication is that this result is due in large part to averting the acute phase. Unfortunately, the

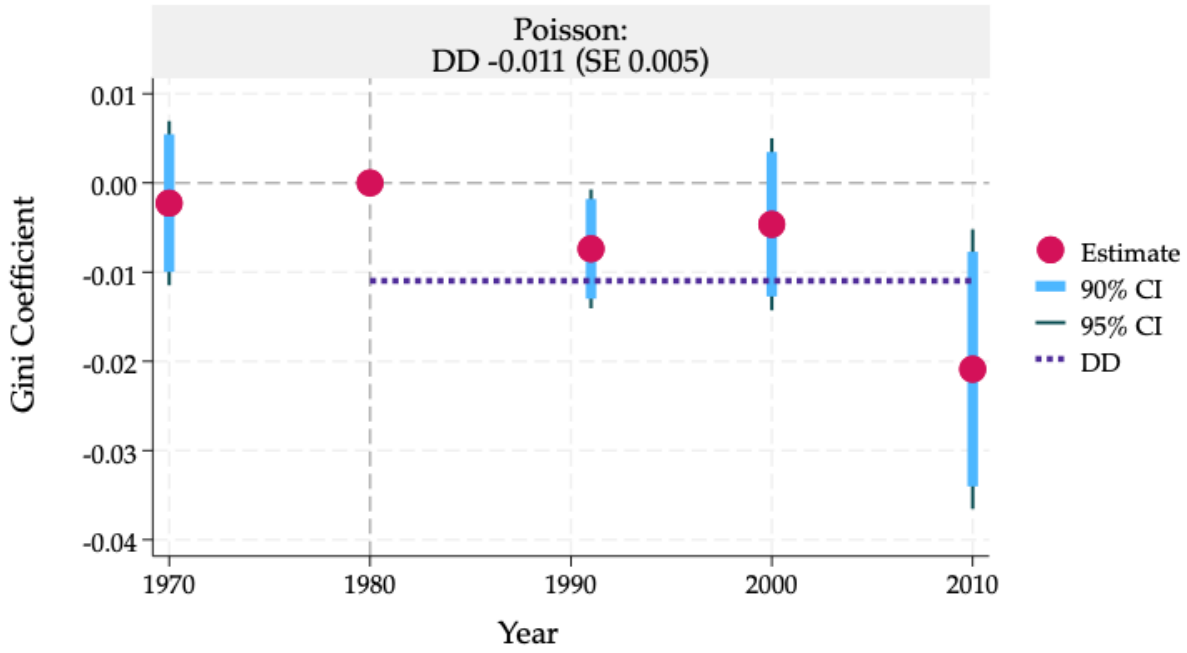
[20] Nonetheless, we reiterate that our approach of comparing unexposed areas to those (eventually) exposed does not suffer from many of these shortcomings.

Figure 4: Effects of Chagas Disease Vector Control on Municipalities



13,152 observations, 1,591 clusters, pre-1985 mean 15,651

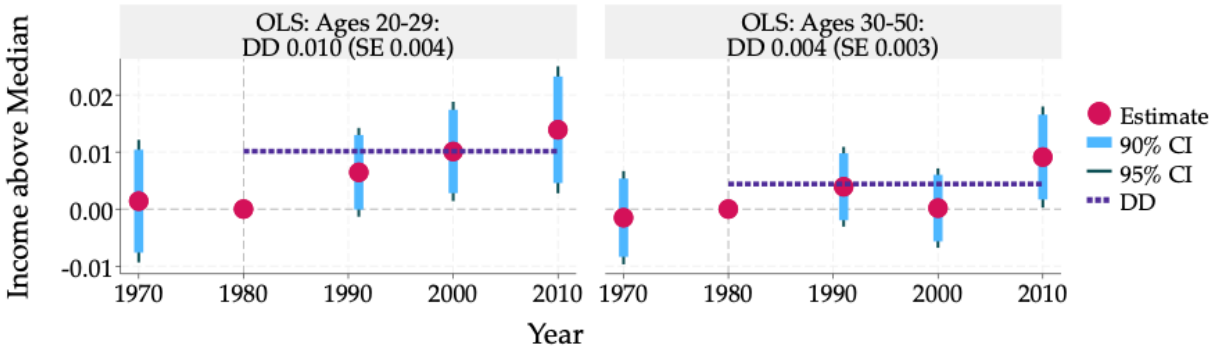
(a) GDP per Capita



8,160 observations, 1,578 clusters, pre-1991 mean 72.95

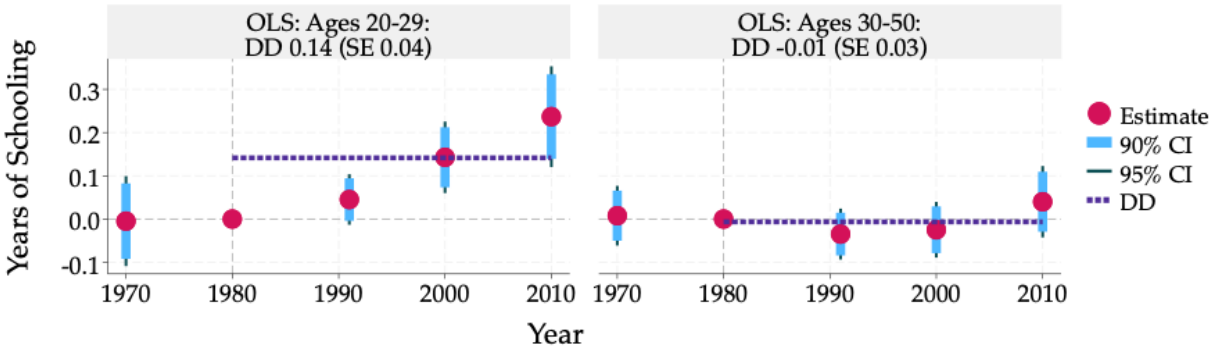
(b) Gini Coefficient

Figure 4: Continued



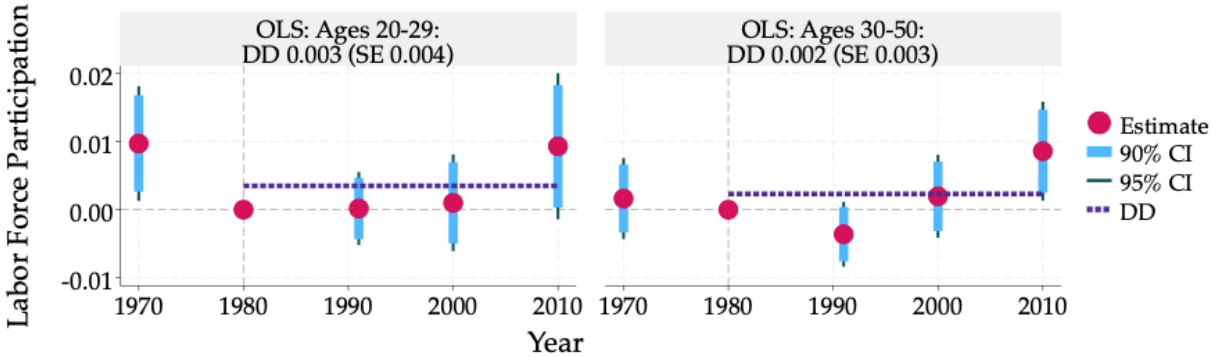
8,160 observations, 1,578 clusters, pre-1991 means: ages 20-29 0.424, ages 30-50 0.486

(c) Income above National Median in Census Sample



8,160 observations, 1,578 clusters, pre-1991 means: ages 20-29 3.90, ages 30-50 2.87

(d) Years of Schooling



8,160 observations, 1,578 clusters, pre-1991 means: ages 20-29 0.597, ages 30-50 0.602

(e) Labor Force Participation Rate

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. GDP per capita is calculated using data from Ipeadata, and all other data are from the IPUMS census samples. Regressions include fixed effects for year and municipality and the interactions of year fixed effects with a vector of predetermined characteristics (shares of the 1980 population that were female, Asian, Black, and Brown, and changes in potential soy and maize yields when switching from traditional to genetically engineered seeds) and state fixed effects. Standard errors are clustered by municipality.

results for ages 30 to 50 in the right panel are more ambiguous: while the 1991 and 2010 dynamic estimates seem to follow the pattern among those aged 20 to 29 and are of similar magnitudes, the estimate for 2000 is null, leading to a smaller and imprecise average effect (0.4 p.p., or 0.8%). It is thus difficult to determine whether older adults also benefited from averting Chagas disease's acute stage or if only reductions in chronic symptoms mattered for their incomes. Nonetheless, these results are evidence of a range of working-age adults moving up the income distribution as a result of Chagas disease control.

We also examine the impacts on years of schooling, as much of the literature on disease control has focused on education as a primary channel for income gains (e.g., Miguel and Kremer, 2004; Bleakley, 2007, 2010). However, in the left panel of Figure 4d, it appears that completed schooling among adults aged 20 to 29 increased more slowly than the share with above-median incomes. Combined with the small average effect (0.14 years, or 3.6%), it suggests that additional time in the classroom was not the only mechanism—and perhaps not the primary one—raising the share of above-median incomes. Along these lines, we find no differential change in completed schooling among older adults in treatment municipalities.[21] Because we would not expect those aged 30 to 50 to return to school even if their health improved, this result helps to rule out additional time in the classroom and the in-migration of more educated individuals as driving the increase in the share of older working-age adults with above-median incomes.

Lastly, in Figure 4e we examine labor force participation, as Chagas disease can cause working-age adults to "fill hospital beds instead of the [labor] force" (World Health Organization, 2010). The absence of parallel trends in the left panel prevents us from drawing conclusions about the effect on young adults' rates, which is unfortunate because it could have shed light on the apparent decline in the right panel in older adults' 1991 labor force participation (e.g., if averted acute stages allowed younger adults to displace these incumbents). Nonetheless, there is a null average effect for older adults due to an equally large and more precise positive estimate for 2010. The latter result is consistent with reductions in chronic symptoms among adults who were children around the time of IRS, allowing them to fill the labor force instead of hospital beds, to paraphrase the WHO quotation above. We focus more directly on health in Section 6.

[21] The near exception is in 2010, when 20- to 29-year-olds in 1991 and 2000 were aged 30 to 50.

5. Effects on Individuals: Long-Run and Intergenerational Impacts

We now take a complementary approach to studying the long-run effects of Chagas disease vector control by examining the impacts on adults who were children around the time that *T. infestans* control began. This approach is taken by other studies on the impacts of reducing disease exposure (e.g., Bleakley, 2010; Cutler et al., 2010; Bütikofer and Salvanes, 2020) and it allows us to examine impacts on racial disparities. We also study the effects of the IRS campaign on treated adults' children. Our results show that it shifted far more non-white than white Brazilians into the top half of the income distribution and that the children of non-white fathers in the treatment group experienced much larger increases in their literacy rates. The implication is that vector control decreased racial inequality in Brazil in the long run and may contribute to further reductions in it in the next generation. Consistent with our previous results, when exploring mechanisms, we find that increases in treated cohorts' years of schooling are unlikely to explain most of the income results, suggesting that a more important channel could be improved long-run health allowing adults treated as children to remain in the labor force.

5.1. Data

To study these impacts on post-treatment cohorts and their children, we use the IPUMS 10% sample of Brazil's 2010 census (Ruggles et al., 2024). It contains information on our outcomes of interest (monthly income and literacy), potential mechanisms (years of schooling and labor force participation), demographic characteristics (age, racial category, and sex), and whether an individual was born in their municipality or state of residence. Because we are focused in this section on the distribution of income rather than average effects, we follow Chen and Roth (2024) by using indicators for whether an individual's income was above the nationwide median in the 2010 census data and whether it was strictly positive. These outcomes thus help us understand whether the poorest or richest individuals from treatment municipalities benefited most from eliminating Chagas disease transmission.

5.2. *Assignment to Treatment and Control Groups*

To examine the effects of additional childhood years free from exposure to Chagas disease, we define our pre-treatment cohorts as those 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. However, as noted in Section 2, both children and adults can experience acute symptoms when (re)infected with *T. cruzi*, and reinfections increase the chance of entering the chronic stage. 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 that primarily impact children like helminthiases and malaria (e.g., Miguel and Kremer, 2004; Bleakley, 2007, 2010; Cutler et al., 2010; Lucas, 2010).

For several reasons, we limit our attention to cohorts born between 1960 and 1980, who were aged 30 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, so age 50 is an appropriate upper bound. In addition, DDT spraying in Brazil began in the late 1950s, so we want all cohorts in our sample to have had the same level of exposure to this intervention. Lastly, the restriction at the bottom of our age range ensures that our sample 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. Among these individuals, we observe the municipalities of birth for just under three-fifths of them; for the remainder, we only know their states of birth. Therefore, we place those with known municipalities of birth into the treatment or control groups (or exclude them from the sample if they were born in Sao Paulo), as discussed in the previous section. We then assign individuals who moved away from their birth municipalities to the treatment group if their birth state had any municipalities with *T. infestans* in 1975-83 or to the control group if their birth state was completely free of the vector.[22]

This categorization means that our treatment group consists of individuals who had at least some degree of exposure to Chagas disease transmission as children and our control group contains only those we know with certainty had none. As such, we interpret our treatment

[22] We create a bin for all individuals born in a state for whom we cannot determine their birth municipality.

effects as resulting from a reduction in the share of children potentially exposed to Chagas disease transmission from around 29%—the fraction of children in the 1980 census living in treatment municipalities—to 0%. We view this interpretation as both intuitive and policy relevant.

5.3. Empirical Strategy

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

$$y_{i,m,c} = \alpha_m + \gamma_c + \sum_{k \neq 1967} \tau_k \cdot \mathbb{1}[\mathbb{P}_i(m \in \text{Treat}) > 0] \cdot \mathbb{1}[c = k] + \mathbf{X}_i\beta + \delta_{s(m)} \times \gamma_c + \epsilon_{i,m,c}, \qquad (2)$$

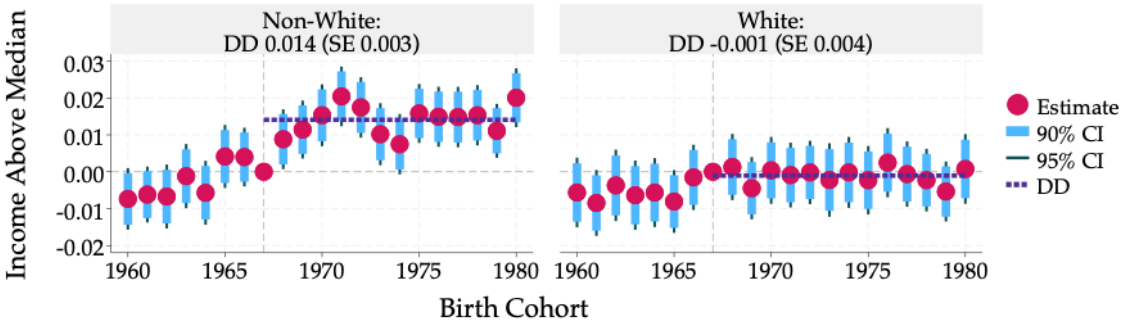
where $y_{i,m,c}$ is an outcome of interest for individual i born in municipality m and of birth cohort c, α_m and γ_c are fixed effects for m and c, $\mathbb{1}[\mathbb{P}_i(m \in \text{Treat}) > 0]$ indicates whether the probability that i was born in a treatment municipality is strictly positive, $\mathbb{1}[c = k]$ 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.

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 29% to 0%, 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 separately by broad racial category (non-white and white) to examine the potential for Chagas disease control to reduce racial inequality without 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.

5.4. Income and Inequality

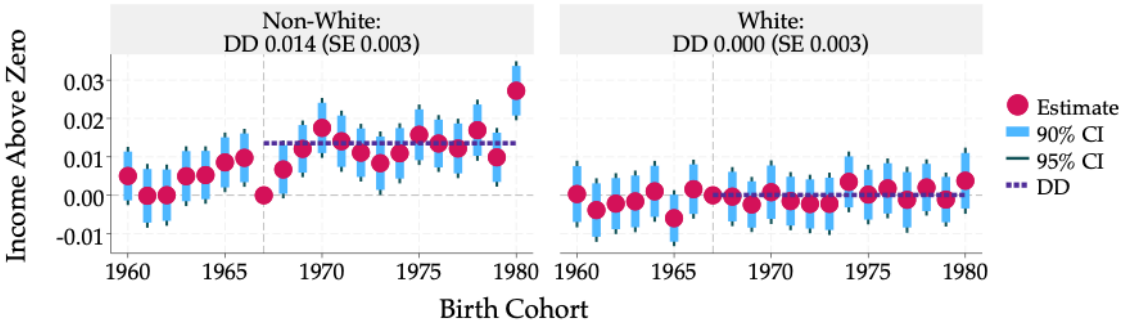
Our first outcome of interest is whether an individual is in the top half of the nationwide income distribution in the 2010 census sample. The left panel of Figure 5a shows that the share earning above the median increased by 1.4 p.p. (2.8%) more among non-white Brazilians potentially exposed to Chagas disease in childhood, and this estimate is precise. Conversely, the right panel

Figure 5: Long-Run and Intergenerational Effects of Chagas Disease Vector Control



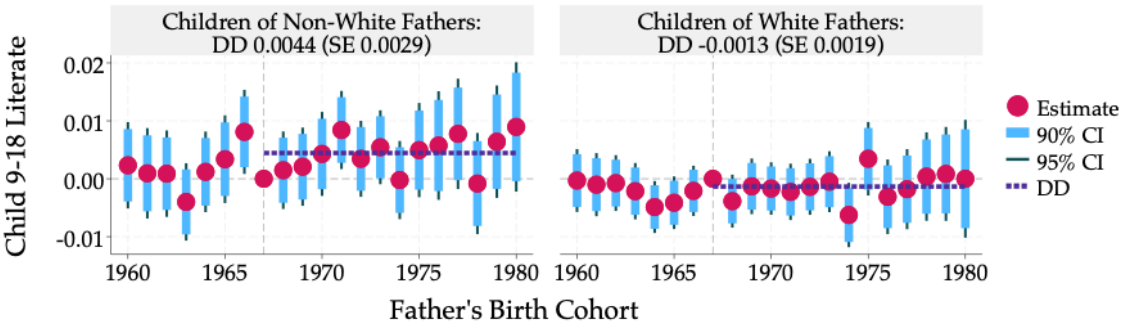
2.23-2.79 million observations, 1,755 clusters, pre-1968 means: non-white 0.509, white 0.654

(a) Income Above Median



2.23-2.79 million observations, 1,755 clusters, pre-1968 means: non-white 0.766, white 0.807

(b) Income Above Zero



0.68-1.05 million observations, 1,752 clusters, pre-1968 father's birth cohort means: non-white 0.9567, white 0.9836

(c) Literacy: Children Ages 9-18

Notes: Graphs show dynamic and post-treatment average (DD) estimates for the respective subgroups, with 90% and 95% 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.

shows that it did not change among their white peers. It appears that much of the increase in non-white adults earning above-median incomes was due to these cohorts moving away from zero reported earnings: the left panel of Figure 5b shows that the share with strictly positive incomes also rose a precisely estimated 1.4 p.p. (1.8%) more. As there is once again no similar effect visible in the right panel for white adults, these findings imply that Chagas disease control helped mitigate racial income inequality by increasing the incomes of the non-white Brazilians who otherwise would have been among the poorest in the country.

At first glance, an important contrast between these results and those in Figures 4a and 4b appears to be in how the effects do not grow monotonically as years of childhood free from Chagas disease exposure increase. However, an important factor that may have interfered with the emergence of such a pattern is age heaping, which is clearly visible in the histogram of birth years in Appendix D1, especially among non-white adults. To the extent that poorer individuals were more likely to report ages (and thus birth years in these data) ending in 0 or 5 and were more exposed to Chagas disease prior to IRS, it is thus not surprising that the estimates seem to have local maxima in these years, particularly in the left panel of Figure 5b. As such, we consider the average effect for all post-treatment cohorts—which is not sensitive to age heaping—to be more informative than any individual dynamic coefficient.

For robustness to the issues raised in the difference-in-differences literature, we use new estimators developed to address them (Arkhangelsky et al., 2021; Callaway and Sant'Anna, 2021; de Chaisemartin and D'Haultfœuille, 2020; Sun and Abraham, 2021) in Appendices D2 and D3.[23] These results are very similar to those above.

5.5. *Next Generation's Literacy*

To study the intergenerational impacts of exposure to vector control during childhood, we attach men's birth municipality and cohort information to the children aged 9 to 18 living in their households in the 2010 census.[24] We then estimate a modified version of equation (2) that uses

[23] Nonetheless, we reiterate that our approach of comparing those never exposed to Chagas disease transmission with those potentially exposed before and after a single treatment time does not suffer from many of these issues.

[24] We limit the sample to exclude those over 18 due to selection into living with their parents after that age. We set 9 years old as the lower bound because literacy rates were still increasing rapidly for 6- to

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 5c shows that it increased by 0.44 p.p. (0.46%)—or non-literacy decreased by that amount (10.2%)—more among the children of non-white fathers who averted exposure to Chagas disease in childhood, though the estimate is slightly imprecise. However, as predicted, there are no effects for the children of white fathers, and the same patterns are also visible when using new difference-in-differences estimators in Appendix D4. Therefore, these results provide evidence that the some of the benefits of Chagas disease vector control were transferred from fathers to their children, highlighting the positive intergenerational impacts of combating NTDs.

5.6. Disentangling Effects of Acute and Chronic Symptoms

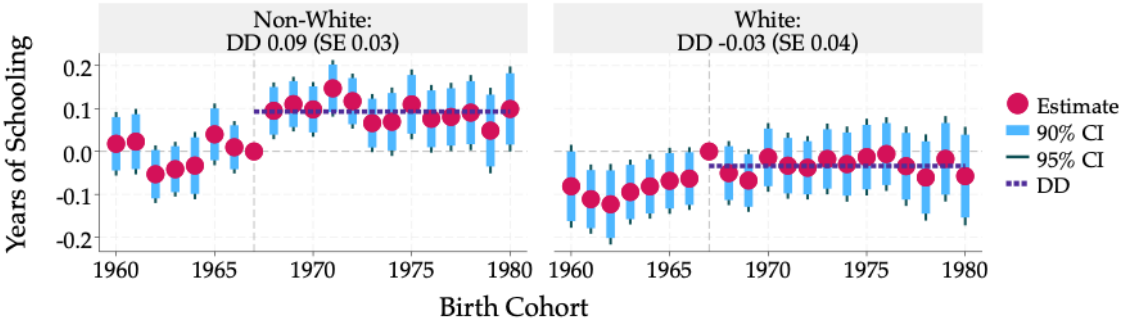
We then turn to studying years of schooling and labor force participation among adults who were children around the time that IRS began. As discussed in the previous section, these outcomes help us understand the impacts of each stage of Chagas disease: acute symptoms during childhood should only have affected schooling while chronic symptoms in adulthood would keep individuals out of the labor force. The left panels of Figure 6a and 6b show that both increased by more for non-white adults treated as children: schooling by 0.09 years (1.8%) and labor force participation by 0.9 p.p. (1.3%). In contrast, the effects are null or much smaller for white adults in the right panels of these figures, in line with our expectations and previous results. As these patterns are also apparent when using the new difference-in-differences estimators in Appendices D5 and D6, the evidence suggests that both mechanisms contributed to the impacts above.

5.6.1. Mediation Analysis

To quantify the contributions of increased schooling and labor force participation to the effects on the income distribution and the next generation's literacy, we conduct a mediation analysis. The first step, in which we calculate the impact of treatment on the mediator of interest $M_{i,m,c}$, is

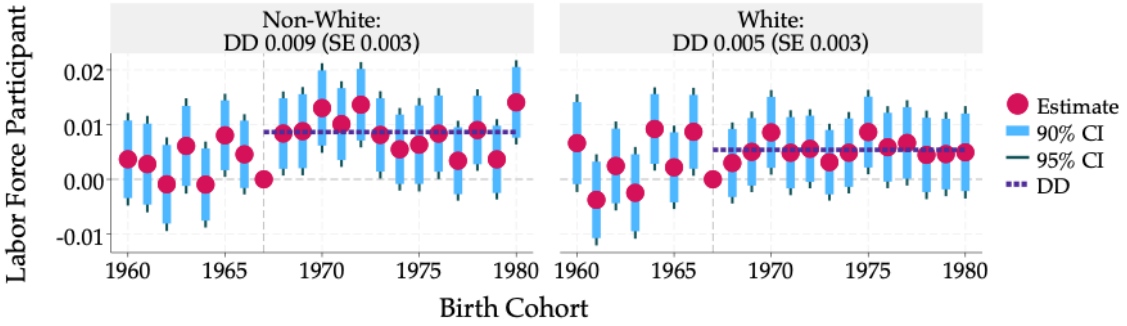
8-year-olds in the 2010 census, so we exclude them to reduce the noise in our estimates.

Figure 6: Mechanisms Underlying Long-Run and Intergenerational Effects



2.23-2.79 million observations, 1,755 clusters, pre-1968 means: non-white 5.06, white 6.67

(a) Years of Schooling



2.23-2.79 million observations, 1,755 clusters, pre-1968 means: non-white 0.712, white 0.771

(b) Labor Force Participant

Notes: Graphs show dynamic and post-treatment average (DD) estimates for the respective subgroups, with 90% and 95% 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.

simply to repeat the estimation of equation (2) with this variable as the outcome,

$$M_{i,m,c} = \alpha_m + \gamma_c + \sum_{k \neq 1967} \tau_k^{\text{Med}} \cdot \mathbb{1}[\mathbb{P}_i(m \in \text{Treat}) > 0] \cdot \mathbb{1}[c = k] + \mathbf{X}_i\beta + \delta_{s(m)} \times \gamma_c + \epsilon_{i,m,c}, \quad (3)$$

and compute the average of the post-treatment τ_k^{Med}, or $\bar{\tau}^{\text{Med}}$. In the second step, we modify equation (2) by adding the mediator to the right-hand side,

$$y_{i,m,c} = \alpha_m + \gamma_c + \sum_{k \neq 1967} \tau_k \cdot \mathbb{1}[\mathbb{P}_i(m \in \text{Treat}) > 0] \cdot \mathbb{1}[c = k] + \mu M_{i,m,c} + \mathbf{X}_i\beta + \delta_{s(m)} \times \gamma_c + \epsilon_{i,m,c}. \quad (4)$$

We then estimate the average of the post-treatment τ_k ($\bar{\tau}$), or the direct impact of treatment on the outcome, and $\mu\bar{\tau}^{\text{Med}}$, which is the effect of the mediator obtained by substituting equation (3) into equation (4). The approach allows us to quantify the share of the total impact $\bar{\tau} + \mu\bar{\tau}^{\text{Med}}$

Table 2: Mediation Analysis of Effects on Non-White Adults and Their Children

Mediating Variable:	Income Above Zero		Income Above Median		Child Literate	
	Years of Schooling (1)	Labor Force Participant (2)	Years of Schooling (3)	Labor Force Participant (4)	Years of Schooling (5)	Labor Force Participant (6)
Mediation Effect	0.0013	0.0044	0.0036	0.0040	0.0006	0.0000
Total Effect	0.0136	0.0136	0.0141	0.0141	0.0050	0.0050
Amount Mediated	10%	32%	25%	28%	11%	1%

Notes: Effects are calculated using the procedure described in the text. Columns (5) and (6) use fathers' values of the mediating variables. Data are from the IPUMS sample of the 2010 census.

that is due to the second term.

The estimated effects in Table 2 Columns (1) through (4) suggest that both mediators played substantive roles in moving treated non-white adults up the income distribution, though the contribution of increased schooling (10% to 25%) was less than that of increased labor force participation (around 30% for both outcomes). Thus, it appears that more of the effects on adults treated as children arose from averting the long-run health effects of Chagas disease than from reducing its acute symptoms in childhood. However, when calculating the impacts on the next generation's literacy in Columns (5) and (6), fathers' years of schooling seem matter more than their labor force participation (11% versus 1%), which makes intuitive sense because literacy is an educational outcome. Taken together, these estimated mediation effects suggest that both the acute and chronic symptoms of this NTD are important, and consistent with the municipality-level results in Section 4, it may be the case that each one compounds the impacts of the other. We study other major impacts of averting chronic Chagas disease symptoms in the next section.

6. Effects on State Public Health Care Systems

If cardiovascular morbidity from chronic Chagas disease was severe enough to reduce the labor force participation of adults exposed to its transmission as children, the costs it imposed on society could have extended beyond income, (racial) inequality, and the next generation's human capital. Specifically, as Brazil has the world's largest government-run health care system (the

Sistema Único de Saúde, or SUS)—which consumes about 4% of GDP and 70% of the population depends on—improvements in adults' heart health could have had important effects on public finances.[25] According to SUS data, circulatory system diseases caused one-tenth of the hospitalizations that it paid for between 2010 and 2019 (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% of GDP).

Therefore, in this section we examine the long-run effects of Chagas disease vector control on circulatory system-related hospital care covered by the SUS and deaths caused by cardiovascular disease. 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, although the impact on deaths is not clear. Nonetheless, the hospitalization results suggest that controlling Chagas disease transmission has yielded substantial savings for the public health care system in Brazil.

6.1. Data and Empirical Strategy

Our outcomes of interest are hospitalizations, spending on hospital care, and deaths, each of which is categorized by International Classification of Diseases (ICD) codes. The first two 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.[26] In addition, we set our omitted year to 1994, or a decade after IRS began.[27] However, as the SUS does not consistently provide municipality-level data for years prior to 1995, we restrict our focus to state-level data from 1991 to 2019.

[25] 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.

[26] 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.

[27] 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.

Along the lines of Section 5, our measure of each state's exposure to Chagas disease vector control is whether any of its population was living in treatment municipalities in 1980. 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 and 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 additional control group, under the assumption that they are subject to the same state-specific, time-varying factors.[28] The specification that we estimate is thus

$$y_{s,t,d} = \alpha_{s,t} + \gamma_{t,d} + \delta_{s,d} + \sum_{k \neq 1994} \tau_k \cdot \mathbb{1}[\mathbb{P}_{s,1980}(m \in \text{Treat}) > 0] \cdot \mathbb{1}[t = k] \cdot \mathbb{1}[d = \text{Circ}] + \eta_{r(s)} \times \gamma_{t,d} + \epsilon_{s,t,d}, \tag{5}$$

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.[29] We also 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 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. Because 85% of the 1980 population outside of Sao Paulo lived in states with any treatment municipality, we frame our results as moving from this number to 0% of the population with vector exposure. To increase precision and policy relevance, we also estimate the average of the post-1994 τ_k. For inference, we compute wild cluster bootstrap confidence intervals after clustering standard errors by the 24 consistent states in the sample (Cameron, Gelbach and Miller, 2008).

[28] 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)

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

However, because we cannot compute these confidence intervals for Poisson regression, we use OLS after log-transforming our state-level data, which never have any zeros to create the associated problems that Chen and Roth (2024) describe. But this solution still suffers from the problem of logs not being scale-invariant. Therefore, in Appendix E1 we use traditional confidence intervals to assess how important the wild cluster bootstrap is for inference in this case, and in Appendix E2 we use Poisson regression to determine how different the point estimates are between these approaches.

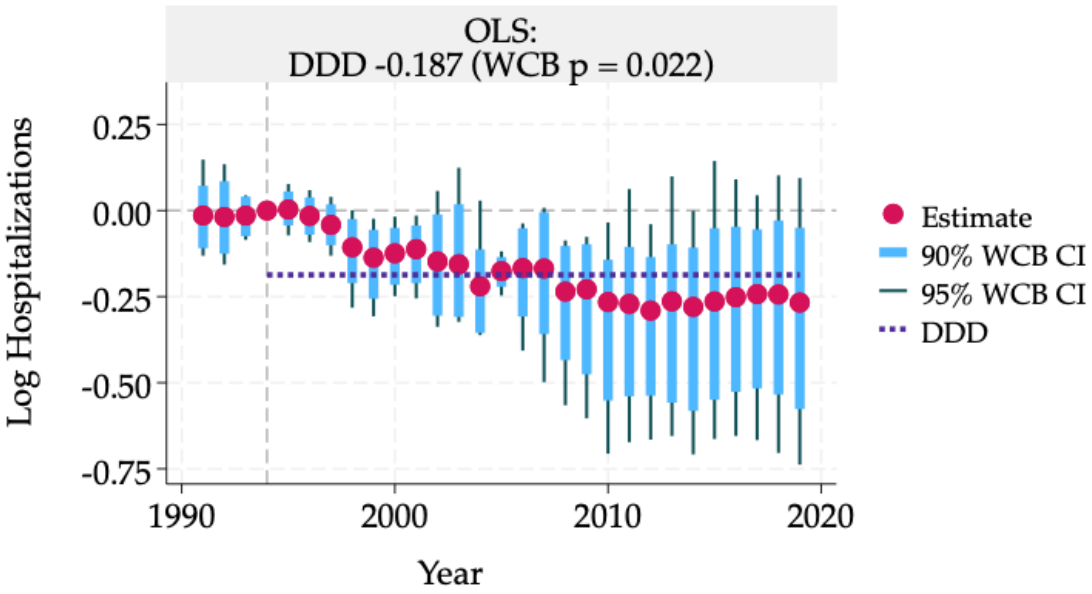
6.2. Circulatory Disease Hospital Care

Consistent with our hypothesis that Chagas disease control would improve cardiovascular outcomes, Figure 7a shows that hospitalizations paid for by the SUS due to circulatory system disease decreased 18.7% 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. Consequently, in Figure 7b, we also find a 16.1% 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. In addition, the confidence intervals (especially the 90% ones) closely resemble those in Appendix E1, and the point estimates are similar when using Poisson regression (Appendix E2) and new difference-in-differences estimators (Appendices E3 and E4). Therefore, these results imply significant benefits for Brazil's public health and public finances, the latter of which we quantify below.

6.3. Circulatory Disease Deaths

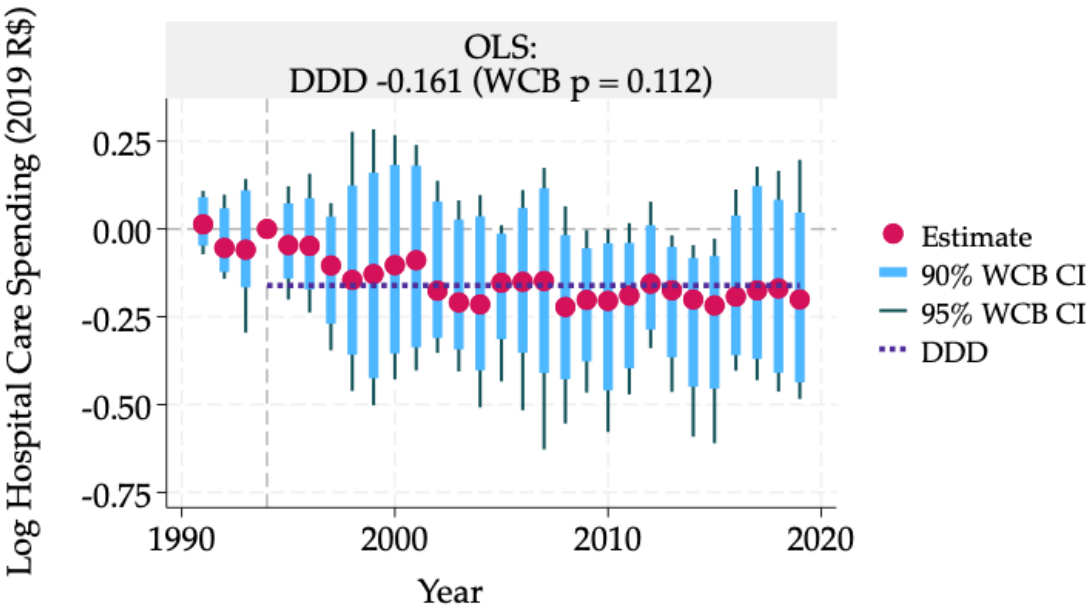
However, the impact on deaths by cause is less clear in Figure 7c. Although the post-treatment average effect is precisely estimated, there is a much larger pre-treatment difference in trends across circulatory and non-circulatory diseases than in the other panels, and the confidence intervals in the late 1990s and early 2000s are still quite wide. Similarly, there are also unclear effects in Appendices E1 through E5. Nonetheless, we view the absence of strong evidence of

Figure 7: Long-Run Effects on Circulatory Disease Outcomes



1,392 observations, 24 clusters, pre-1995 mean 0.24 million

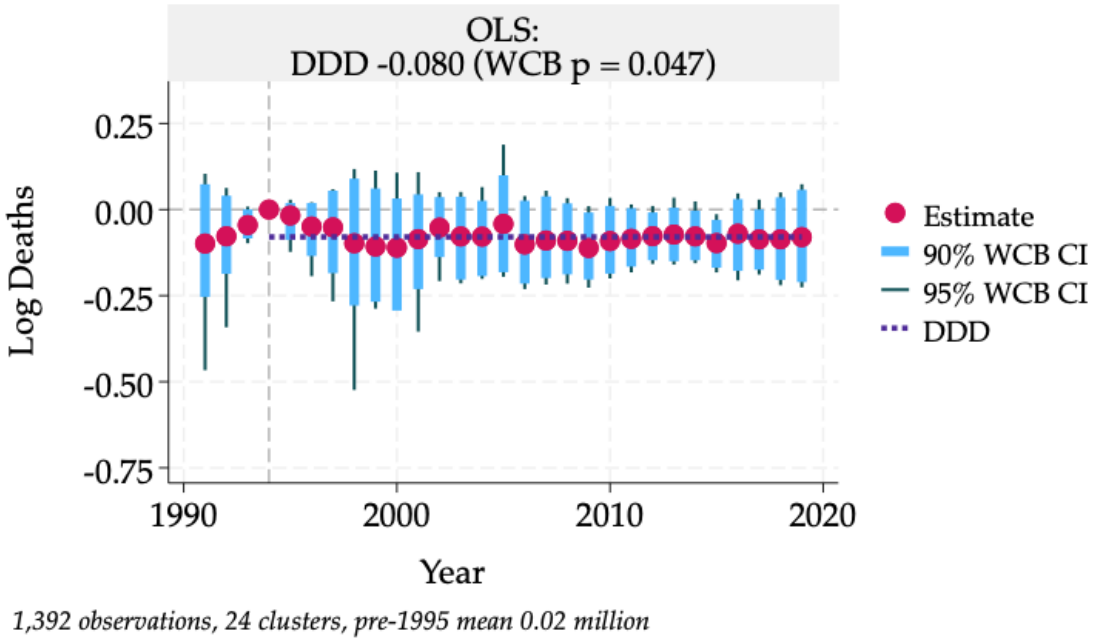
(a) Log Hospitalizations



1,392 observations, 24 clusters, pre-1995 mean 307 million

(b) Log Hospital Care Spending

Figure 7: Continued



(c) Log Deaths

Notes: Graphs show dynamic and post-treatment average (DDD) estimates for the respective outcomes, 90% and 95% 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 p-values are clustered by state.

clear impact 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 that there should be a meaningful gap between the two events.

7. Cost-Benefit Analyses and Extrapolation

Given the substantial benefits of Chagas disease vector control for individuals' incomes (Section 5) and states' public health care systems (Section 6), we provide two simple cost-benefit analyses to understand the economic viability of controlling neglected tropical diseases, especially those causing chronic health problems. In these exercises, we calculate an internal rate of return (IRR) due to increases in income and decreases in hospital care spending —i.e., not considering will-

ingness to pay for better health—of 23.9% and an infinite marginal value of public funds (MVPF) arising from the health spending effects. We also extrapolate our percentage effects on Brazilian municipalities to all Latin American countries to suggest that eliminating Chagas disease transmission could lead to meaningful reductions in the region's disparities and underdevelopment.

7.1. Internal Rate of Return

In our first cost-benefit analysis, we calculate the internal rate of return (IRR), or the discount rate required for a net present value of zero. On the cost side, Dias (1986) reported that spending on Chagas disease control in 1985 was Cr$ 500 million, which was the only number we could find from this period. To avoid interpretation issues stemming from hyperinflation in the mid-1980s through early 1990s, we use data from IBGE to calculate it as a share of nominal GDP (0.0038%) and assume that spending on Chagas disease vector control was at that level from 1984 through the pausing of IRS in 1986.[30] We then take World Bank data on Brazil's 1984-2019 GDP in constant 2015 USD, convert these values into 2019 R$ using the July 1, 2015 exchange rate (from the Banco Central do Brasil) and the GDP deflator (also from the World Bank), and calculate what spending on IRS was in each year according to our assumptions.

On the benefit side, we include only the effects on the incomes of adults who were exposed as children to IRS in this period and on hospital care spending. We therefore exclude impacts that we cannot directly measure in our data: namely, an individual's willingness to pay to avoid hospitalizations due to Chagas disease's chronic symptoms—not to mention its acute and subclinical effects—which is a conservative assumption because it is likely substantial.[31] For incomes, we assume that an additional 1.4% of non-white adults from treatment municipalities were shifted from earning no income to earning the median income in the sample (see Figures 5a and 5b), which was 2019 R$ 908. We also assume that this amount was constant for each cohort of approximately 675,000 non-white Brazilians from treatment municipalities entering the labor force

[30] Dias (1986) noted that public health authorities intended to spend a constant amount in real terms in these years.

[31] The Global Burden of Disease Collaborative Network (2024) estimates a disability weight—which takes values from 0 (full health) to 1 (death)—for atrial fibrillation and flutter due to Chagas disease of 0.22. The description of this condition is that a person "has periods of rapid and irregular heartbeats and occasional fainting." Disability weights for its other symptoms fall between this value and 0.05 (acute Chagas disease and controlled, medically managed heart failure).

at age 16 through 2019.[32] To calculate averted circulatory disease hospital care spending, we use the fact that the SUS spends approximately 0.1% of GDP on it each year and multiply it by the 16% average reduction estimated in Figure 7b to get savings of 0.016% of GDP in each year from 1995 to 2019.

As a result, we estimate an IRR of 23.9%. For comparison, Hamory et al. (2021) calculated an IRR of 37% in a 20-year follow-up of deworming in western Kenya by comparing consumption gains to the costs of administering the drugs and hiring additional teachers resulting from the increase in schooling, and Bütikofer and Salvanes (2020) estimated an IRR between 3.2% and 8.5% for Norway's mid-1900s tuberculosis testing and vaccination campaign. These differences likely emerge from the differences in upfront costs across these interventions. Specifically, deworming pills are extremely cheap and relatively easy to distribute to schoolchildren, whereas IRS against *T. infestans* requires large quantities of insecticides, and tuberculosis testing and vaccination are even more expensive. Thus, heavy discounting offsets much of the savings from controlling Chagas disease even though they comprise a non-trivial fraction of GDP each year. To illustrate this point, note that the IRR would be 21.6% if we only considered the income gains. Nonetheless, failing to consider the chronic health effects of eliminating Chagas disease transmission means understating its return by 2.3 p.p. (about one-tenth), so this omission could lead to suboptimal allocations of scarce funds to promote economic development.

7.2. Marginal Value of Public Funds

We also use the assumptions above to calculate the marginal value of public funds (MVPF), or the benefits to recipients divided by the net cost to the government. Any program that fully recovers its cost (in discounted terms) has an infinite MVPF, which is often the case with those targeting low-income children (Hendren and Sprung-Keyser, 2020). Because the IRR when considering only the hospital care savings is 9.1%—well above the standard 5% discount rate used in studies of developing economies (Haacker, Hallett and Atun, 2020)—the implication is that using this

[32] Because the costs of spraying and the savings on hospital care spending are not easily translated into per-person amounts, we convert the effects on an individual's income into an aggregate number. We calculate the average cohort size using the 2010 IPUMS census sample by taking the number of non-white individuals from the 1968-80 cohorts in the treatment group, scaling by it 10, and assuming a uniform distribution across years of birth.

rate yields an MVPF of infinity.[33] This result notably arises without raising additional revenue, as the marginal tax rate at the median income in the 2010 census data was 0%.[34] Therefore, from developing countries' governments' perspective, an intervention to improve the health of their poorest citizens may be more attractive if it reduces the long-run burden on their public health care systems, as gains in these individuals' very low incomes—or low state capacity and tax compliance—might not result in the collection of additional revenue.

7.3. Extrapolating Effects across Latin America

To assess the implications of controlling Chagas disease beyond Brazil, we extrapolate the estimated effects on Brazilian municipalities' output per person and inequality to all of Latin America. We use data from the World Health Organization (2015) on the share of each country's population that is still exposed to transmission (see Appendix F1 for a map), the region-wide average of which is 13%. Following Bleakley (2007, 2010), we perform a back-of-the-envelope calculation by multiplying World Bank data on 2019 GDP per capita and Gini coefficients by this share and our Poisson regression estimates in Section 4.[35]

With the appropriate caveats in mind, Table 3 presents these extrapolated impacts. The analysis suggests that, if Chagas disease transmission were eliminated across Latin America, the region's GDP per capita could be about 1.5% higher and its Gini coefficient could be around 0.15% lower. Naturally, there is significant variation across countries depending on the shares exposed, implying that countries like Ecuador (29% exposed), Guyana and Suriname (25% exposed), and Mexico (21% exposed) would experience large effects while unexposed wealthier ones (Chile and Uruguay) would not.[36] It is thus important to note that although these extrapolated effects seem small on average, the benefits would mostly accrue to the poorest and most unequal countries

[33] Similarly, Hendren and Sprung-Keyser (2020) estimated an infinite MVPF arising from the long-run health care savings generated by a childhood Medicaid eligibility expansion (Wherry et al., 2018).

[34] For marginal tax rates in Brazil in 2010, see: https://www.gov.br/receitafederal/pt-br/assuntos/meu-imposto-de-renda/tabelas/2010.

[35] While this extrapolation provides valuable insights, it has important limitations. For example, general equilibrium effects, such as changes in labor market dynamics, could influence the magnitude of these estimates, either by amplifying (e.g., via human capital spillovers) or dampening them (e.g., from higher labor supply). These factors imply that we cannot say whether our extrapolated estimates are upper or lower bounds on what would be the true effects.

[36] Along the lines of the estimates in Appendix A1, the percent exposed to Chagas disease has a correlation coefficient of -0.44 with GDP per capita and 0.30 with the Gini coefficient.

Table 3: Extrapolated Impacts of Controlling Chagas Disease across Latin America

Country	Population Exposed (%)	GDP per Capita ($)	Extrapolated Change ($)	Gini Coefficient	Extrapolated Change
Argentina	5	10,076	56	42.9	-0.02
Belize	22	4,983	122		
Bolivia	6	3,552	24	41.6	-0.03
Brazil	13	8,876	128	53.5	-0.08
Chile	0	14,699	0	44.4	0.00
Colombia	11	6,419	78	51.3	-0.06
Costa Rica	5	12,762	71	48.2	-0.03
Ecuador	29	6,223	200	45.7	-0.15
El Salvador	15	4,168	69	38.8	-0.06
Guatemala	10	4,639	51	48.3	-0.05
Guyana	25	6,610	183		
Honduras	15	2,574	43	48.2	-0.08
Mexico	21	9,950	232	46.7	-0.11
Nicaragua	11	1,924	23	46.2	-0.06
Panama	13	15,774	228	49.8	-0.07
Paraguay	20	5,384	120	45.7	-0.10
Peru	4	7,023	31	41.6	-0.02
Suriname	25	6,854	190		
Uruguay	0	17,688	0	39.7	0.00
Average	13	7,904	115	45.8	-0.07

Notes: Percent exposed is the share of the population exposed to Chagas disease vectors from the World Health Organization (2015). GDP per capita is in US dollars for 2019 and the Gini coefficient is the most recently reported value (scaled by 100) between 2010 and 2019, both from the World Bank. Estimated changes in GDP per capita and the Gini coefficient are calculated by multiplying a country's value by its percent exposed to Chagas disease vectors and the respective Poisson regression estimates for Brazilian municipalities in Figures 4a and 4b.

within the region, which would represent meaningful progress in narrowing the gaps between Latin America and North America or Europe in their levels of development and equality.

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 benefits to Brazil's campaign to control the main vector of Chagas disease, which has both acute and chronic phases like many other NTDs, and important long-run benefits beyond individuals' labor market returns. In particular, we found that controlling Chagas disease led to substantial short- and long-run increases in GDP per capita and reductions in inequality in treated municipalities. Furthermore, exposure to vector control in childhood raised adult incomes for non-white Brazilians, helping to increase the speed of racial convergence in a country with wide disparities in this dimension. We also found evidence that improved long-run health (as captured by labor force participation) played a role at least as large as educational attainment in driving this result. In addition, we showed that controlling this NTD may help to interrupt the intergenerational transmission of poverty by increasing the literacy of the children of these non-white adults.

Because circulatory system disease causes a substantial share of hospitalizations (10%) and spending (20%) covered by Brazil's publicly-run health care system, which consumes around 4% 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. As a result, simple cost-benefit analyses considering only the increases in income, reductions in health care spending, and costs of spraying against the main vector finds an internal rate of return of 23.9% and an infinite marginal value of public funds. We interpret these results as evidence for Chagas disease control having a significant impact on Brazil's public and fiscal health in the long run, which are other important impacts not previously examined in the literature.[37]

Thus, these results present a more complete picture of the economic consequences of NTD control for developing countries, the vast majority of which can be controlled via environmental management programs like the IRS campaign studied in this paper. Whether our results generalize beyond this malady that exclusively afflicts the Americas is an open question we leave to

[37] However, because these benefits generally materialize many years later, the results raise important questions for the political economy of disease control given some policymakers' short time horizons. We view this matter as an important area of future research.

future research. Nonetheless, we believe that this paper has identified novel areas through which health can generate inclusive growth in developing countries, helping to strengthen justifications for controlling transmission of not just this NTD—which an estimated 8 million people throughout the Western Hemisphere suffer from and another 75 million are exposed to—but also all of the others that cause chronic health problems among the poorest billion people on the planet.

References

Alsan, Marcella. 2015. "The Effect of the TseTse Fly on African Development." *American Economic Review*, 105(1): 382–410. [2, 6]

Alsan, Marcella, and Marianne Wanamaker. 2018. "Tuskegee and the Health of Black Men." *Quarterly Journal of Economics*, 133(1): 407–455. [6]

Arkhangelsky, Dmitry, Susan Athey, David A. Hirshberg, Guido W. Imbens, and Stefan Wager. 2021. "Synthetic Differnce-in-Differences." *American Economic Review*, 111(12): 4088–4118. [16, 24, 52, 53, 55, 57, 59, 61, 63, 67, 68, 69]

Attanasio, Orazio, Florencia Lopez-Boo, Diana Perez-Lopez, and Sarah Anne Reynolds. 2024. "Inequality in the Early Years in LAC: A Comparative Study of Size, Persistence, and Policies." Inter-American Development Bank Working Paper IDB-WP-01562. [5]

Bleakley, Hoyt. 2007. "Disease and Development: Evidence from Hookworm Eradication in the American South." *Quarterly Journal of Economics*, 122(1): 73–117. [6, 19, 21, 35]

Bleakley, Hoyt. 2010. "Malaria Eradication in the Americas: A Retrospective Analysis of Childhood Exposure." *American Economic Journal: Applied Economics*, 2(2): 1–45. [6, 13, 19, 20, 21, 35]

Briceño-León, Roberto, and Jorge Méndez Galván. 2007. "The Social Determinants of Chagas Disease and the Transformations of Latin America." *Memórias do Instituto Oswaldo Cruz*, 102: 109–112. [1]

Bustos, Paula, Bruno Caprettini, and Jacopo Ponticelli. 2016. "Agricultural Productivity and Structural Transformation." *American Economic Review*, 106(6): 1320–1365. [15]

Bütikofer, Aline, and Kjell G Salvanes. 2020. "Disease Control and Inequality Reduction: Evidence from a Tuberculosis Testing and Vaccination Campaign." *The Review of Economic Studies*, 87(5): 2087–2125. [6, 13, 20, 21, 34]

Callaway, Brantly, and Pedro H. C. Sant'Anna. 2021. "Differnce-in-Differences with Multiple Time Periods." *Journal of Econometrics*, 225(2): 200–230. [3, 16, 24, 52, 53, 55, 57, 59, 61, 63, 67, 68, 69]

Cameron, A. Colin, Jonah B. Gelbach, and Douglas L. Miller. 2008. "Bootstrap-Based Improvements for Inference with Clustered Errors." *Review of Economics and Statistics*, 90(3): 414–427. [29]

Castro, Marcia C, Adriano Massuda, Gisele Almeida, Naercio Aquino Menezes-Filho, Monica Viegas Andrade, Kenya Valéria Micaela de Souza Noronha, Rudi Rocha, James Macinko, Thomas Hone, and Renato Tasca, et al. 2018. "Brazil's Unified Health System: The First 30 Years and Prospects for the Future." *Lancet*, 394(10195): 345–356. [29]

Chagas, Carlos. 1909. "Nova Especie Morbida do Homem, Produzida por um Trypanozoma (Trypanozoma Cruzi)." *Brazil-Medico*, 23(16): 161. [7, 8]

Chancel, Lucas, Thomas Piketty, Emmanuel Saez, and Gabriel Zucman. 2021. *World Inequality Report 2022.* World Inequality Lab. [1]

Chen, Jiafeng, and Jonathan Roth. 2024. "Logs with Zeros? Some Problems and Solutions." *Quarterly Journal of Economics*, 139(2): 891–936. [4, 15, 20, 30]

Costa Passos, Afonso Dinis, and Antônio Carlos Silveira. 2011. "Síntese dos Resultados dos Inquéritos Nacionais." *Revista da Sociedade Brasileira de Medicina Tropical*, 44(Suppl. 2): 47–50. [9, 50]

Coura, José Rodrigues, and Pedro Albajar Viñas. 2010. "Chagas Disease: A New Worldwide Challenge." *Nature*, 465(7301): S6–S7. [1, 8]

Coura, José Rodrigues, and João Carlos Pinto Dias. 2009. "Epidemiology, Control and Surveillance of Chagas Disease - 100 Years after its Discovery." *Memórias do Instituto Oswaldo Cruz*, 104(Suppl. 2): 31–40. [9]

Cutler, David, Winnie Fung, Michael Kremer, Monica Singhal, and Tom Vogl. 2010. "Early-Life Malaria Exposure and Adult Outcomes: Evidence from Malaria Eradication in India." *American Economic Journal: Applied Economics*, 2(2): 72–94. [6, 13, 20, 21]

Deaton, Angus. 2003. "Health, Inequality, and Economic Development." *Journal of Economic Literature*, 41(1): 113–158. [6]

de Chaisemartin, Clément, and Xavier D'Haultfœuille. 2020. "Two-Way Fixed Effects Estimators with Heterogeneous Treatment Effects." *American Economic Review*, 110(9): 2964–2996. [3, 16, 24, 52, 53, 55, 56, 57, 58, 59, 60, 61, 62, 63, 64, 67, 68, 69]

Delaporte, François. 2012. *Chagas Disease: History of a Continent's Scourge.* Fordham University Press. [1]

Depetris-Chauvin, Emilio, and David N Weil. 2018. "Malaria and Early African Development: Evidence from the Sickle Cell Trait." *The Economic Journal*, 128(610): 1207–1234. [6]

Derenoncourt, Ellora, and Claire Montialoux. 2021. "Minimum Wages and Racial Inequality." *Quarterly Journal of Economics*, 136(1): 169–228. [6]

Derenoncourt, Ellora, François Gérard, Lorenzo Lagos, and Claire Montialoux. 2021. "Racial Inequality, Minimum Wage Spillovers, and the Informal Sector." Unpublished. [6]

De Rosa, Mauricio, Ignacio Flores, and Marc Morgan. 2020. "Inequality in Latin America Revisited: Insights from Distributional National Accounts." World Inequality Lab Issue Brief 2020/09. [1]

Dias, J. C. P. 1987. "Control of Chagas Disease in Brazil." *Parasitology Today*, 3(11): 336–341. [9, 51]

Dias, João Carlos Pinto. 1986. "Programa de Controle da Doença de Chagas no Brasil em 1986." *Revista da Sociedade Brasileira de Medicina Tropical*, 19(3): 129–133. [33]

Dillon, Andrew, Jed Friedman, and Pieter Serneels. 2021. "Health Information, Treatment, and Worker Productivity." *Journal of the European Economic Association*, 19(2): 1077–1115. [6]

Eberhard, Fanny E., Sarah Cunze, Judith Kochmann, and Sven Klimpel. 2020. "Modelling the Climatic Suitability of Chagas Disease Vectors on a Global Scale." *eLife*, 9: e52072. [1, 2, 6, 46]

Eslava, Francisco, and Felipe Valencia Caicedo. 2023. "Origins of Latin American Inequality." Inter-American Development Bank Working Paper IDB-WP-01492. [5]

Farmer, Paul. 2001. *Infections and Inequalities: The Modern Plagues.* Berkeley:University of California Press. [6]

Franco-Paredes, Carlos, Anna Von, Alicia Hidron, Alfonso J. Rodríguez-Morales, Ildefonso Tellez, Maribel Barragán, Danielle Jones, Cesar G. Náquira, and Jorge Mendez. 2007. "Chagas Disease: An Impediment in Achieving the Millennium Development Goals in Latin America." *BMC International Health and Human Rights*, 7(1): 1–6. [1, 8]

Global Burden of Disease Collaborative Network. 2024. *Global Burden of Disease Study 2021.* Seattle:Institute for Health Metrics and Evaluation. [33]

Goodman-Bacon, Andrew. 2021. "Differences-in-Differences with Variation in Treatment Timing." *Journal of Econometrics*, 225(2): 254–277. [3, 10]

Haacker, Markus, Timothy B. Hallett, and Rifat Atun. 2020. "On Discount Rates for Economic Evaluations in Global Health." *Health Policy and Planning*, 35(1): 107–114. [5, 34]

Hamory, Joan, Edward Miguel, Michael Walker, Michael Kremer, and Sarah Baird. 2021. "Twenty-Year Economic Impacts of Deworming." *Proceedings of the National Academy of Sciences*, 118(14): e2023185118. [6, 34]

Hendren, Nathaniel, and Ben Sprung-Keyser. 2020. "A Unified Welfare Analysis of Government Policies." *Quarterly Journal of Economics*, 135(3): 1209–1318. [34, 35]

Hernández, Daisy. 2021. *The Kissing Bug: A True Story of a Family, an Insect, and a Nation's Neglect of a Deadly Disease.* Portland:Tin House. [6]

Hotez, Peter J. 2011. "The Neglected Tropical Diseases and the Neglected Infections of Poverty: Overview of Their Common Features, Global Disease Burden and Distribution, New Control Tools, and Prospects for Disease Elimination." In *The Causes and Impacts of Neglected Tropical and Zoonotic Diseases: Opportunities for Integrated Intervention Strategies*. Washington, DC:National Academies Press. [2]

Hotez, Peter J., Eric Dumonteil, Miguel Betancourt Cravioto, Maria Elena Bottazzi, Roberto Tapia-Conyer, Sheba Meymandi, Unni Karunakara, Isabela Ribeiro, Rachel M. Cohen, and Bernard Pecoul. 2013. "An Unfolding Tragedy of Chagas Disease in North America." *PLOS Neglected Tropical Diseases*, 7(10): e2300. [1, 8]

Houweling, Tanja AJ, Henrike E Karim-Kos, Margarete C Kulik, Wilma A Stolk, Juanita A Haagsma, Edeltraud J Lenk, Jan Hendrik Richardus, and Sake J de Vlas. 2016. "Socioeconomic Inequalities in Neglected Tropical Diseases: A Systematic Review." *PLoS Neglected Tropical Diseases*, 10(5): e0004546. [1, 8]

Irish, Amanda, Jeffrey D Whitman, Eva H Clark, Rachel Marcus, and Caryn Bern. 2022. "Updated Estimates and Mapping for Prevalence of Chagas Disease Among Adults, United States." *Emerging Infectious Diseases*, 28(7): 1313. [6]

Khan, M. Gabriel. 2011. "Chagas Disease." In *Encyclopedia of Heart Diseases*. . 2 ed., 295–299. New York:Springer. [7]

Lucas, Adrienne M. 2010. "Malaria Eradication and Educational Attainment: Evidence from Paraguay and Sri Lanka." *American Economic Journal: Applied Economics*, 2(2): 46–71. [6, 21]

Médicos Sin Fronteras. 2013. *Chagas: Una Tragedia Silenciosa / A Silent Tragedy.* Buenos Aires:Losada. [1]

Miguel, Edward, and Michael Kremer. 2004. "Worms: Identifying Impacts on Education and Health in the Presence of Treatment Externalities." *Econometrica*, 72(1): 159–217. [6, 19, 21]

Mora-Garcia, Claudio A. 2018. "Can Benefits from Malaria Eradication be Increased? Evidence from Costa Rica." *Economic Development and Cultural Change*, 66(3): 585–628. [6]

Murdock, George P., and Douglas R. White. 1969. "Standard Cross-Cultural Sample." *Ethnology*, 8(4): 329–369. [5, 47]

Nunes, Maria Carmo Pereira, Andrea Beaton, Harry Acquatella, Carolyn Bern, Ann F. Bolger, Luis E. Echeverría, Walderez O. Dutra, Joaquim Gascon, Carlos A. Morillo, and Jamary Oliveira-Filho, et al. 2018. "Chagas Cardiomyopathy: An Update of Current Clinical Knowledge and Management: A Scientific Statement from the American Heart Association." *Circulation*, 138(2): e169–e209. [8]

Olden, Andreas, and Jarle Møen. 2022. "The Triple Difference Estimator." *Econometrics Journal*, 25(3): 531–553. [29]

Olivo Freites, Christian, Hendrik Sy, Amal Gharamti, Nelson I. Agudelo Higuita, Carlos Franco-Paredes, José Antonio Suárez, and Andrés F Henao-Martínez. 2022. "Chronic Chagas Disease—The Potential Role of Reinfections in Cardiomyopathy Pathogenesis." *Current Heart Failure Reports*, 19(5): 279–289. [8]

O'Donnell, Owen, Eddy Van Doorslaer, and Tom Van Ourti. 2015. "Health and Inequality." In *Handbook of Income Distribution*. Vol. 2, 1419–1533. Elsevier. [6]

Rassi, A., J. C. P Dias, J. A. Marin-Neto, and A. Rassi. 2009. "Challenges and Opportunities for Primary, Secondary, and Tertiary Prevention of Chagas' Disease." *Heart*, 95(7): 524–534. [7, 49]

Rassi, Anis, Anis Rassi, and William C. Little. 2000. "Chagas' Heart Disease." *Clinical Cardiology*, 23(12): 883–889. [8]

Ruggles, Steven, Lara Cleveland, Rodrigo Lovaton, Sula Sarkar, Matthew Sobek, Derek Burk, Dan Ehrlich, Quinn Heimann, and Jane Lee. 2024. *Integrated Public Use Microdata Series, International: Version 7.5.* Minneapolis, MN:Minnesota Population Center. [10, 14, 20]

Santos, Emily F., Ângelo A. O. Silva, Leonardo M. Leony, Natália E. M. Freitas, Ramona T. Daltro, Carlos G. Regis-Silva, and Rodrigo P Del-Rei, et al. 2020. "Acute Chagas Disease in Brazil from 2001 to 2018: A Nationwide Spatiotemporal Analysis." *PLOS Neglected Tropical Diseases*, 14(8): e0008445. [1, 8]

Schofield, C. J. 1988. "Biosystematics of the Triatominae." In *Biosystematics of Haematophagous Insects*. , ed. M. W. Service. Oxford, UK:Clarendon Press. [7]

Schofield, C. J., and J. C. P. Dias. 1999. "The Southern Cone Initiative against Chagas Disease." In *Advances in Parasitology*. Vol. 42, , ed. J. R. Baker, R. Muller and D. Rollinson, 1–27. San Diego:Academic Press. [7, 9]

Silveira, Antônio Carlos. 2011. "O Inquérito Triatomínico (1975-1983)." *Revista da Sociedade Brasileira de Medicina Tropical*, 44(Suppl. 2): 26–32. [10, 11]

Sun, Liyang, and Sarah Abraham. 2021. "Estimating Dynamic Treatment Effects in Event Studies with Heterogeneous Treatment Effects." *Journal of Econometrics*, 225(2): 175–199. [3, 16, 24, 52, 53, 56, 58, 60, 62, 64, 67, 68, 69]

Telles, Edward E., Stanley R. Bailey, Shahin Davoudpour, and Nicholas C. Freeman. 2023. "Racial and Ethnic Inequality in Latin America." Inter-American Development Bank Working Paper IDB-WP-01529. [1, 5, 8]

Wherry, Laura R., Sarah Miller, Robert Kaestner, and Bruce D. Meyer. 2018. "Childhood Medicaid Coverage and Later-Life Health Care Utilization." *Review of Economics and Statistics*, 100(2): 287–302. [35]

Woo-Mora, L. Guillermo. 2024. "Unveiling the Cosmic Race: Skin Tone Disparities in Latin America." World Inequality Lab Working Paper 2022/02. [1]

World Health Organization. 2010. *Working to Overcome the Global Impact of Neglected Tropical Diseases: First WHO Report on Neglected Tropical Diseases.* Geneva:WHO Press. [1, 3, 19]

World Health Organization. 2015. "Chagas Disease in Latin America: An Epidemiological Update Based on 2010 Estimates." *Weekly Epidemiological Record (Relevé Épidémiologique Hebdomadaire)*, 90(6): 33–44. [5, 35, 36, 70]

Zingales, Bianca, Michael A. Miles, David A. Campbell, Michel Tibayrenc, Andrea M. Macedo, Marta M.G. Teixeira, and Alejandro G. Schijman. 2012. "The Revised *Trypanosoma cruzi* Subspecific Nomenclature: Rationale, Epidemiological Relevance and Research Applications." *Infection, Genetics and Evolution*, 12(2): 240–253. [8]

Online Appendix for:

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

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University of Chicago

February 19, 2025

Contents

Appendix A. Additional Results: Introduction

A1. Chagas Disease Suitability, GDP Per Capita, and Inequality Across Countries

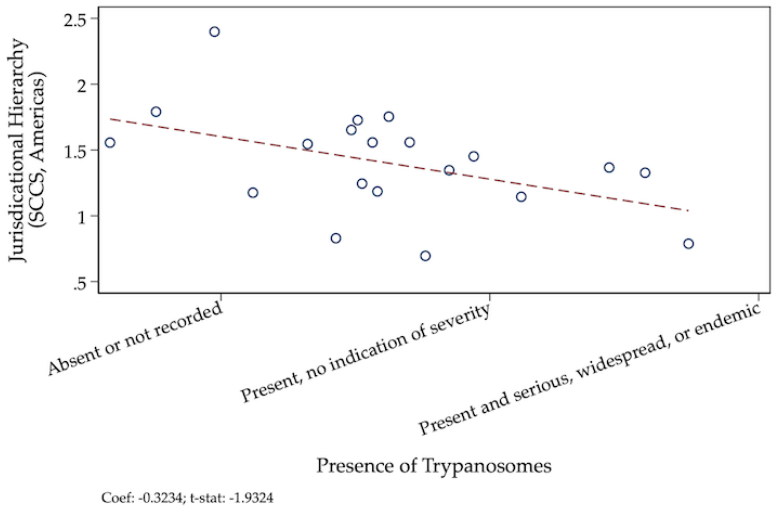
Table A1: Continent-Specific Effects of Chagas Disease [1]

	GDP per Capita		Gini Coefficient	
	(1)	(2)	(3)	(4)
Poisson Regression Coefficients				
Chagas Suitability	-0.017	-0.017	0.002	0.001
	(0.005)	(0.005)	(0.001)	(0.001)
Chagas Suitability × **1**[Americas]	-0.021	-0.024	0.005	0.005
	(0.009)	(0.009)	(0.001)	(0.001)
Continent FE	x	x	x	x
Geographic Controls	x	x	x	x
Disease Controls		x		x
Observations	184		151	
Mean	18,911		37.76	
Standard Deviation	20,045		7.51	

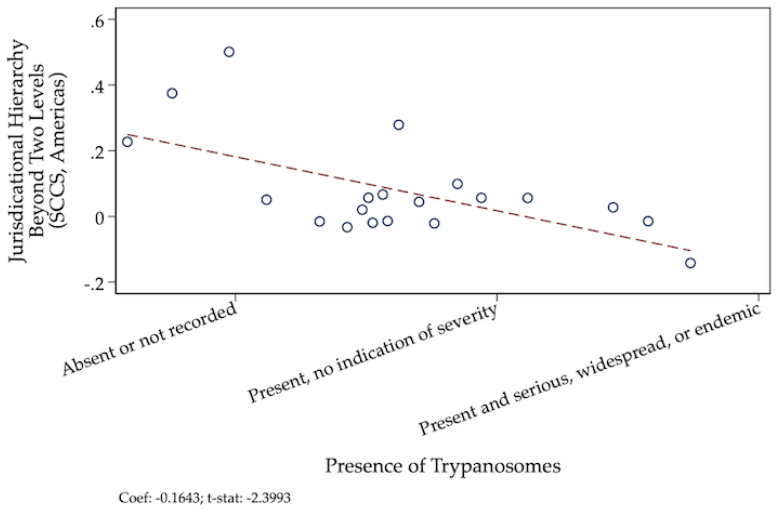
Notes: Observations are countries. Robust standard errors in parentheses. The outcome in columns (1) and (2) is the average 2010-19 GDP per capita (PPP, current international dollars), and the outcome in columns (3) and (4) is the average 2010-19 Gini coefficient (scaled by 100), both from the World Bank. Chagas suitability is a 0 to 100 measure of the ecological suitability for Chagas disease vectors from Eberhard et al. (2020). 1[Americas] is an indicator variable equal to one if a country is in North or South America. Geographic controls include centroid latitude, centroid longitude, average rainfall, average temperature, elevation, area, and agricultural suitability. Disease controls include malaria and tsetse fly suitability.

A2. Precolonial Presence of Trypanosomes and Jurisdictional Hierarchy

Figure A1: Precolonial Presence of Trypanosomes and Jurisdictional Hierarchy [1]



(a) Jurisdictional Hierarchy



(b) Centralization

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

Appendix B. Additional Figures: Chagas Disease & Its Control in Brazil

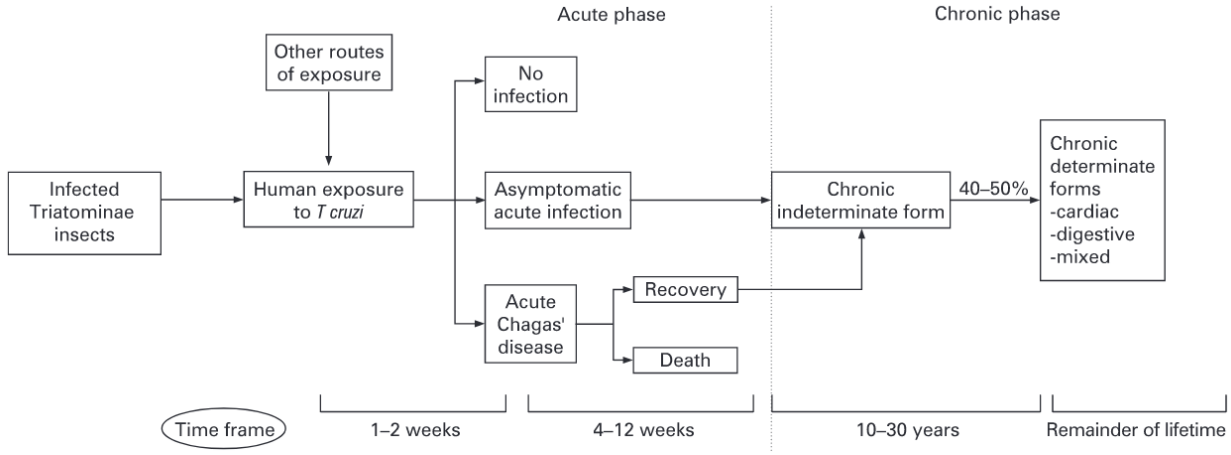
B1. Image of Triatoma infestans

Figure B1: Image of *Triatoma infestans* [7]

Notes: Image shows *Triatoma infestans*, the main vector in Brazil prior to the post-1983 Chagas disease control campaign. These 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 others.

B2. Phases of Chagas Disease

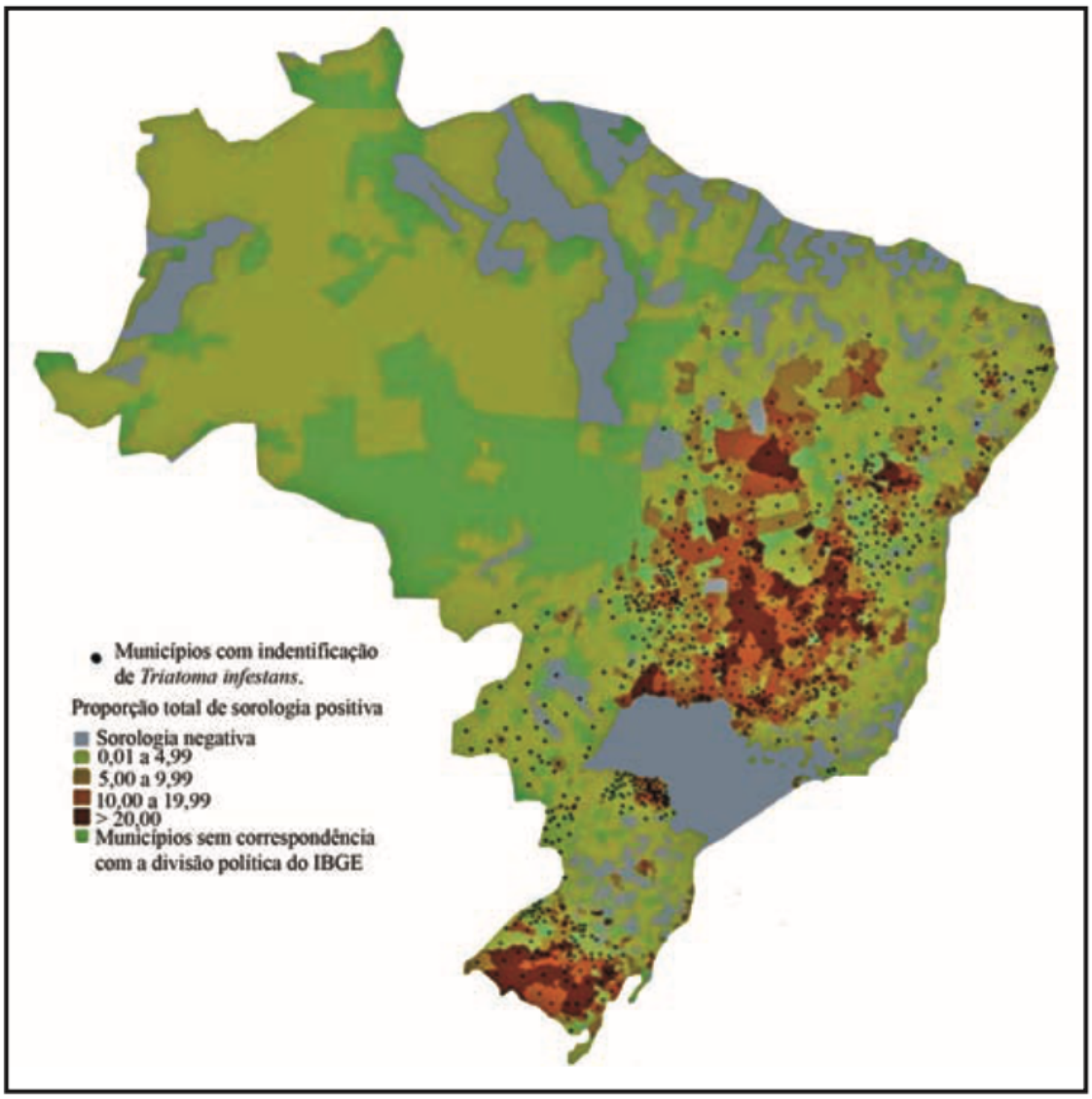
Figure B2: Phases of Chagas Disease [7]



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

B3. T. cruzi Seroprevalence, 1975-83

Figure B3: *T. cruzi* Seroprevalence, 1975-83 [9]



Notes: Figure reproduces a map of *T. cruzi* Seroprevalence prior to the Chagas control campaign across municipalities from Costa Passos and Silveira (2011). Black dots represent municipalities in which *T. infestans* were present, gray shading indicates 0% *T. cruzi*, prevalence, darker colors from yellow to brown indicate higher prevalence (see the ranges given in the legend), and green indicates municipalities whose boundaries changed over time.

B4. Images of Indoor Residual Spraying

Figure B4: Images of Indoor Residual Spraying [9]

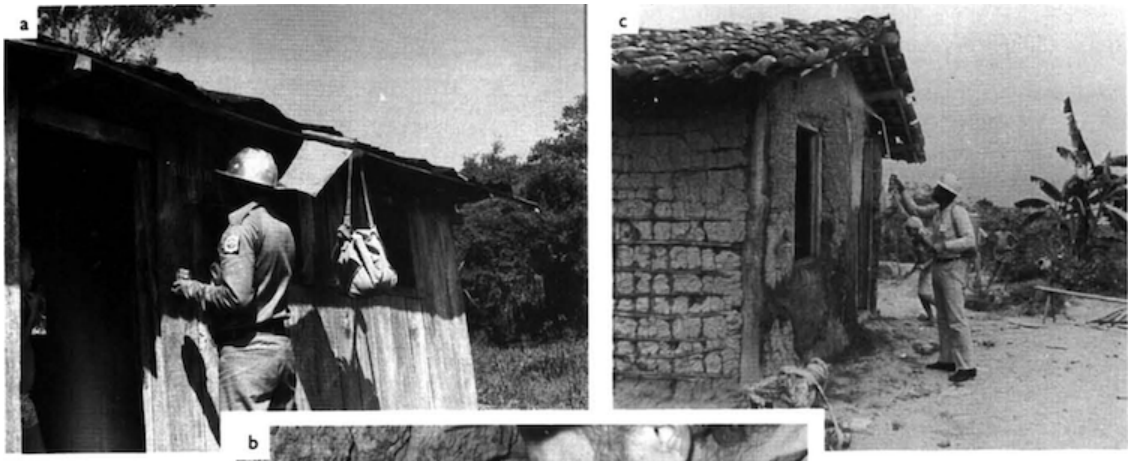


Fig. 2. Chagas disease control activities begin with a geographical reconnaissance of the target area (a) with each house inspected for bugs by trained field personnel (b). During the attack phase, all houses are sprayed while those that were positive for bugs are then resprayed 3–6 months later (c).

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

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.

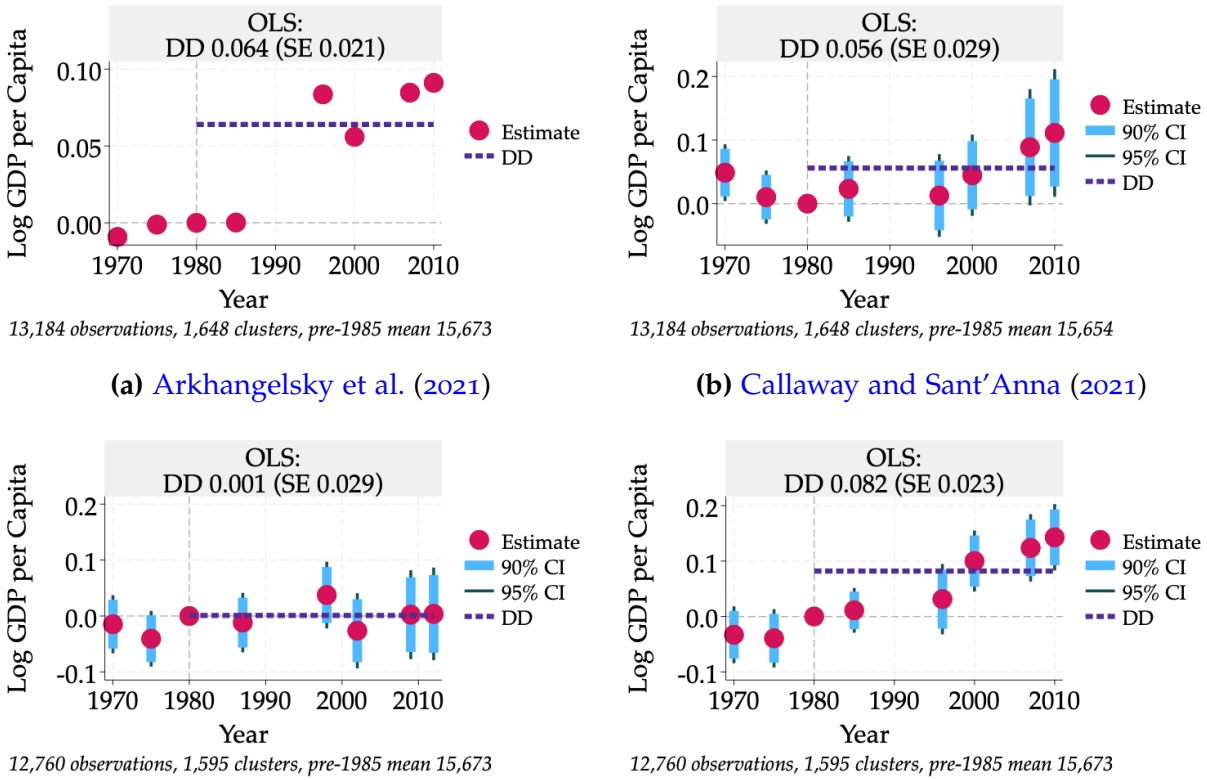
2 *The Attack Phase*. The initial spraying of an area is known as 'attack one' or massive attack, during which all houses and outbuildings are sprayed regardless of whether or not they were found to be infested. A second selective spraying is then carried out 3–6 months later only in houses known to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

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

Appendix C. Additional Results: Effects on Municipalities

C1. GDP per Capita Results Using New Difference-in-Differences Estimators

Figure C1: GDP per Capita Results Using New Estimators [16]



(a) Arkhangelsky et al. (2021)

(b) Callaway and Sant'Anna (2021)

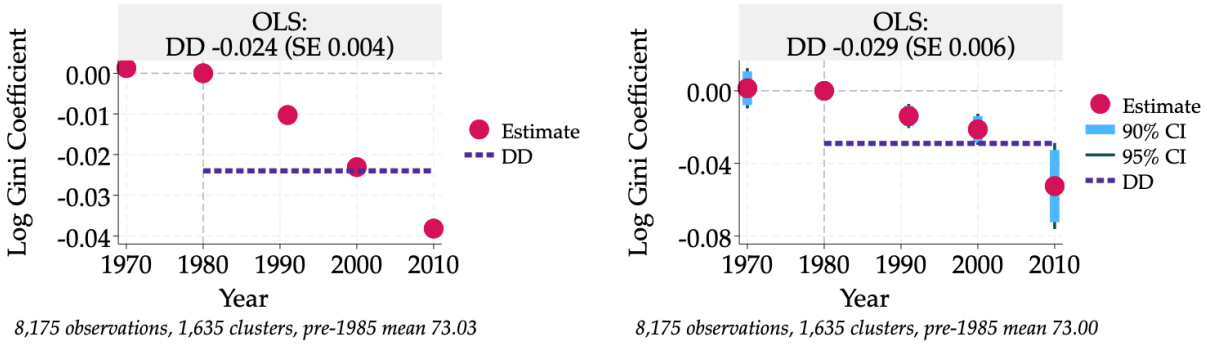
(c) de Chaisemartin and D'Haultfœuille (2020)

(d) Sun and Abraham (2021)

Notes: Observations are municipality-years. Graphs show dynamic and post-treatment average (DD) estimates when using a given estimator, with 90% and 95% confidence intervals for dynamic estimates except when using the Arkhangelsky et al. (2021) estimator. Data are from Ipeadata. All regressions include fixed effects for year and municipality, and those using the de Chaisemartin and D'Haultfœuille (2020) and Sun and Abraham (2021) estimators include the interactions of year fixed effects with a vector of predetermined characteristics (shares of the 1980 population that were female, Asian, Black, and Brown, and changes in potential soy and maize yields when switching from traditional to genetically engineered seeds) and state fixed effects. Standard errors are clustered by municipality.

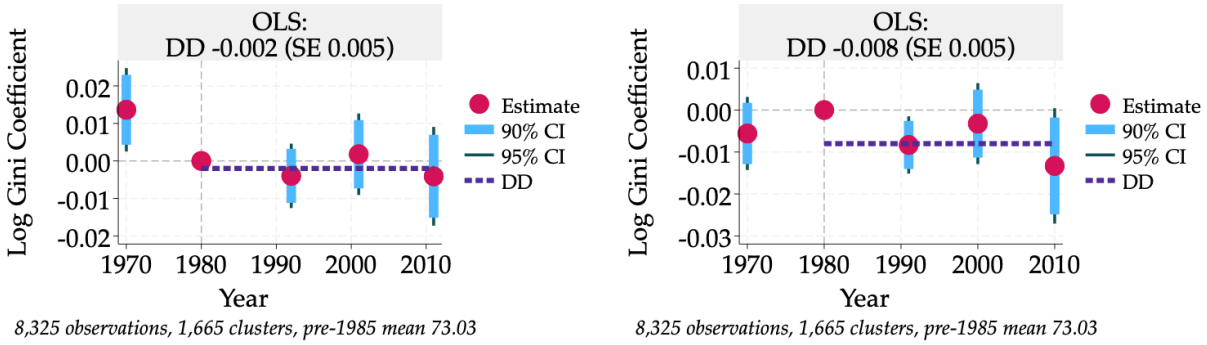
C2. Gini Coefficient Results Using New Difference-in-Differences Estimators

Figure C2: Gini Coefficient Results Using New Estimators [16]



(a) Arkhangelsky et al. (2021)

(b) Callaway and Sant'Anna (2021)



(c) de Chaisemartin and D'Haultfœuille (2020)

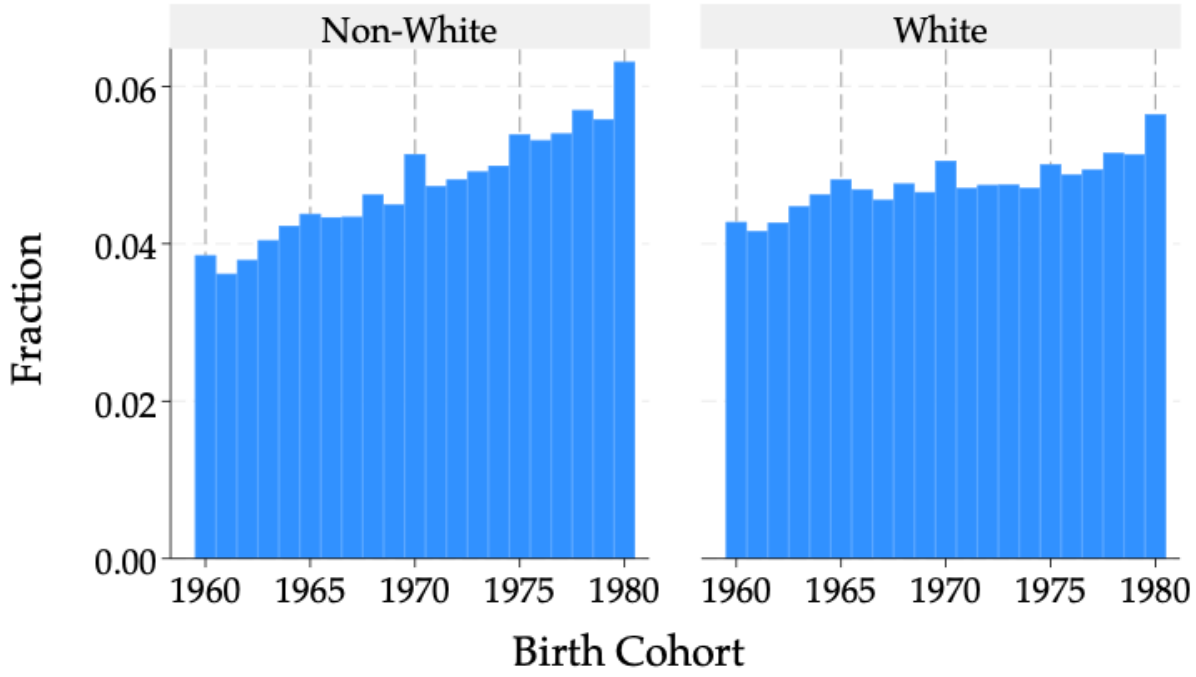
(d) Sun and Abraham (2021)

Notes: Observations are municipality-years. Graphs show dynamic and post-treatment average (DD) estimates when using a given estimator, with 90% and 95% confidence intervals for dynamic estimates except when using the Arkhangelsky et al. (2021) estimator. Data are from the IPUMS census samples. All regressions include fixed effects for year and municipality, and those using the de Chaisemartin and D'Haultfœuille (2020) and Sun and Abraham (2021) estimators include the interactions of year fixed effects with a vector of predetermined characteristics (shares of the 1980 population that were female, Asian, Black, and Brown, and changes in potential soy and maize yields when switching from traditional to genetically engineered seeds) and state fixed effects. Standard errors are clustered by municipality.

Appendix D. Additional Results: Effects on Individuals

D1. Age Heaping in Individual-Level Data

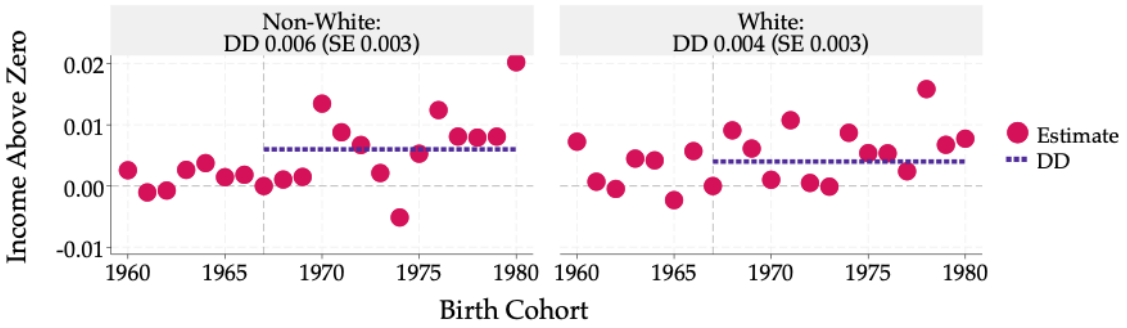
Figure D1: Histogram of Birth Years [22]



Notes: Data are from the IPUMS sample of the 2010 census.

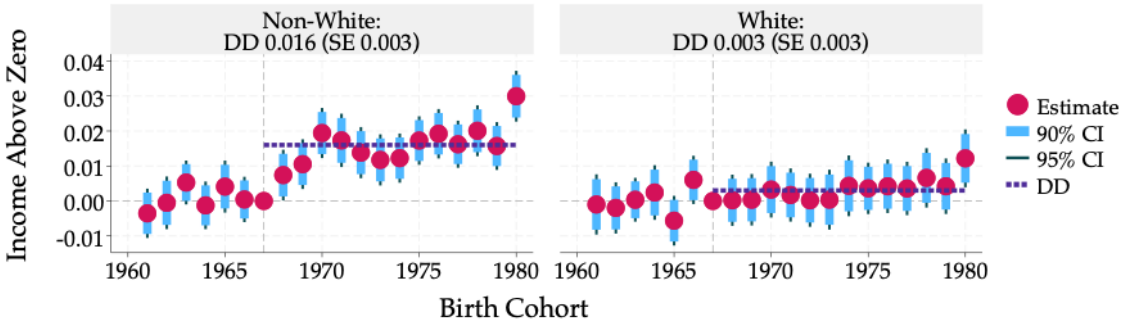
D2. Income Above Zero Results Using New Difference-in-Differences Estimators

Figure D2: Income Above Zero Results Using New Estimators [22]



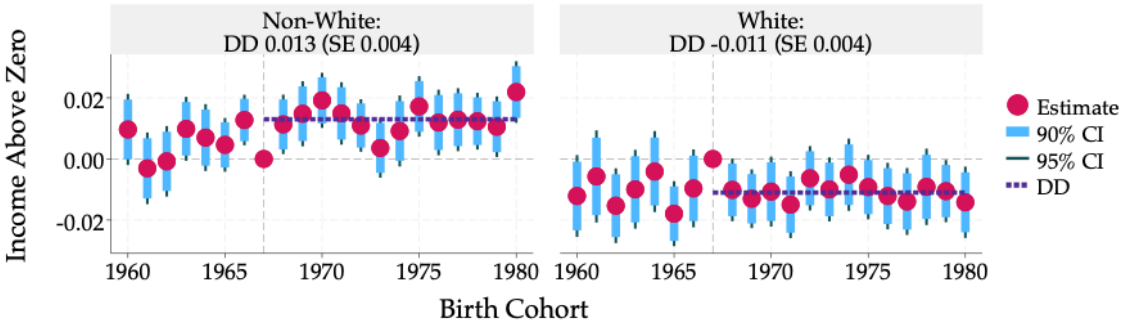
34,167-35,091 observations, 1,627 clusters, pre-1968 means: non-white 0.766, white 0.807

(a) Arkhangelsky et al. (2021)



2.23-2.79 million observations, 1,755 clusters, pre-1968 means: non-white 0.766, white 0.807

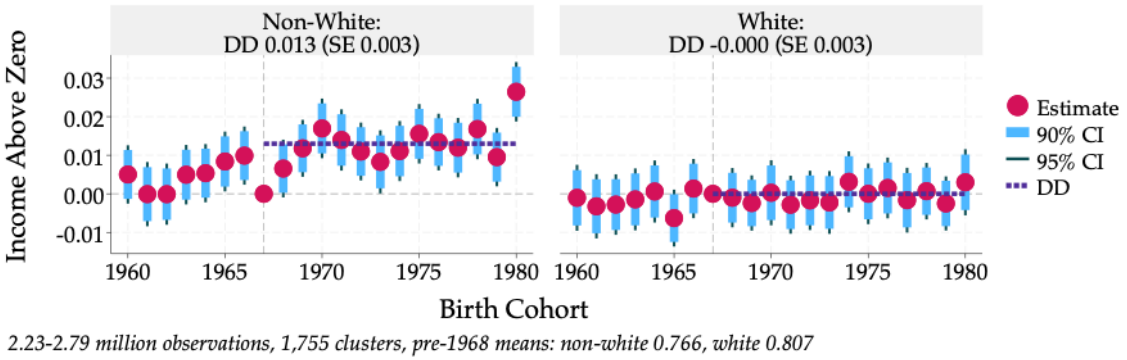
(b) Callaway and Sant'Anna (2021)



2.16-2.52 million observations, 1,755 clusters, pre-1968 means: non-white 0.766, white 0.807

(c) de Chaisemartin and D'Haultfœuille (2020)

Figure D2: Continued

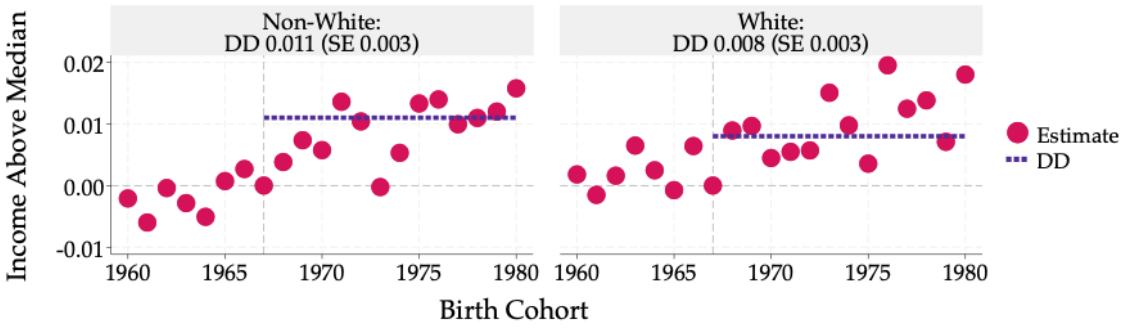


(d) Sun and Abraham (2021)

Notes: Graphs show dynamic and post-treatment average (DD) estimates for the respective subgroups when using a given estimator, with 90% and 95% confidence intervals for dynamic estimates in all but the top panel, which uses data collapsed to the municipality level. Data are from the IPUMS sample of the 2010 census. All regressions include fixed effects for birth cohort and birth municipality, and those using the de Chaisemartin and D'Haultfœuille (2020) and Sun and Abraham (2021) estimators include state-by-year and racial category fixed effects. Standard errors are clustered by birth municipality.

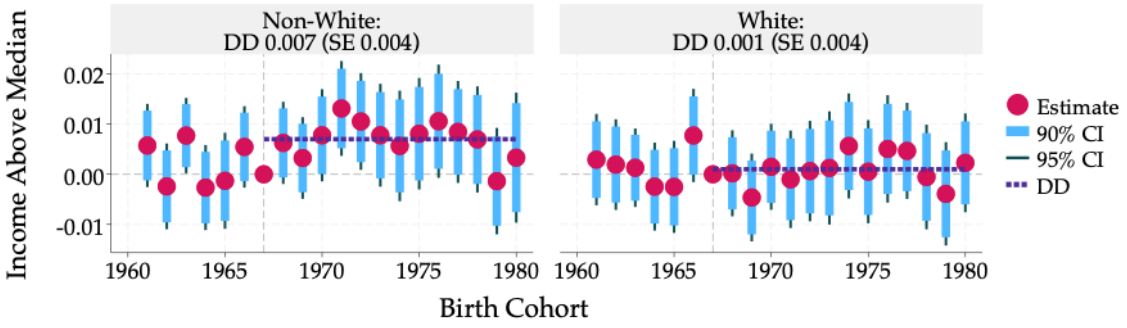
D3. Income Above Median Results Using New Difference-in-Differences Estimators

Figure D3: Income Above Median Results Using New Estimators [22]



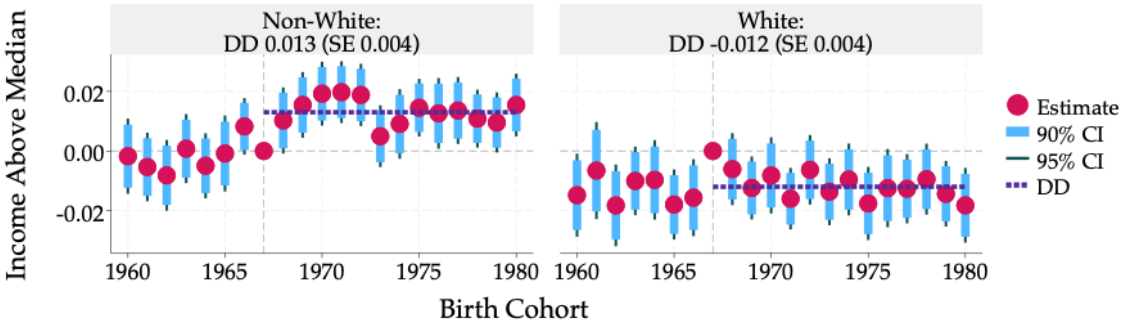
34,167-35,091 observations, 1,627 clusters, pre-1968 means: non-white 0.509, white 0.655

(a) Arkhangelsky et al. (2021)



2.23-2.79 million observations, 1,755 clusters, pre-1968 means: non-white 0.509, white 0.654

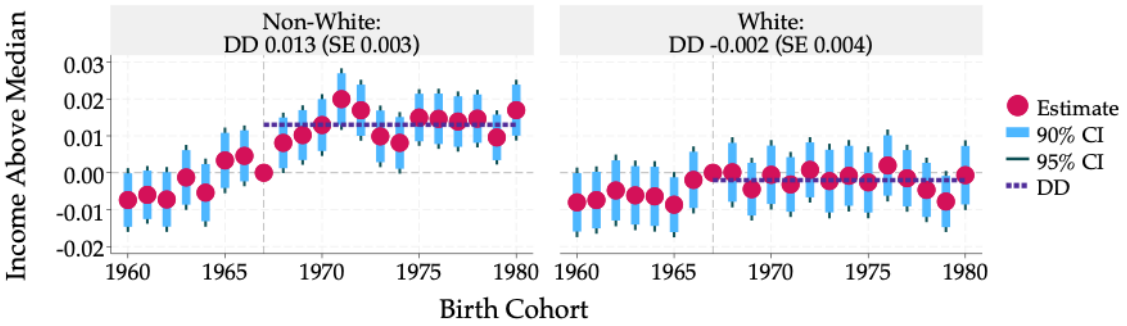
(b) Callaway and Sant'Anna (2021)



2.16-2.52 million observations, 1,755 clusters, pre-1968 means: non-white 0.509, white 0.654

(c) de Chaisemartin and D'Haultfœuille (2020)

Figure D3: Continued

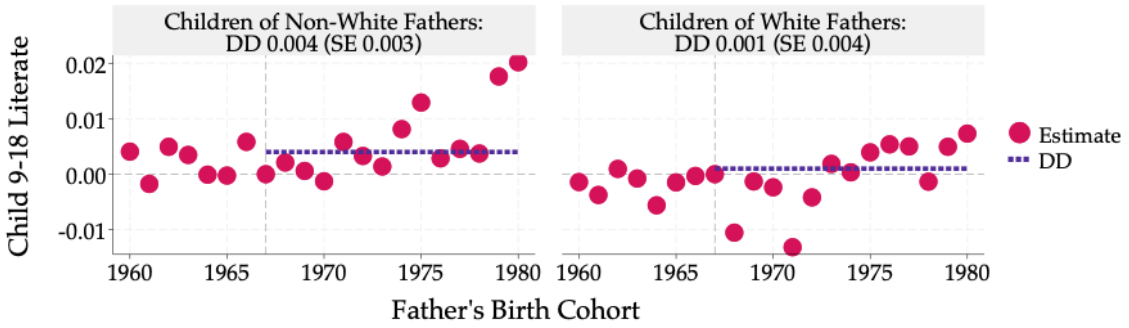


(d) Sun and Abraham (2021)

Notes: Graphs show dynamic and post-treatment average (DD) estimates for the respective subgroups when using a given estimator, with 90% and 95% confidence intervals for dynamic estimates in all but the top panel, which uses data collapsed to the municipality level. Data are from the IPUMS sample of the 2010 census. All regressions include fixed effects for birth cohort and birth municipality, and those using the de Chaisemartin and D'Haultfœuille (2020) and Sun and Abraham (2021) estimators include state-by-year and racial category fixed effects. Standard errors are clustered by birth municipality.

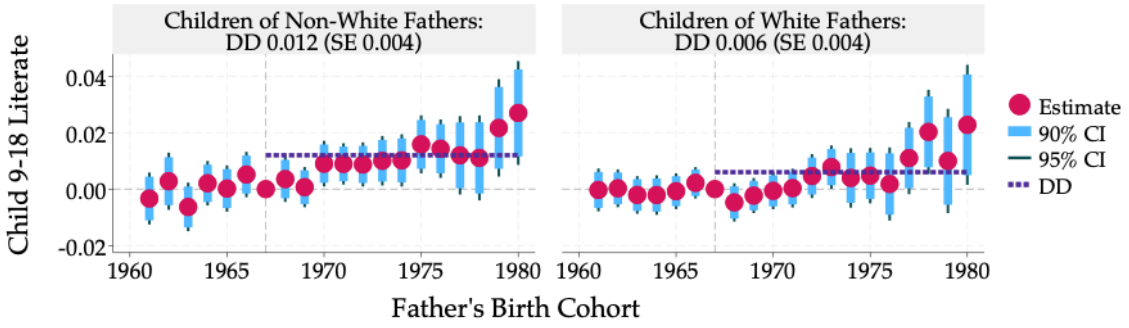
D4. Next Generation Literacy Results Using New Difference-in-Differences Estimators

Figure D4: Next Generation Literacy Results Using New Estimators [24]



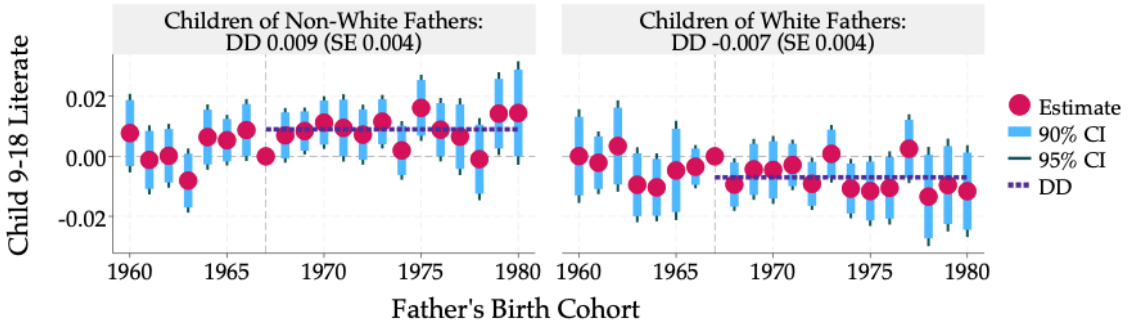
21,252-27,573 observations, 1,012 clusters, pre-1968 father's birth cohort means: non-white 0.921, white fathers 0.965

(a) Arkhangelsky et al. (2021)



0.87-1.34 million observations, 1,755 clusters, pre-1968 father's birth cohort means: non-white 0.921, white 0.963

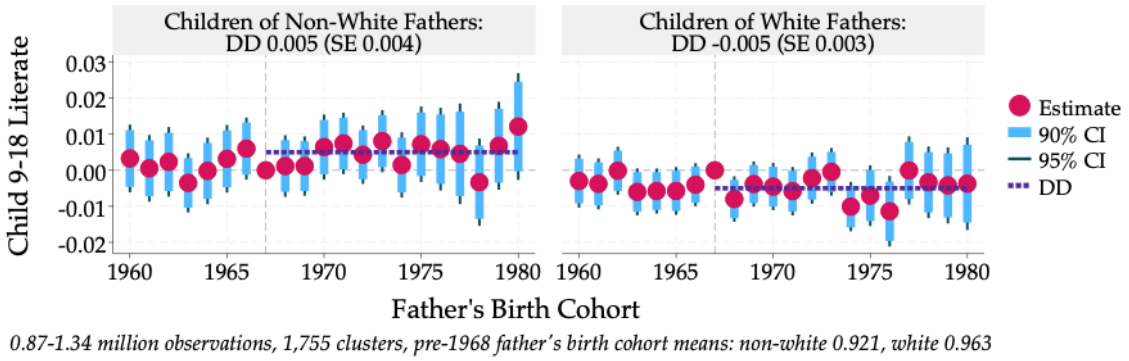
(b) Callaway and Sant'Anna (2021)



0.80-1.17 million observations, 1,755 clusters, pre-1968 father's birth cohort means: non-white 0.921, white 0.963

(c) de Chaisemartin and D'Haultfœuille (2020)

Figure D4: Continued

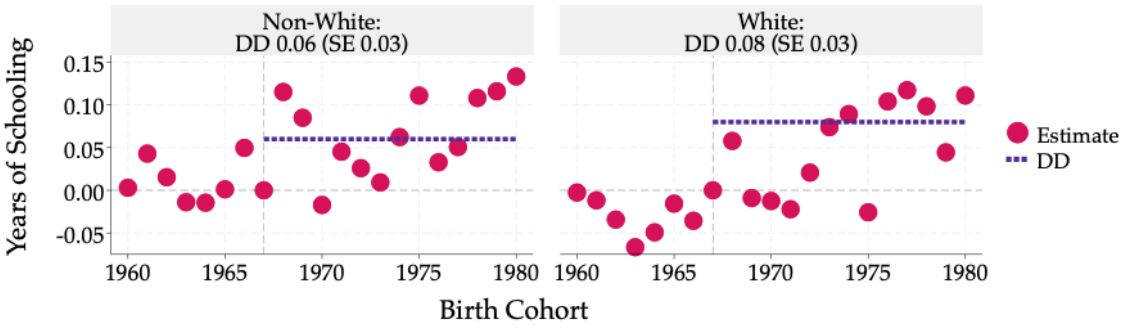


(d) Sun and Abraham (2021)

Notes: Graphs show dynamic and post-treatment average (DD) estimates for the respective subgroups when using a given estimator, with 90% and 95% confidence intervals for dynamic estimates in all but the top panel, which uses data collapsed to the municipality level. Data are from the IPUMS sample of the 2010 census. All regressions include fixed effects for birth cohort and birth municipality, and those using the de Chaisemartin and D'Haultfœuille (2020) and Sun and Abraham (2021) estimators include state-by-year and racial category fixed effects. Standard errors are clustered by birth municipality.

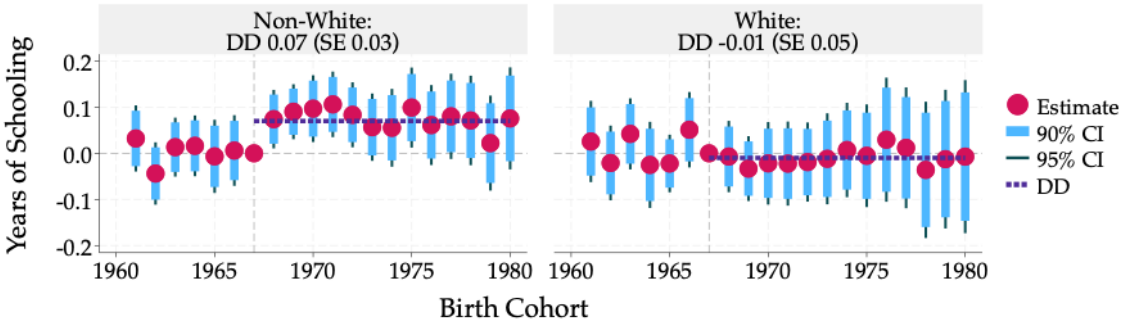
D5. Years of Schooling Results Using New Difference-in-Differences Estimators

Figure D5: Years of Schooling Results Using New Estimators [25]



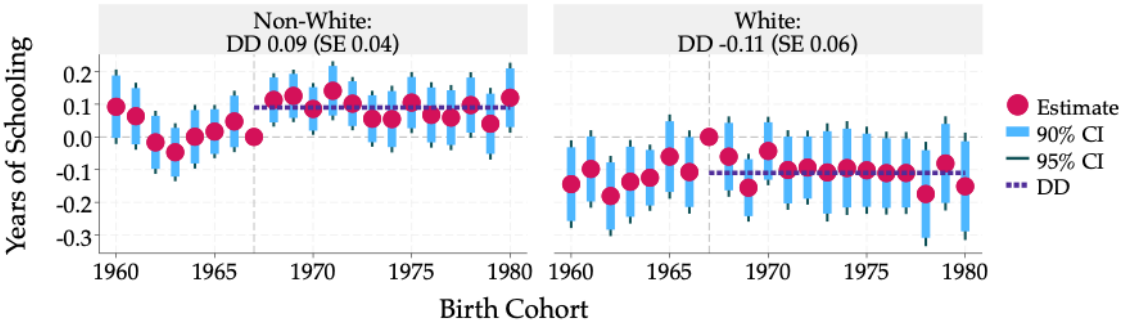
34,167-35,091 observations, 1,627 clusters, pre-1968 means: non-white 5.06, white 6.68

(a) Arkhangelsky et al. (2021)



2.23-2.79 million observations, 1,755 clusters, pre-1968 means: non-white 5.06, white 6.67

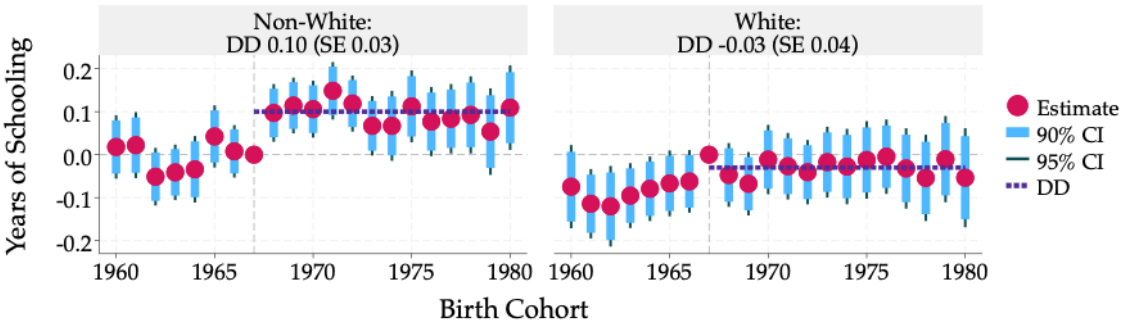
(b) Callaway and Sant'Anna (2021)



2.16-2.52 million observations, 1,755 clusters, pre-1968 means: non-white 5.06, white 6.67

(c) de Chaisemartin and D'Haultfœuille (2020)

Figure D5: Continued

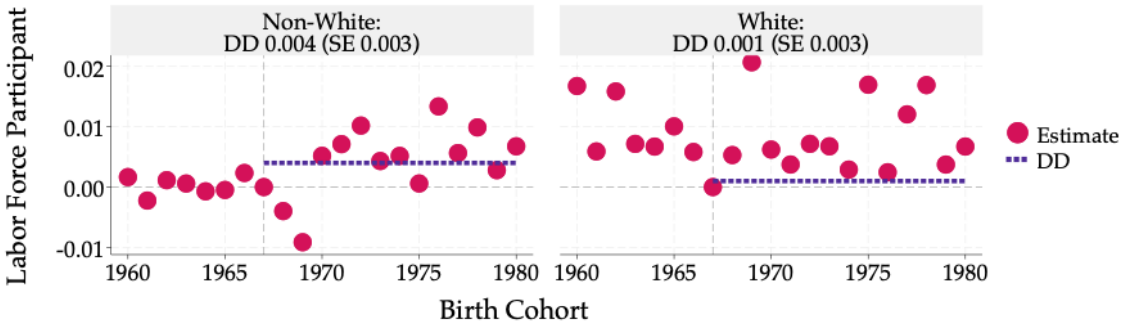


(d) Sun and Abraham (2021)

Notes: Graphs show dynamic and post-treatment average (DD) estimates for the respective subgroups when using a given estimator, with 90% and 95% confidence intervals for dynamic estimates in all but the top panel, which uses data collapsed to the municipality level. Data are from the IPUMS sample of the 2010 census. All regressions include fixed effects for birth cohort and birth municipality, and those using the de Chaisemartin and D'Haultfœuille (2020) and Sun and Abraham (2021) estimators include state-by-year and racial category fixed effects. Standard errors are clustered by birth municipality.

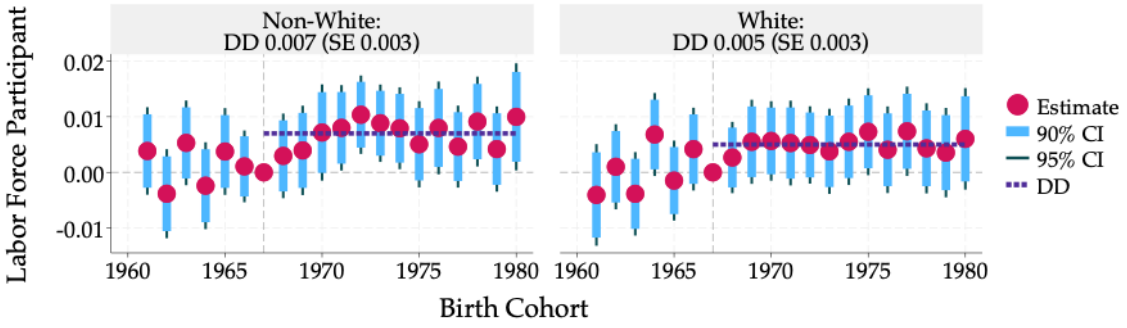
D6. Labor Force Participation Results Using New Difference-in-Differences Estimators

Figure D6: Labor Force Participation Results Using New Estimators [25]



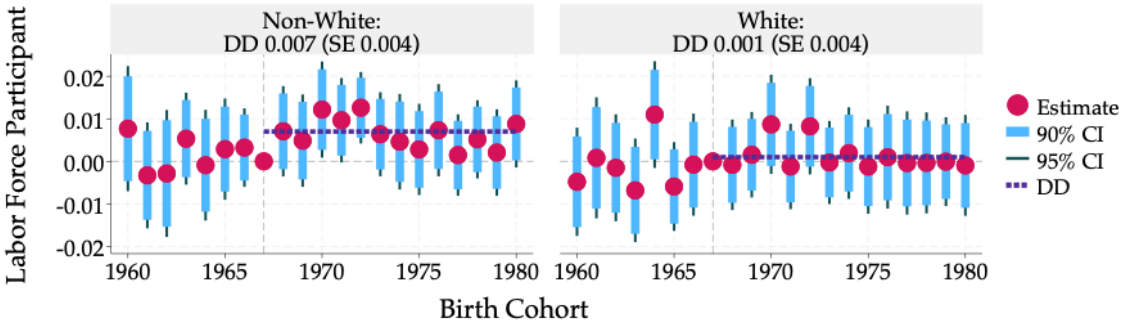
34,167-35,091 observations, 1,627 clusters, pre-1968 means: non-white 0.712, white 0.771

(a) Arkhangelsky et al. (2021)



2.23-2.79 million observations, 1,755 clusters, pre-1968 means: non-white 0.712, white 0.771

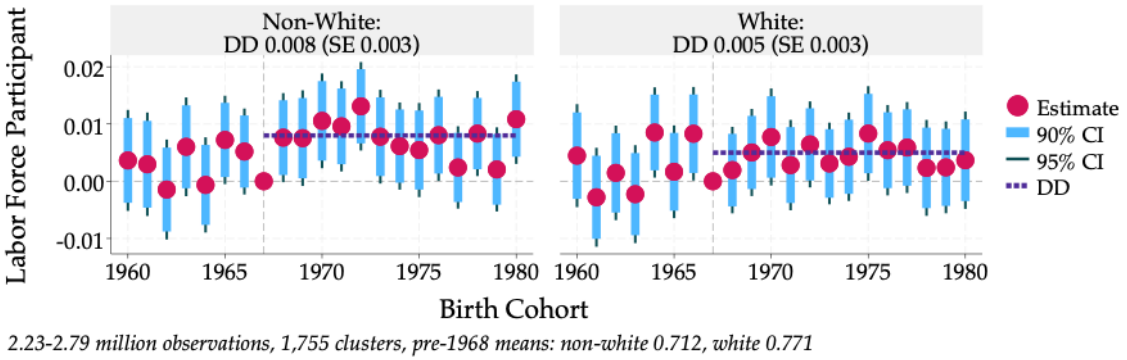
(b) Callaway and Sant'Anna (2021)



2.16-2.52 million observations, 1,755 clusters, pre-1968 means: non-white 0.712, white 0.771

(c) de Chaisemartin and D'Haultfœuille (2020)

Figure D6: Continued



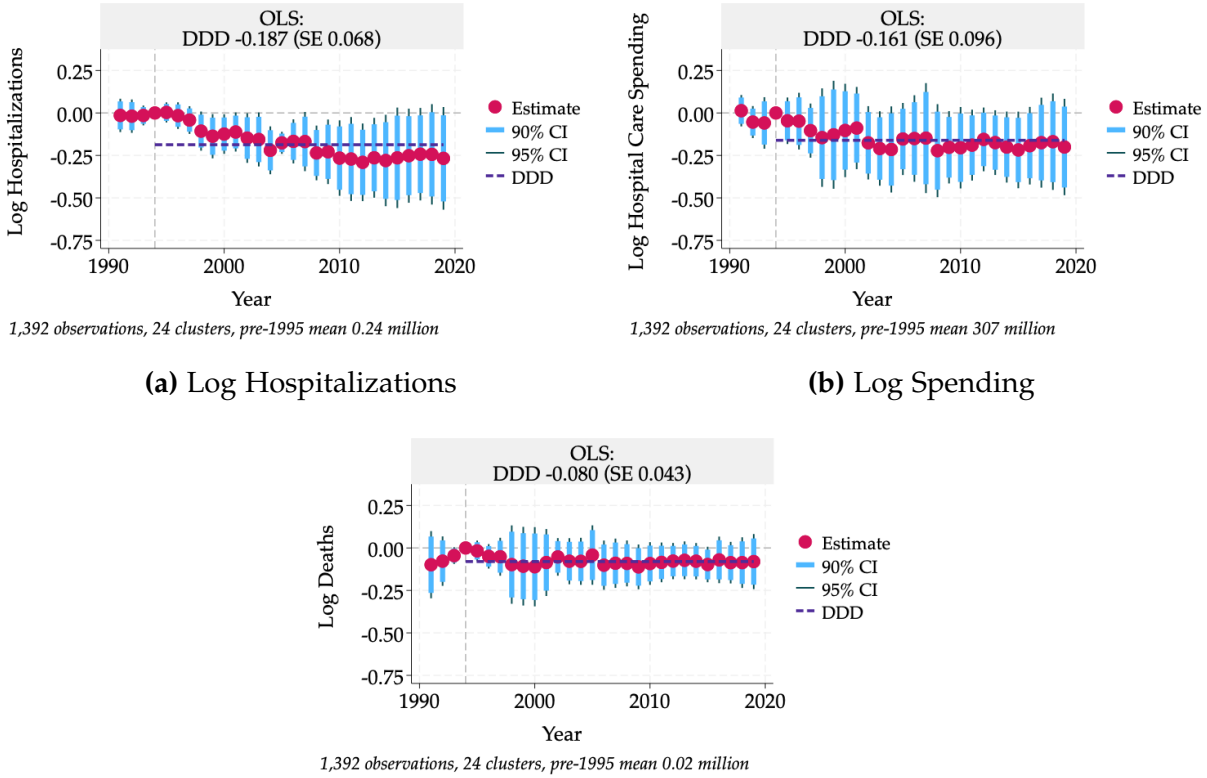
(d) Sun and Abraham (2021)

Notes: Graphs show dynamic and post-treatment average (DD) estimates for the respective subgroups when using a given estimator, with 90% and 95% confidence intervals for dynamic estimates in all but the top panel, which uses data collapsed to the municipality level. Data are from the IPUMS sample of the 2010 census. All regressions include fixed effects for birth cohort and birth municipality, and those using the de Chaisemartin and D'Haultfœuille (2020) and Sun and Abraham (2021) estimators include state-by-year and racial category fixed effects. Standard errors are clustered by birth municipality.

Appendix E. Additional Results: Effects on Public Health Care

E1. Health Care Results Using Traditional Confidence Intervals

Figure E1: Health Care Results Using Traditional Confidence Intervals [30]



(a) Log Hospitalizations

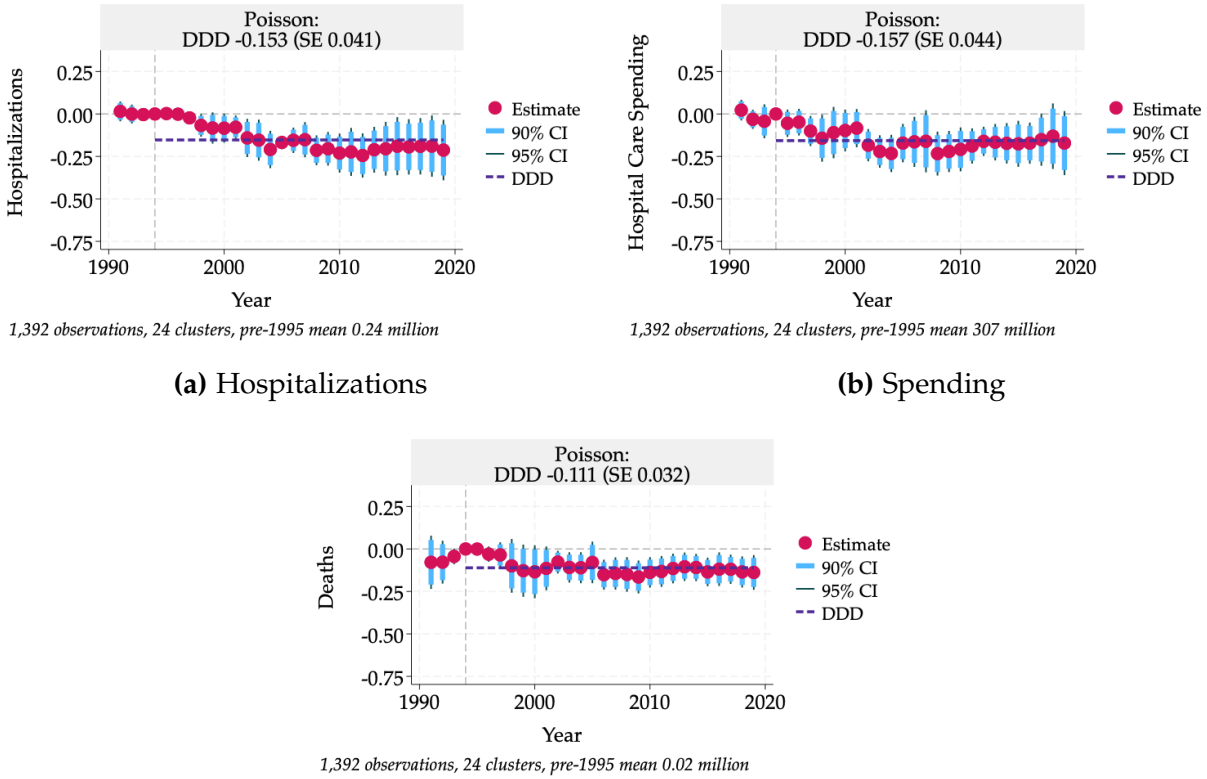
(b) Log Spending

(c) Log Deaths

Notes: Graphs show dynamic and post-treatment average (DDD) estimates for the respective outcomes, with 90% and 95% 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. Standard errors are clustered by state.

E2. Health Care Results Using Poisson Regression and Traditional Confidence Intervals

Figure E2: Health Care Results Using Poisson Regression and Traditional Confidence Intervals [30]



(a) Hospitalizations

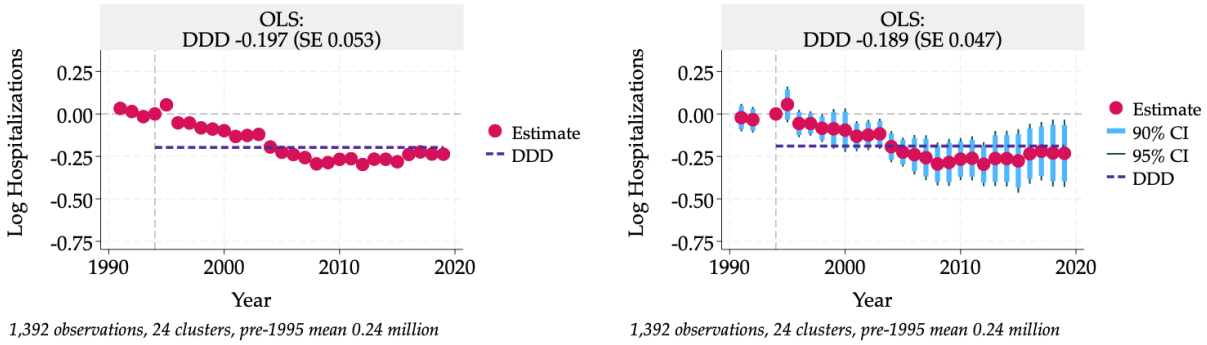
(b) Spending

(c) Deaths

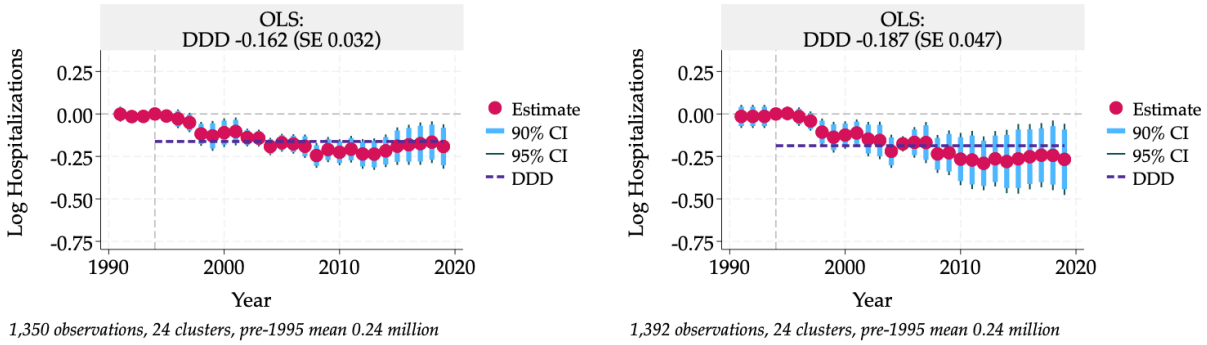
Notes: Graphs show dynamic and post-treatment average (DDD) estimates for the respective outcomes, with 90% and 95% confidence intervals for dynamic estimates. Data are from DATASUS. All regressions include fixed effects for state-year, year-disease category, and state-disease category and the interaction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

E3. Hospitalization Results Using New Difference-in-Differences Estimators

Figure E3: Hospitalization Results Using New Estimators [30]



(a) Arkhangelsky et al. (2021) **(b)** Callaway and Sant'Anna (2021)

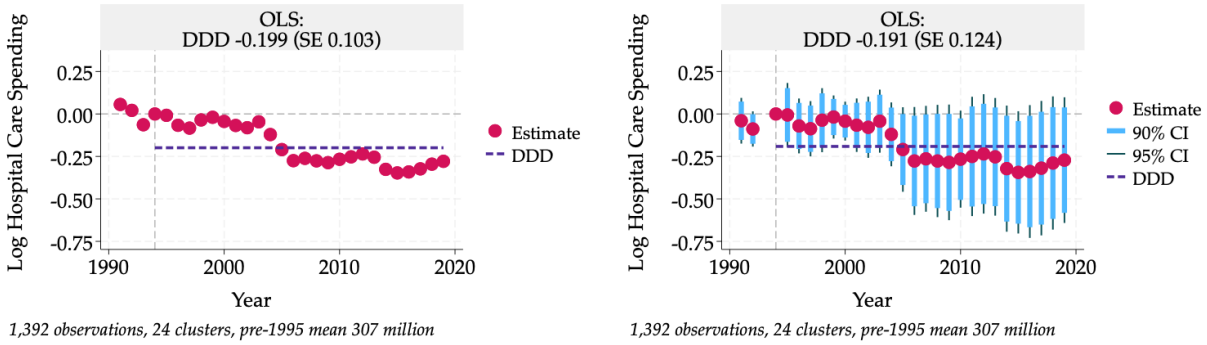


(c) de Chaisemartin and D'Haultfœuille (2020) **(d)** Sun and Abraham (2021)

Notes: Graphs show dynamic and post-treatment average (DDD) estimates for the respective outcomes, with 90% and 95% confidence intervals for dynamic estimates in all but the top panel. Data are from DATASUS. All regressions include fixed effects for state-year, year-disease category, and state-disease category, and those using the de Chaisemartin and D'Haultfœuille (2020) and Sun and Abraham (2021) estimators include the interaction of region and year-disease category fixed effects. Standard errors are clustered by state.

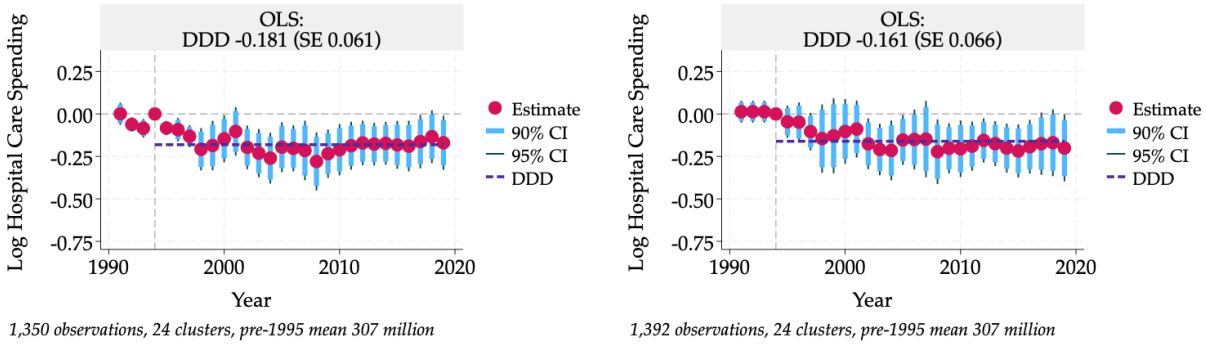
E4. Hospital Care Spending Results Using New Difference-in-Differences Estimators

Figure E4: Hospital Care Spending Results Using New Estimators [30]



(a) Arkhangelsky et al. (2021)

(b) Callaway and Sant'Anna (2021)



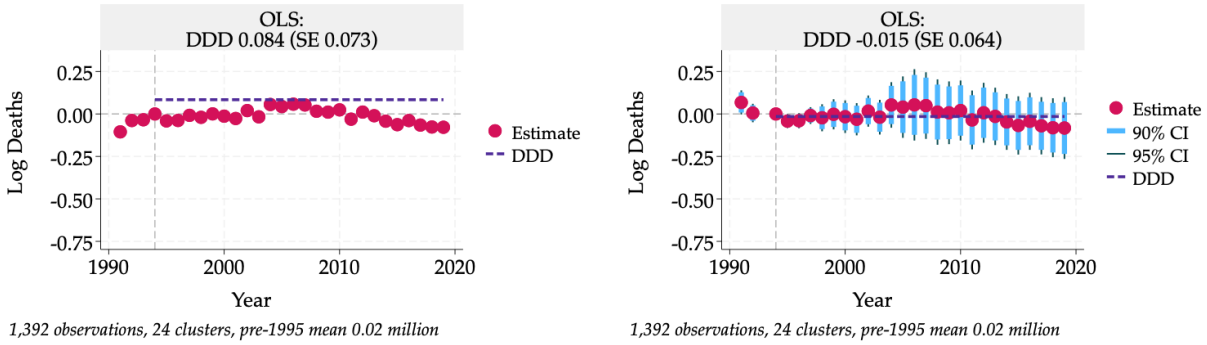
(c) de Chaisemartin and D'Haultfœuille (2020)

(d) Sun and Abraham (2021)

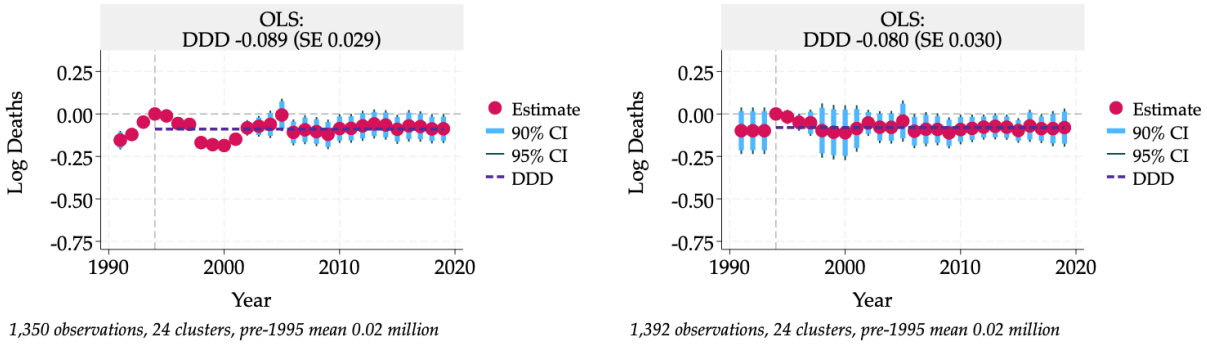
Notes: Graphs show dynamic and post-treatment average (DDD) estimates for the respective outcomes, with 90% and 95% confidence intervals for dynamic estimates in all but the top panel. Data are from DATASUS. All regressions include fixed effects for state-year, year-disease category, and state-disease category, and those using the de Chaisemartin and D'Haultfœuille (2020) and Sun and Abraham (2021) estimators include the interaction of region and year-disease category fixed effects. Standard errors are clustered by state.

E5. Death Results Using New Difference-in-Differences Estimators

Figure E5: Death Results Using New Estimators [30]



(a) Arkhangelsky et al. (2021) **(b)** Callaway and Sant'Anna (2021)



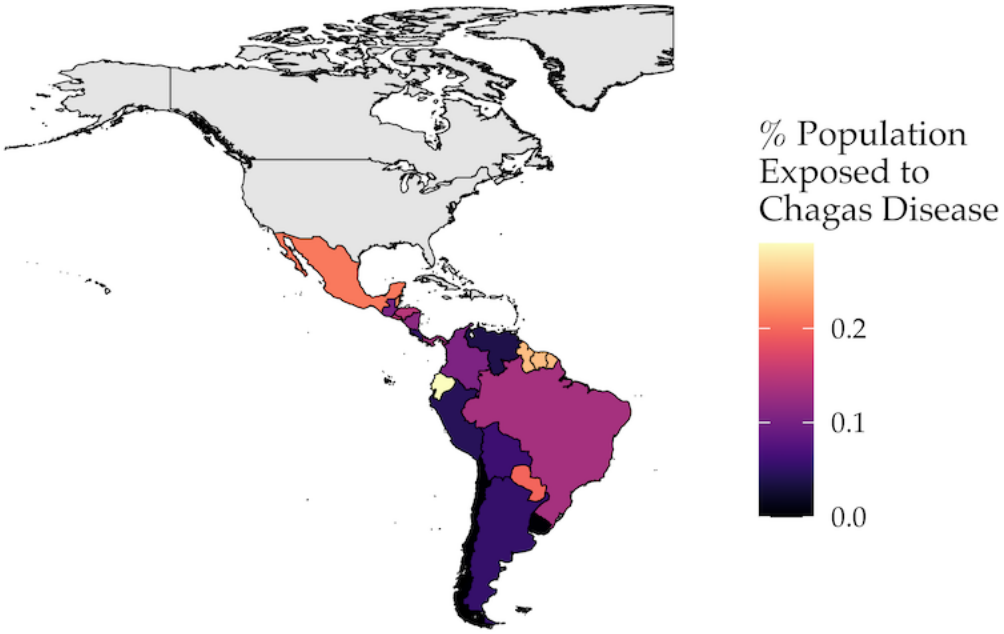
(c) de Chaisemartin and D'Haultfœuille (2020) **(d)** Sun and Abraham (2021)

Notes: Graphs show dynamic and post-treatment average (DDD) estimates for the respective outcomes, with 90% and 95% confidence intervals for dynamic estimates in all but the top panel. Data are from DATASUS. All regressions include fixed effects for state-year, year-disease category, and state-disease category, and those using the de Chaisemartin and D'Haultfœuille (2020) and Sun and Abraham (2021) estimators include the interaction of region and year-disease category fixed effects. Standard errors are clustered by state.

Appendix F. Additional Figures: Cost-Benefit Analyses and Extrapolation

F1. Estimated Population Exposure to Chagas Disease

Figure F1: Estimated Population Exposure to Chagas Disease [35]



Notes: Map shows the estimated percent of the population in each Latin American country exposed to Chagas disease from the World Health Organization (2015). Countries without estimates are in light gray.