

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

Jon Denton-Schneider[†]

Clark University

Eduardo Montero[‡]

University of Chicago

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Abstract

Neglected tropical diseases (NTDs) afflict the world's poorest people and often lead to decades-long health problems. Combating them could thus reduce inequality, the intergenerational transmission of poverty, and burdens on healthcare systems in developing economies. We show that these novel benefits arose from Brazil's initial campaign to eliminate Chagas disease (1984-89), an NTD that occurs almost entirely among poor, non-white, and rural Latin Americans and can cause chronic heart problems. Exploiting the pre-treatment presence of its main vector, we find that Chagas disease control increased municipalities' GDP per capita by 9.8% and reduced their Gini coefficients by 1.1% in the long run. Furthermore, having a childhood free of exposure to this NTD raised non-white adults' incomes by 1 percentile in the national distribution (2 to 4%) and their daughters' literacy rates by 0.5 percentage points (0.5%). Coinciding with the expected reduction in chronic symptoms, we also show that public spending on circulatory disease hospital care declined by 16%, contributing to an internal rate of return of 25% 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.

Keywords: Tropical Disease, Racial Inequality, Intergenerational Mobility, Health Spending

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

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[†]Clark University, Department of Economics, 950 Main St, Worcester, MA 01610. Email: jdentonschneider@clarku.edu. Website: jondentonschneider.com

[‡]University of Chicago, Harris School of Public Policy, 1307 E 60th St, Chicago, IL 60637. Email: emontero@uchicago.edu. Website: www.eduardo-montero.com.

[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](#))

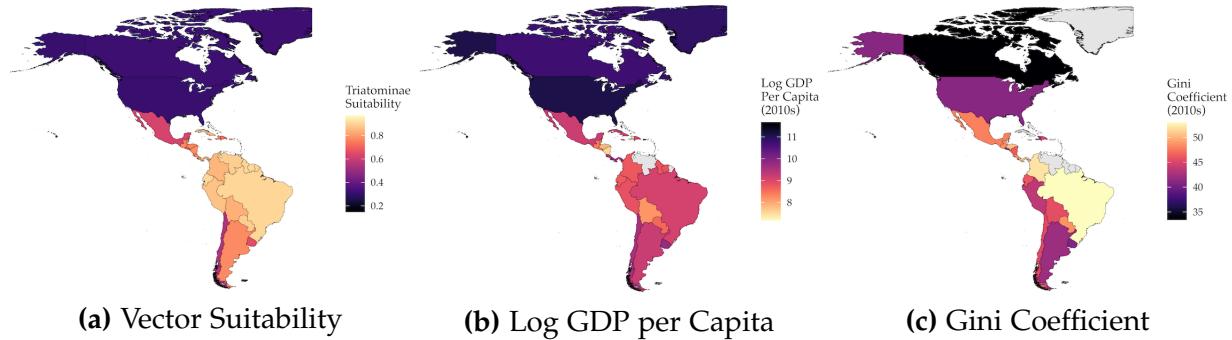
1. Introduction

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 (racial) disparities and underdevelopment is Chagas disease, which afflicts 8 million predominantly non-white people in Latin America and to which another 75 million are exposed ([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 can 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 by using the start of Brazil's campaign to eliminate vectorial transmission of Chagas disease (1984-89) and a difference-in-differences approach. 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



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. To explore this further, we follow [Alsan \(2015\)](#) in testing for a continent-specific relationship.² Using Poisson regressions, we examine the impact of the suitability index and its interaction with an indicator for being in the Americas on these outcomes. Table 1 shows that they are very strongly related within the Western Hemisphere, particularly for income inequality.³

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: as we describe in Section 2, 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 transmission of Chagas disease. Specifically, we study the consequences of the first years (1984-89) of indoor residual spraying (IRS) against the main Chagas disease vector in Brazil—a country that Figure 1 shows has high vector suitability, a middle income, and very

² [Alsan \(2015\)](#) studies African trypanosomiasis, or sleeping sickness. Chagas disease is known as American trypanosomiasis, but only because it is also caused by parasites of the genus *Trypanosoma*. However, there is no overlap in these species, their vectors, or the diseases' symptoms.

³ In addition, we show in Appendix A1 that a greater recorded presence of this parasite in precolonial Latin America was associated with lower levels of centralization in the Standard Cross-Cultural Survey ([Murdock and White, 1969](#)), suggesting that it may have hindered the formation of more complex societies.

Table 1: Continent-Specific Effects of Chagas Disease

	GDP per Capita		Gini Coefficient	
	(1)	(2)	(3)	(4)
<i>Poisson Regression Coefficients</i>				
Chagas Suitability	-0.017 (0.005)	-0.017 (0.005)	0.002 (0.001)	0.001 (0.001)
Chagas Suitability $\times 1[\text{Americas}]$	-0.021 (0.009)	-0.024 (0.009)	0.005 (0.001)	0.005 (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. \(2020\)](#). $1[\text{Americas}]$ 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.

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.

In Section 3, we describe our basic difference-in-differences strategy that combines cross-municipality and cross-year variation in exposure to the main Chagas disease vector. For the former, our focus is on comparing municipalities never infested by the vector with those that were rid of it by 1989. Following recent developments in the difference-in-differences literature ([Goodman-Bacon, 2021](#)), we exclude municipalities that were treated later and those in the state of São Paulo, which was wealthy enough to conduct its own vector control in the 1960s with a more expensive insecticide. With respect to time variation, we lack information on when in the 1984-89 period a municipality was treated, so we make the conservative assumption that it occurred uniformly in 1984.

We first study the long-run impacts of eliminating Chagas disease transmission on income and inequality across municipalities in Section 4. We compare treatment and control municipal-

ties using population censuses and data on GDP per capita between 1970 and 2010. We find that after 1984, treatment municipalities experienced larger increases in their residents' average percentiles in the national income distribution (0.7 percentage points [p.p.], or 2% of the mean) and their GDP per capita (9.8%), and had larger decreases in their Gini coefficients (1.1%).⁴ When examining mechanisms, we find no substantive increases in years of schooling. Instead, to paraphrase the [World Health Organization \(2010\)](#) quote above, the evidence is consistent with individuals filling the labor force instead of hospital beds: we find that the share of individuals working for a wage or salary—which we view as a proxy for being able to work consistently—increased by 1.8 p.p. (6%) more in treatment municipalities. Importantly, all of these effects arose in the first post-treatment year and grew more pronounced over time, suggesting that reducing chronic Chagas disease augmented the benefits of averting its acute symptoms.

In Section 5, 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: incomes of non-white Brazilians from treatment municipalities increased by 1 percentile (2 to 4%) more than for non-white adults from the control group, but there were no differences for white Brazilians across these groups. This effect resulted from around 1.5 p.p. (2 to 4%) of non-white individuals shifting from reporting no income to one above the median.⁵ We also show that the literacy of daughters of non-white men from treatment municipalities increased by 0.5 percentage points (0.5%), suggesting that Chagas disease control could contribute to breaking intergenerational cycles of (racial) poverty. Consistent with our municipality-level results, we find little evidence that these results are due to treated adults having completed more schooling. Instead, we find that formal employment increased 2 p.p. (6% to 9%) more among treated non-white Brazilians.

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, as circulatory diseases account for 10% of hospitalizations and 20% of

⁴ We use income ranks across time rather than levels to avoid problems arising from Brazil's hyperinflation in the mid-1980s and early 1990s.

⁵ We use these transformations of income rather than its log following the recommendations of [Chen and Roth \(2024\)](#). The problems they identify are especially important when the extensive margin effect is large, as is the case in our results.

its spending on hospital care. In Section 6, we therefore examine hospitalizations, person-days spent in the hospital, and 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%), hospital days (15%), and spending on hospital care (16%) resulting from circulatory system problems than from all others. 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.

In Section 7, 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 25%. A key lesson from this exercise is that including the hospital care savings increases the IRR by 2 p.p. (8%), 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.⁶ As such, another important lesson is that if developing country governments 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 popula-

⁶ It would take a discount rate above 9% 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](#)).

tion exposed to transmission (World Health Organization, 2015) and the percent effects on these outcomes for Brazilian municipalities estimated in Section 4. Our calculations suggest that on average, this intervention could increase Latin American incomes per person by 2% and reduce Gini coefficients by 0.3%. Notably, greater benefits would accrue to countries in the region with higher shares exposed to Chagas disease, which are precisely the countries that are more underdeveloped and unequal today.

Our results therefore contribute to several literatures. The first is the 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; Depetrise-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 scarce resources dedicated to promoting economic development.

Second, our study contributes to the broader literature on health and inequality (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 Bütkofer and Salvanes (2020), who find that Norway's control of tuberculosis (TB)—another disease that primarily affects 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 these benefits obtain when controlling an NTD in a developing country setting, there is a racial dimension to the ensuing reduction in inequality, and averting the NTD's chronic stage results in substantial cost savings and compounds the effects of averting its acute symptoms.

Lastly, our results add to our understanding of the comparative development of Latin America. A growing literature 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 its unique development trajectory. We also provide quasi-experimental evidence that eliminating Chagas disease transmission can have important and novel long-run benefits for the region such 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 global health challenge.

2. Chagas Disease and Its Control in Brazil

2.1. Vectors and Disease Progression

The parasite *Trypanosoma cruzi* causes Chagas disease, also known as American trypanosomiasis. Around 90% 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 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

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*.

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%).

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

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*.

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

Due to this "development of suitable vector control methods ... [and] 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 visited millions of homes across the endemic region to conduct indoor residual spraying (IRS).¹¹ However, the program was paused in 1986 due to outbreaks of dengue fever in coastal (i.e., tourist) areas, leading public health officials to divert sprayers to the control of this mosquito-borne disease. Triatomine bugs thus reestablished themselves in some recently-sprayed municipalities ([Dias, 1987](#); [Schofield and Dias, 1999](#)), but there was nonetheless a clear decline in the presence of *T. infestans* before Chagas disease vector control restarted in 1989 (see Appendix B4 from [Coura and Dias, 2009](#)). The program continued into the 21st century, and by 2006, the Pan American Health Organization certified that Brazil had interrupted transmission of Chagas disease by *T. infestans*.

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

¹¹ See Appendix B5 for images from [Dias \(1987\)](#) of insecticide being sprayed on rural homes with many cracks and spaces in their roofs and walls for *T. infestans* to colonize.

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. Treatment, Control, and Excluded Municipalities

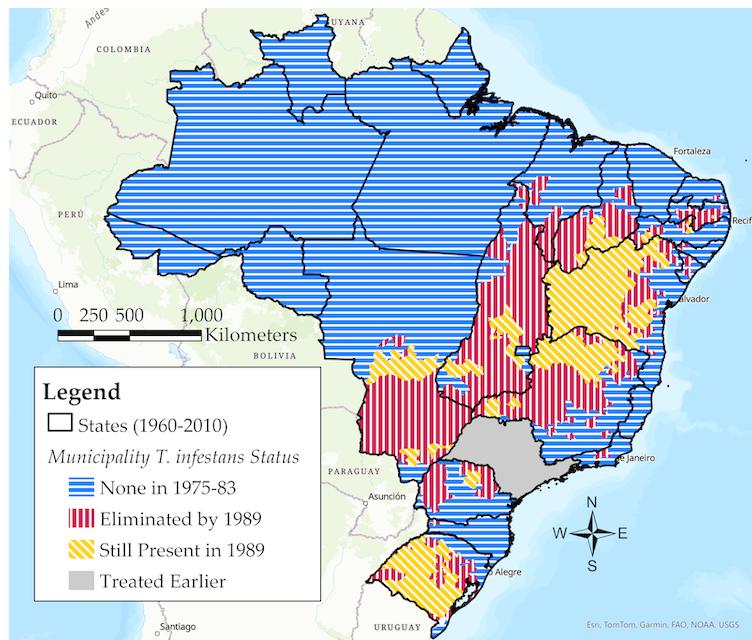
To determine which municipalities to compare, we combine the maps in Appendix B4 showing the presence of *T. infestans* in the entomological surveys before (1975–83) and after IRS began but had been interrupted (1989). Figure 2 shows the resulting four categories of municipalities. Our control group consists of those in which the vector was not found prior to spraying, so we assume that they were never exposed to transmission of Chagas disease via *T. infestans*.¹² Our treatment group is comprised of municipalities in which the vector was present before IRS began but had been eliminated by 1989. We thus exclude from our sample those where the vector was observed again in that year (i.e., treated much later, where we also lack information on the treatment date) and the state of São Paulo (treated earlier).¹³ We thus examine the effects of eliminating vectorial transmission of Chagas disease over the longest time horizon and limit our comparisons to a single treatment time (Goodman-Bacon, 2021).

Table 2 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 vulnerable to vector infestation—which we define as having a roof made of wood, straw, or scraps of material, walls made of uncoated lathe and plaster or straw, or a floor made of dirt—than the white

¹² 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 underestimate the true effects.

¹³ Note that the first of these groups includes municipalities where the vector may have been temporarily eliminated but the interruption in spraying allowed it to return. Subsequent control campaigns eliminated Chagas disease transmission for this group, but we lack information on when it occurred.

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



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

population, consistent with racial disparities in exposure to Chagas disease transmission.¹⁴ The means in Panel C imply a correlation between greater exposure to Chagas disease and worse economic development outcomes. It is also associated with somewhat greater within-municipality income inequality (Panel D).

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

We cannot observe when in the 1984-89 period vectorial transmission of Chagas disease was eliminated in a municipality, so we assume that it occurred uniformly throughout the treatment group in 1984. The graph of hospitalizations for acute Chagas disease in Figure 3 suggests that this assumption is conservative, as IRS appeared to reduce this measure of *T. cruzi* transmission

¹⁴ The fact that a much greater share of the population in municipalities where the vector was still present in 1989 were poorer and living in vulnerable housing suggests that it could have been more difficult to eliminate there. One implication is that the benefits of eliminating transmission estimated by comparing our treatment and control groups might underestimate the effects on the later-treated group. However, the consequences for the cost-benefit analysis are more ambiguous, as it was likely more expensive to eliminate *T. infestans* from these areas.

Table 2: Summary Statistics, 1980

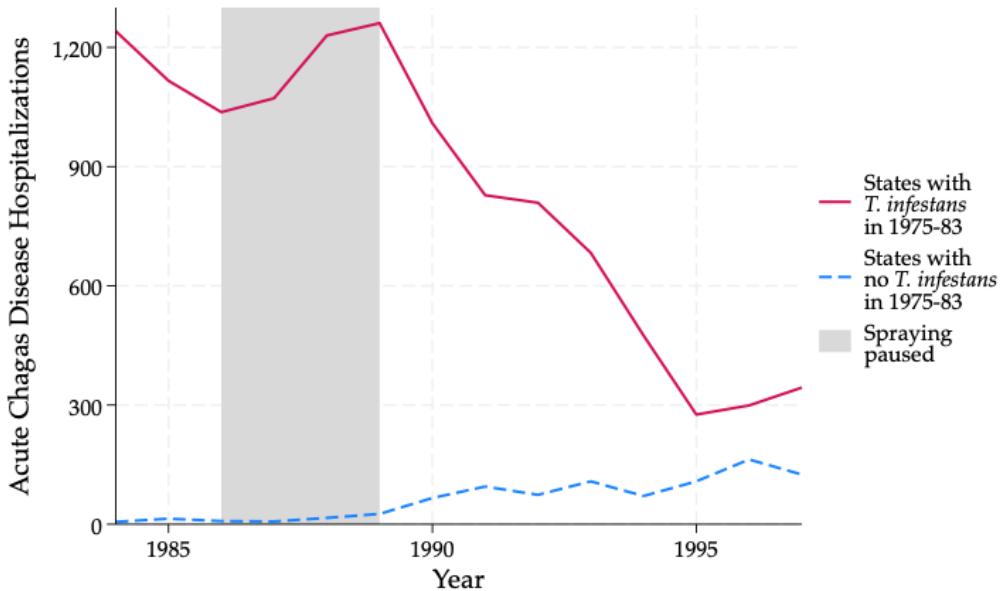
	Municipalities by 1975-89 <i>T. infestans</i> Status			
	None (Control)	Eliminated (Treatment)	Still Present (Excluded)	Treated Earlier (Excluded)
<i>Panel A. Demographics</i>				
Age	24.01 (19.12)	24.34 (19.16)	24.23 (19.45)	25.95 (18.93)
Female	0.507	0.502	0.502	0.501
Asian	0.002	0.003	0.003	0.019
Black	0.065	0.056	0.060	0.045
Brown	0.473	0.352	0.393	0.182
White	0.460	0.589	0.544	0.754
<i>Panel B. Living in Housing Vulnerable to Infestation</i>				
Non-White	0.341	0.353	0.463	0.056
White	0.112	0.116	0.173	0.026
<i>Panel C. Labor Market Outcomes and Human Capital</i>				
Income Percentile	36.80 (36.71)	36.72 (36.42)	34.14 (35.45)	48.01 (39.66)
Years of Schooling: Adults 19+	3.70 (3.86)	3.43 (3.62)	2.98 (3.43)	4.68 (3.90)
Literate: Children 10-18	0.768	0.802	0.752	0.955
Wage/Salary Worker	0.320	0.306	0.247	0.464
<i>Panel D. Income Inequality</i>				
Gini Coefficient	76.30 (4.02)	76.67 (4.66)	77.56 (3.21)	72.31 (3.17)
Observations	16,431,739	4,554,538	2,057,166	6,181,009
Municipalities	1,156	402	173	309

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

but it did not immediately fall to zero.¹⁵ When studying the impacts on municipalities and states, our final pre-treatment year is thus 1983 when using annual data and 1980 when comparing across decennial censuses. However, in our examination of the effects of additional childhood years free from exposure to Chagas disease (e.g., Bleakley, 2010; Cutler et al., 2010; Bütkofer 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).

¹⁵ 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 control group municipalities (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.

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 cohorts or municipalities. 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 incomes and reduced inequality, and that these effects become more pronounced after 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. We also find no substantive increases in schooling but clear increases in employment rates, suggesting that improved health is much more important in explaining the effects on income and inequality.

4.1. Data and Empirical Strategy

For most of our outcomes, we collapse individual-level data 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 We examine the average rank in a given census wave's monthly income distribution, the Gini coefficient for incomes within a municipality, average years of schooling, and the share of the population that were wage or salary workers.¹⁷ We consider the first two outcomes to be the main ones of interest and the last two as potential mechanisms (i.e., increased education and the ability to hold a regular job due to improved health). In addition, we calculated municipalities' GDP per capita (in constant 2019 R\$) in 8 years in this period using data from Ipeadata.¹⁸ However, because the 1970 census does not contain information on race (and the same is true for all GDP per capita estimates), we cannot assess parallel trends assumptions within subgroups.¹⁹

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

¹⁷ We use income percentiles instead of a log transformation for two reasons: as we discuss in Section 5.1.1, OLS estimates using outcomes in logs are not unit-invariant, making them difficult to interpret as percentage effects ([Chen and Roth, 2024](#)), and percentiles help us avoid problems stemming from Brazil's hyperinflation and currency changes from the mid-1980s to the early 1990s. For wage and salary workers, we only consider those with legal contracts to be in this category, and we calculate the share in the entire population (i.e., including the unemployed and those not in the labor force).

¹⁸ 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.

¹⁹ In Section 5, we use individual-level data from 2010 to examine impacts by racial group and sex.

Instead, we use averages from each municipality m in year t to estimate

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

where $y_{m,t}$ is an outcome of interest for municipality m in year 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 year k , $\mathbf{X}_{m,1980}$ is a vector of m 's characteristics in 1980 (the female, Asian, Black, and Brown shares of the population), $\delta_{s(m)}$ is a fixed effect for m 's state, and $\epsilon_{m,t}$ is the idiosyncratic error term. We interact $\mathbf{X}_{m,1980}$ and $\delta_{s(m)}$ with γ_t to control for 1980 characteristics' time-varying influences and state-specific trends across years.

The coefficients of interest are the τ_k , which measure the difference in an outcome in a given census year across the treatment and control groups, relative to the size of that difference in 1980. 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 OLS (income percentile, years of schooling, and wage or salary work) and Poisson regression (Gini coefficient) with standard errors clustered by municipality, and we weight observations by their 1980 population to account for differences in municipality sizes.²⁰ 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 years. 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).²¹

4.2. Cross-Census Results

We first study the effects of IRS on the average percentile in the national monthly income distribution within a municipality, which avoids estimation and interpretation problems due to Brazil's hyperinflation between the mid-1980s and early 1990s. The top-left panel of Figure 4a shows

²⁰ We use Poisson regression when examining the Gini coefficient to recover a percentage effect for our cross-country extrapolation in Section 7.3.

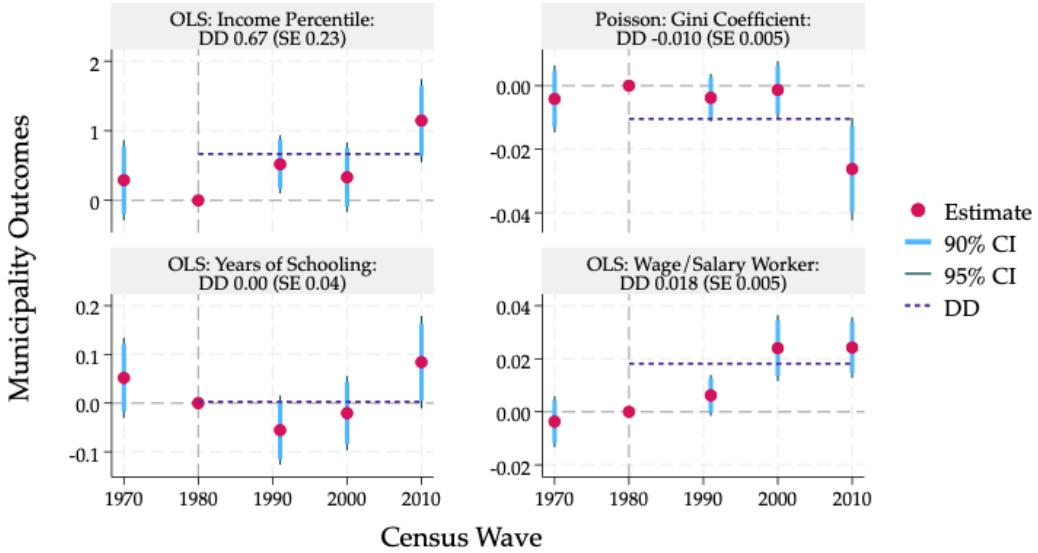
²¹ Nonetheless, we emphasize that our approach of comparing unexposed areas to those exposed at one treatment time (i.e., non-staggered) does not suffer from many of these shortcomings.

that the mean ranking increased 0.67 p.p. (2%) more in treatment municipalities over this period, and this estimate is precise. Next, to examine income inequality within municipalities, we use each municipality-year's Gini coefficient, which is another unit-invariant measure. Using Poisson regression, the top-right panel shows that it decreased by a precisely estimated 1.1% more in the treatment group, providing further evidence that Chagas disease control reduced income inequality in Brazil. In addition, the patterns in both of these panels is consistent with effects that manifested soon after vector control began and became larger with time.

To understand the mechanisms driving these results, we also examine the effects of Chagas disease control on years of schooling. While much of the literature on the effects of NTD control has focused on increased education as a primary mechanism (e.g., [Miguel and Kremer, 2004](#); [Bleakley, 2007, 2010](#)), the bottom left panel of Figure [4a](#) shows very limited evidence of differential increases in schooling over this period in this section. Instead, we view reductions in its chronic symptoms—which “can cause young adults to develop heart conditions, so that they fill hospital beds instead of the [labor] force” ([World Health Organization, 2010](#))—as a more promising candidate. Because they manifest 10 or more years after infection and over two decades had passed between the start of IRS and the 2010 census, it is very much possible that these symptoms would have begun to manifest before 2010 in the absence of the 1984-89 vector control program. However, we cannot test this proposition directly in the census data, as they lack information on an individual’s health status

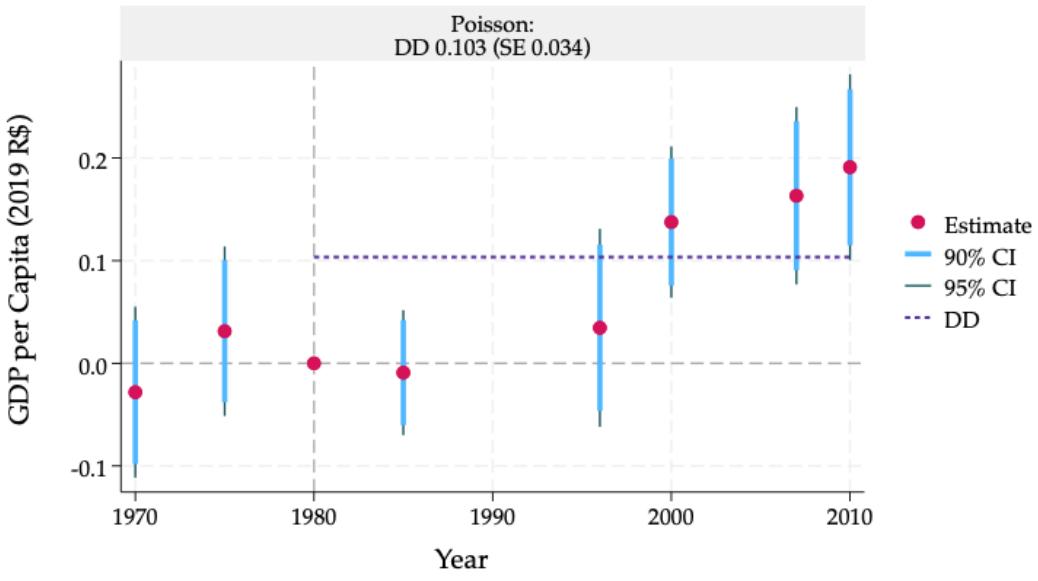
As an indirect test, we take inspiration from the WHO quotation above and examine whether an individual is working for a wage or salary (excluding those without a legal contract). We do so because poor long-run cardiovascular health might preclude someone from holding such regular employment, instead leaving them to work on their own account, without a legal contract, or as an unpaid family worker. The bottom right panel of Figure [4a](#) shows that rates of wage or salary work increased 1.8 p.p. (6%) more in treatment municipalities. Our interpretation is that these results point to health (observed indirectly through the ability to hold such jobs) as a more consequential channel than schooling. We focus directly on the long-run health channel in Section [6](#).

Figure 4: Effects of Chagas Disease Vector Control on Municipalities



7,365 observations, 1,473 clusters, pre-1991 means: income percentile 41.21, Gini coefficient 69.22, years of schooling 3.00, wage/salary worker 0.329

(a) Cross-Census Results



(b) GDP per Capita

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

4.3. GDP per Capita

Lastly, we use Poisson regression to study how estimated output per person changed across treatment and control municipalities.²² Figure 4b shows that GDP per capita increased by 9.8% more in treatment municipalities over this period, and this effect is precisely estimated. As before, the impact of IRS on this outcome increased over time. Taken alongside the evidence using collapsed census data, these results suggest that reductions in Chagas disease transmission led to important improvements in standards of living across Brazilian municipalities and reductions in income inequality within them.

4.4. Robustness

In Appendices C1 and C2, we test the robustness of our results to new difference-in-differences estimators. The patterns in these estimates are very similar to those above: treated municipalities experienced substantial increases in incomes and decreases in inequality, with little evidence of increases in years of schooling.

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

In this section, we take a complementary approach to studying the long-run effects of Chagas disease vector control by studying the impacts on adults who were children around the time that *T. infestans* control began. This is the approach taken by other studies on the impacts of reducing NTDs (e.g., Bleakley, 2010; Cutler et al., 2010; Bütkofer and Salvanes, 2020), allowing us to compare our estimates with related studies and to examine impacts on racial disparities. We also study the effects of the IRS campaign on treated adults' children. Our results show that it raised incomes substantially more for non-white than white Brazilians, and that the daughters of non-white fathers experienced larger increases in their literacy rates than those with white fathers. The implication is that vector control decreased racial inequality in Brazil in the long run and across generations. Consistent with our previous results, when exploring mechanisms, we

²² Once again, we use this approach instead of OLS and percentiles because we want to estimate percent effects on GDP per capita when extrapolating across Latin America.

once again find that increases in treated cohorts' years of schooling are unlikely to explain most of the income results, suggesting that movement into formal sector jobs and improved long-run health could be more important channels.

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, literacy, years of schooling, and formal employment), demographic characteristics (age, racial category, and sex), and whether an individual was born in their municipality or state of residence.

5.1.1. Alternatives to Log Income

When incomes can be zero, Chen and Roth (2024) show that using $\log(1 + \text{income})$ as the outcome does not yield an average percentage effect because the results depend on the units used.²³ We use several of their recommended transformations in the main text: the percentile in the national income distribution (as in Section 4), an indicator for having a strictly positive income, and an indicator for having an above-median income. The first transformation allows us to examine the average effects on treatment groups while the other two shed light on whether the poorest or richest within them benefit most. Nonetheless, to maintain comparability with previous studies of disease control that used $\log(1 + y)$ as an outcome (e.g., Bleakley, 2010; Cutler et al., 2010; Bütkofer and Salvanes, 2020), in Appendix D1 we present estimates using this transformation. We also include results using Poisson regression in Appendix D2, which allows us to estimate the average effect on the level of income as a percentage of the mean in the control group.²⁴

²³ This problem arises because the percentage change when moving from zero to a strictly positive value is not defined. As we show below, the extensive margin effect is an important element of our results.

²⁴ The main text features OLS results using percentiles instead of Poisson regression for greater comparability with our cross-census results in Section 4, as Brazil's hyperinflation in the late 1980s and early 1990s makes estimates using the latter approach difficult to interpret. In addition, the new difference-in-differences estimators discussed below are not suited for Poisson regression, so using them for robustness tests necessitates transforming the outcomes in our preferred ways.

5.2. Assignment to Treatment and Control Groups

To examine the effects of additional childhood years free from exposure to Chagas disease (e.g., Bleakley, 2010; Cutler et al., 2010; Bütkofer and Salvanes, 2020), 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. But as noted in Section 2, both children and adults can experience symptoms of the acute stage of Chagas disease again when reinfected with *T. cruzi*, which also increases the chances of developing its chronic manifestations. Therefore, the interpretation of our individual-level results is closer to those for childhood exposure to tuberculosis (e.g., Bütkofer and Salvanes, 2020), which also affects all ages, than to diseases like malaria that primarily impact children (e.g., Bleakley, 2010; Lucas, 2010; Cutler et al., 2010).

For several reasons, we limit our attention to cohorts born between 1960 and 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. In addition, DDT spraying began in the late 1950s, so we want to avoid comparing cohorts with different levels of exposure to that public health intervention. Lastly, the restriction at the other end of our age range ensures that our sample 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.

We can observe municipalities of birth for just under three-fifths of the sample, but 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, as discussed in the previous section. We then assign to those with unknown municipalities of birth the probability in the 1980 census that a 6- to 10-year-old child of their race, sex, and state of birth was living in a treatment municipality. Because we know with certainty that just over 45% of the sample was born in a control municipality, we put anyone with a strictly positive probability into the treatment group, which is nearly identical to performing a median split.

This decision means that we compare anyone who could have been born in a treatment municipality against those we are certain were not. As such, we interpret our treatment effects

as resulting from a reduction in the share of children potentially exposed to Chagas disease transmission from around 45%—the average of the probability values in the treatment group—to 0%.²⁵ We view this interpretation as both intuitive and policy relevant. It also allows us to avoid making the much stronger assumptions required in a continuous-treatment difference-in-differences framework (Callaway, Goodman-Bacon and Sant'Anna, 2024).

5.3. Empirical Strategy

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

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

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.²⁶

The coefficients of interest are the τ_k , which measure the difference in an outcome for a given birth cohort as the share of children potentially exposed to Chagas disease transmission is reduced from about 45% to 0%, 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) and sex to examine the potential for Chagas disease control to reduce inequality along these dimensions without imposing the additional assumptions necessary for—or risking the greater imprecision in—a triple-differences framework. Instead, we simply discuss and compare patterns across groups, which is facilitated by estimating the average of the post-treatment τ_k , though it requires that we impose a single treatment effect across these cohorts.

²⁵ We thus use an intent-to-treat design, as we cannot observe additional information about childhood.

²⁶ We create a bin for all individuals born in a given state for whom we cannot observe more detailed information about their birthplace.

5.3.1. Robustness

We use new difference-in-differences estimators (Arkhangelsky et al., 2021; Callaway and Sant'Anna, 2021; de Chaisemartin and D'Haultfœuille, 2020; Sun and Abraham, 2021) in Appendices D₃ through D₈ to test the robustness of our results to issues raised in this literature.²⁷

5.4. Income and Inequality

Our first outcome of interest is the percentile in the national distribution of monthly income as reported in the census for July 2010. Figure 5c shows that while these rankings mostly evolved in parallel across treatment and control groups for cohorts that turned 16 before IRS began, the income percentiles of non-white men and women who were exposed to treatment increased by around 1 point more (2% to 4% of the respective means) than those of their non-white peers from control municipalities. These estimates are also very precise. In contrast, there are no similar treatment-control divergences in the income rankings of white Brazilians. As such, these results are consistent with our hypothesis that Chagas disease control reduced racial income disparities.

To further examine inequality, we create indicator variables for whether an individual has an income greater than zero or greater than the median. Figures 5a and 5b show that these probabilities rose by more for non-white cohorts exposed to treatment: 1.1 p.p. among men and 1.5 p.p. among women for having any income (1% to 2%), and effectively identical amounts for having an above-median income (1.2 and 1.7 p.p., or 2% to 4%). As each estimate is precise and there are no similar effects for white adults, these results imply that Chagas disease control helped mitigate racial income inequality by increasing the incomes of poorer non-white Brazilians.

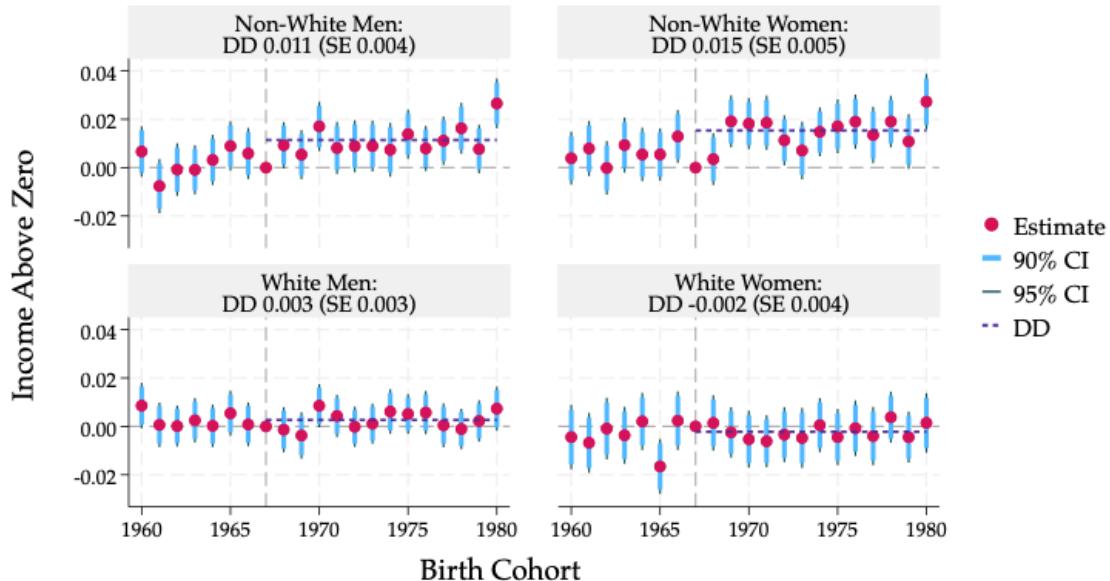
5.5. Next Generation's Literacy

To study the impacts on the children of men exposed to vector control during childhood, we attach men's birth municipality and cohort information to the children aged 10 to 18 living in their households in the 2010 census.²⁸ We then estimate a modified version of equation (2) that

²⁷ Nonetheless, we reiterate that our approach of comparing unexposed areas to those exposed at one treatment time does not suffer from many of these shortcomings.

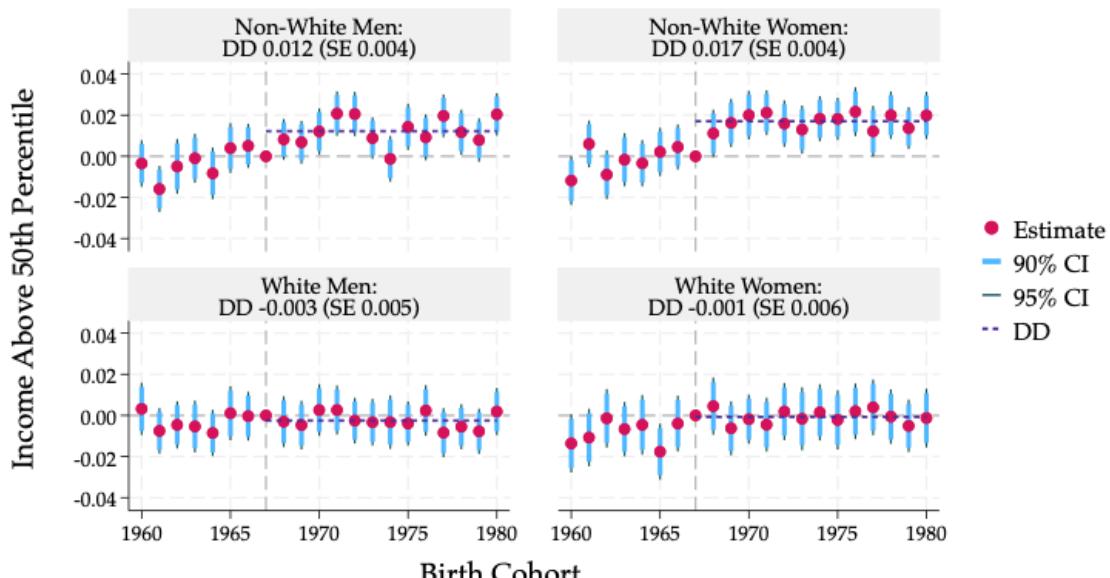
²⁸ We limit the sample to exclude those older than 18 due to selection into living with their parents after that age. We set 10 years old as the lower bound because the data show that children are still becoming

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



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

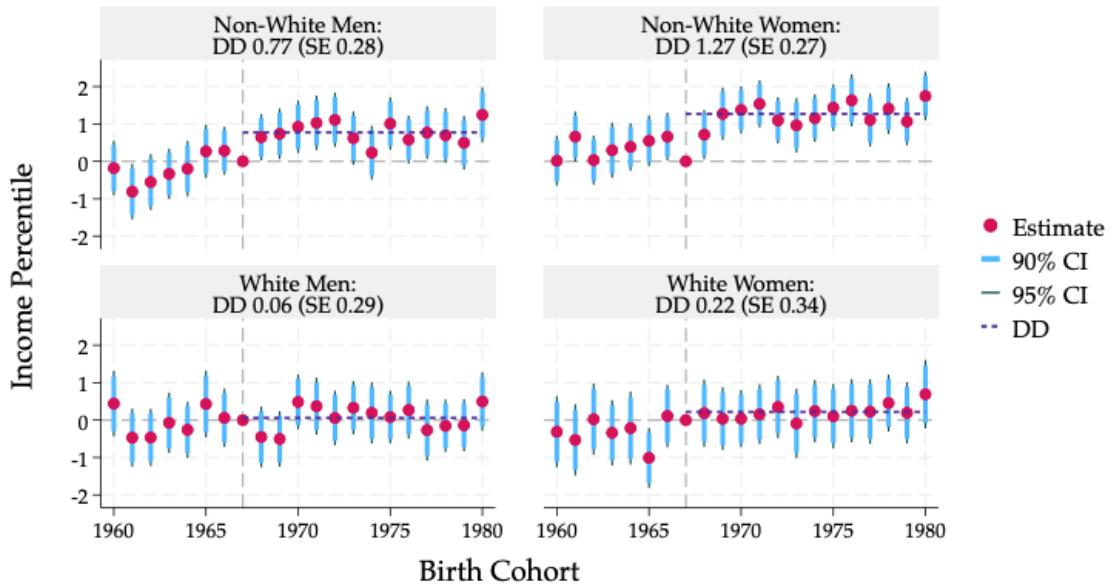
(a) Income Above Zero



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

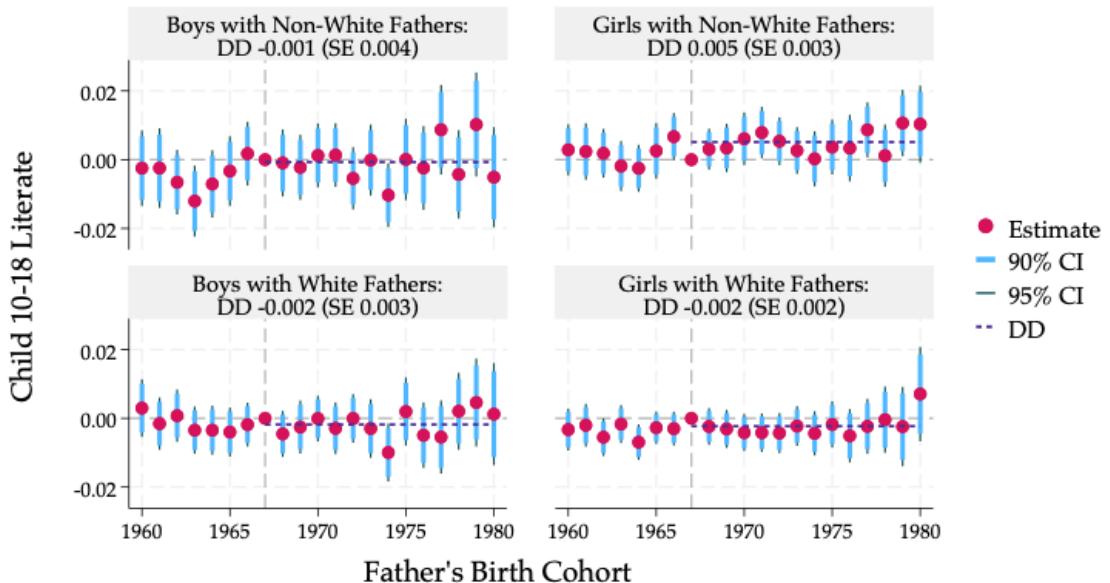
(b) Income Above Median

Figure 5: Continued



1.01-1.32 million observations, 1,582 clusters, pre-1968 birth cohort means: non-white men 49.07, non-white women 35.41, white men 62.96, white women 44.25

(c) Percentile in National Income Distribution



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

(d) Literacy: Children Ages 10-18

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

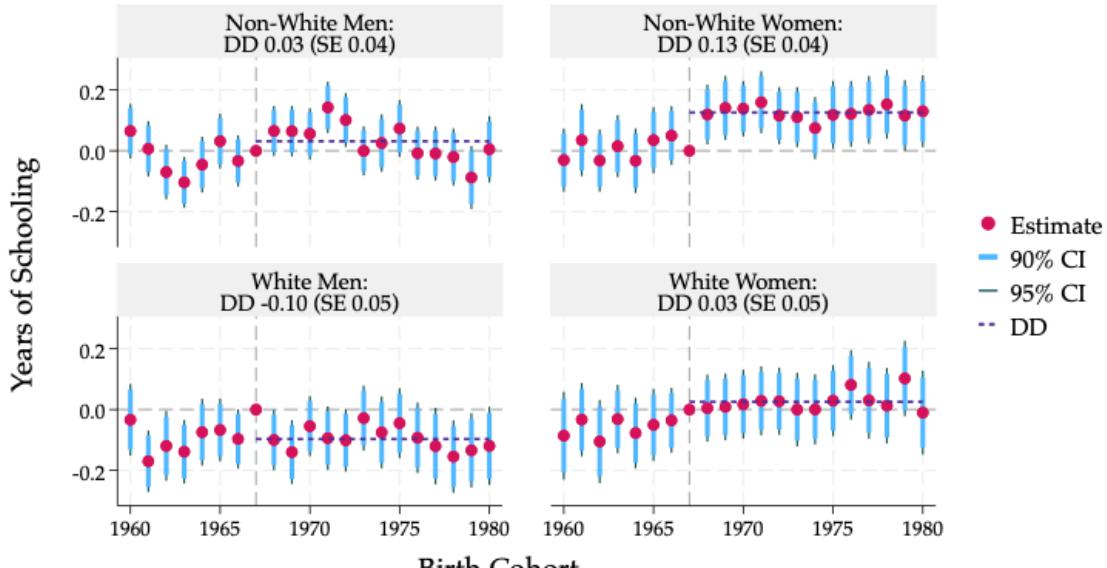
uses fathers' birth municipalities and cohorts, and we add children's age and age squared to the vector of controls. Given the children's ages, our outcome of interest is their literacy, which we view as a measure of their knowledge and thus their future labor market prospects. Notably, even though Brazil had made extensive progress toward universal child literacy between 1980 (see Table 2) and 2010, Figure 5d shows that it increased by a precisely estimated 0.5 p.p. (0.5%) more for the daughters of non-white fathers who were children in treatment municipalities after IRS began. As predicted, there are no effects for the children of white fathers, although the same is true for the sons of non-white fathers (perhaps due to greater noise in the estimates). Nonetheless, these results provide evidence that some of the benefits of Chagas disease vector control were transferred from parents to their female children, highlighting the positive intergenerational impacts of combating NTDs.

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

We use the 2010 census data and the same identification strategy as in Section 5.4 to study years of schooling among adults who were children around the time that Chagas disease vector control began. Figure 6a shows that the results are not particularly consistent with time in the classroom having a predominant role, similar to our findings in Section 4.2. Specifically, there is a precisely estimated effect for non-white women (0.13 years, or 2.4%), but it is small and noisy for non-white men (0.03 years, or 0.6%). While the results also show a clear null effect for white women, at first glace there appears to be a negative effect for white men (-0.10 years, or -1.6%), but it is an artifact of noise in the omitted cohort that pushes down pre-treatment estimates by the same magnitude. Therefore, our view is that only the estimate for non-white women is credible, but it is still quite small.

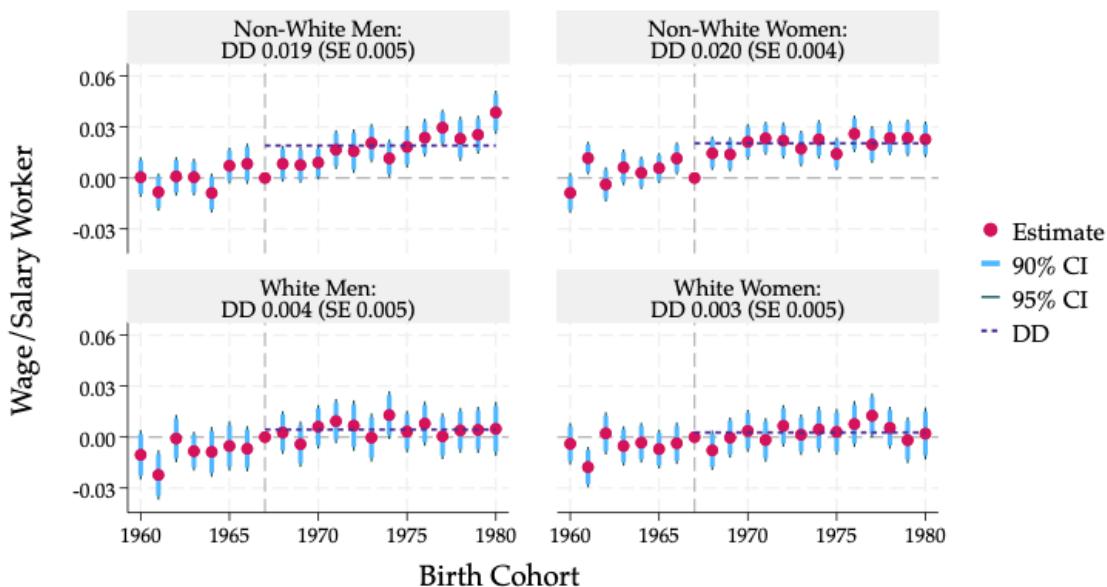
To support this interpretation, we perform a back-of-the-envelope calculation of the upper bound of years of schooling's contribution to the income results. Using the Mincerian return to schooling in Brazil of 15.7% (as reported in Psacharopoulos and Patrinos, 2018)—which almost certainly overstates the causal effect of schooling on income—the 0.13-year increase in non-white women's schooling can account for at most a 2.0-p.p. increase in their incomes, or less than literate between ages 6 and 9, so we exclude them to reduce the noise in our estimates.

Figure 6: Mechanisms Underlying Long-Run and Intergenerational Effects



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

(a) Years of Schooling



1.01-1.32 million observations, 1,582 clusters, pre-1968 birth cohort means: non-white men 0.328, non-white women 0.230, white men 0.374, white women 0.313

(b) Wage or Salary Worker

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.

one-third of the estimated 6.6% increase when using Poisson regression (see Appendix D2). For non-white men, taking the imprecise 0.03-year estimate at face value generates at most a 0.5-p.p. increase in their incomes, which is just over 10% of the Poisson regression estimate. As such, increased time in school is unlikely to explain most of the long-run increases in incomes and reductions in racial disparities arising from Chagas disease control.

Instead, consistent with findings in Section 4, Figure ?? shows that non-white men and women from treatment municipalities are 1.0 to 2.3 p.p. (2% to 5%) more likely to be in formal employment, and these effects are precisely estimated. Once again, there are no similar effects among their white peers. We interpret these results as indirectly suggesting that long-run improvements in health are a more important mechanism than years of schooling in this setting, in contrast to other studies using a similar individual-level empirical strategy (e.g., Bleakley, 2007, 2010; Lucas, 2010; Bütkofer and Salvanes, 2020). We directly examine the long-run health channel, albeit with a place-based approach, in Section 6.

6. Effects on State Public Health Care Systems

If cardiovascular morbidity from chronic Chagas disease was severe enough to reduce the productivity of adults exposed to its transmission as children, the costs it imposed on society could have extended beyond income and (racial) inequality. Specifically, as Brazil has the world's largest government-run health care system (the *Sistema Único de Saúde*, or SUS)—which consumes about 4% of GDP and 70% of the population depends on—improvements in adults' heart health could have had important effects on public finances.²⁹ 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 cardiovascular-related hospital care covered by the SUS. Using a triple-differences strategy comparing circulatory and non-circulatory system-related causes, we show that hospitalizations

²⁹ 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>.

and spending resulting from the former decreased more in states more exposed to treatment. As a result, 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, person-days spent in the hospital, spending on hospital care, and deaths, each of which is by International Classification of Diseases (ICD) code. The first three of these measures are from the SUS's Hospital Information System (SIH/SUS), and we deflate the spending data so that figures are in 2019 BRL. The data on deaths are from the SUS's Mortality Information System (SIM). Given that Chagas disease is highly under-diagnosed and its chronic effects manifest primarily as cardiovascular problems 10 or more years after infection (see Section 2), we focus on all diseases of the circulatory system.³⁰ In addition, we set our omitted year to 1994, or a decade after IRS began.³¹ However, as the SUS does not consistently provide municipality-level data for years prior to 1995, our focus in the main text is on state-level data from 1991 to 2019.

In a similar vein as our approach in Section 5, our measure of each state's exposure to Chagas disease vector control is the share of its 1980 population living in treatment municipalities. However, because the SUS is a heavily decentralized system with transfers of responsibilities and funds to states and municipalities (Castro et al., 2018), there are likely confounders that vary across both state and year (e.g., public health priorities, non-hospital care spending) in violation of the difference-in-differences common trends assumption. To address this complication, we use a triple-differences strategy using all other non-circulatory system disease categories as the additional control group, under the assumption that they are subject to the same state-specific, time-varying factors.³² The specification that we estimate is thus

³⁰ 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.

³¹ 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.

³² 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)

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

where $y_{s,t,d}$ is state s 's outcome in year t for disease category d , $\alpha_{s,t}$, $\gamma_{t,d}$, and $\delta_{s,d}$ are fixed effects for state-year, year-disease category, and state-disease category, $\mathbb{1}[\mathbb{P}_{s,1980}(m \in \text{Treat}) > 0]$ indicates whether s had any of its population living in treatment municipalities in 1980, $\mathbb{1}[d = \text{Circ}]$ indicates whether d is circulatory system diseases, $\eta_{r(s)}$ is a fixed effect for s 's region, and all other variables are analogous to those in previous specifications.³³ As in equation (1), we include the interaction of $\eta_{r(s)}$ and $\gamma_{t,d}$ to control for region-disease category-specific trends.

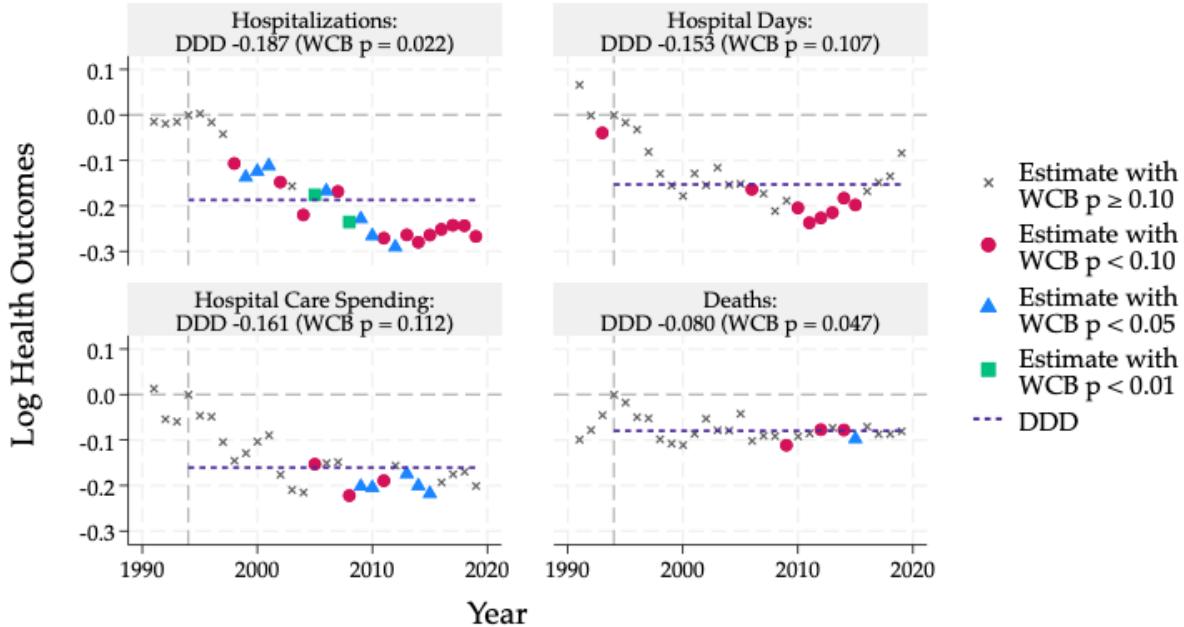
This strategy first estimates the differences in outcomes due to circulatory system diseases in a given year between states with any individuals living in treatment municipalities in 1980 and those that did not, relative to the size of that difference in 1994. Then we compare this double-difference to the analogous one for non-circulatory diseases. Because the mean value of this probability is approximately 25 p.p., we frame our results as moving from one-quarter 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). But because we cannot compute them for Poisson regression in this case, we log-transform our state-level data, which never have any zeros to create the problems that Chen and Roth (2024) describe. Likely as a result, using traditional confidence intervals with log outcomes and Poisson regression in Appendices E1 and E2 yield very similar plots, which also resemble those produced using new difference-in-differences estimators in Appendix E3.

6.2. Circulatory Disease Hospital Care

Consistent with our hypothesis that Chagas disease control would improve cardiovascular outcomes, the top-left panel of Figure 7 shows that hospitalizations paid for by the SUS due to circulatory system disease decreased by 19% 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

³³ Brazilian states are grouped into five regions: North, Northeast, Southeast, South, and Center-West.

Figure 7: Long-Run Effects on Circulatory Disease Outcomes



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

Notes: Graphs show dynamic and post-treatment average (DDD) estimates for the respective outcomes, with shapes and colors denoting wild cluster bootstrap (WCB) p-value bins. 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.

divergence began close to the year predicted in the medical literature. Similarly, in the top-right panel, SUS-covered days spent in the hospital as a result of cardiovascular problems decreased by 15% more, and this estimate is also precise. Consequently, we also find a 16% greater decrease in the SUS's spending on circulatory disease hospital care relative to spending on care due to other causes. While the dynamic and post-treatment average effects in the bottom-left panel are noisier than those in the top panels, the patterns over time are effectively identical, which we consider further evidence of the spending effect. Therefore, these results 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 the bottom-right panel. Although the post-treatment average effect is precisely estimated, there is a much larger pre-treatment difference

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

7. Cost-Benefit Analyses and Extrapolation

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 willingness to pay for better health—of 23% 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 of the 1984-89 vector control program, 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 1986.³⁴ We then take World Bank data on Brazil's 1984-2019 GDP in constant 2015 USD, convert these values into 2019 R\$ using the

³⁴ [Dias \(1986\)](#) noted that public health authorities intended to spend a constant amount in real terms in these years.

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.³⁵ For incomes, we take the 1-percentile increase for non-white men and women in post-treatment cohorts (see Figure 5c) and assume this gap between treatment and control municipalities was constant from each cohort of approximately 675,000 non-white Brazilians from treatment municipalities entering the labor force at age 16 through 2019.³⁶ As the average income percentile for non-white pre-treatment cohorts was 42, moving to the 43rd percentile in the 2010 census data yields an increase of 2019 R\$ 144/year (1.3%). 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 7 to get savings of 0.016% of GDP in each year from 1995 to 2019.

As a result, we estimate an IRR of 16%. 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. While both helminthiases and Chagas disease are NTDs, the gap between the returns to controlling them arises from the extremely low upfront costs of deworming compared to IRS against *T. infestans*, and from it taking more than a decade for circulatory disease hospital care spending to be averted. Thus, the heavy discounting outweighs the fact that the

³⁵ 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).

³⁶ 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.

savings comprise a non-trivial fraction of GDP each year. To illustrate this point, note that the IRR would be 13% if we only considered the income gains because we assume that they began immediately. Nonetheless, failing to consider the chronic health effects of eliminating Chagas disease transmission means understating its return by 3 p.p. (nearly one-fifth), 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 just over 5.4%—above the standard 5% discount rate used in studies of developing economies ([Haacker, Hallett and Atun, 2020](#))—the implication is that using this rate yields an MVPF of infinity.³⁷ This result notably arises without raising additional revenue, as the marginal tax rate at the 42nd income percentile in the 2010 census data (2019 R\$ 908) was 0%.³⁸ 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

³⁷ 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](#)).

³⁸ For marginal tax rates in Brazil in 2010, see: <https://www.gov.br/receitafederal/pt-br/assuntos/meu-imposto-de-renda/tabelas/2010>.

calculation by multiplying World Bank data on 2019 GDP per capita and Gini coefficients by this share and our Poisson regression estimates in Section 4.³⁹

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 2.4% 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.⁴⁰ 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 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

³⁹ 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.

⁴⁰ Along the lines of the estimates in Table 1, 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	Percent Exposed	GDP per Capita	Estimated Change	Gini Coefficient	Estimated Change
Argentina	5	10,076	98	42.9	-0.06
Belize	22	4,983	200		
Bolivia	6	3,552	38	41.6	-0.06
Brazil	13	8,876	213	53.5	-0.18
Chile	0	14,699	0	44.4	-0.00
Colombia	11	6,419	121	51.3	-0.13
Costa Rica	5	12,762	119	48.2	-0.06
Ecuador	29	6,223	325	45.7	-0.33
El Salvador	15	4,168	110	38.8	-0.14
Guatemala	10	4,639	86	48.3	-0.12
Guyana	25	6,610	299		
Honduras	15	2,574	68	48.2	-0.18
Mexico	21	9,950	374	46.7	-0.24
Nicaragua	11	1,924	40	46.2	-0.13
Panama	13	15,774	372	49.8	-0.16
Paraguay	20	5,384	190	45.7	-0.22
Peru	4	7,023	56	41.6	-0.05
Suriname	25	6,854	310		
Uruguay	0	17,688	0	39.7	-0.00
Average	13	7,904	159	45.8	-0.13

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.

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, potentially helping to increase the speed of racial convergence in a country with wide disparities in this dimension. We also found little evidence that educational attainment drove this result; instead, the results are more consistent with an important role for improved long-run health allowing individuals to hold formal employment.

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 16% 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.⁴¹

Thus, these results present a more complete picture of the economic consequences of NTD control for developing countries. Whether they generalize beyond this malady that exclusively afflicts the Americas is an open question we leave to 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 6 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.

⁴¹ However, because these benefits generally materialize many years later, the results raise important questions for the political economy of disease control: policymakers with short time horizons might not invest in NTD control despite their large benefits. We see these questions as important avenues for future research.

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Online Appendix for:

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

Jon Denton-Schneider

Clark University

Eduardo Montero

University of Chicago

December 5, 2024

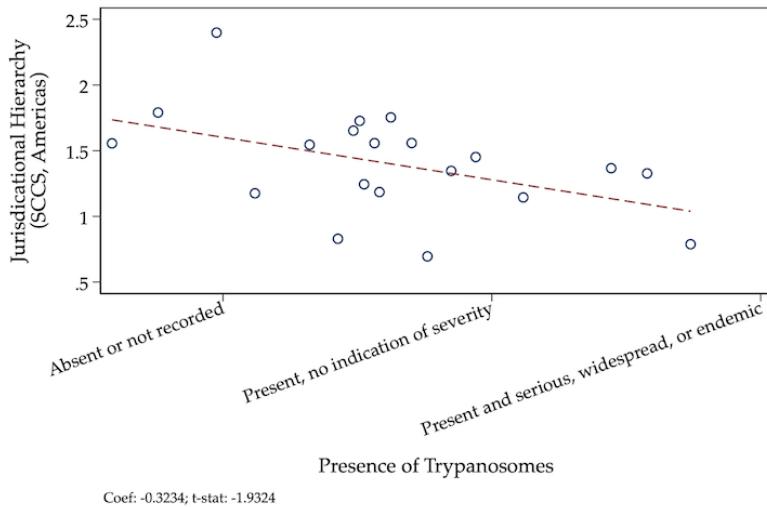
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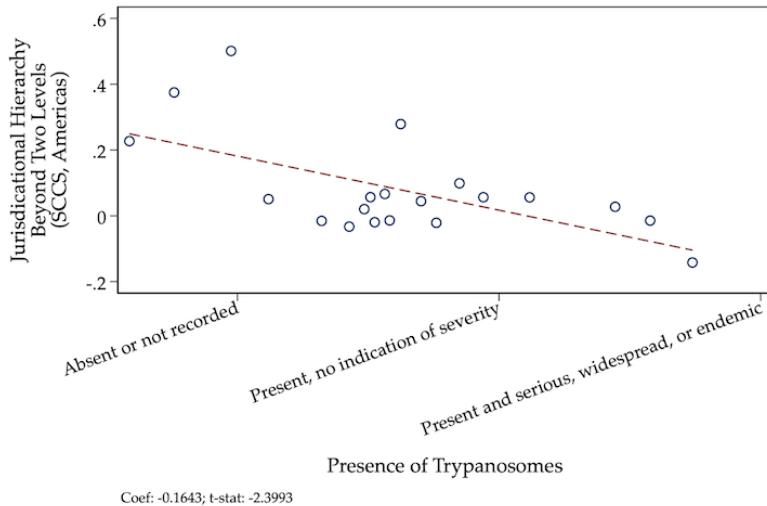
Appendix A. Additional Figures: Introduction

A1. 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 and Its Control in Brazil

B1. Image of *Triatoma infestans*

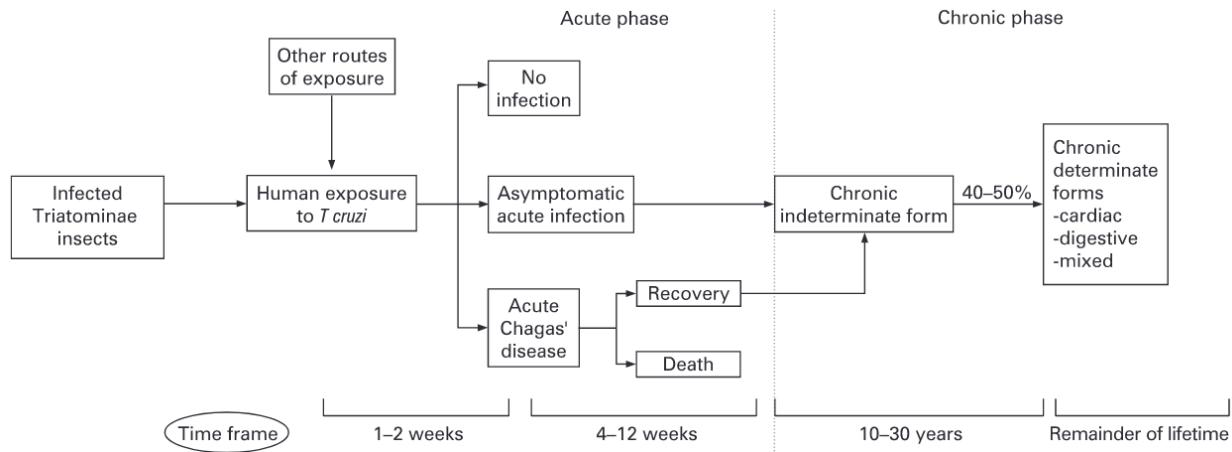
Figure B1: Image of *Triatoma infestans* [7]



Notes: Image shows *Triatoma infestans*, the main vector in Brazil prior to the 1984-89 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

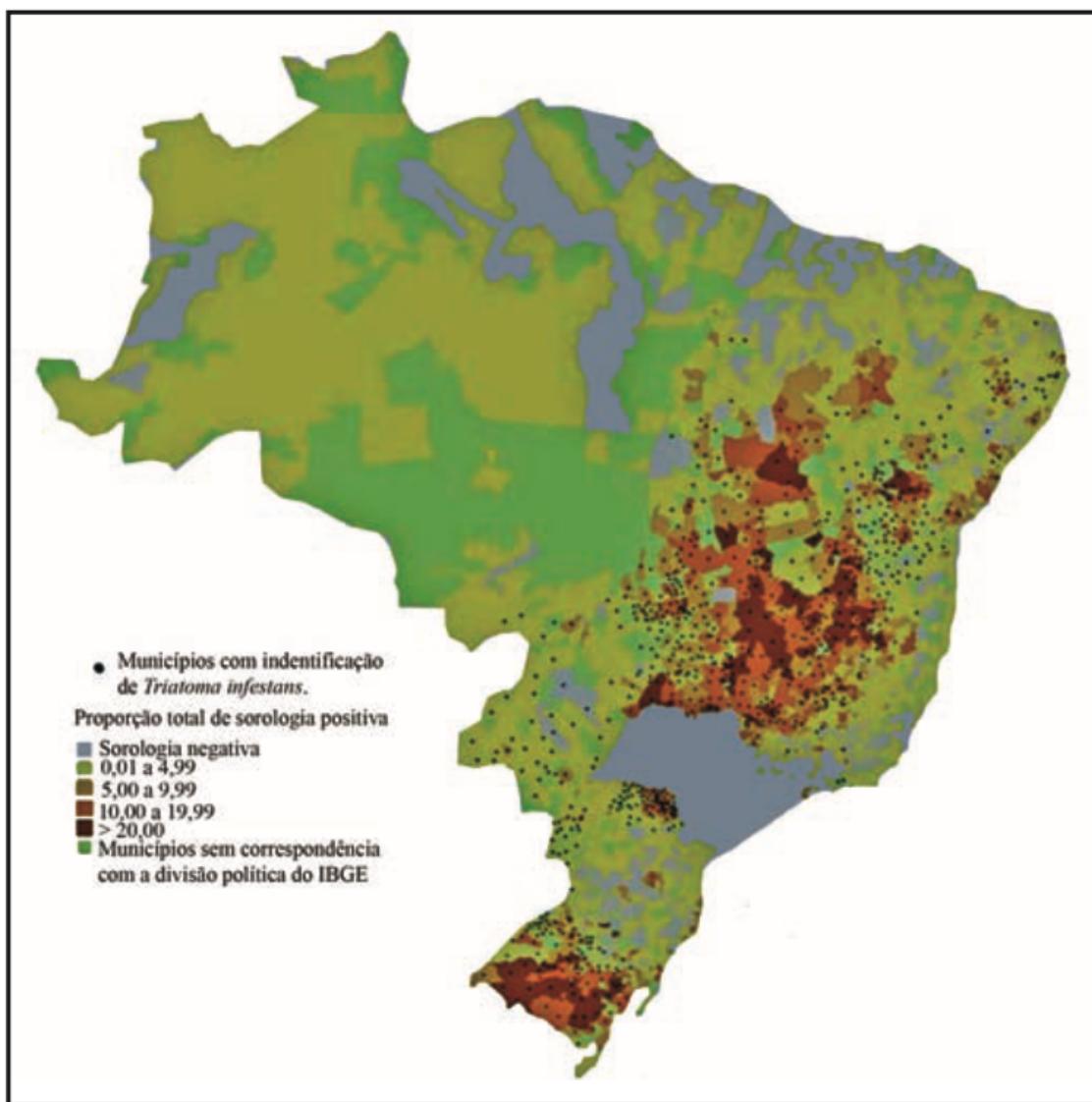
Figure B2: Phases of Chagas Disease [7]



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

B3. *T. cruzi* Seroprevalence, 1975-83

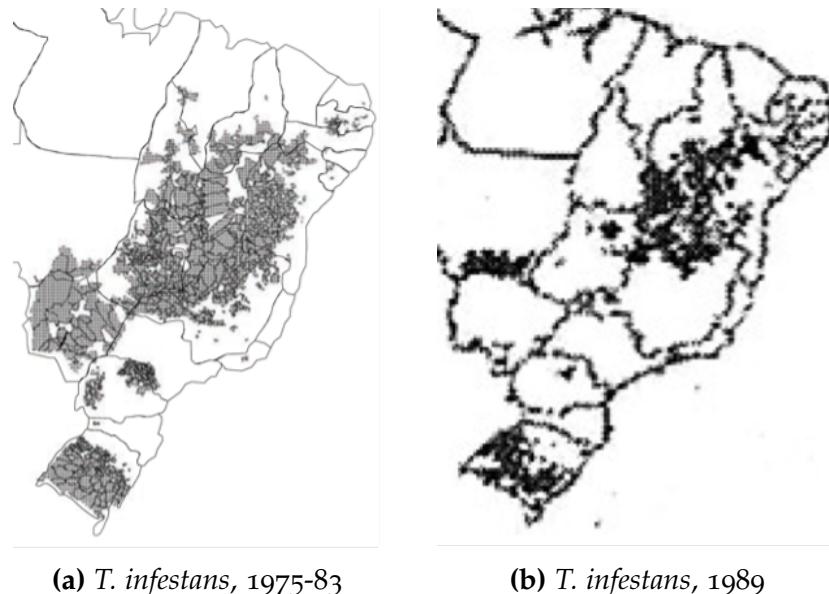
Figure B3: *T. cruzi* Seroprevalence, 1975-83 [8]



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

B4. Vector Control Progress, 1975-89

Figure B4: Vector Control Progress, 1975-89 [8]



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

(b) *T. infestans*, 1989

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

B5. Images of Indoor Residual Spraying

Figure B5: Images of Indoor Residual Spraying [8]



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.

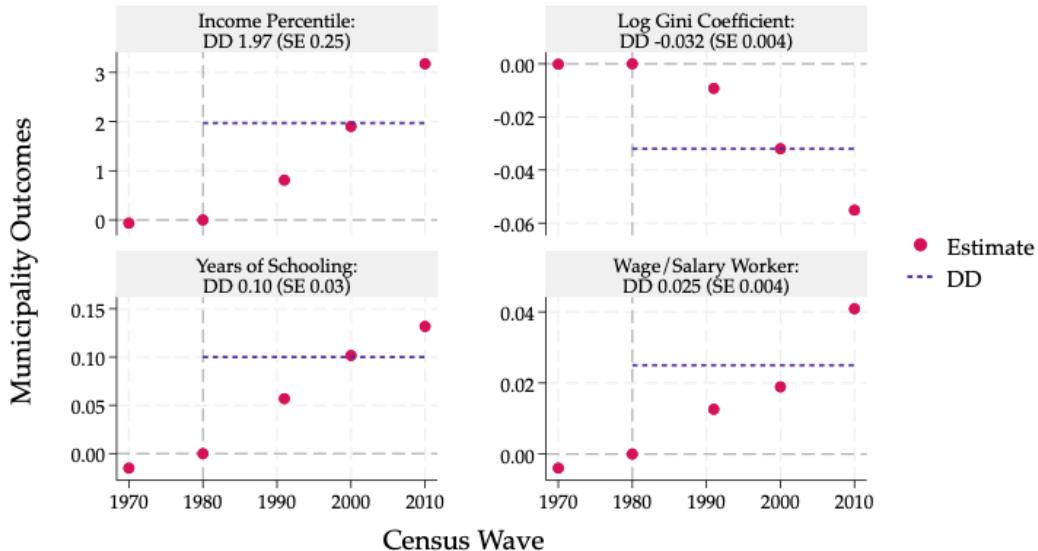
2 *The Attack Phase.* The initial spraying of an area is known as 'attack one' or massive attack, during which all houses and outbuildings are sprayed regardless of whether or not they were found to be infested. A second selective spraying is then carried out 3–6 months later only in houses known to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

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

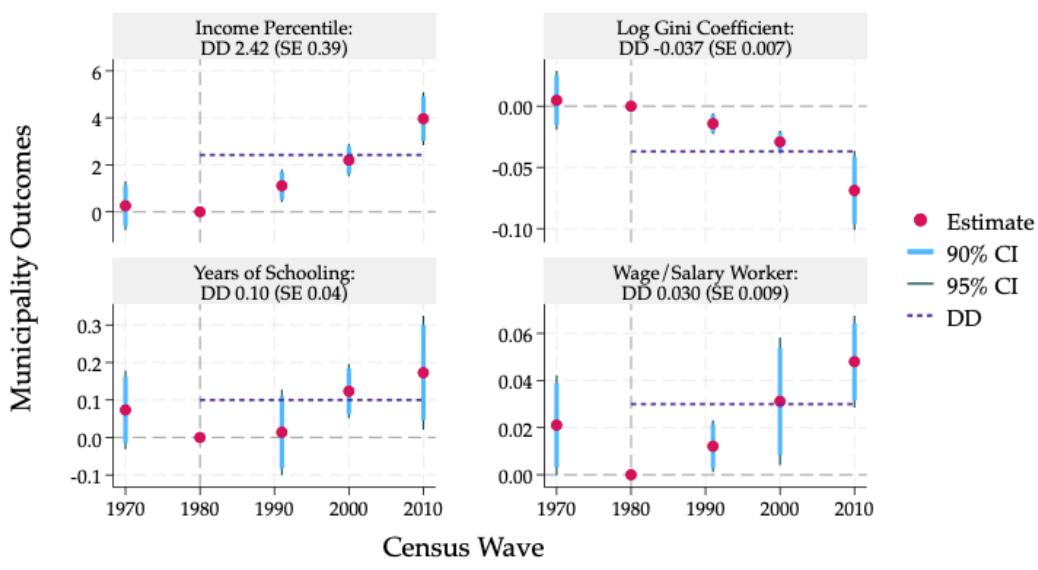
Appendix C. Additional Results: Effects on Municipalities

C1. Municipality Results Using New Difference-in-Differences Estimators

Figure C1: Municipality Results Using New Estimators [14]

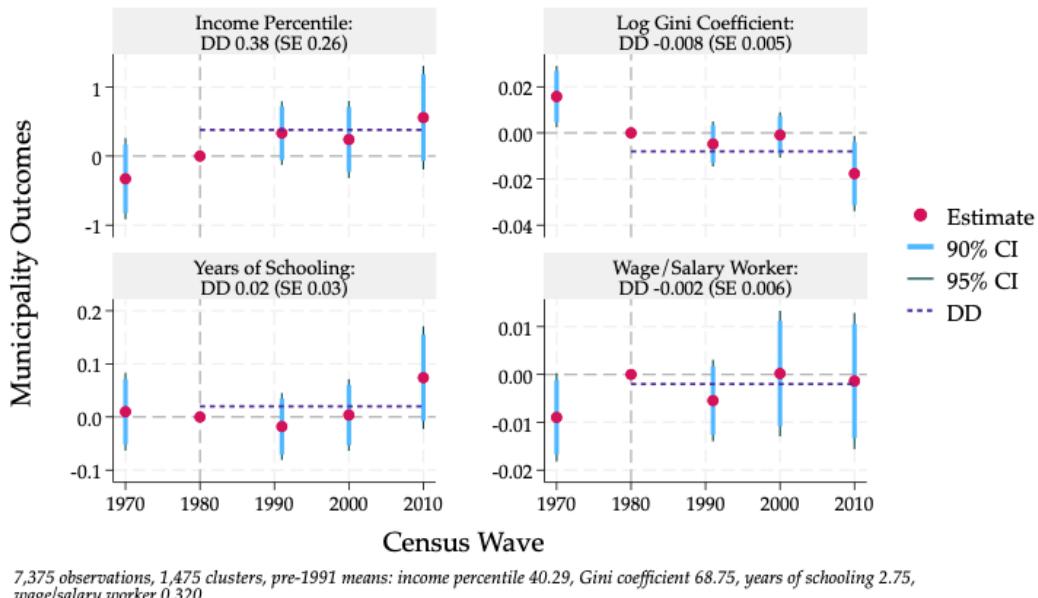


(a) Arkhangelsky et al. (2021)

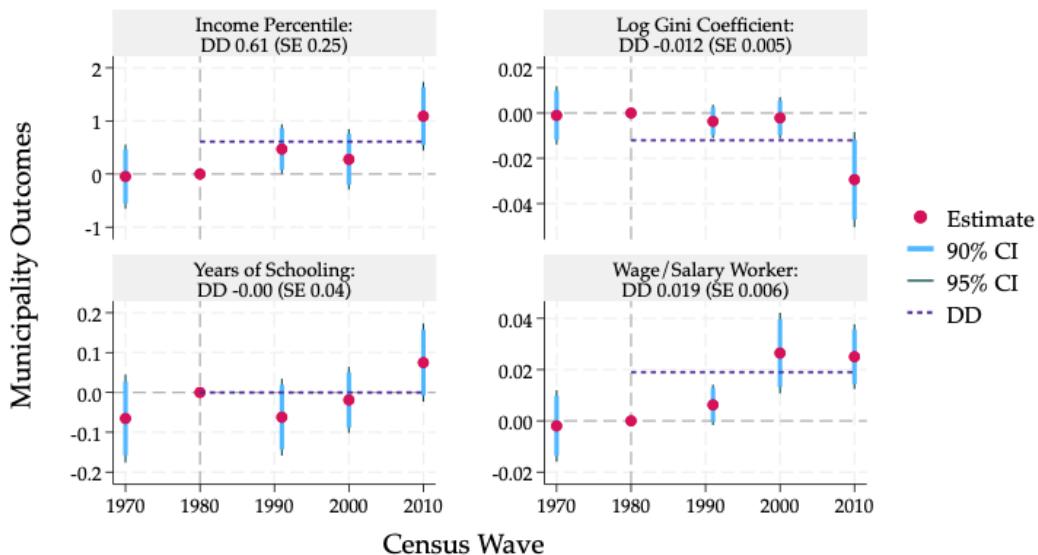


(b) Callaway and Sant'Anna (2021)

Figure C1: Continued



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

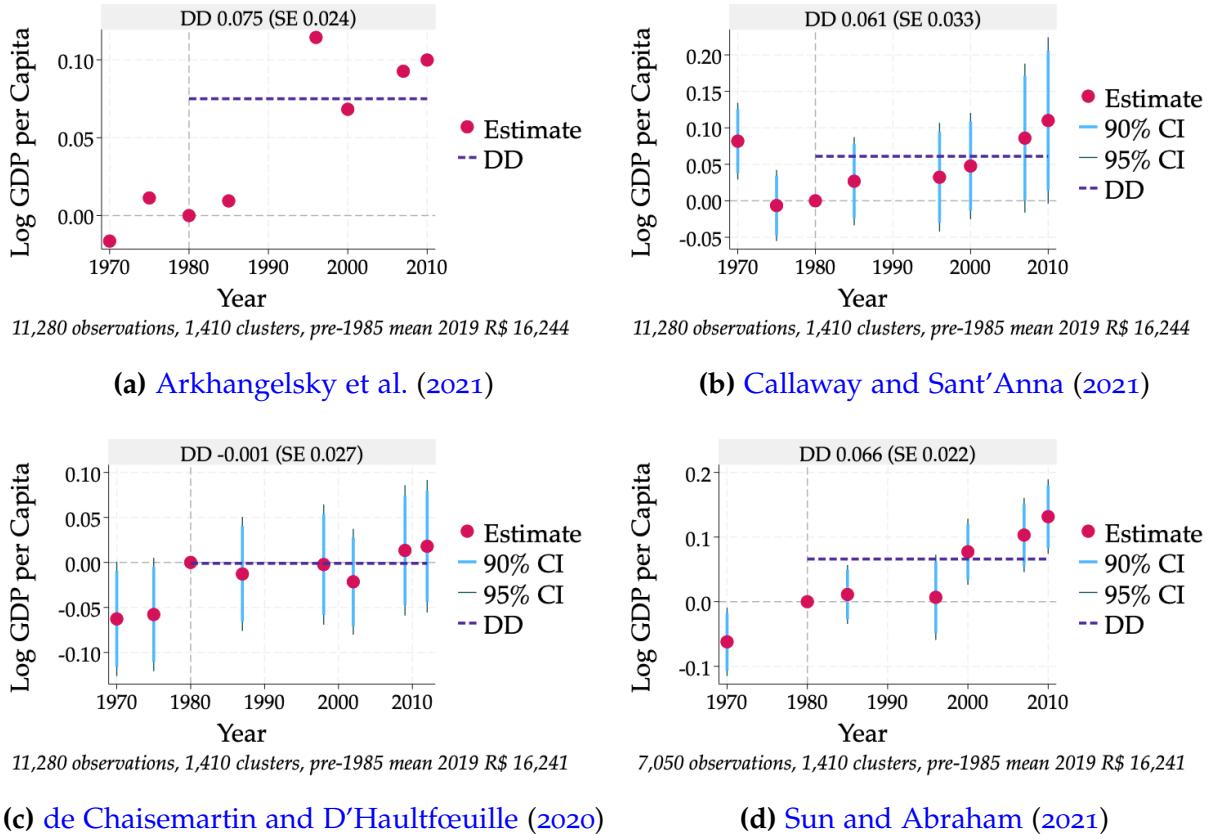


(d) Sun and Abraham (2021)

Notes: Observations are municipality-years. Graphs show dynamic and post-treatment average (DD) estimates for the respective outcomes when using a given estimator, with 90% and 95% confidence intervals for dynamic estimates in all but the top panel. Data are from the IPUMS samples of the 1970, 1980, 1991, 2000, and 2010 censuses. 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 1980 characteristics (shares of the population that were female, Asian, Black, and Brown) and state fixed effects. Standard errors are clustered by municipality.

C2. GDP per Capita Results Using New Difference-in-Differences Estimators

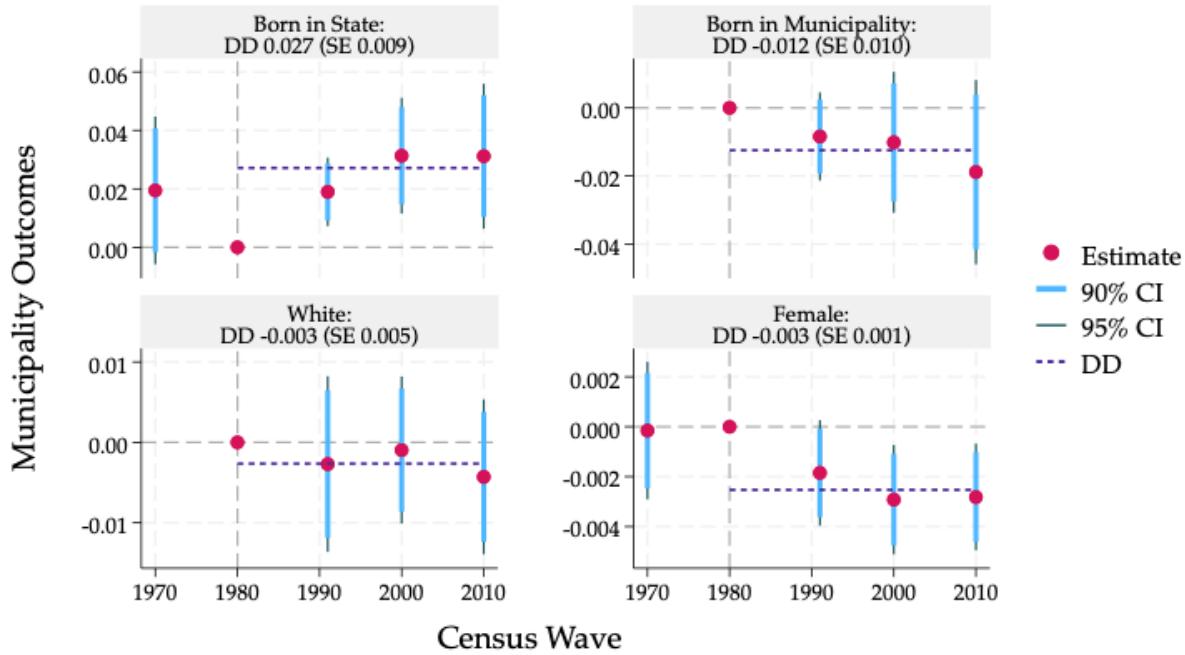
Figure C2: GDP per Capita Results Using New Estimators [14]



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 in all but the top panel. 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 1980 characteristics (shares of the population that were female, Asian, Black, and Brown) and state fixed effects. Standard errors are clustered by municipality.

C3. Municipality Composition Results

Figure C3: Municipality Composition Results [14]



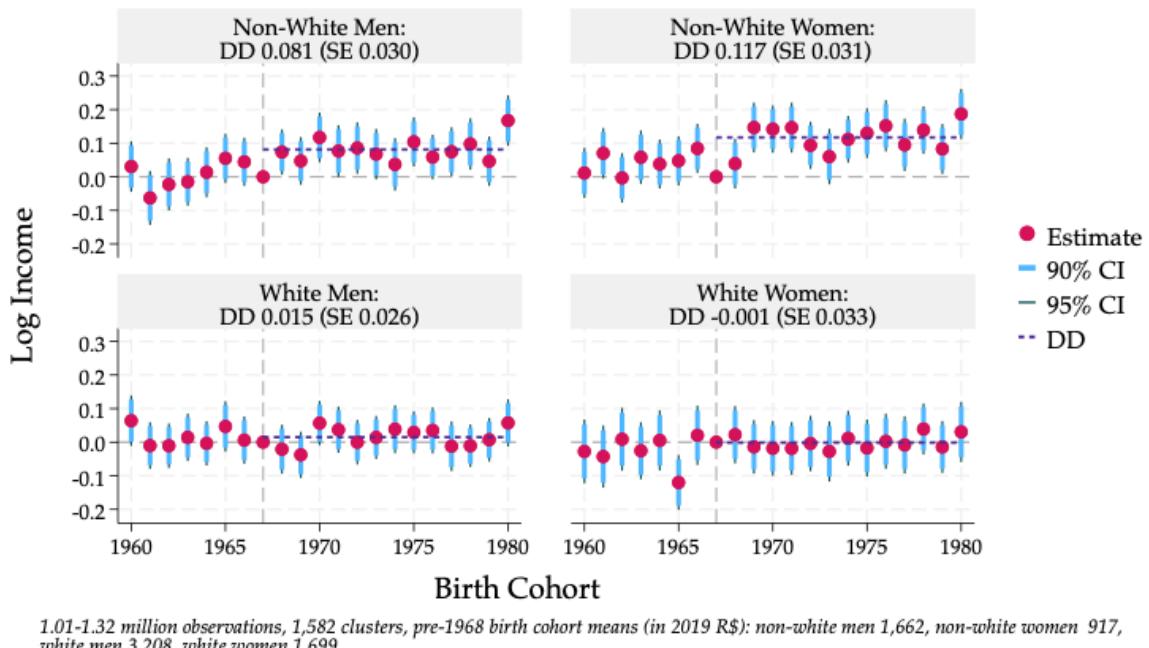
5,892-7,365 observations, 1,473 clusters, pre-1991 means: born in state 0.752, born in municipality 0.462, white 0.512, female 0.505

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

Appendix D. Additional Results: Effects on Individuals

D1. Income Results Using Log Transformation

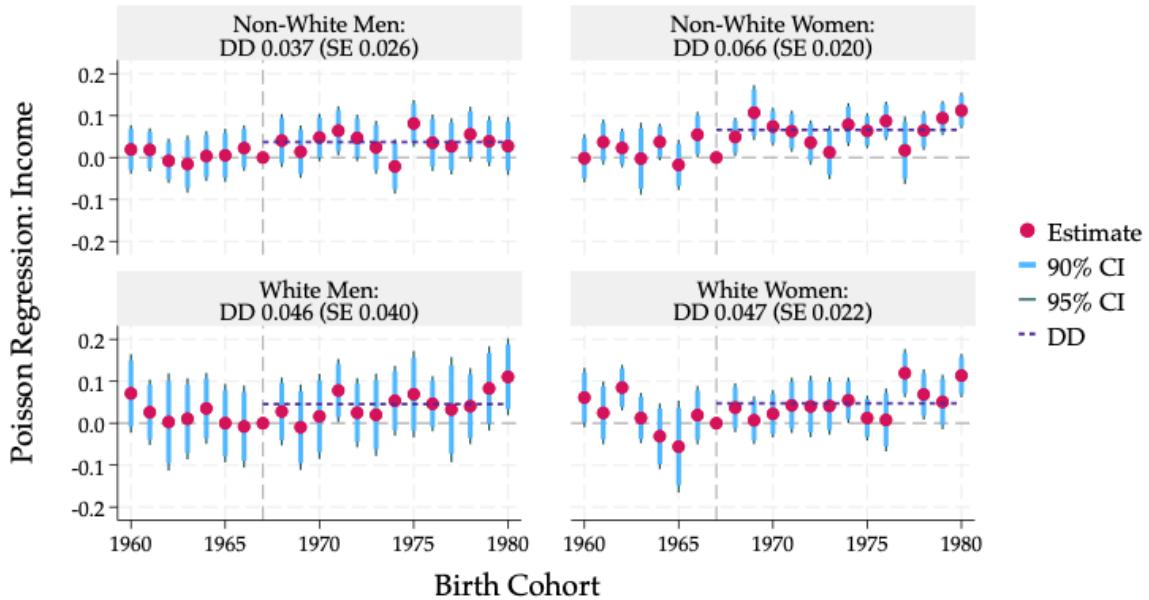
Figure D1: Income Results Using Log Transformation [19]



Notes: Graphs show dynamic and post-treatment average (DD) estimates, 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.

D2. Income Results Using Poisson Regression

Figure D2: Income Results Using Poisson Regression [19]

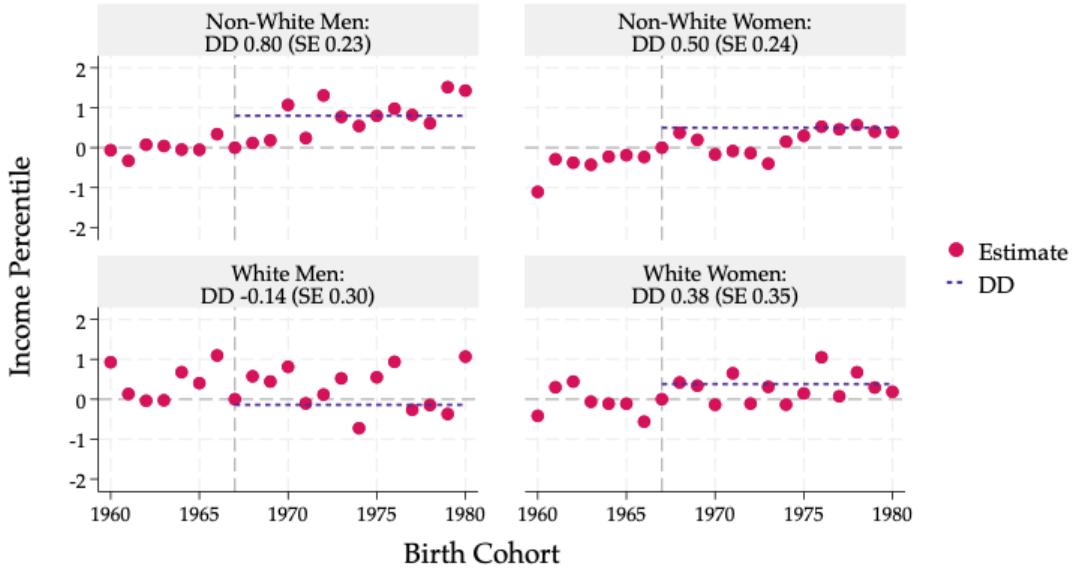


1.01-1.32 million observations, 1,582 clusters, pre-1968 birth cohort means (in 2019 R\$): non-white men 1,662, non-white women 917, white men 3,208, white women 1,699

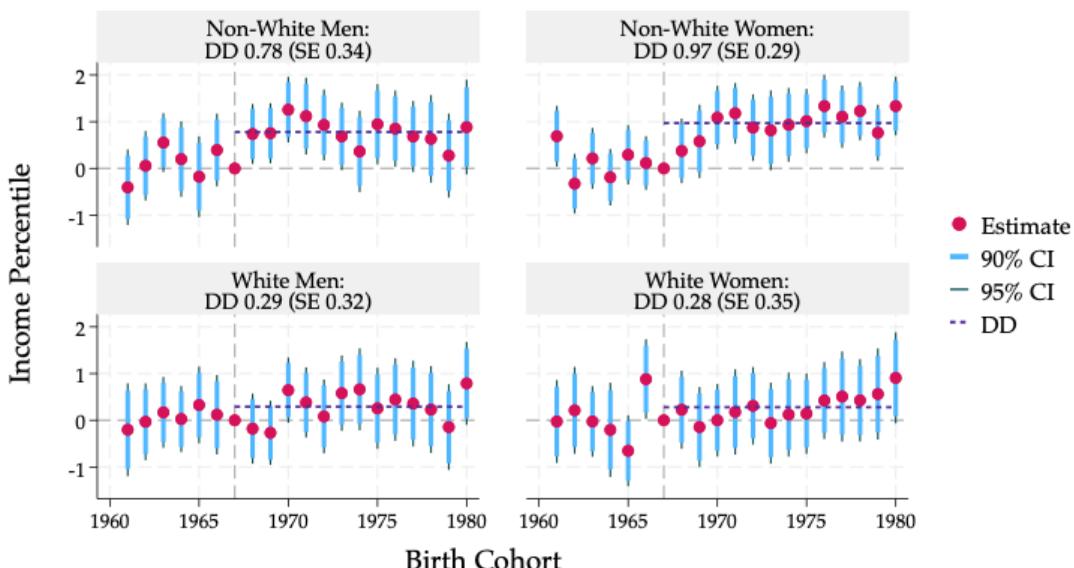
Notes: Graphs show dynamic and post-treatment average (DD) estimates, with 90% and 95% confidence intervals for dynamic estimates. Data are from the IPUMS sample of the 2010 census. All regressions include fixed effects for birth cohort, birth municipality, and racial category. Standard errors are clustered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

D3. Income Percentile Results Using New Difference-in-Differences Estimators

Figure D3: Income Percentile Results Using New Estimators [22]

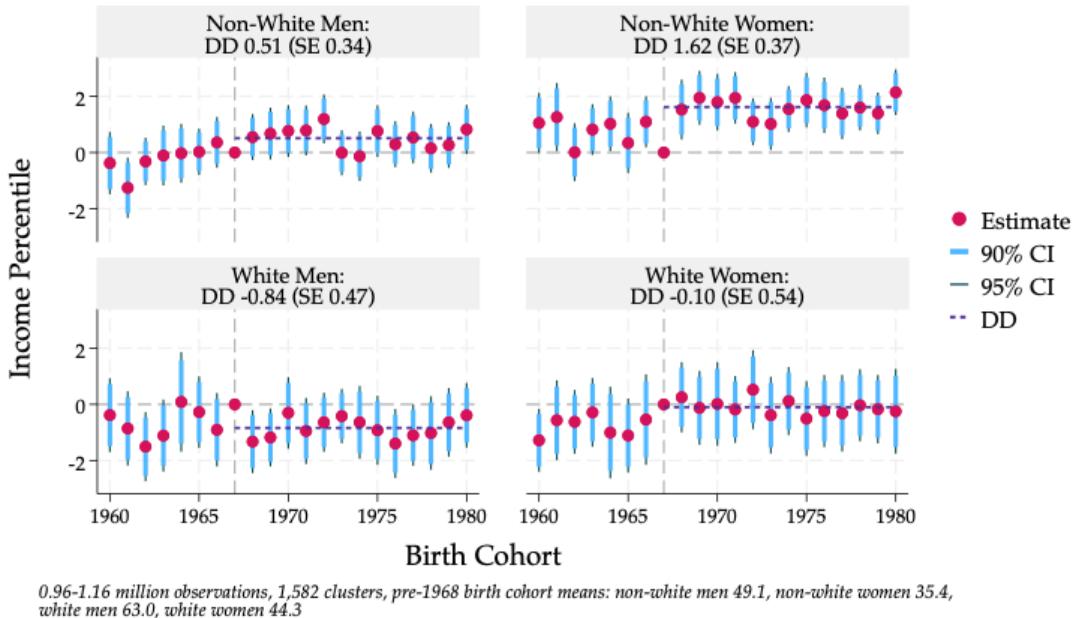


(a) Arkhangelsky et al. (2021)

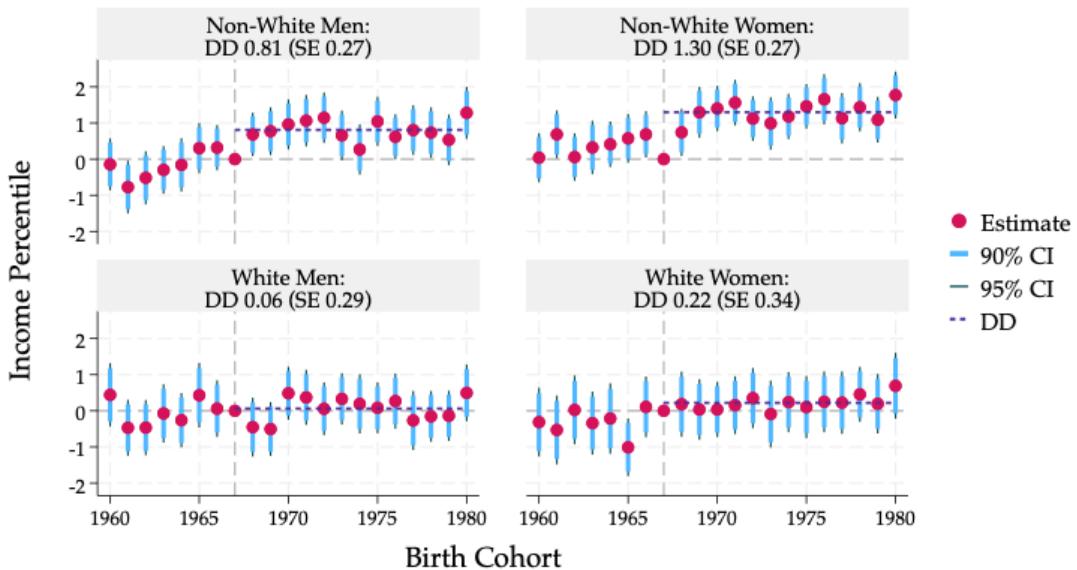


(b) Callaway and Sant'Anna (2021)

Figure D3: Continued



(c) [de Chaisemartin and D'Haultfœuille \(2020\)](#)

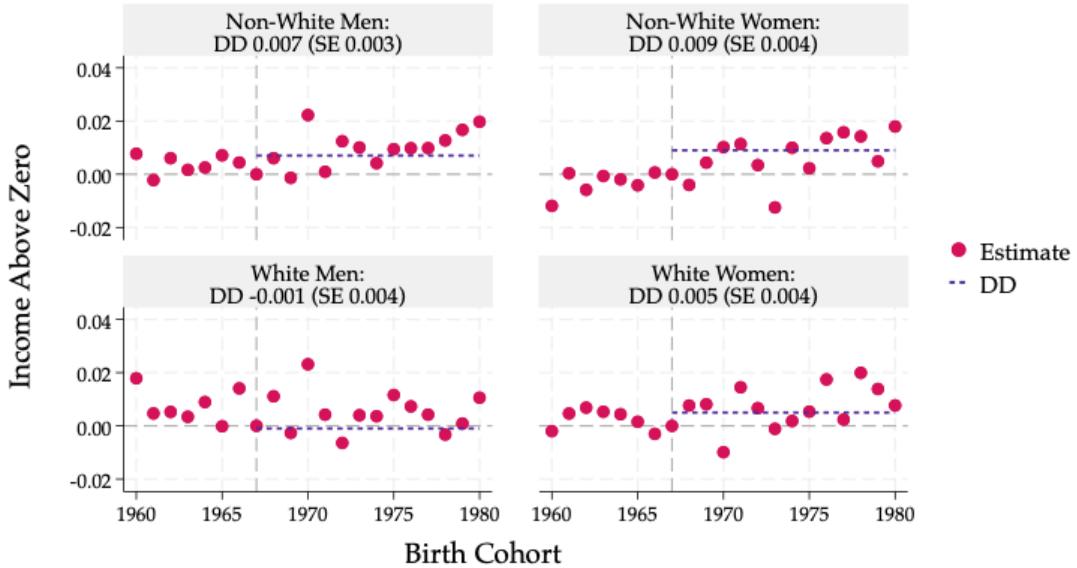


(d) [Sun and Abraham \(2021\)](#)

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.

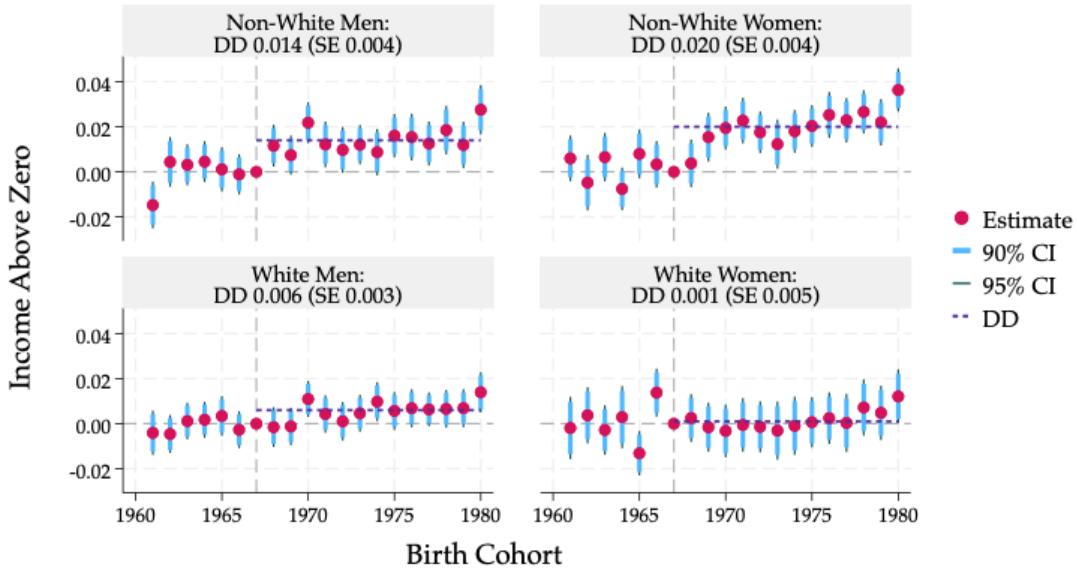
D4. Income Above Zero Results Using New Difference-in-Differences Estimators

Figure D4: Income Above Zero Results Using New Estimators [22]



26,628-30,009 observations, 1,282 clusters, pre-1968 birth cohort means: non-white men 0.807, non-white women 0.727, white men 0.890, white women 0.732

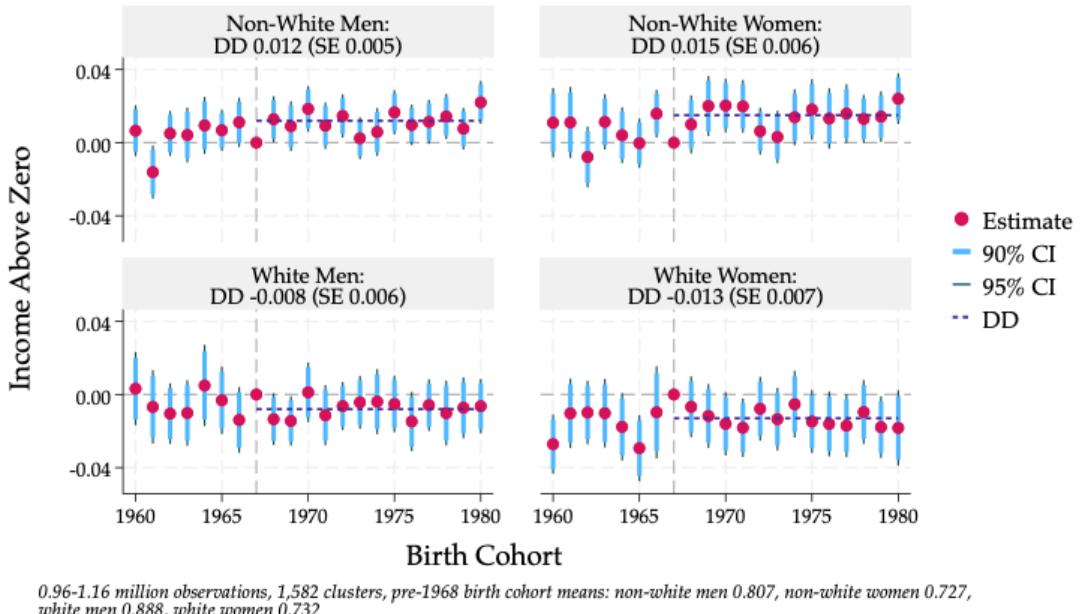
(a) Arkhangelsky et al. (2021)



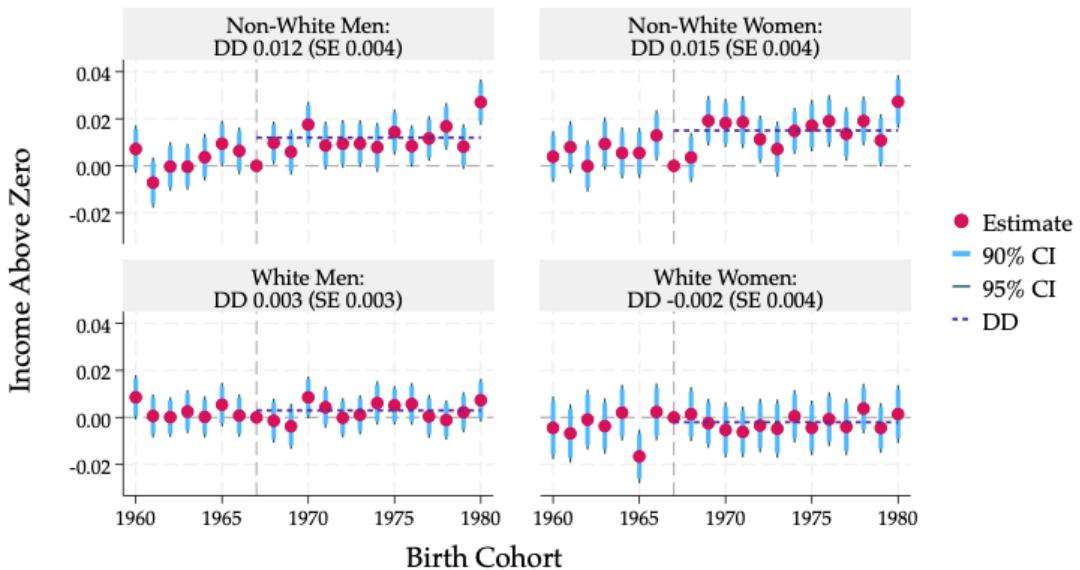
1.01-1.32 million observations, 1,582 clusters, pre-1968 birth cohort means: non-white men 0.807, non-white women 0.727, white men 0.888, white women 0.732

(b) Callaway and Sant'Anna (2021)

Figure D4: Continued



(c) [de Chaisemartin and D'Haultfœuille \(2020\)](#)

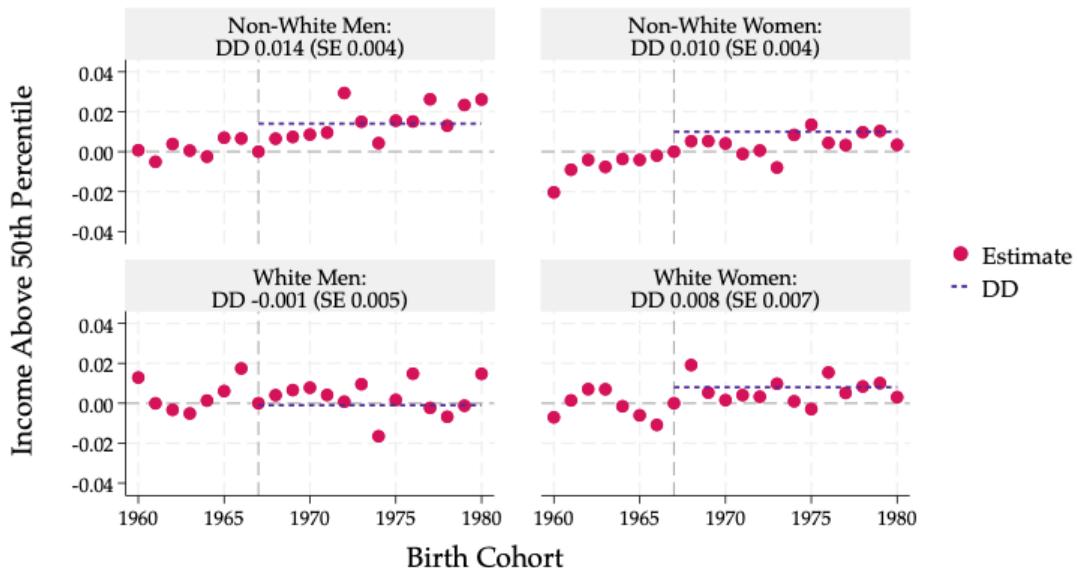


(d) [Sun and Abraham \(2021\)](#)

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.

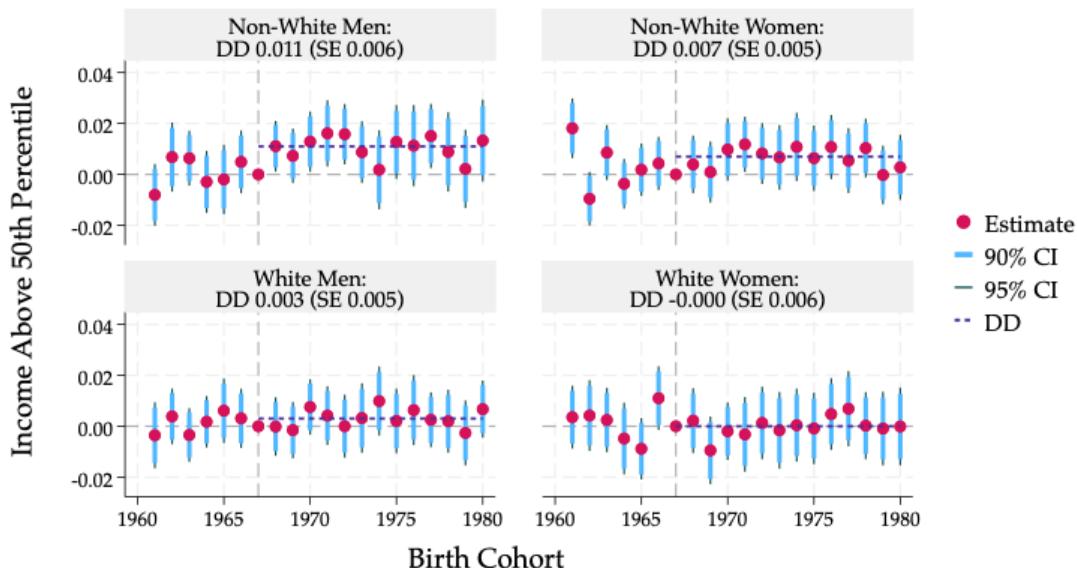
D5. Income Above 50th Percentile Results Using New Difference-in-Differences Estimators

Figure D5: Income Above 50th Percentile Results Using New Estimators [22]



26,628-30,009 observations, 1,282 clusters, pre-1968 birth cohort means: non-white men 0.624, non-white women 0.407, white men 0.784, white women 0.549

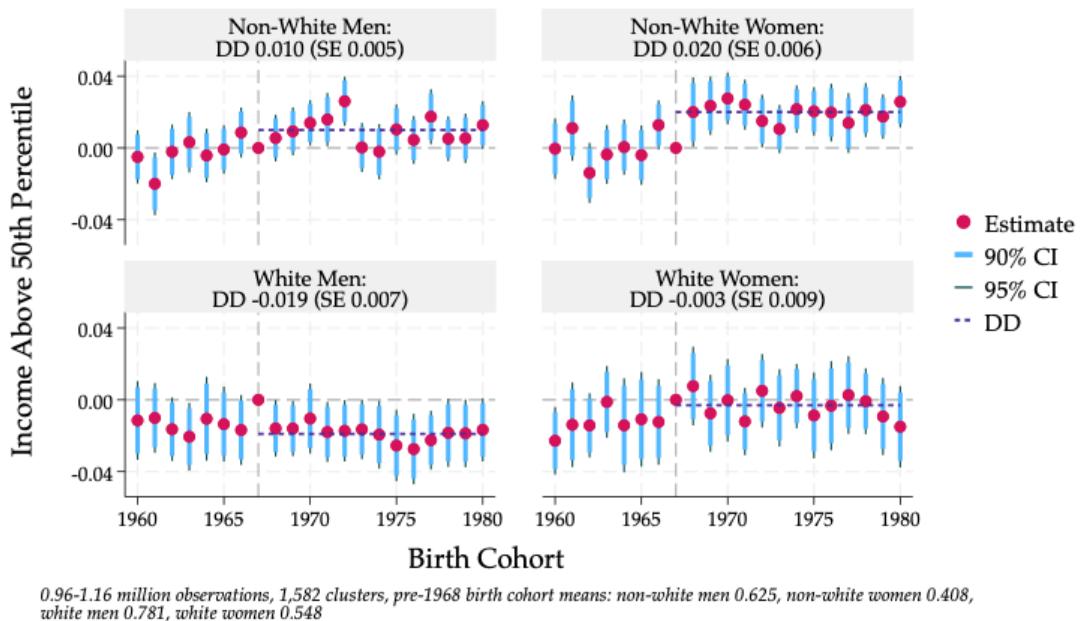
(a) Arkhangelsky et al. (2021)



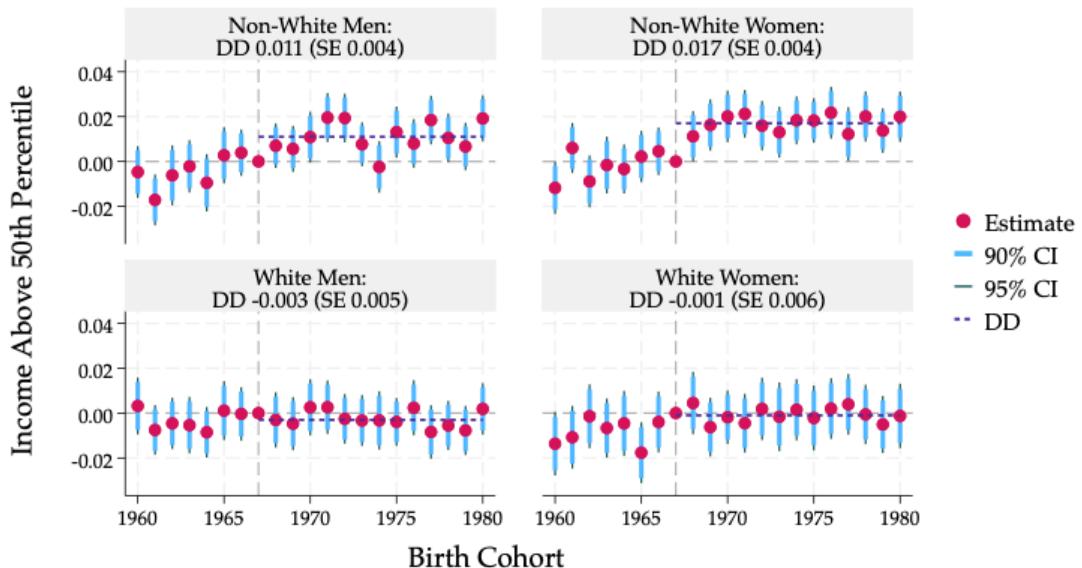
1.01-1.32 million observations, 1,582 clusters, pre-1968 birth cohort means: non-white men 0.625, non-white women 0.408, white men 0.781, white women 0.548

(b) Callaway and Sant'Anna (2021)

Figure D5: Continued



(c) [de Chaisemartin and D'Haultfœuille \(2020\)](#)

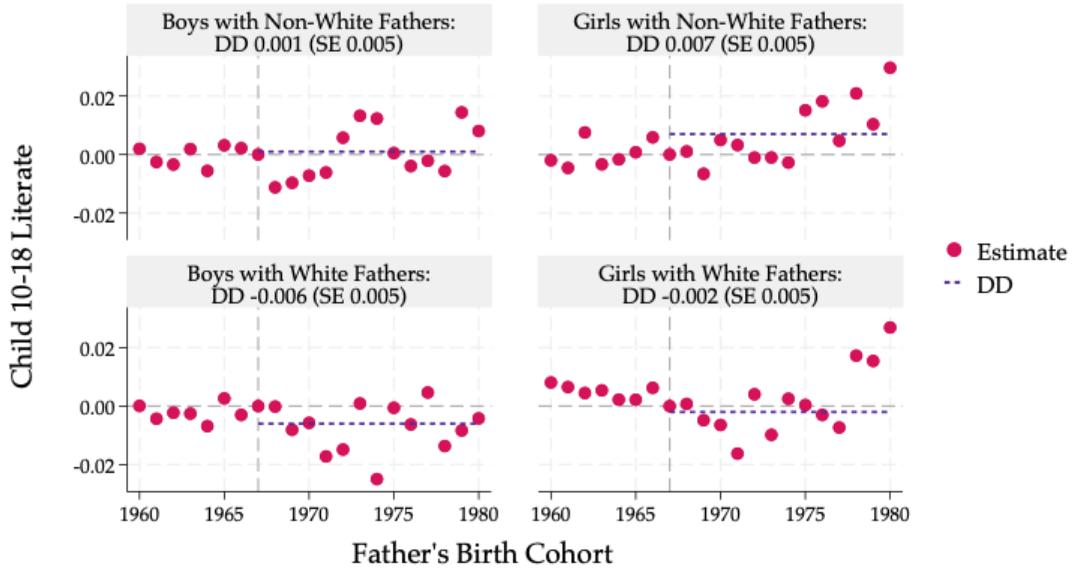


(d) [Sun and Abraham \(2021\)](#)

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.

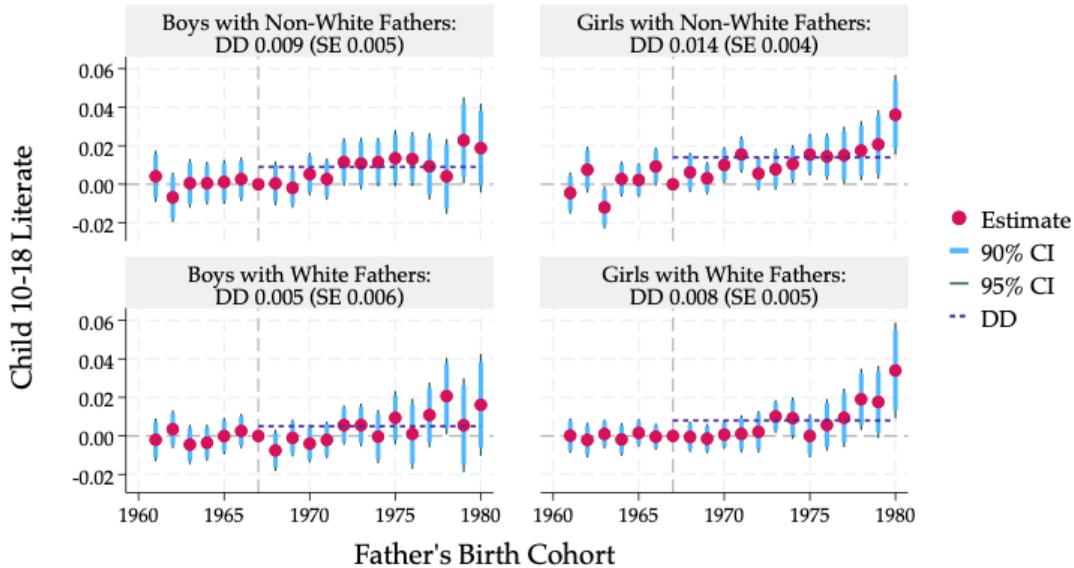
D6. Next Generation Literacy Results Using New Difference-in-Differences Estimators

Figure D6: Next Generation Literacy Results Using New Estimators [22]



12,747-20,622 observations, 607 clusters, pre-1968 father's birth cohort means: boys with non-white fathers 0.905, girls with non-white fathers 0.935, boys with white fathers 0.961, girls with white fathers 0.971

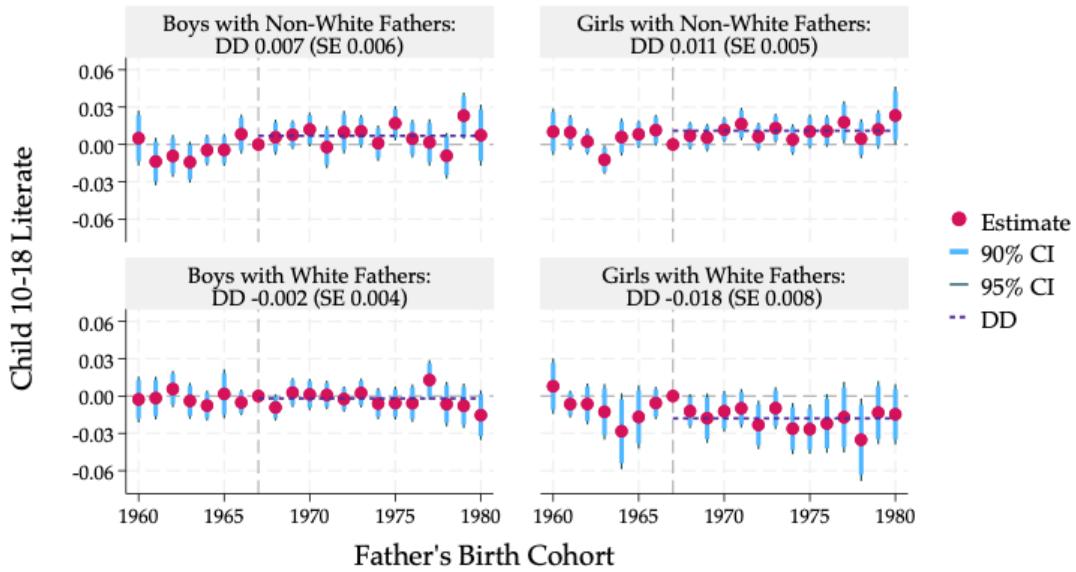
(a) Arkhangelsky et al. (2021)



0.36-0.62 million observations, 1,582 clusters, pre-1968 father's birth cohort means: boys with non-white fathers 0.906, girls with non-white fathers 0.936, boys with white fathers 0.957, girls with white fathers 0.969

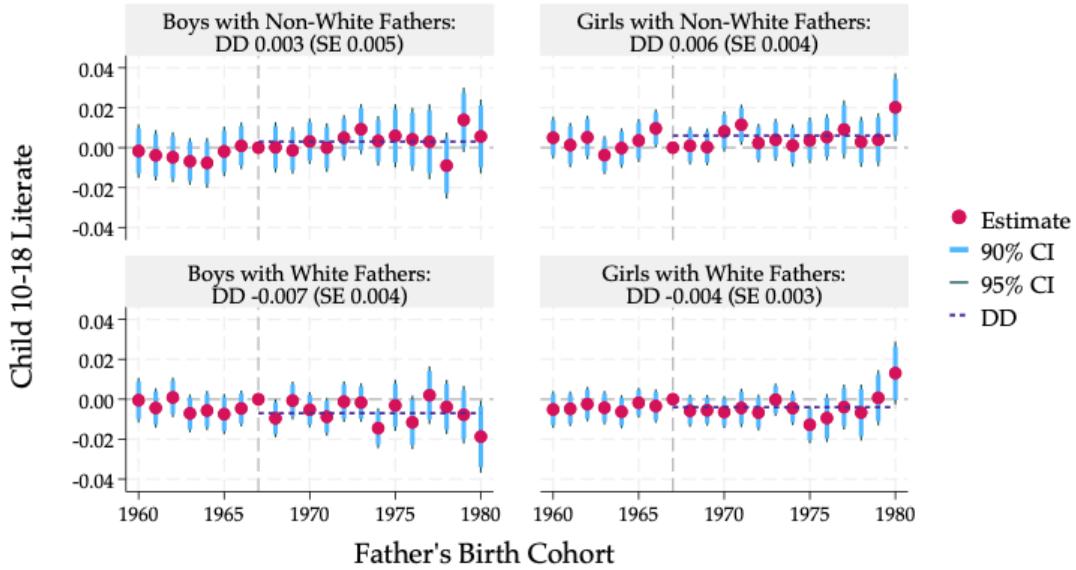
(b) Callaway and Sant'Anna (2021)

Figure D6: Continued



0.32-0.51 million observations, 1,582 clusters, pre-1968 father's birth cohort means: boys with non-white fathers 0.906, girls with non-white fathers 0.936, boys with white fathers 0.957, girls with white fathers 0.969

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



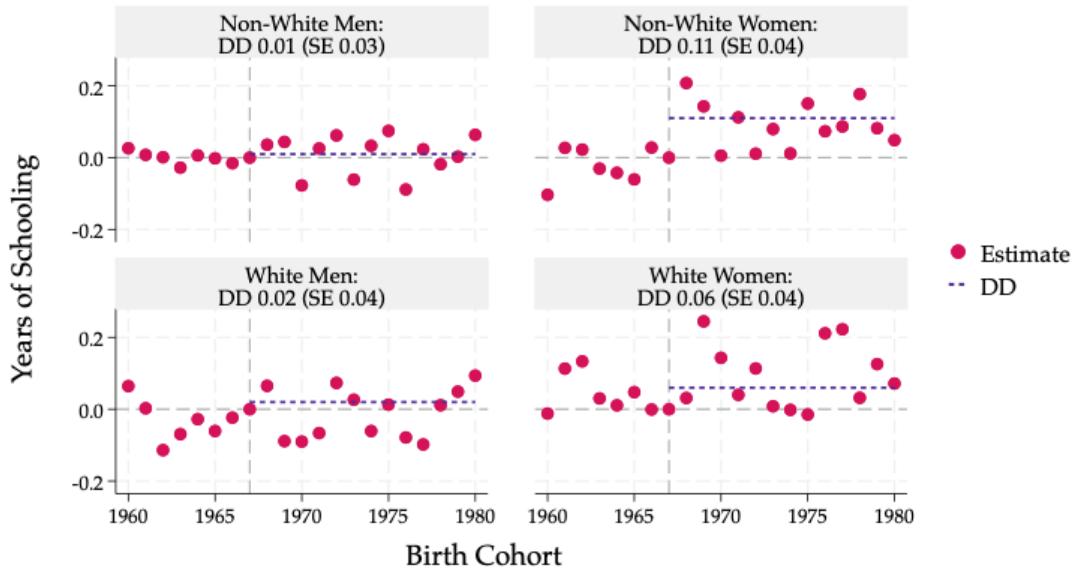
0.36-0.62 million observations, 1,582 clusters, pre-1968 father's birth cohort means: boys with non-white fathers 0.906, girls with non-white fathers 0.936, boys with white fathers 0.957, girls with white fathers 0.969

(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.

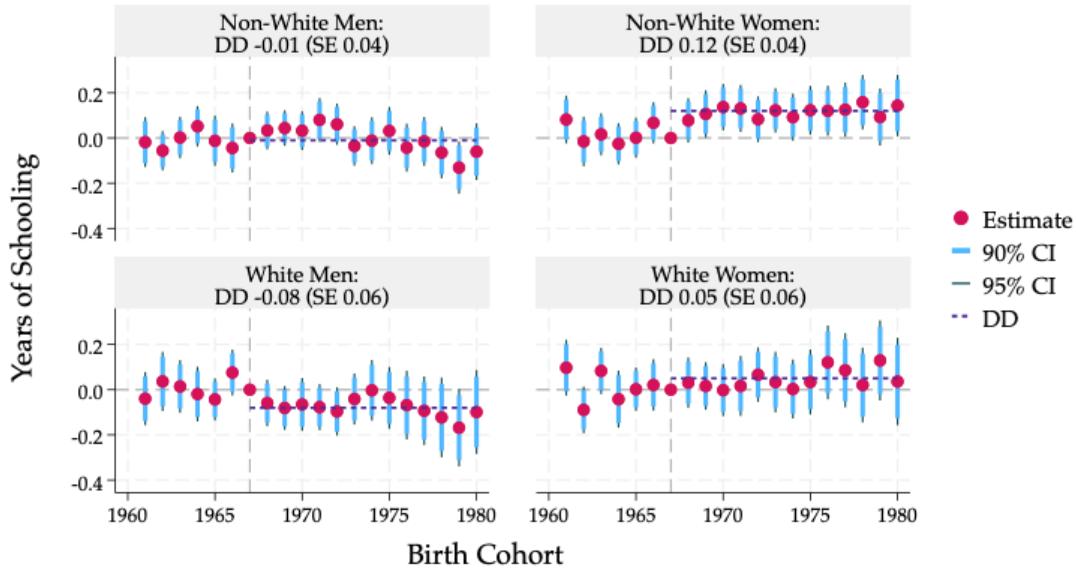
D7. Years of Schooling Results Using New Difference-in-Differences Estimators

Figure D7: Years of Schooling Results Using New Estimators [25]



26,628-30,009 observations, 1,282 clusters, pre-1968 birth cohort means: non-white men 4.80, non-white women 5.42, white men 6.46, white women 6.97

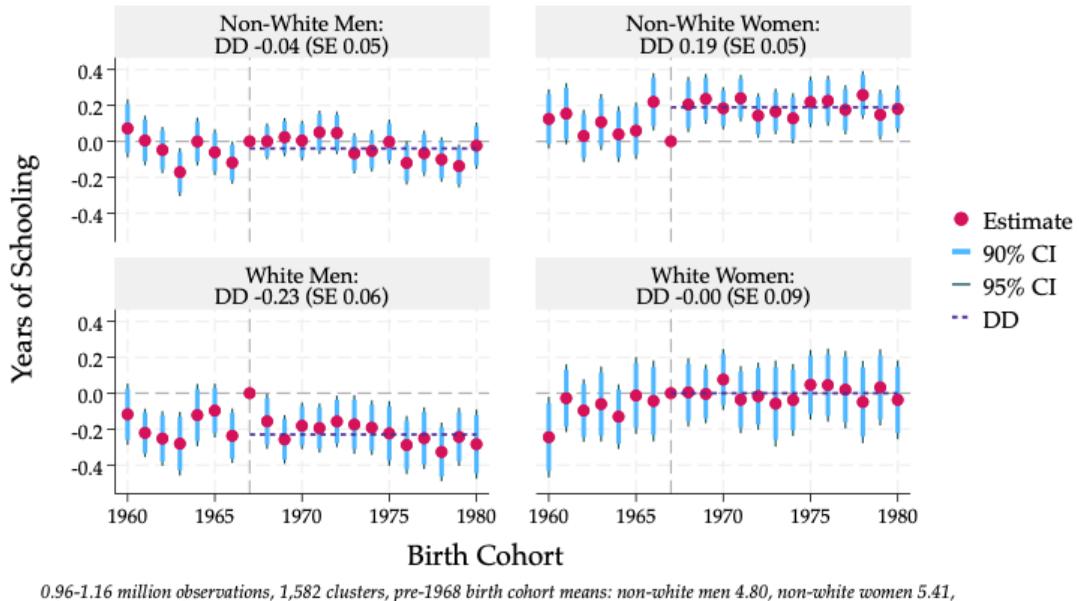
(a) Arkhangelsky et al. (2021)



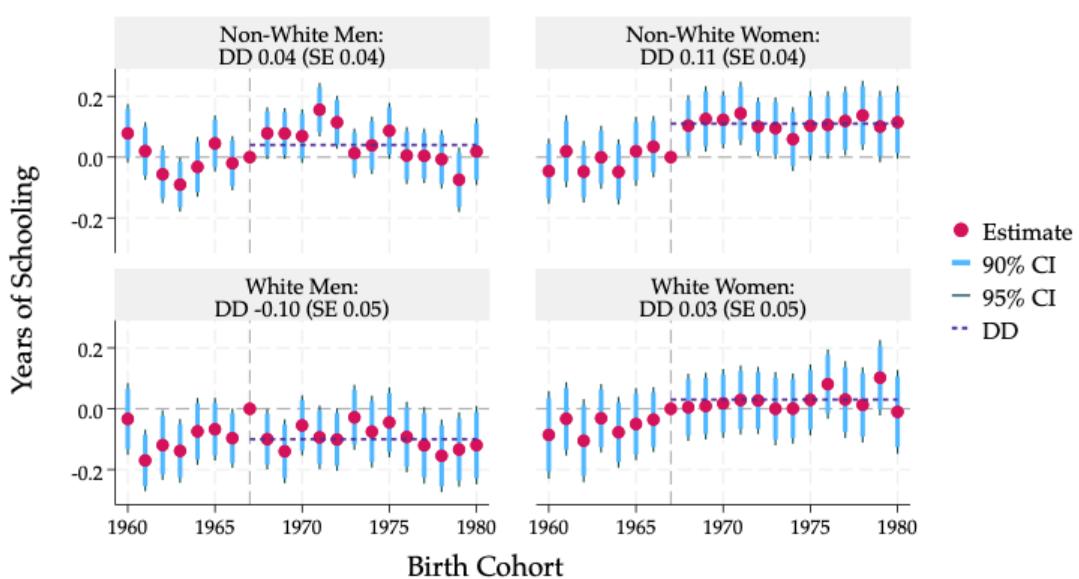
1.01-1.32 million observations, 1,582 clusters, pre-1968 birth cohort means: non-white men 4.80, non-white women 5.41, white men 6.44, white women 6.96

(b) Callaway and Sant'Anna (2021)

Figure D7: Continued



(c) [de Chaisemartin and D'Haultfœuille \(2020\)](#)

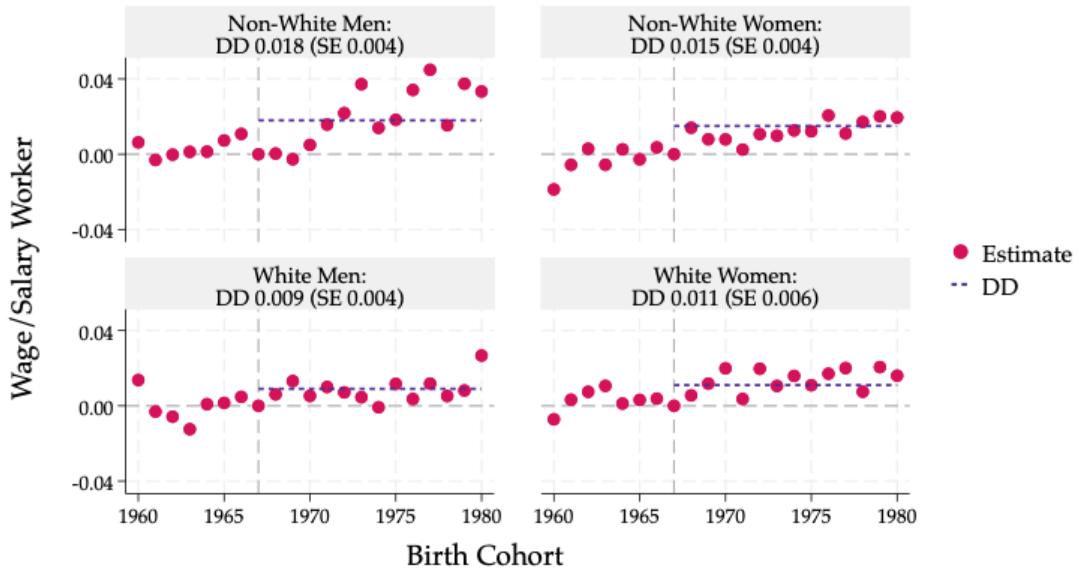


(d) [Sun and Abraham \(2021\)](#)

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.

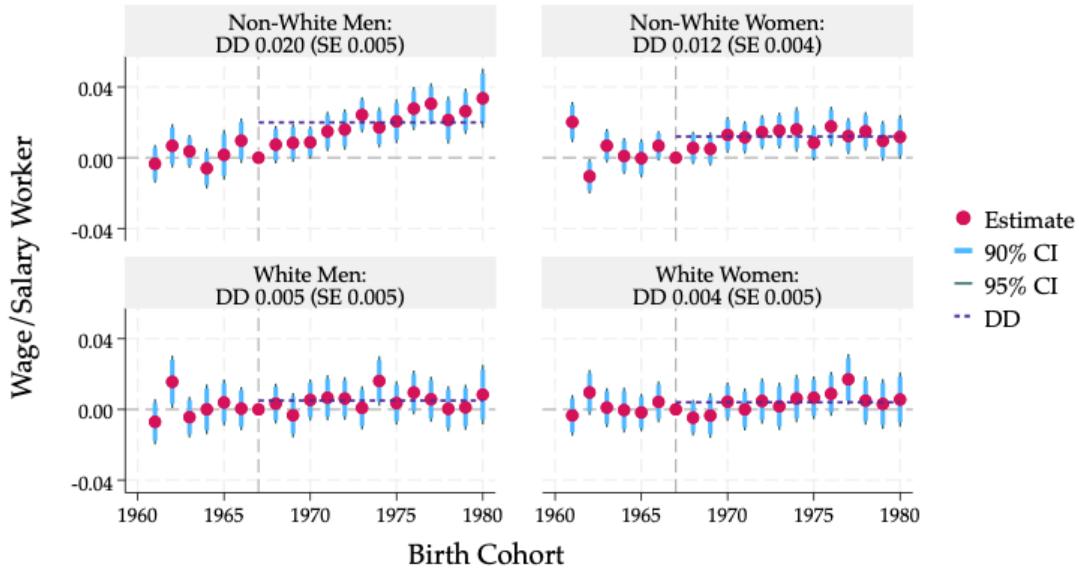
D8. Wage or Salary Worker Results Using New Difference-in-Differences Estimators

Figure D8: Formal Employment Results Using New Estimators [25]



26,628-30,009 observations, 1,282 clusters, pre-1968 birth cohort means: non-white men 0.328, non-white women 0.230, white men 0.376, white women 0.315

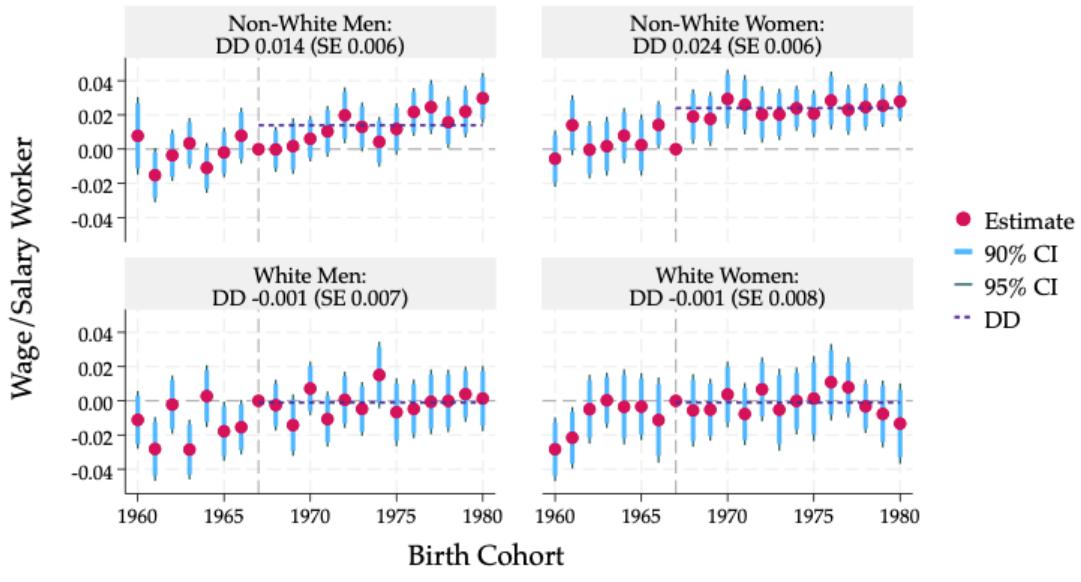
(a) Arkhangelsky et al. (2021)



1.01-1.32 million observations, 1,582 clusters, pre-1968 birth cohort means: non-white men 0.328, non-white women 0.230, white men 0.374, white women 0.313

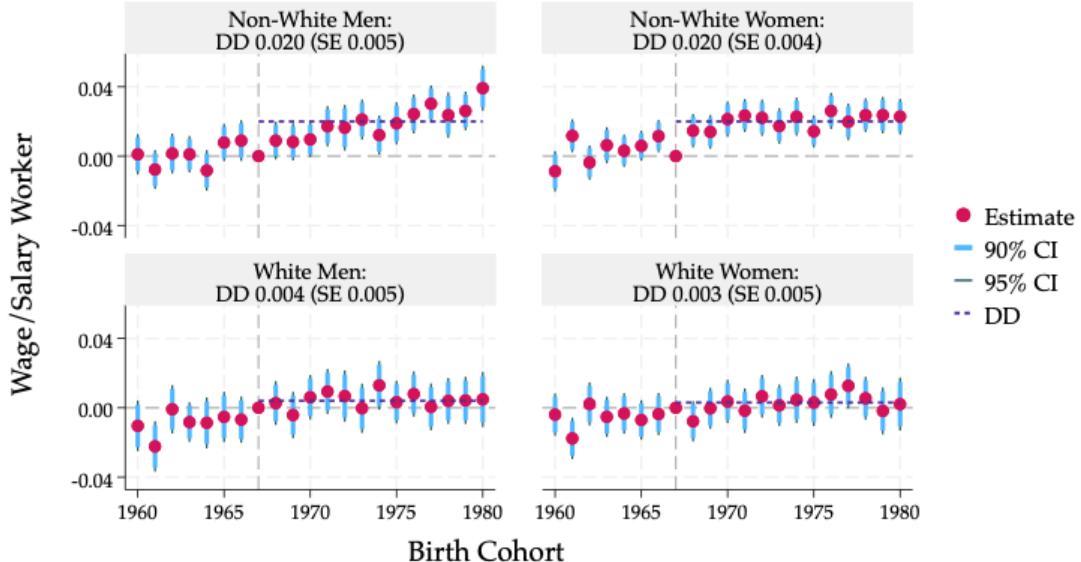
(b) Callaway and Sant'Anna (2021)

Figure D8: Continued



0.96-1.16 million observations, 1,582 clusters, pre-1968 birth cohort means: non-white men 0.328, non-white women 0.230, white men 0.374, white women 0.313

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



1.01-1.32 million observations, 1,582 clusters, pre-1968 birth cohort means: non-white men 0.328, non-white women 0.230, white men 0.374, white women 0.313

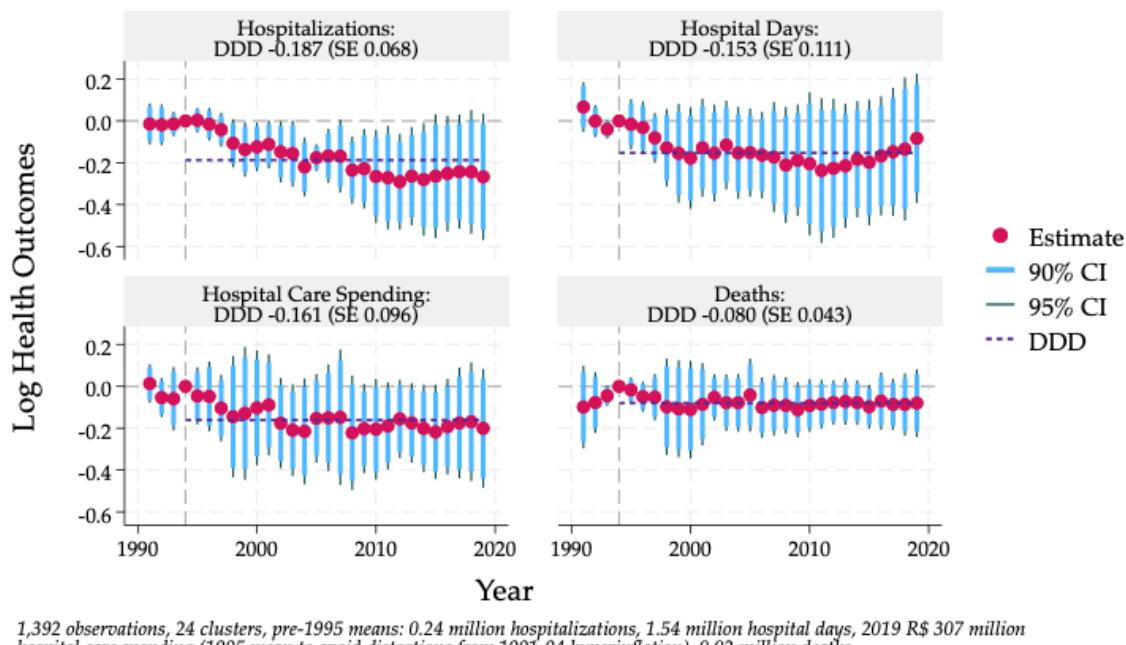
(d) Sun and Abraham (2021)

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 State Public Health Care Systems

E1. Circulatory Disease Results Using Traditional Confidence Intervals

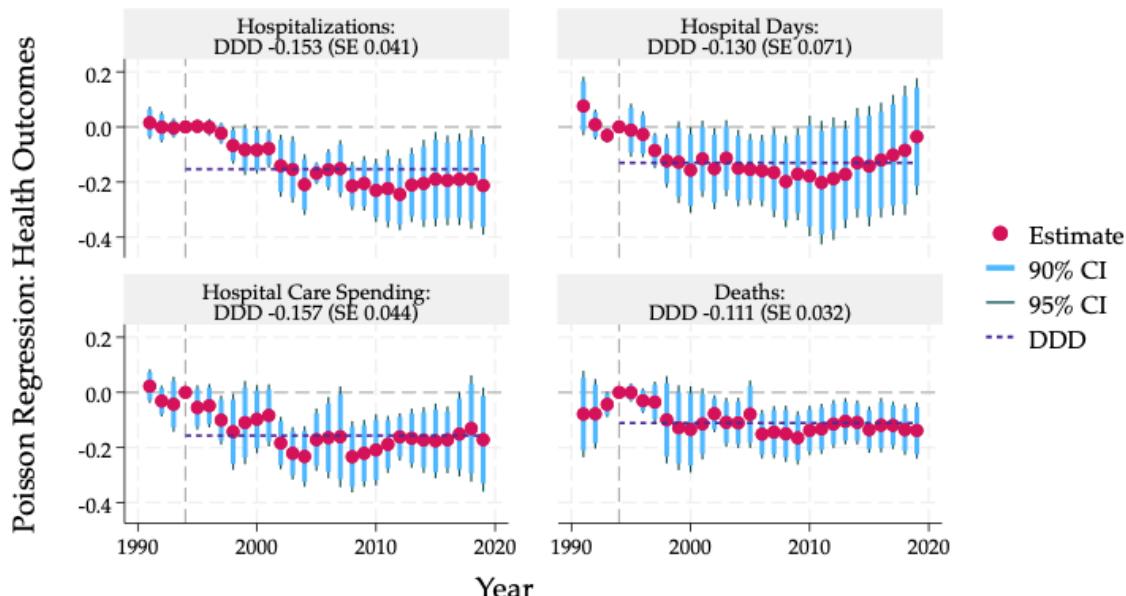
Figure E1: Circulatory Disease Results Using Traditional Confidence Intervals [28]



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. Circulatory Disease Results Using Poisson Regression and Traditional Confidence Intervals

Figure E2: Circulatory Disease Results Using Poisson Regression and Traditional Confidence Intervals [28]

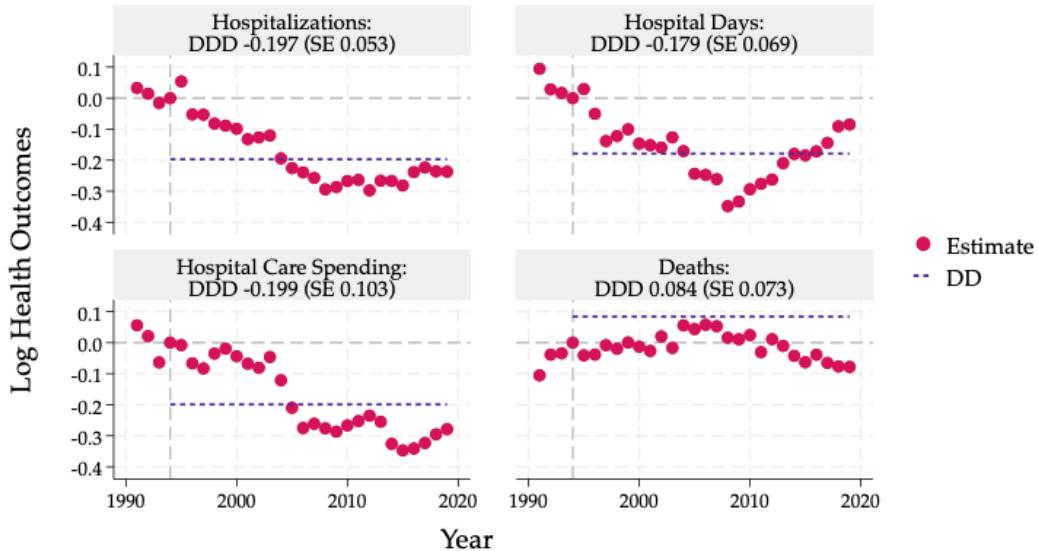


1,392 observations, 24 clusters, pre-1995 means: 0.24 million hospitalizations, 1.54 million hospital days, 2019 R\$ 307 million hospital care spending (1995 mean to avoid distortions from 1991-94 hyperinflation), 0.02 million deaths

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

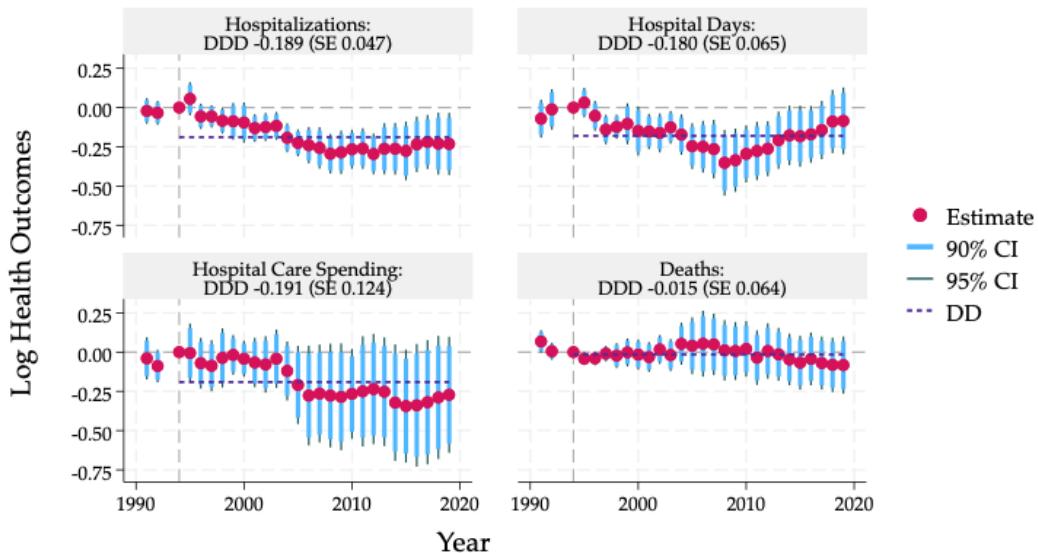
E3. Circulatory Disease Results Using New Difference-in-Differences Estimators

Figure E3: Circulatory Disease Results Using New Estimators [29]



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

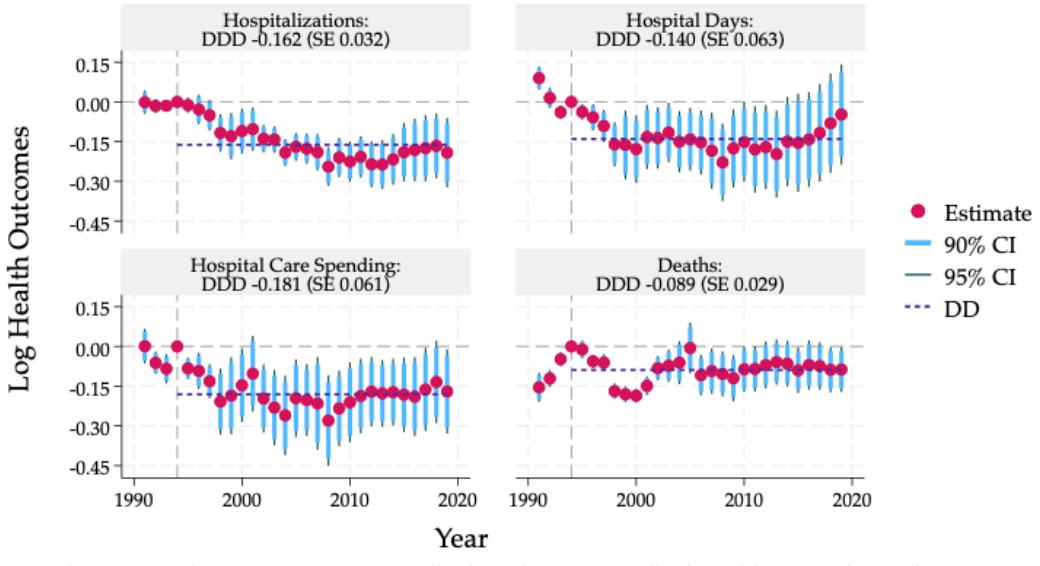
(a) Arkhangelsky et al. (2021)



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

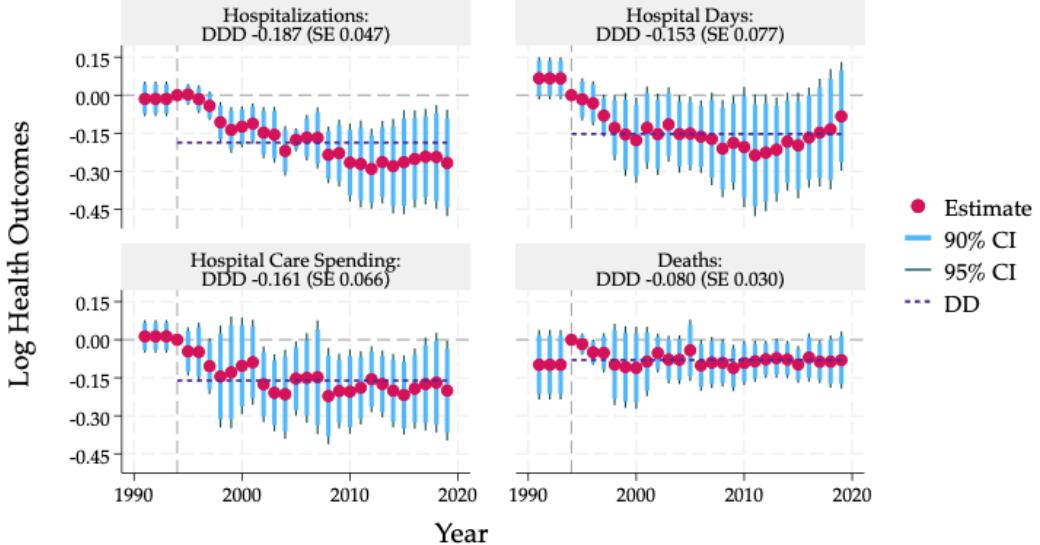
(b) Callaway and Sant'Anna (2021)

Figure E3: Continued



1,350 observations, 24 clusters, pre-1995 means: 0.24 million hospitalizations, 1.54 million hospital days, 2019 R\$ 307 million hospital care spending (1995 mean to avoid distortions from 1991-94 hyperinflation), 0.02 million deaths

(c) [de Chaisemartin and D'Haultfœuille \(2020\)](#)



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

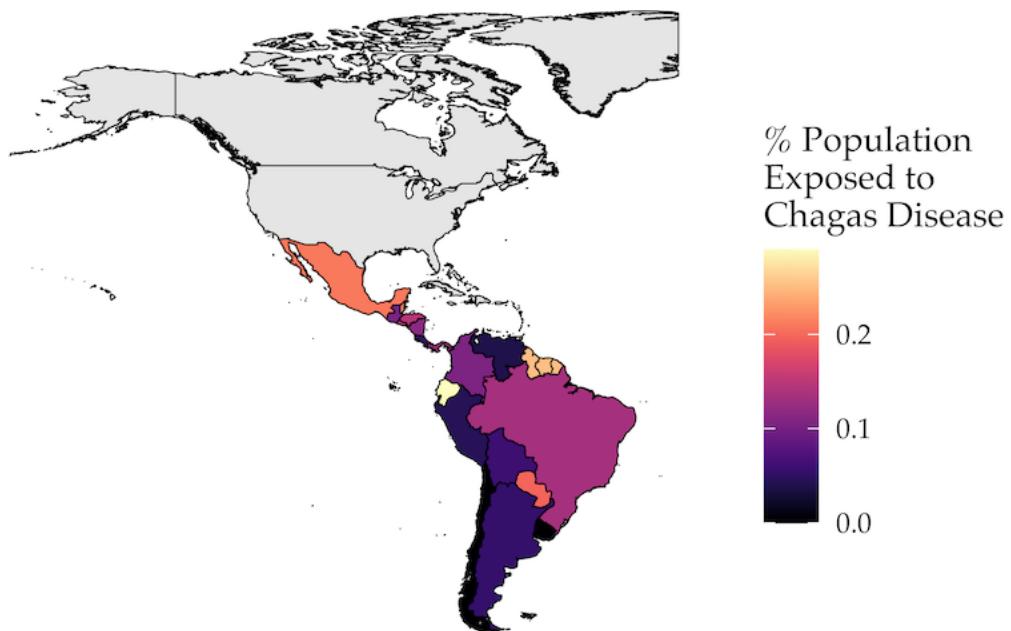
(d) [Sun and Abraham \(2021\)](#)

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 [33]



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.