

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

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Abstract

Poor, non-white Latin Americans disproportionately suffer from Chagas disease, a neglected tropical disease that causes acute symptoms and chronic heart problems decades later. We show that Brazil's post-1983 vector control campaign raised GDP per capita and lowered Gini coefficients in treated municipalities within two decades. Eliminating childhood exposure decreased racial income inequality in adulthood and increased literacy rates in the next generation. The primary mechanism was improved adult health, which also reduced public spending on circulatory disease hospitalizations. Controlling illnesses afflicting the poor, particularly those with chronic effects, can thus foster inclusive growth while strengthening fiscal health in developing economies.

Keywords: Neglected Diseases, Racial Inequality, Intergenerational Mobility, Health Spending

JEL Classification: D31, H51, I14, I15, J15, J62, O15

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[A] tragedy that makes no sound, patients that [cannot] pay, [an] illness that will not sell. Chagas disease holds no allure for the pharmaceutical industry, for politicians or the press. It selects its victims from among the poor. It bites and, slowly, relentlessly, they waste away.

—Eduardo Galeano ([Médicos Sin Fronteras, 2013](#))

1. Introduction

Few regions match Latin America's unequal income distribution, where the top decile captures a majority of national income and white individuals earn over two times more than their darker-skinned peers ([Chancel et al., 2021](#); [Telles et al., 2023](#); [Woo-Mora, 2025](#)). Historians and public health scholars have long suggested that one potential contributor to this middle-income region's disparities and underdevelopment is Chagas disease, which afflicts 8 million predominantly non-white people in Latin America and another 75 million are at risk of contracting ([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 often labeled a "neglected disease of poor, rural, and forgotten populations" because its vectors—triatomine "kissing bugs"—much more easily infest housing made of substandard materials ([Coura and Viñas, 2010](#); [Houweling et al., 2016](#)).¹ It can cause weeks of acute symptoms (e.g., fever, fatigue, and headaches), and 10 to 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).

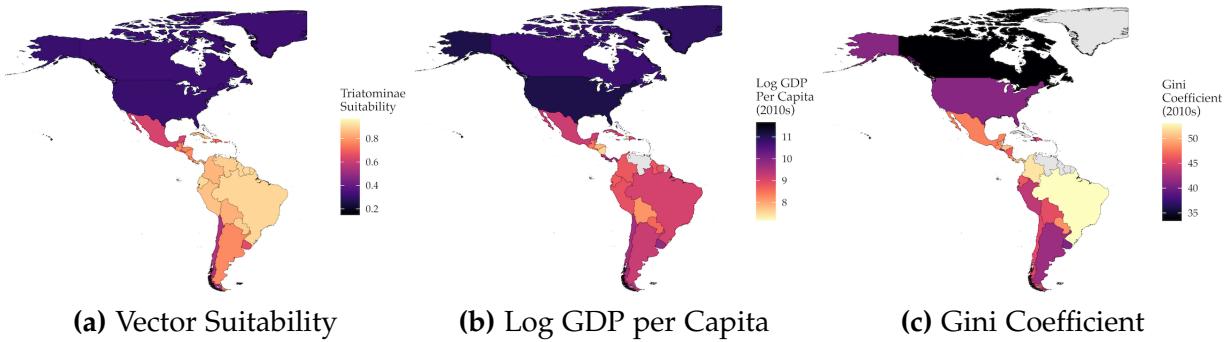
While the most studied tropical illnesses (e.g., malaria and parasitic worm infections) reduce incomes by limiting children's human capital, Chagas disease's most important manifestations arise decades later, meaning that any schooling effects it has should be dwarfed by its impacts on prime-age labor supply. It is therefore emblematic of many neglected tropical diseases (NTDs), which disproportionately afflict the poorest people in the poorest countries and often result in chronic health issues, so their development impacts can be missed or misattributed unless studied over the long run ([Hotez, 2011](#)). Understanding how these maladies influence development is essential not only for accurately measuring the returns to their control, but also for uncovering alternative pathways through which NTDs can perpetuate inequality and underdevelopment. Already, chronic and other non-communicable diseases consume the bulk of health spending in low- and middle-income countries, and they are projected to dominate the global disease burden within the next decade—making it even more urgent to understand the development impacts of chronic health conditions like Chagas disease's adult-onset symptoms ([NCD Alliance, 2024](#)).

In this paper, we study whether controlling Chagas disease can contribute to inclusive growth in a developing economy. We begin by showing suggestive evidence that it could: country-level measures of the ecological suitability for Chagas disease vectors are strongly associated with lower GDP per capita and higher Gini coefficients, but only within the Americas.² We then

¹ Additionally, lack of access to adequate health education, health care, and environmental management strengthen the link between poverty and Chagas disease ([Hotez et al., 2013](#)).

² This continent-specific relationship mirrors the one documented by [Alsan \(2015\)](#) for African try-

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.

investigate the causal impacts of reducing Chagas disease transmission looking within Brazil—a country that Figure 1 shows has high vector suitability, a middle income, and extremely high inequality—using a difference-in-differences design that exploits the 1984 start of its campaign to eliminate vectorial transmission of Chagas disease.

Specifically, we study the consequences of indoor residual spraying (IRS) against the main Chagas disease vector, which was made possible by the invention of pyrethroid insecticides and a 1975–83 nationwide entomological survey measuring its presence at the municipal level. Our basic difference-in-differences strategy combines within-state, cross-municipality, and cross-year variation in exposure to this vector, comparing those where it was never present against those in which it was found before IRS began. Following recent developments in the difference-in-differences literature ([Goodman-Bacon, 2021](#)), we exclude municipalities in the state of São Paulo, which was wealthy enough to have conducted its own vector control program in the 1960s with a much less cost-effective insecticide. With respect to time variation, detailed information on when each municipality was treated is unavailable, so we make the conservative assumption that treatment occurred uniformly in 1984.

We first examine the long-run impacts of eliminating Chagas disease transmission on income and inequality across municipalities, comparing our treatment and control groups using data on GDP per capita and from population censuses between 1970 and 2010. We find that in treatment municipalities over the 26 years after 1984, output per person rose about 20% more (equivalent to 0.7% faster annual growth) and average incomes in census data increased by 1 percentile (about 2.5% of the pre-campaign mean), while Gini coefficients fell by about 1.8% more (a 0.1% faster annual decline). Importantly, these effects were apparent in the first post-treatment census

panosomiasis (sleeping sickness). Chagas disease is also known as American trypanosomiasis because it is caused by parasites of the genus *Trypanosoma* as well, but there is no overlap in these species, their vectors, or the symptoms they cause.

and grew more pronounced over time, suggesting that averting Chagas disease's acute stage had detectable impacts that were then compounded by reductions in chronic symptoms. When examining mechanisms, we find that human capital gains over 26 years were modest (0.13 years, or 4% of the pre-treatment mean) and were not apparent before 2010, so neither their size nor their timing can explain the results above. Instead, an explanation more consistent with the evidence is that more prime-age adults were filling the labor force instead of hospital beds.

We complement the municipality-level results by examining the long-run effects 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: non-white adults born in treatment municipalities ranked 1 percentile (2.5% of the mean for pre-treatment cohorts) higher in the national income distribution and earned about 10% more, whereas there were no detectable changes for white adults, consistent with their much lower degree of vector exposure. We also show that the literacy of children of non-white men from treatment municipalities increased by 0.5 percentage points (p.p., or 0.5%) more, suggesting that Chagas disease control could weaken intergenerational poverty traps. In line with our municipality-level results, we find that schooling gains for these adults were modest (about 0.1 years, or 1.8%) but the share earning positive incomes rose by 1.9 p.p. (2.3%), indicating that much of the improvement came from higher labor force participation. Mediation analyses corroborate that for non-white adults, the extensive margin labor supply response explained a far larger share of income gains than schooling, reflecting a reduction in chronic symptoms enabling more prime-age adults to work.

If this decrease in chronic Chagas disease morbidity was the primary driver of the increase in non-white incomes, it could also have resulted in substantial savings for Brazil's universal health care system (known as SUS), as circulatory diseases account for 10% of the hospitalizations it pays for and 20% of its hospital care expenditures. We therefore examine hospitalizations covered by SUS and the amount spent on them 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. Our triple-differences results show that by 2005-10, there had been greater reductions in hospitalizations (19%) and hospital care spending (16%) resulting from circulatory system disease compared to all other categories. 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 in the disease's chronic stage.

We conduct two simple cost-benefit analyses comparing the costs of IRS against the main Chagas disease vector with 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 20%. One lesson from this exercise is that excluding the hospital care savings reduces the IRR by 1 p.p. (5%), 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.³ As such, another important lesson is that if developing countries are considering interventions to improve the health of their poorest citizens but do not expect to collect taxes from them (e.g., due to high informality or incomes below a minimum level), they can still recoup their investments in controlling NTDs causing chronic health problems because doing so will reduce future health care burdens.

Lastly, we conduct an extrapolation exercise to shed light on the potential 26-year impacts of eliminating Chagas disease transmission on disparities and underdevelopment across Latin America. We multiply each country's GDP per capita and Gini coefficient by the share of its population exposed to transmission ([World Health Organization, 2015](#)) and the percent effects on these outcomes for Brazilian municipalities. Our calculations suggest that on average, this intervention could increase Latin American incomes per person by 2% and reduce Gini coefficients by 0.2%. Notably, greater benefits would accrue to the countries with higher shares exposed to Chagas disease, which are precisely those that are more underdeveloped and unequal today.

Our results contribute to several literatures. By documenting how the chronic burden of an NTD can inhibit growth and perpetuate racial and spatial disparities, we extend the literature on health and development beyond its extant focus on acute pathogens primarily affecting children and provide new evidence that fighting adult-onset diseases can be a powerful but underappreciated lever for inclusive growth. In this way, we complement the growing body of research in economics on disease and long-run development, which documents how illnesses reduce childhood human capital and thus incomes later in life (e.g., [Miguel and Kremer, 2004](#); [Bleakley, 2007, 2010](#); [Cutler et al., 2010](#); [Lucas, 2010](#); [Mora-Garcia, 2018](#); [Bütikofer and Salvanes, 2020](#); [Hamory et al., 2021](#)). We show that suppressing Chagas disease, which afflicts adults with its short- and long-run symptoms, raises municipalities' GDPs per capita and lowers Gini coefficients primarily by keeping adults working rather than increasing children's schooling. These findings thus add to the literature on health's macroeconomic effects, or lack thereof ([Acemoglu and Johnson, 2007](#); [Ashraf, Lester and Weil, 2009](#); [Weil, 2014](#)), in which controlling diseases affecting prime-age adults has impacts shortly after treatment ([Tompsett, 2020](#); [Carney and Denton-Schneider, 2025](#)).

Second, we demonstrate that controlling a chronic infectious disease can narrow income gaps and weaken intergenerational poverty traps in a developing economy, adding to our understanding of the effects of health on inequality (and vice versa) and the transmission of socioeconomic status from parents to children (e.g., [Farmer, 2001](#); [Deaton, 2003](#); [O'Donnell, Van Doorslaer and Van Ourti, 2015](#); [Bütikofer, Løken and Salvanes, 2019](#); [Abrahamsson et al., 2025](#)). Our closest

³ It would take a discount rate of nearly 8% for there to be a net cost to the government, which is well above the standard 5% rate in studies of developing countries ([Haacker, Hallett and Atun, 2020](#)).

antecedent for the inequality and intergenerational mobility impacts of disease control is the study of Norway's 1948 tuberculosis campaign by [Bütikofer and Salvanes \(2020\)](#), who showed that eradicating TB raised adult earnings and mobility for children from high-incidence areas, with larger gains for those from poorer backgrounds. We show that controlling an NTD with chronic symptoms in a racially stratified middle-income country yields mobility gains comparable to Norway's TB campaign as well as large public health savings, demonstrating that such interventions can both promote equality and pay for itself in a developing economy.⁴

Third, by measuring the public finance dividend from controlling an adult-onset NTD, we highlight an important fiscal channel in the disease and development literature. Reductions in chronic symptoms lowered hospitalizations and spending on circulatory disease care by roughly one-sixth, savings that on their own yield an IRR of almost 8% and an infinite MVPF. In other words, interventions targeting chronic diseases can pay for themselves through savings to the public health system, a particularly relevant result as such conditions absorb more of health budgets in developing countries ([NCD Alliance, 2024](#); [Duhon et al., Forthcoming](#)) and because their governments might be unable to recoup such investments by collecting additional taxes.

Finally, by studying Chagas disease, a malady deeply entrenched in the socioeconomic fabric of Latin America, we offer a novel perspective on the region's unique development trajectory. A growing body of work has sought to understand the region's disappointing growth and pronounced inequality (see [Eslava and Valencia Caicedo, 2023](#); [Telles et al., 2023](#); [Attanasio et al., 2024](#)), while other studies have highlighted how pathogen environments shaped long-run development elsewhere (e.g., [Alsan, 2015](#); [Depetris-Chauvin and Weil, 2018](#)). We contribute quasi-experimental evidence from Latin America that eliminating Chagas disease transmission can have important long-run benefits such as higher rates of inclusive growth in the region. Nonetheless, our results also have relevance beyond Latin America: as climate change and increased migration bring the parasite and its vectors into non-endemic areas in the United States and Europe ([Eberhard et al., 2020](#); [Hernández, 2021](#); [Irish et al., 2022](#)), it becomes ever more important to understand the consequences of this increasingly globalized health challenge.

The rest of the paper is organized as follows. Section 2 provides background on Chagas disease and describes Brazil's mid-1980s vector control campaign. Section 3 presents our data and difference-in-differences empirical strategy. Section 4 reports our main municipality- and individual-level results, and Section 5 examines the underlying mechanisms and the implications of reduced chronic Chagas disease morbidity for Brazil's universal public health care system. Finally, Section 6 discusses the broader implications of the findings, including cost-benefit analyses and an extrapolation of the impacts across Latin America. We conclude with Section 7.

⁴ Moreover, our results imply that in a country with a long history of skin color-based disparities, disease control can be a race-neutral policy that nonetheless reduces racial income inequality, similar to minimum wage policies (e.g., [Derenoncourt and Montialoux, 2021](#); [Derenoncourt et al., 2021](#)). As such, it can be a simple, relatively inexpensive, and politically palatable tool to help mitigate mutually reinforcing health and socioeconomic gaps between white and non-white individuals.

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, which live in cracks in roofs and walls and transmit the parasite humans when they emerge at night to take blood meals.⁵ In Brazil, the most important vector species was *Triatoma infestans*, thought to have been responsible for 80% of infections (Schofield and Dias, 1999).⁶ Appendix A1 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 A2 shows the progression of Chagas disease from a patient's exposure to *T. cruzi* through the rest of their life. The acute stage begins after 1 to 2 weeks of incubation, lasts 1 to 3 months, and is characterized by nonspecific symptoms such as malaise and fever (Rassi et al., 2009). People of any age can experience these symptoms, though children generally become more acutely ill than adults and more than 40% of infections are asymptomatic (Khan, 2011). Individuals then enter the disease's chronic stage. Around half of *T. cruzi* carriers have no subsequent symptoms, but for the others, after 10 to 30 years, the usual outcome is that the heart muscle becomes enlarged and fibrous (Rassi, Rassi and Little, 2000).⁷ This cardiomyopathy makes it more difficult for the heart to pump blood and causes most of the morbidity and mortality from Chagas disease (Nunes et al., 2018). Importantly, reinfection can lead to a reoccurrence of acute stage symptoms, and more reinfections increase the likelihood of eventually experiencing symptomatic chronic Chagas disease (Olivo Freites et al., 2022).

2.2. Association with Poverty and Race

In announcing the discovery of *T. cruzi*, Chagas (1909) noted that its vector often inhabited fissures in the unplastered walls of poor rural patients' homes. He was thus the first of many to link exposure to the parasite with poverty, especially poor-quality housing, outside of cities (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 Chagas disease symptoms can be a cause of it as well:

Many people forgotten by [society] are ... the same people who encounter infection

⁵ The other 10% of transmission occurs through blood transfusions and the placenta.

⁶ 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*.

⁷ 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*.

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 ([Woo-Mora, 2025](#)), it is therefore not surprising that mixed-race ("Brown") Brazilians—who predominate in the rural interior of the country—comprise 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. Patterns Across the Americas: Vector Suitability, Income, and Inequality

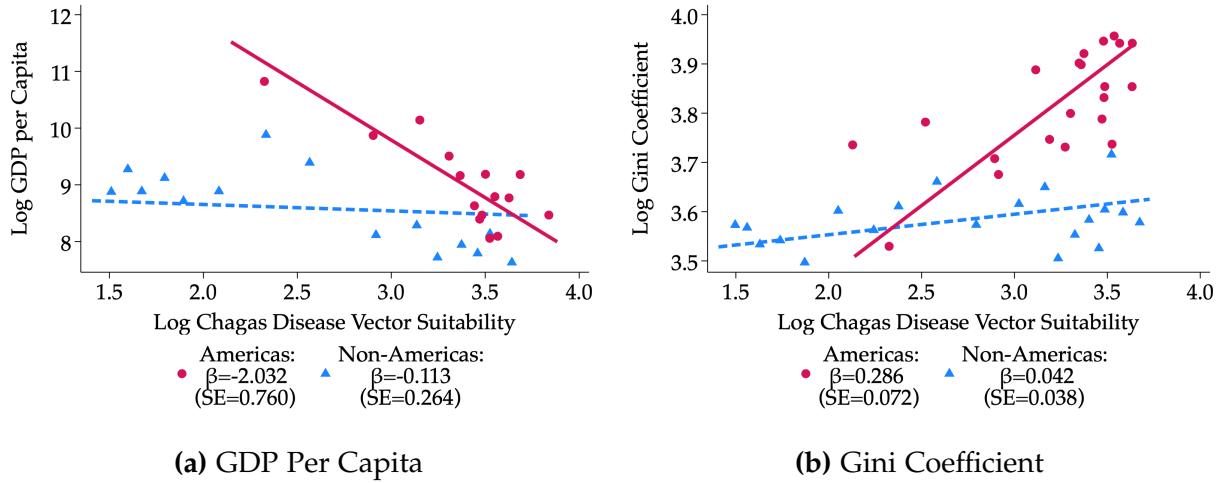
Exposure to Chagas disease varies considerably across the Western Hemisphere. To document cross-country patterns between Chagas disease risk and contemporary development, we draw on the ecological suitability raster for its vectors produced by [Eberhard et al. \(2020\)](#). We calculate country-level suitabilities and merge them with World Bank data on average 2010–2019 GDP per capita (PPP) and Gini coefficients (see Figure 1 for maps of these measures in the Americas).

Figures 2a and 2b show binned scatterplots of the associations between Chagas disease vector suitability and these measures of income and inequality. We present the relationships separately for countries in the Americas and those elsewhere, conditioning on a rich set of geographic and ecological covariates. The figures reveal a striking pattern: outside the Western Hemisphere, vector suitability is essentially orthogonal to both GDP per capita and inequality. Within the Americas, however, higher suitability is associated with lower incomes and greater inequality. Specifically, the fitted slopes imply that a 10% increase in ecological suitability for Chagas disease vectors in the Americas is associated with what converts into about an 18% lower GDP per capita and a 3% higher Gini coefficient.

Although the cross-country patterns are descriptive and merely correlational, they point to two key facts. First, within the Americas, higher Chagas disease suitability is systematically linked to lower incomes and greater inequality, indicating that the disease may be an important but as-yet overlooked driver of the region's development gaps. Second, the size of the association is large enough to imply substantive potential gains from vector control.⁸ These observations motivate our shift to a within-country analysis in Brazil, where we can identify the causal impacts of reducing Chagas disease transmission by exploiting its mid-1980s vector control program.

⁸ In Appendix A3, we provide evidence that Chagas disease may also have affected the region's historical development: a greater recorded presence of the parasite in precolonial Latin America is associated with lower levels of centralization in the Standard Cross-Cultural Survey ([Murdock and White, 1969](#)).

Figure 2: Americas-Specific Effects of Chagas Disease Vector Suitability



Notes: Figure presents binned scatterplots (Cattaneo et al., 2024). The outcome in the left panel is the log of the average 2010-19 GDP per capita (PPP), and the outcome in the right panel is the average 2010-19 Gini coefficient, both from the World Bank (2024). The explanatory variable is a 0 to 100 measure of the ecological suitability for Chagas disease vectors from Eberhard et al. (2020). *Americas* includes countries in North or South America (35 for GDP per capita, 21 for Gini); *Non-Americas* includes all other countries (152 for GDP per capita, 130 for Gini). All regressions include continent fixed effects and geographic controls: the explanatory variables from Eberhard et al. (2020)—temperature seasonality (BIO4), maximum temperature of the warmest month (BIO5), minimum temperature of the coldest month (BIO6), precipitation of the wettest month (BIO13), precipitation of the driest month (BIO14), and precipitation seasonality (BIO15)—and a quadratic in latitude and longitude. Estimated slopes and robust standard errors correspond to the fitted lines within and outside of the Americas.

2.4. Eradication of Vectorial Transmission in Brazil

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

Due to this “development of suitable vector control methods … [and] demonstrations that

vector control was feasible,” the Brazilian government launched a national effort against Chagas disease in 1975 (Dias, 1987, p. 338). The first stage consisted of serological and entomological surveys lasting until the early 1980s. They found a national rural *T. cruzi* prevalence rate of roughly 4-5%, but this average masked pronounced spatial and socioeconomic heterogeneity. In some municipalities, the share of infected residents exceeded 30% and prevalence reached 9% in the most heavily infested states (Minas Gerais and Rio Grande do Sul), where poverty and mud-walled housing were most common (Camargo et al., 1984; Dias, 1987). Overall, vectors were detected in just over one-third of Brazilian municipalities, and the highest infection burdens fell on the most deprived, predominantly non-white rural communities.⁹

After the surveys concluded, thousands of public health personnel began visiting millions of homes across the endemic region in 1984 to conduct indoor residual spraying (IRS) against *T. infestans*.¹⁰ However, outbreaks of dengue fever in coastal (i.e., tourist) areas in 1986 resulted in a 3-year diversion of public health personnel to the control of this mosquito-borne disease.¹¹ IRS restarted in 1989 and continued into the 21st century, and by 2006, the Pan American Health Organization certified that Brazil had interrupted transmission of Chagas disease by *T. infestans*.

3. Data and Empirical Strategy

In our main analysis of the effects of eliminating Chagas disease transmission, we use a difference-in-differences strategy comparing outcomes across municipalities (of birth) and years (of birth). We now define our treatment and control groups and pre- and post-treatment years, give an overview of the data we use, and detail our empirical specifications.

3.1. Control, Treatment, and Excluded Municipalities

We digitized the map of Brazilian municipalities in which *T. infestans* were present in the 1975-83 entomological surveys (Silveira, 2011). Figure 3 shows these data. Our control group consists of those in which the vector was not found prior to spraying, so we assume that they were never exposed to transmission of Chagas disease via *T. infestans*.¹² Our treatment group consists of all municipalities in which the vector was present before IRS began.¹³ Lastly, we exclude the state

⁹ Appendix A4 shows a municipality-level serology map from Passos and Silveira (2011).

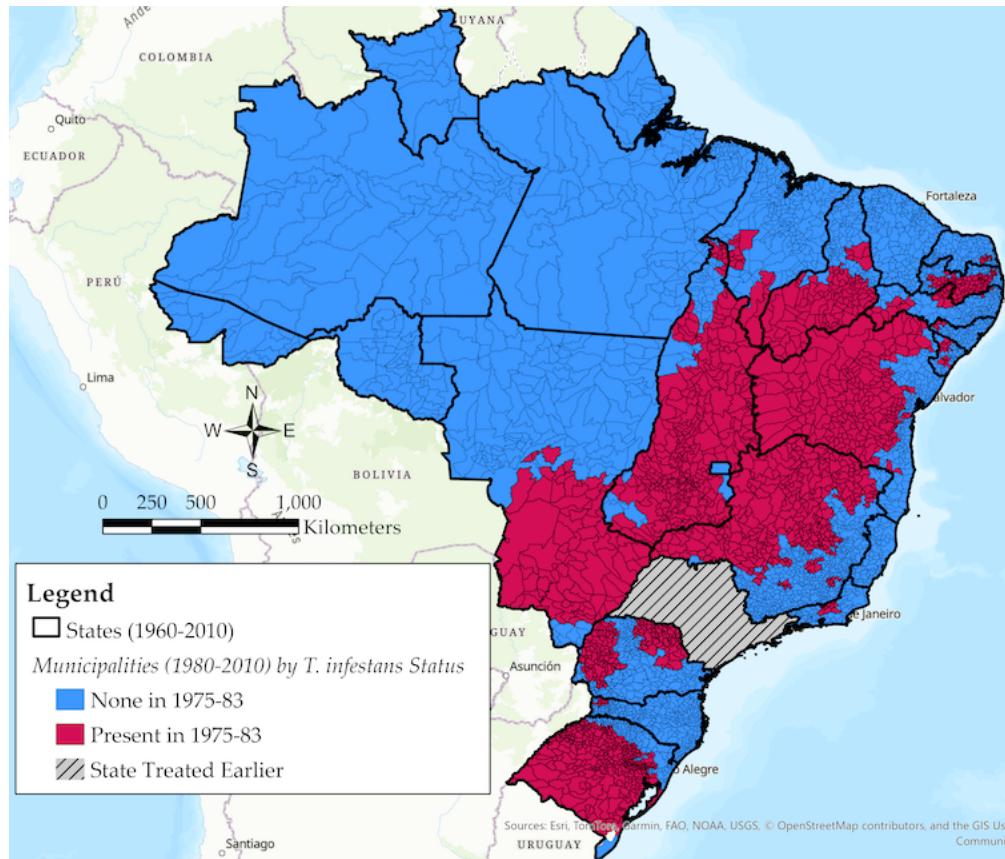
¹⁰ See Appendix A5 for images from Dias (1987) of insecticide being sprayed on rural homes with many cracks and spaces in their roofs and walls amenable to colonization by triatomine bugs.

¹¹ The bugs thus reestablished themselves in some recently-sprayed municipalities (Dias, 1987; Schofield and Dias, 1999), though the map in Coura and Dias (2009) shows a clear decline in the presence of *T. infestans* by 1989. However, we could not find any record of which municipalities had not yet had IRS and which ones were treated before 1986 but had the bugs return during the pause in spraying.

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 understate the true effects.

¹³ Campaign records do not indicate whether the vector was successfully eliminated from a municipality in the early phase of IRS (1984-86), though we do know that it was eventually eliminated in every case. We include all of these municipalities in our treatment group to be conservative.

Figure 3: Brazilian Municipalities by *T. infestans* Status, 1975–83



Notes: Map shows consistent municipalities without *T. infestans* in 1975–83 (control group) in blue and those in which it was present in that period (treatment group) in red. The state of São Paulo (gray with diagonal lines) was treated earlier and is thus excluded from our sample. Data are from [Silveira \(2011\)](#).

of São Paulo because of its earlier vector control program, so we avoid treatment heterogeneity problems in our difference-in-differences estimation ([Goodman-Bacon, 2021](#)).

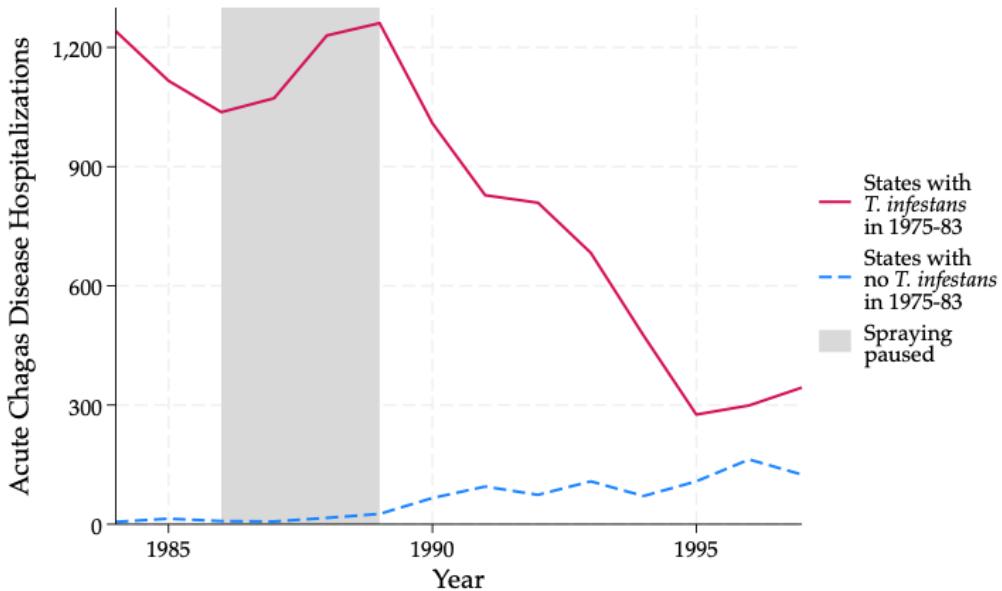
Table 1 presents summary statistics for these groups using individual-level data from the IPUMS 25% sample of the 1980 census ([Ruggles et al., 2024](#)) and municipalities' estimated GDP per capita from Ipeadata. Notably, the share of white Brazilians was lower in control municipalities than in treatment 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 population, consistent with racial disparities in exposure to Chagas disease transmission. In addition, the data in Panels C through E reflect lower incomes and lower levels of schooling in treatment municipalities, and these disparities were especially stark for non-white Brazilians.

Table 1: Summary Statistics, 1980

	Municipalities by 1975-83 <i>T. infestans</i> Status		
	None (Control)	Present (Treatment)	Treated Earlier (Excluded)
<i>Panel A. Demographics</i>			
Age	24.01 (19.12)	24.31 (19.25)	25.95 (18.93)
Female	0.507	0.502	0.501
Asian	0.002	0.003	0.019
Black	0.065	0.057	0.045
Brown	0.473	0.365	0.182
White	0.460	0.575	0.754
<i>Panel B. Living in Housing Vulnerable to Infestation</i>			
Non-White	0.341	0.390	0.056
White	0.112	0.133	0.026
<i>Panel C. Income and Inequality within Municipalities</i>			
Log GDP per Capita (2019 R\$)	9.58 (0.90)	9.50 (0.81)	10.59 (0.53)
Log Gini Coefficient	4.33 (0.05)	4.34 (0.06)	4.28 (0.04)
<i>Panel D. Labor Market Outcomes: Ages 20-50</i>			
Income Percentile: Non-White	44.37 (35.37)	41.19 (34.74)	57.88 (36.44)
Income Percentile: White	49.74 (39.04)	47.69 (38.34)	57.10 (39.80)
Income Above Zero: Non-White	0.625	0.594	0.724
Income Above Zero: White	0.630	0.618	0.679
Living in Birth State: Non-White	0.815	0.831	0.461
Living in Birth State: White	0.764	0.820	0.701
<i>Panel E. Human Capital</i>			
Years of Schooling: Non-White Ages 20-50	3.04 (3.45)	2.34 (3.03)	3.89 (3.35)
Years of Schooling: White Ages 20-50	5.25 (4.14)	4.59 (3.80)	5.55 (3.98)
Literate: Non-White Ages 9-18	0.658	0.641	0.916
Literate: White Ages 9-18	0.867	0.872	0.962
Observations	16,431,739	6,611,704	6,181,009
Consistent 1980-2010 Municipalities	1,156	575	309

Notes: Means for variables of interest are displayed, with standard deviations in parentheses for continuous measures. Variable definitions are given in the text and municipality groupings correspond to those in Figure 3. Log GDP per capita data are from Ipeadata and all other variables are from the IPUMS sample of the 1980 Brazilian census.

Figure 4: Hospitalizations Due to Acute Chagas Disease, 1984-97



Notes: Graph shows hospitalizations due to acute Chagas disease from 1984 to 1997 in states with any treatment municipality (solid red line) and those with only municipalities in the control group (dashed blue line). See Figure 3 for a map of these municipalities. Gray shading indicates years in which indoor residual spraying against *T. infestans* was paused. Data are from DATASUS.

3.2. Treatment Timing and Omitted Years

Because the historical records do not report municipality-specific IRS start dates, we assume that it occurred uniformly throughout the treatment group in 1984. The graph of hospitalizations for acute Chagas disease in Figure 4 suggests that this assumption is conservative, as IRS appeared to reduce this measure of *T. cruzi* transmission but it was not immediately eliminated. As a result, we may overstate how long it takes for the initial benefits of vector control to manifest.

When studying the impacts on municipalities, our final pre-treatment year when comparing across decennial censuses is thus 1980. For our individual-level analysis, in which we examine the effects of additional childhood years free from exposure to Chagas disease, our final pre-treatment year is 1983 and we assume that childhood ended at the age when school attendance rates were (just under) 50%, which was 16 in the 1980 census. Therefore, our pre-treatment cohorts were born in or before 1967. The implication is that these cohorts were too old to have benefited from *T. infestans* control. However, as noted in Section 2.1, both children and adults can experience acute symptoms when (re)infected with *T. cruzi*, and reinfections increase the chance of having a symptomatic chronic stage. Therefore, the interpretation of our individual-level results is closer to those for childhood exposure to tuberculosis (e.g., Bütkofer and Salvanes, 2020), which can also affect all ages, than to diseases that primarily impact children like helminthiases and malaria (e.g., Miguel and Kremer, 2004; Bleakley, 2007, 2010; Cutler et al., 2010; Lucas, 2010).

3.3. Municipality-Level Data and Estimating Equation

Our first main outcome is municipalities' log GDP per capita (in constant 2019 R\$) in census years from 1970 to 2010 using data from Ipeadata.¹⁴ In addition, we collapsed individual-level data for adults aged 20 to 50 into municipality-level means across five Brazilian censuses: two 25% samples from pre-treatment years (1970 and 1980) and three 10% samples from post-treatment years (1991, 2000, and 2010), all of which are from IPUMS (Ruggles et al., 2024).¹⁵ Doing so allows us to calculate our other main outcome, which is the log of a municipality's income Gini coefficient in each year.¹⁶ As complementary measures of development, we also examine municipalities' average percentiles in the national income distribution within a census year and their average years of schooling.¹⁷

To study the effects of Chagas disease control on the evolution of these outcomes in municipality m in year t , our estimating equation is

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

where $y_{m,t}$ is an outcome of interest for m in t , α_m and γ_t are fixed effects for m and t , $\mathbb{1}[m \in \text{Treat}]$ indicates whether m is in the treatment group (i.e., the vector was present in 1975-83), $\mathbb{1}[t = k]$ is an indicator for whether t was the given year k , $\delta_{s(m)}$ is a fixed effect for m 's state, and $\epsilon_{m,t}$ is the idiosyncratic error term. We interact $\delta_{s(m)}$ with γ_t , which limits comparisons to municipalities within the same state and thus accounts for state-specific trends. Our preferred specification contains only these municipality, year, and state-by-year fixed effects because interpreting the effects is very intuitive: only treatment and control municipalities in the same state are compared.

As a robustness check, we also estimate specifications that contain a vector of pre-treatment municipality-level characteristics, $\mathbf{X}_{m,1980}$ — m 's average age and age squared, racial shares (Asian, Black, Brown, and White), and share female, all measured in 1980; and its ecological suitability for genetically engineered (GE) soy and maize (Bustos, Caprettini and Ponticelli, 2016)—each of which is interacted with γ_t . Doing so controls for municipalities' exposure to shocks based on these factors, which is an important adjustment but nonetheless complicates the interpretation by adding conditional statements to descriptions of the identifying variation.¹⁸

¹⁴ These data are constructed using census information and are available for 1970, 1980, 2000, and 2010. Additional years that require any interpolation from Ipeadata on economic activity or population between census waves are excluded. Data are not available for 1991.

¹⁵ We match 1970 municipalities to consistent IPUMS 1980-2010 municipalities containing their centroids.

¹⁶ We use logs of GDP per capita and the Gini coefficient so effect sizes are approximate percentage changes, which are more intuitively applied in our cross-country extrapolations in Section 6.3.

¹⁷ Income percentiles are a scale invariant measure that allows us to abstract from currency changes and measurement error in local price deflators. For context, between the mid-1980s and mid-1990s, Brazil experienced extremely high inflation—and even hyperinflation—and changed its currency five times.

¹⁸ Baseline demographic characteristics are important to adjust for on their own, but their interactions with year fixed effects also control for a municipality's exposure to group-specific shocks such as the expansion of pensions for rural women in 1991 and racial university admissions quotas beginning in 2001.

The coefficients of interest are the τ_k , which measure within-state differences in an outcome in a given year across the treatment and control groups relative to the size of that difference in 1980. We estimate equation (1) using OLS with standard errors clustered by municipality, and we weight observations by their 1980 population to account for differences in municipality sizes at baseline. We also use newly developed difference-in-differences estimators to test whether the issues identified in this literature drive our results (Arkhangelsky et al., 2021; Callaway and Sant'Anna, 2021; de Chaisemartin and D'Haultfœuille, 2020; Sun and Abraham, 2021).

3.4. Individual-Level Data and Estimating Equation

To study the impacts of childhood years free of exposure to Chagas disease transmission on post-treatment cohorts as well as on their children, we use the IPUMS 10% sample of Brazil's 2010 census (Ruggles et al., 2024). It contains our outcomes of interest (monthly income, years of schooling, and children's literacy), demographic characteristics (age, racial group, and sex), and whether someone was born in their municipality of residence (and if not, it lists their birth state).

For several reasons, we limit our attention to cohorts born between 1960 and 1980, who were aged 30 to 50 in 2010. First, in that year, workers could retire with social security benefits after having paid into the system for 30 to 35 years, so age 50 is an appropriate upper bound. In addition, DDT spraying in Brazil began in the late 1950s, so we want all cohorts in our sample to have had the same level of exposure to this intervention. Lastly, the restriction at the bottom of our age range ensures that our sample contains prime-age adults with varying exposure to Chagas disease transmission in childhood and who had already made substantial progress along their lifetime earnings trajectories.

Among these individuals, we observe the municipalities of birth for just under three-fifths of them; for the remainder, we only know their states of birth. Therefore, we place those with known municipalities of birth into the treatment or control groups (or exclude them from the sample if they were born in São Paulo), as discussed above. We then assign individuals who were no longer living in their birth municipalities to the treatment group if their birth state had any municipalities with *T. infestans* in 1975–83 or to the control group if their birth state was completely free of the vector.¹⁹

This categorization means that our treatment group consists of individuals who had at least some degree of exposure to Chagas disease transmission as children and our control group contains only those we know with certainty had none.²⁰ As such, our treatment effects capture the result of reducing the share of children potentially exposed to Chagas disease transmission from around 29% (the fraction of children in the 1980 census living in treatment municipalities) to 0%. We view this interpretation as both intuitive and policy relevant.

¹⁹ We create a bin for all individuals born in a state for whom we cannot observe birth municipalities.

²⁰ Using a binary indicator for a strictly positive “dosage” is the simple approach to estimating average level treatment effects suggested by Callaway, Goodman-Bacon and Sant'Anna (2024).

Our individual-level estimation strategy modifies equation (1) to be

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

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 , $\delta_{s(m)}$ is a fixed effect for m 's state, \mathbf{X}_i is a vector of individual-level covariates (fixed effects for female sex and Asian, Black, and Brown racial categories), and $\epsilon_{i,m,c}$ is the idiosyncratic error term. As above, we interact $\delta_{s(m)}$ with γ_c to limit comparisons across treatment and control birth municipalities in the same state. Our preferred specification contains only these fixed effects, but for robustness, we also include birth municipalities' GE crop suitabilities Z_m interacted with γ_c to account for exposure to shocks related to this agricultural technology change.

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 was reduced from about 29% to 0%, relative to the size of that difference for the 1967 cohort. Using OLS with standard errors clustered by municipality of birth, we estimate them separately for non-white and white Brazilians to examine whether this intervention reduced racial inequality. Because Appendix B1 shows substantial heaping of birth years, especially for non-white adults, we report in our tables the average effects for 3 multiple-cohort bins (i.e., 1968-72, 1973-77, and 1978-80), which also allows us to be more parsimonious. However, our event study plots show effects for individual cohorts, and, as above, we use the newly developed estimators as well.

3.5. Threats to Identification

The main identification assumption in our difference-in-differences design is that, absent the IRS campaign, treated and control municipalities would have continued along parallel epidemiological and socioeconomic trends. Therefore, both sets of municipalities must have been in epidemiological steady states—one with persistent transmission, the other without—leading up to the 1984 intervention. This logic mirrors the identification strategy in other studies exploiting the control of a disease, in which long-run endemic prevalence before a sudden public health shock helps to justify the parallel trends assumption (e.g., Bleakley, 2007; Lucas, 2010; Cutler et al., 2010; Bütkofer and Salvanes, 2020). Nonetheless, for all outcomes, we use event study plots to assess whether outcomes evolved in parallel across treatment and control municipalities before IRS. We also use the Rambachan and Roth (2023) procedure to test if the estimate for the first post-treatment year or multi-cohort bin remains statistically significant after correcting for violations of the parallel trends assumption.

Relative to prior work on other diseases, which mostly focuses on pathogens with short-run symptoms affecting children, the acute (shortly after infection) and chronic symptoms (10

to 30 years later) of Chagas disease have important implications for identification and studying mechanisms. First, within the post-IRS period, we should see certain health improvements arrive with a lag: while acute symptoms should decline immediately after transmission is interrupted, circulatory conditions should fall a decade or more later and deaths well after that. Second, beyond short-run gains from reductions in acute symptoms, economic impacts should increase with time since IRS as chronic Chagas disease is averted, implying larger municipality-level gains in later decades and larger cohort effects for individuals with more years of childhood free from Chagas disease exposure. As discussed in Sections 4.1 and 5.4, the timing of divergences in economic and health trends largely matches these predictions.

A remaining concern is that IRS coincided with other place-specific interventions or shocks. For instance, if it coincided with other public health campaigns, our results would not be solely attributable to the elimination of vectorial *T. cruzi* transmission. However, we did not find any historical accounts or budget records suggesting that large-scale health or infrastructure programs were begun preferentially in *T. infestans* areas, mitigating concerns that other forces drive the results. Moreover, competing explanations would have to both co-vary with pre-IRS vector presence and lead to effects with the same dynamic patterns. In other words, any such program would have to (i) begin at the same time as IRS (ii) in the same locations within a state as IRS and (iii) lead to the same time lags in economic and health trends as reductions in acute and then chronic symptoms would generate. Nevertheless, we also test whether our results are robust to controls for potential place-specific shocks that happened during our period of study.

4. Main Results

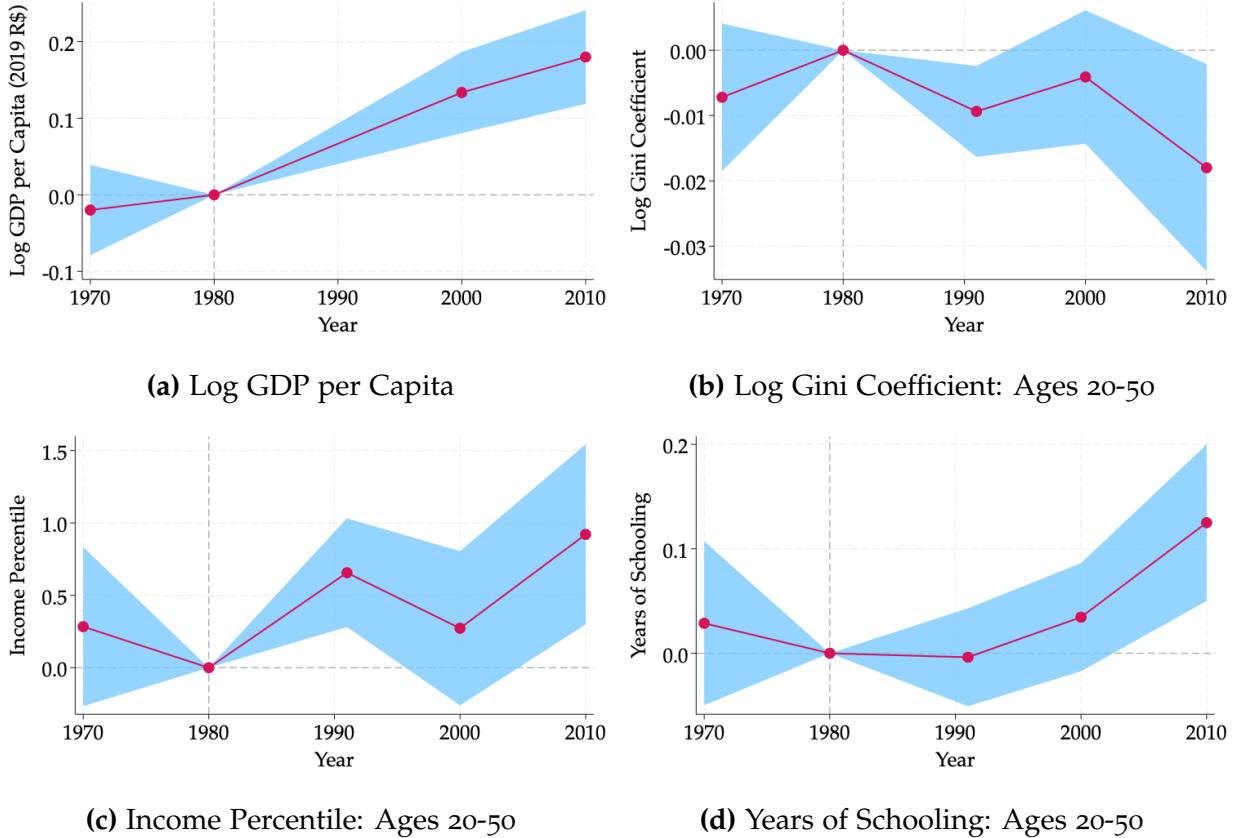
We first study the long-run effects of Chagas disease vector control by comparing municipality-level outcomes across our treatment and control groups. We show that it led to increased GDP per capita and reduced income inequality. We then take a complementary approach and examine outcomes among adults who were children around the time of treatment. We find that IRS increased incomes for non-white adults only, implying that vector control decreased racial inequality in Brazil in the long run. Lastly, because of the narratives linking Chagas disease to intergenerational poverty traps in Section 2.2, we test whether vector control affected the outcomes of the children of the adults studied above. We find at least suggestive evidence of increased literacy among those born to non-white fathers, indicating further reductions in racial inequality in the next generation. All three sets of results became more pronounced over time or were larger for younger cohorts, suggesting that reductions in chronic Chagas disease augmented the positive shorter-run benefits of averting its acute symptoms.

Table 2: Effects of Chagas Disease Vector Control on Municipalities

	(1)	(2)	(3)	(4)
<i>Panel A. Log GDP per Capita</i>				
Treat \times 2000	0.044 (0.034)	0.134 (0.027)	0.104 (0.026)	0.077 (0.028)
Treat \times 2010	0.106 (0.050)	0.180 (0.031)	0.145 (0.029)	0.113 (0.032)
Average Annual Change 1984-2010 (%/year)	0.4	0.7	0.6	0.4
<i>Panel B. Log Gini Coefficient</i>				
Treat \times 1991	-0.016 (0.004)	-0.009 (0.004)	-0.004 (0.003)	-0.005 (0.003)
Treat \times 2000	-0.027 (0.005)	-0.004 (0.005)	0.003 (0.005)	-0.002 (0.005)
Treat \times 2010	-0.054 (0.010)	-0.018 (0.008)	-0.007 (0.007)	-0.014 (0.007)
Average Annual Change 1984-2010 (%/year)	-0.2	-0.1	-0.0	-0.1
<i>Panel C. Income Percentile</i>				
Treat \times 1991	1.02 (0.33)	0.68 (0.19)	0.37 (0.19)	0.46 (0.19)
Treat \times 2000	1.77 (0.36)	0.31 (0.27)	-0.11 (0.23)	0.21 (0.24)
Treat \times 2010	2.99 (0.51)	0.97 (0.32)	0.52 (0.28)	0.93 (0.29)
Effect through 2010 as % of Pre-1984 Mean	7.6	2.5	1.3	2.4
<i>Panel D. Years of Schooling</i>				
Treat \times 1991	0.01 (0.02)	-0.00 (0.02)	0.00 (0.02)	-0.01 (0.02)
Treat \times 2000	0.12 (0.04)	0.04 (0.03)	0.03 (0.03)	0.02 (0.03)
Treat \times 2010	0.13 (0.07)	0.13 (0.04)	0.14 (0.04)	0.10 (0.04)
Effect through 2010 as % of Pre-1984 Mean	4.1	4.1	4.3	3.2
Municipality FE and Year FE	x	x	x	x
State FE \times Year FE		x	x	x
1980 Demographics \times Year FE			x	x
GE Crop Suitabilities \times Year FE				x
Observations in Panel A	6,533	6,525	6,525	6,525
Observations in Panels B-D	8,170	8,160	8,160	8,160
Consistent Municipalities	1,580	1,578	1,578	1,578

Notes: Observations are municipality-years. GDP per capita is calculated using data from Ipeadata for available census years and all other data are from IPUMS. 1980 demographics are average age and age squared, shares in each racial group (Asian, Black, Brown, and White), and the share female, and the GE crops are soy and maize. Standard errors in parentheses are clustered by consistent municipalities.

Figure 5: Event Study Plots for Effects on Municipalities



Notes: Graphs show estimates with 95% confidence intervals. Regressions include year, municipality, and state-by-year fixed effects, as in Table 2 Column (2). Standard errors are clustered by municipality.

4.1. Effects on Municipalities

Table 2 presents the evolution of the four key municipality-level outcomes—log GDP per capita, log Gini coefficients, income percentiles, and years of schooling—across treatment and control municipalities. Our baseline estimates in Panel A Column (2) show that, relative to control municipalities in the same state, treated municipalities experienced what converts into an additional increase in GDP per capita of about 14% by 2000 and 20% by 2010, corresponding to a 0.7% faster annual growth rate after 1984. These effects are precisely estimated and Figure 5a shows that they do not appear to be a continuation of non-parallel trends between 1970 and 1980.

Turning to inequality, the estimates in Panel B Column (2) show that Gini coefficients declined by 0.9% more in treatment municipalities in the same state by 1991 and 1.8% more by 2010. Both of these estimates are precise, though there was a slight decrease in the decline between 1991 and 2000. Nonetheless, spreading the effect by 2010 over 26 years implies an approximately 0.1% faster annual decline in the Gini coefficient in treated municipalities. As with log GDP per capita, Figure 5b shows that log Gini coefficients evolved roughly in parallel across the treatment and

control groups prior to the start of IRS.

Panel C presents the estimates for income percentiles. In Column (2), the average income rank in treated municipalities rose an additional 0.7 income percentiles by 1991 and 1 percentile by 2010 (about 2.5% of the pre-IRS mean) relative to control municipalities in the same state. Figure 5c shows the event-study plot for income percentiles: they also evolved in parallel prior to the IRS campaign before diverging after it began. The pattern in average income percentiles closely matches that of log Gini coefficients—Figures 5b and 5c appear to be reflections of each other over the x -axis—which provides reassurance that the compression in incomes did not arise from reductions at the top of the distribution. Furthermore, the gradual widening of post-campaign effects is consistent with the long-run nature of Chagas disease, as reductions in chronic symptoms should have amplified the benefits of avoiding the acute phase.

Finally, Panel D reports the estimated impacts on years of schooling. Column (2) shows that it increased in treatment municipalities by an imprecisely estimated 0.04 years more by 2000 and a precisely estimated 0.13 years more by 2010, and Figure 5d shows that these post-treatment schooling effects are unlikely to have been a continuation of differential trends prior to treatment. However, far more notable is the contrast between this small increase in completed schooling that was detectable only in 2010 and the effects on income and inequality that both were substantially larger and had emerged much earlier. This disconnect suggests that other channels not related to time in school—most plausibly higher labor force participation and improved worker productivity due to averted Chagas disease—may have driven the bulk of the improvements in income and inequality. We unpack these alternative mechanisms in Section 5.

4.1.1. Robustness

Taken together, these results—which we estimated by comparing treatment and control municipalities in the same state—provide clear evidence that eliminating Chagas disease transmission led to important improvements in standards of living across Brazilian municipalities and reductions in inequality within them. We probe their robustness in Columns (3) and (4) by controlling for the time-varying effects of pre-treatment demographics and GE soy and maize potential yields. Across outcomes, the estimates in Columns (2) and (4) are similar in size and precision, indicating that our results do not arise from the differential effects of any state-level shocks, demographic group-specific policies, or shifts in agricultural technologies over our period of study.

To address concerns about difference-in-differences estimation, in Appendices C1 through C4 we present event study plots using new techniques. The point estimates are generally quite similar to those in Figure 5, though the confidence intervals are often wider given their greater data requirements. We also test in these appendices the robustness of the first post-treatment estimate to violations of the parallel trends assumption. None has its statistical significance affected by a linear violation or even one that deviates from linearity by a full standard error of the estimated coefficient for 1970.

Table 3: Effects of Chagas Disease Vector Control on Adults Exposed as Children

	(1)	(2)	(3)	(4)
<i>Panel A. Income Percentile: Non-White</i>				
Treat × 1968-72 Cohorts	0.85 (0.23)	0.99 (0.21)	1.04 (0.21)	0.98 (0.21)
Treat × 1973-77 Cohorts	0.97 (0.25)	0.93 (0.22)	0.94 (0.23)	0.88 (0.24)
Treat × 1978-80 Cohorts	1.30 (0.25)	1.06 (0.24)	1.15 (0.24)	1.06 (0.25)
Effect on 1978-80 Cohorts as % of Pre-1968 Cohorts' Mean	3.1	2.5	2.7	2.5
<i>Panel B. Income Percentile: White</i>				
Treat × 1968-72 Cohorts	0.14 (0.21)	0.02 (0.23)	0.07 (0.23)	0.04 (0.23)
Treat × 1973-77 Cohorts	0.36 (0.25)	0.11 (0.25)	0.15 (0.24)	0.12 (0.24)
Treat × 1978-80 Cohorts	0.73 (0.33)	0.13 (0.25)	0.29 (0.25)	0.30 (0.25)
Effect on 1978-80 Cohorts as % of Pre-1968 Cohorts' Mean	1.4	0.2	0.5	0.6
<i>Panel C. Log Income: Non-White</i>				
Treat × 1968-72 Cohorts	0.089 (0.024)	0.096 (0.023)	0.099 (0.023)	0.095 (0.023)
Treat × 1973-77 Cohorts	0.098 (0.024)	0.090 (0.024)	0.091 (0.024)	0.087 (0.025)
Treat × 1978-80 Cohorts	0.141 (0.025)	0.119 (0.024)	0.125 (0.025)	0.118 (0.025)
<i>Panel D. Log Income: White</i>				
Treat × 1968-72 Cohorts	0.008 (0.021)	-0.005 (0.023)	-0.001 (0.023)	-0.003 (0.023)
Treat × 1973-77 Cohorts	0.021 (0.022)	0.005 (0.024)	0.007 (0.024)	0.004 (0.024)
Treat × 1978-80 Cohorts	0.056 (0.027)	0.009 (0.024)	0.021 (0.024)	0.023 (0.025)
<i>Panel E. Years of Schooling: Non-White</i>				
Treat × 1968-72 Cohorts	0.10 (0.03)	0.12 (0.03)	0.11 (0.03)	0.11 (0.03)
Treat × 1973-77 Cohorts	0.09 (0.04)	0.08 (0.04)	0.08 (0.03)	0.06 (0.04)
Treat × 1978-80 Cohorts	0.13 (0.05)	0.09 (0.05)	0.08 (0.05)	0.06 (0.05)
Effect on 1978-80 Cohorts as % of Pre-1968 Cohorts' Mean	2.6	1.8	1.6	1.2

Table 3: Continued

	(1)	(2)	(3)	(4)
<i>Panel F. Years of Schooling: White</i>				
Treat × 1968-72 Cohorts	0.01 (0.03)	-0.04 (0.03)	-0.04 (0.03)	-0.04 (0.03)
Treat × 1973-77 Cohorts	0.06 (0.05)	-0.02 (0.04)	-0.02 (0.04)	-0.01 (0.04)
Treat × 1978-80 Cohorts	0.10 (0.08)	-0.04 (0.05)	-0.05 (0.05)	-0.03 (0.05)
Effect on 1978-80 Cohorts as % of Pre-1968 Cohorts' Mean	1.5	-0.6	-0.7	-0.4
Birth Municipality FE and Cohort FE	x	x	x	x
Birth State FE × Cohort FE		x	x	x
Demographic FE			x	x
GE Crop Suitabilities × Cohort FE				x
Observations in Panels A, C, and E	2,790,838	2,790,838	2,790,838	2,790,838
Observations in Panels B, D, and F	2,234,657	2,234,657	2,234,657	2,234,657
Consistent Municipalities	1,755	1,755	1,755	1,755

Notes: Data are from the IPUMS 2010 census sample. Demographic fixed effects are for sex and racial group (Asian, Black, and Brown), and GE crops are soy and maize. Standard errors in parentheses are clustered by consistent birth municipalities.

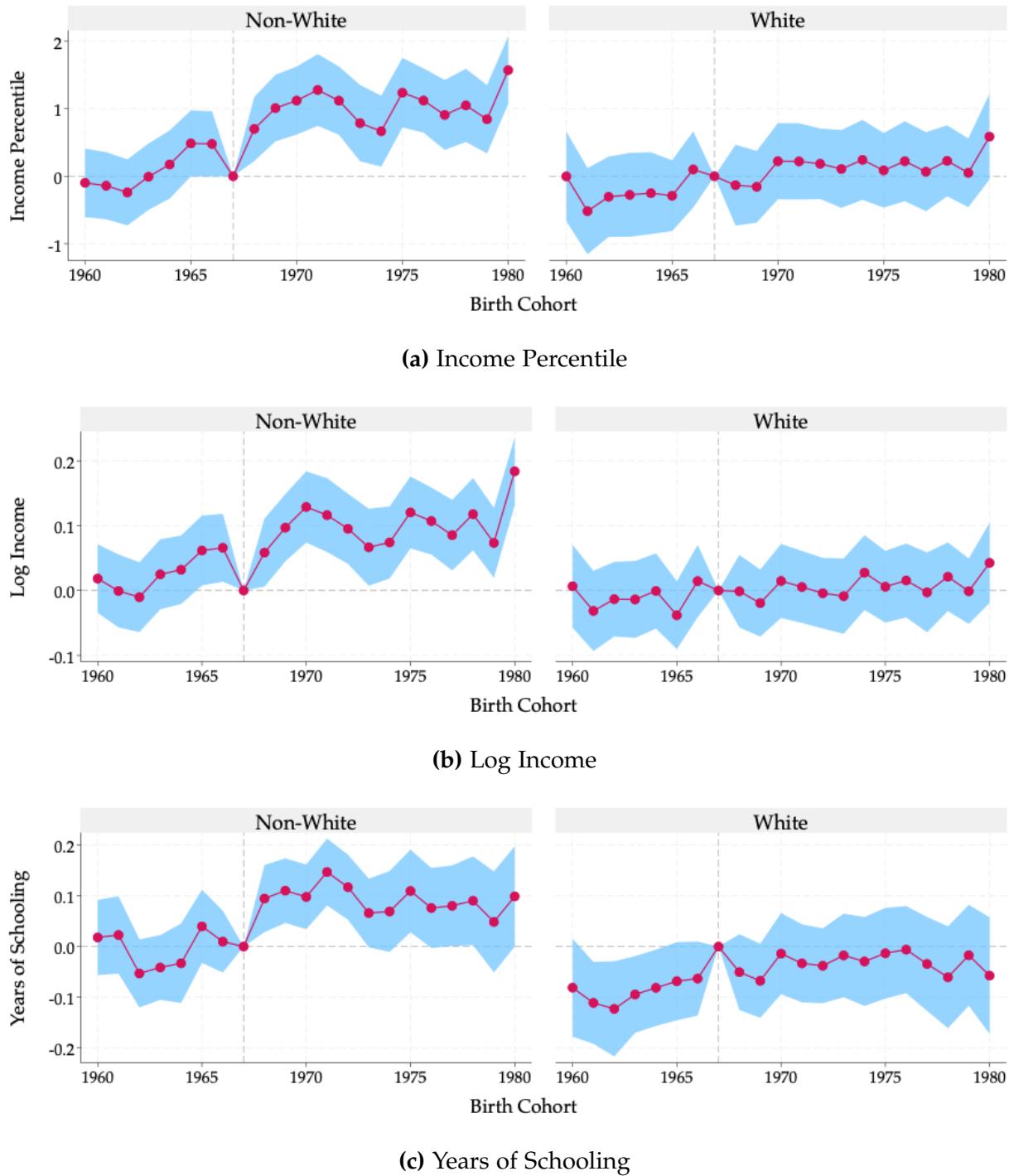
4.2. Effects on Individuals: Adults Treated in Childhood

Next, we turn to individual-level data and examine income percentiles and log incomes to capture the long-run earnings impact of childhood Chagas disease control exposure.²¹ Table 3 Panels A and B report estimated effects on income percentiles for non-white and white cohorts. In our preferred specification in Column (3), we find that non-white cohorts from treatment municipalities moved up the income distribution by about 1 percentile more (around 2.5% of the mean for pre-1968 cohorts) than non-white cohorts from control municipalities, whereas there were no significant differences across groups among white cohorts. Figure 6a presents the corresponding event study plots, showing that treated and control groups followed parallel pre-IRS trends.

In Panels C and D, Column (3) shows similarly stark differences for log income: the traditional interpretation is that earnings for treated non-white cohorts grew around 10% more while estimates for white adults remain near zero across all cohorts. Figure 6b visualizes this pattern and again shows similar pre-treatment trends. Notably, the magnitude of the non-white earnings effect generated by moving from 29% to zero exposure to the main Chagas disease vector falls

²¹ Log(1+income) is the standard transformation in previous disease eradication studies, so it allows us to compare these effects to those of hookworm, malaria, and tuberculosis control. Yet it is sensitive to the large mass at zero (Chen and Roth, 2024); we therefore first examine percentiles because they avoid that issue while preserving the distributional meaning, and they complement the municipality-level results.

Figure 6: Event Study Plots for Effects on Adults Exposed as Children



Notes: Graphs show estimates with 95% confidence intervals. Regressions include cohort, birth municipality, birth state-by-cohort, sex, and racial category fixed effects, as in Table 3 Column (3). Standard errors are clustered by consistent birth municipalities.

squarely within the range of other estimates in the disease eradication literature.²²

These results largely match the municipality-level income percentile results from Section 4.1 and suggest that these gains were concentrated among non-white individuals. However, at first glance, an important contrast appears to be in how the effects do not grow monotonically as years of childhood free from Chagas disease exposure increase. But it is important to recall the clear patterns of age heaping shown in Appendix B1, especially among non-white adults. To the extent that poorer individuals were more likely to report ages ending in 0 or 5 and were more exposed to Chagas disease prior to IRS, it is thus not surprising that the estimates seem to have local maxima in these years, particularly in the left panels of Figures 6a and 6b. As such, we consider any individual cohort's estimate to be less informative than the effects in Panels A and C for binned post-treatment cohorts, which are less sensitive to age heaping. They show that the impacts were largely increasing in years of childhood free from Chagas disease exposure.

Finally, Column (3) in Panels E and F shows modest effects on years of schooling for non-white individuals—0.21 years for the youngest cohorts, which is comparable to the municipal-level gains of at most 0.15 years by 2010 among those aged 20-50 in Section 4.1—and null estimates for white Brazilians. Figure 6c shows that the non-white effects emerged after parallel pre-campaign trends. However, these schooling gains seem far too small to explain the 1-percentile and roughly 10% effects on incomes above. This discrepancy suggests that other channels—particularly increased labor force participation and a reduced chronic disease burden—may have been the primary drivers of the income and inequality impacts. We formally test this hypothesis in Section 5.

4.2.1. Robustness

These results show that Chagas disease vector control reduced racial inequality among post-treatment cohorts. In Column (4), we test whether they were driven by differential exposure to the introduction of an extremely important agricultural technology, GE crops. The estimates for non-white cohorts in Panels A, C, and E are largely unchanged in terms of size and precision. For robustness to issues with difference-in-differences estimation, we use the procedures developed to address them in Appendices D1 through D3, and again the conclusions do not change. We also show that the estimates are robust to a violation of the parallel trends assumption deviating from linearity by up to half a standard error of the estimated effect for pre-treatment cohorts.

4.3. Effects on Individuals: Next Generation

To examine intergenerational impacts, we use children's literacy as the outcome, as these cohorts were too young to have entered prime working ages in 2010. We attach men's information to the

²² These estimates include a 17% effect from hookworm elimination in the US South (Bleakley, 2007), 12–40% effects from malaria campaigns in the Americas (Bleakley, 2010), and a 7% impact from tuberculosis control in Norway (Bütikofer and Salvanes, 2020).

Table 4: Effects of Chagas Disease Vector Control on Next Generation

	(1)	(2)	(3)	(4)
<i>Panel A. Literacy: Children 9-18 with Non-White Fathers</i>				
Treat \times 1968-72 Cohorts	0.0028 (0.0021)	0.0033 (0.0028)	0.0039 (0.0028)	0.0035 (0.0029)
Treat \times 1973-77 Cohorts	0.0055 (0.0028)	0.0042 (0.0032)	0.0048 (0.0031)	0.0049 (0.0032)
Treat \times 1978-80 Cohorts	0.0113 (0.0049)	0.0051 (0.0043)	0.0048 (0.0041)	0.0054 (0.0041)
Effect on 1978-80 Cohorts as % of Pre-1968 Cohorts' Mean	1.2	0.5	0.5	0.6
<i>Panel B. Literacy: Children 9-18 with White Fathers</i>				
Treat \times 1968-72 Cohorts	-0.0020 (0.0019)	-0.0021 (0.0018)	-0.0021 (0.0018)	-0.0020 (0.0018)
Treat \times 1973-77 Cohorts	0.0015 (0.0027)	-0.0015 (0.0021)	-0.0017 (0.0021)	-0.0012 (0.0021)
Treat \times 1978-80 Cohorts	0.0075 (0.0043)	0.0008 (0.0030)	0.0003 (0.0029)	0.0010 (0.0030)
Effect on 1978-80 Cohorts as % of Pre-1968 Cohorts' Mean	0.8	0.1	0.0	0.1
Birth Municipality FE and Cohort FE	x	x	x	x
Birth State FE \times Cohort FE		x	x	x
Demographic FE			x	x
GE Crop Suitabilities \times Cohort FE				x
Observations in Panel A	1,054,274	1,054,274	1,054,274	1,054,274
Observations in Panel B	682,440	682,440	682,440	682,440
Consistent Municipalities	1,753	1,753	1,753	1,753

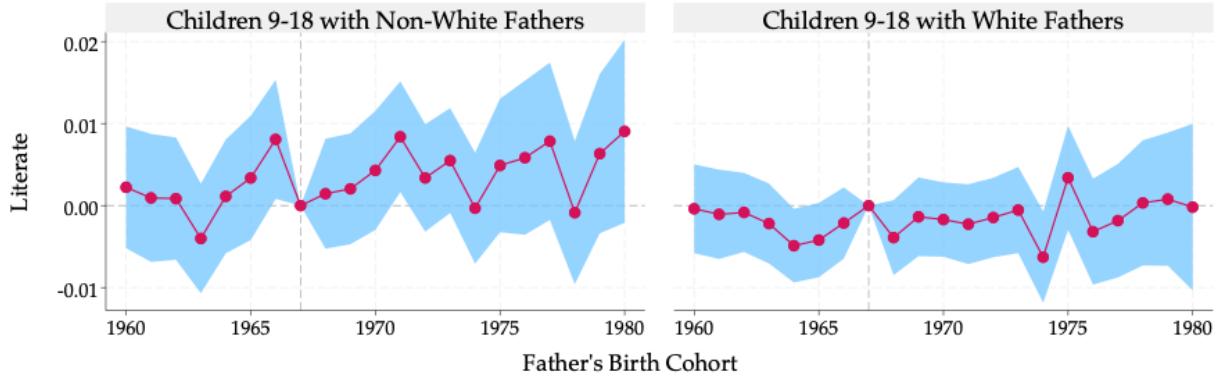
Notes: Data are from the IPUMS 2010 census sample. Birth municipality, cohort, and state fixed effects and GE crop suitabilities are based on fathers and demographic fixed effects (age, sex, and racial group) are based on children. Standard errors in parentheses are clustered by consistent birth municipalities.

children aged 9 to 18 living in their households in the 2010 census.²³ We then modify equation (2) to use fathers' values of the variables and add fixed effects for a child's age and sex.

Even with Brazil's extensive progress toward universal child literacy between 1980 (see Table 1) and 2010, Table 4 Panel A Column (3) shows that it increased by 0.4-0.5 p.p. (0.5% of the mean) more among the children of non-white fathers in treated cohorts, though the estimates are slightly imprecise. As predicted, there are no effects for the children of white fathers, and Figure 7 shows a lack of differential pre-treatment trends in both cases. Importantly, the results for children of non-white fathers become slightly stronger in Column (4) and are broadly robust to using the new difference-in-differences estimators in Appendix D4. But given the extensive

²³ We exclude those over 18 due to selection into living with their parents after that age. We set 9 years old as the lower bound because literacy rates were still increasing rapidly for 6- to 8-year-olds in the 2010 census, so we exclude them to reduce the noise in our estimates.

Figure 7: Event Study Plots for Effects on Next Generation



Notes: Graphs show estimates with 95% confidence intervals. Regressions include fixed effects for father's cohort, birth municipality, and birth state-by-cohort as well as fixed effects for child's age, sex, and racial category, as in Table 4 Column (3). Standard errors are clustered by consistent birth municipalities.

noise in the estimates for pre-treatment non-white cohorts, the result is sensitive to a linear violation of parallel trends. Nonetheless, we view this result as at least suggestive evidence that some benefits of Chagas disease vector control were transferred from fathers to their children, highlighting the potential for NTD control to improve the lives of the next generation.

5. Mechanisms

The main results presented in Section 4 showed large income gains and inequality reductions stemming from the IRS campaign despite small increases in schooling. We now unpack the channels behind these patterns and proceed in three steps. First, we document that vector control sharply increased the share of adults reporting earning non-zero incomes in treated municipalities and cohorts. Second, we conduct mediation analyses to show that this extensive margin labor supply response explains a far larger share of the income gains than schooling, pointing to enhanced work capacity and productivity due to better health in adulthood as the likely mechanisms. Third, we corroborate this interpretation by examining health outcomes in a triple-differences framework: hospitalizations attributed to circulatory system diseases fell by more than those due to all other causes right when chronic Chagas disease symptoms should have begun decreasing, implying that adult health improvements were indeed an important channel.

5.1. Effects on Municipalities

We begin by studying the effects of IRS on the share of adults in a municipality with incomes above zero in each census sample. Table 5 Panel A Column (2) shows that it largely increased by more over time in treatment municipalities, reaching an additional 2.2 p.p. by 2010 (3.8% of the

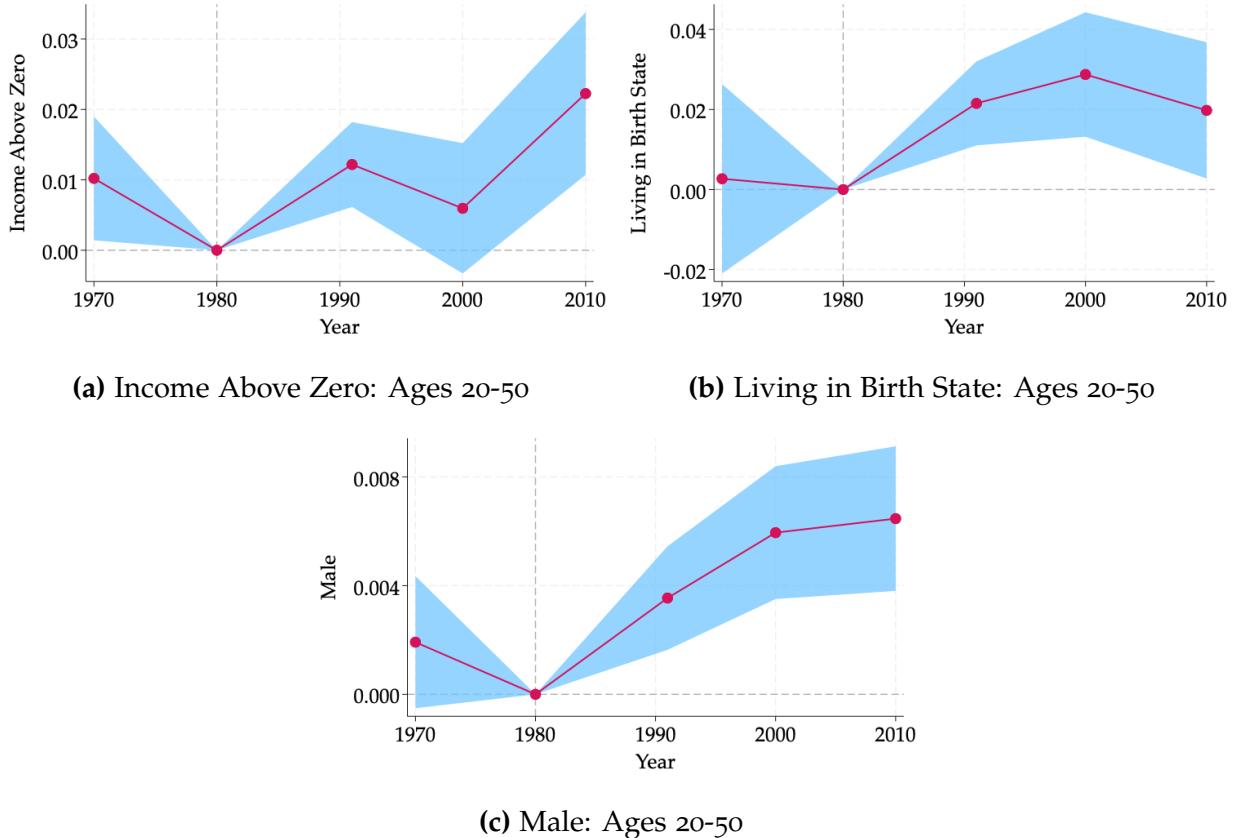
Table 5: Mechanisms for Municipality-Level Results

	(1)	(2)	(3)	(4)
<i>Panel A. Income Above Zero</i>				
Treat × 1991	0.016 (0.005)	0.012 (0.003)	0.007 (0.003)	0.007 (0.003)
Treat × 2000	0.028 (0.006)	0.006 (0.005)	-0.000 (0.004)	0.004 (0.004)
Treat × 2010	0.042 (0.008)	0.022 (0.006)	0.015 (0.005)	0.018 (0.005)
Effect through 2010 as % of Pre-1984 Mean	7.2	3.8	2.6	3.0
<i>Panel B. Living in Birth State</i>				
Treat × 1991	-0.004 (0.008)	0.022 (0.005)	0.019 (0.005)	0.020 (0.006)
Treat × 2000	-0.001 (0.012)	0.029 (0.008)	0.027 (0.008)	0.028 (0.008)
Treat × 2010	-0.013 (0.013)	0.020 (0.009)	0.024 (0.008)	0.024 (0.009)
Effect through 2010 as % of Pre-1984 Mean	-1.7	2.5	3.0	3.1
<i>Panel C. Male</i>				
Treat × 1991	0.002 (0.001)	0.004 (0.001)	0.002 (0.001)	0.002 (0.001)
Treat × 2000	0.002 (0.001)	0.006 (0.001)	0.004 (0.001)	0.004 (0.001)
Treat × 2010	0.002 (0.001)	0.006 (0.001)	0.004 (0.001)	0.004 (0.001)
Effect through 2010 as % of Pre-1984 Mean	0.4	1.3	0.8	0.8
Municipality FE and Year FE	x	x	x	x
State FE × Year FE		x	x	x
1980 Demographics × Year FE			x	x
GE Crop Suitabilities × Year FE				x
Observations	8,170	8,160	8,160	8,160
Consistent Municipalities	1,580	1,578	1,578	1,578

Notes: Observations are municipality-years. Data are from the IPUMS sample of the 1980 census. 1980 demographics are average age and age squared, shares in each racial category (Asian, Black, Brown, and White), and the share female, and GE crops are soy and maize ([Bustos, Caprettini and Ponticelli, 2016](#)). Standard errors in parentheses are clustered by consistent municipalities.

pre-IRS mean). Figure 8a shows the event study plot, which closely resembles the Gini coefficient and income percentile results in Figures 5b and 5c. The likely implication is that by immediately reducing acute Chagas disease symptoms and eventually decreasing circulatory system disease, IRS led more adults to enter into and hold down regular employment.

Figure 8: Event Study Plots for Municipality-Level Mechanisms



Notes: Graphs show estimates with 95% confidence intervals. Regressions include year, municipality, and state-by-year fixed effects, as in Table 5 Column (2). Standard errors are clustered by municipality.

Next, we examine whether there were changes in shares of the population that were living in their birth state and that were male, as IRS could have made these places more attractive.²⁴ Column (2) in Panels B and C show that within a decade of the start of IRS, these fractions had grown by more in treatment municipalities, respectively increasing an additional 2.0 p.p. (2.5% of the pre-treatment mean) and 0.6 p.p. (1.3%) by 2010. As such, vector control may have mitigated a push factor driving prime-aged men's migration out of these areas. In combination with long-run improvements in health pulling more adults into regular work, these effects could explain much of the income and inequality results. But given that Figures 8b and 8c do not resemble the event study plots for Gini coefficients and income percentiles as nicely, these compositional changes may have been less important. We test this notion formally in mediation analyses below.

As before, the estimates change little when controlling for GE crop suitabilities in Column (4). Appendices E1 through E3 also show their strong robustness to using new estimators and even large deviations from linear violations of parallel pre-treatment trends.

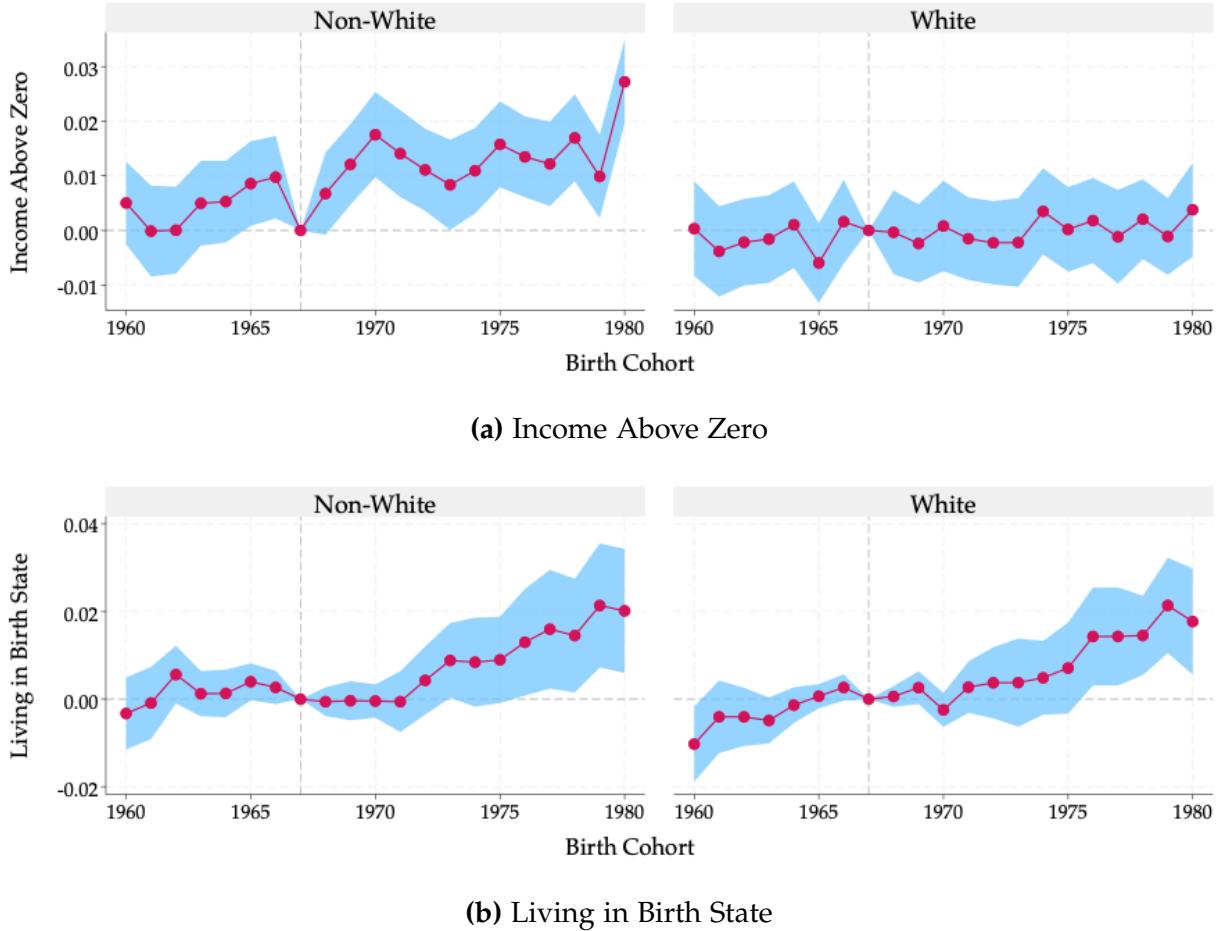
²⁴ Living in one's municipality of birth and racial group were not recorded in the 1970 census.

Table 6: Mechanisms for Individual-Level Results

	(1)	(2)	(3)	(4)
<i>Panel A. Income Above Zero: Non-White</i>				
Treat × 1968-72 Cohorts	0.012 (0.003)	0.012 (0.003)	0.012 (0.003)	0.012 (0.003)
Treat × 1973-77 Cohorts	0.014 (0.003)	0.012 (0.003)	0.012 (0.003)	0.012 (0.003)
Treat × 1978-80 Cohorts	0.020 (0.003)	0.018 (0.003)	0.018 (0.004)	0.018 (0.004)
Effect on 1978-80 Cohorts as % of Pre-1968 Cohorts' Mean	2.6	2.3	2.3	2.3
<i>Panel B. Income Above Zero: White</i>				
Treat × 1968-72 Cohorts	0.000 (0.003)	-0.002 (0.003)	-0.001 (0.003)	-0.001 (0.003)
Treat × 1973-77 Cohorts	0.001 (0.003)	0.000 (0.003)	0.000 (0.003)	0.000 (0.003)
Treat × 1978-80 Cohorts	0.005 (0.003)	0.000 (0.003)	0.002 (0.003)	0.002 (0.003)
Effect on 1978-80 Cohorts as % of Pre-1968 Cohorts' Mean	0.6	0.0	0.2	0.2
<i>Panel C. Living in Birth State: Non-White</i>				
Treat × 1968-72 Cohorts	-0.000 (0.004)	0.001 (0.002)	0.000 (0.002)	0.001 (0.002)
Treat × 1973-77 Cohorts	0.008 (0.008)	0.011 (0.005)	0.011 (0.005)	0.012 (0.006)
Treat × 1978-80 Cohorts	0.016 (0.009)	0.019 (0.007)	0.019 (0.007)	0.020 (0.007)
Effect on 1978-80 Cohorts as % of Pre-1968 Cohorts' Mean	2.0	2.3	2.3	2.5
<i>Panel D. Living in Birth State: White</i>				
Treat × 1968-72 Cohorts	0.003 (0.003)	0.001 (0.002)	0.001 (0.002)	0.002 (0.002)
Treat × 1973-77 Cohorts	0.006 (0.007)	0.009 (0.005)	0.009 (0.005)	0.009 (0.005)
Treat × 1978-80 Cohorts	0.013 (0.006)	0.018 (0.005)	0.018 (0.005)	0.018 (0.005)
Effect on 1978-80 Cohorts as % of Pre-1968 Cohorts' Mean	1.6	2.2	2.2	2.2
Birth Municipality FE and Cohort FE	x	x	x	x
Birth State FE × Cohort FE		x	x	x
Demographic FE			x	x
GE Crop Suitabilities × Cohort FE				x
Observations in Panels A and C	2,790,838	2,790,838	2,790,838	2,790,838
Observations in Panels B and D	2,234,657	2,234,657	2,234,657	2,234,657
Consistent Municipalities	1,755	1,755	1,755	1,755

Notes: Data are from the IPUMS 2010 census sample. Demographic fixed effects are for sex and racial group (Asian, Black, and Brown), and GE crops are soy and maize. Standard errors in parentheses are clustered by consistent birth municipalities.

Figure 9: Event Study Plots for Individual-Level Mechanisms



Notes: Graphs show estimates with 95% confidence intervals. Regressions include cohort, birth municipality, birth state-by-cohort, sex, and racial category fixed effects, as in Table 6 Column (3). Standard errors are clustered by consistent birth municipalities.

5.2. Effects on Individuals

Building on the municipality-level findings, we examine two of the same mechanisms—earning positive incomes and living in one's birth state—across cohorts and racial groups in Table 6. In Panels A and C, the results for non-white adults in Column (3) show larger increases for post-treatment cohorts, respectively reaching 1.8 p.p. (2.3% of the mean for pre-treatment cohorts) and 1.9 p.p. (also 2.3%) for those born in 1978-80. In contrast, there are null effects on white adults earning positive incomes in Panel B but similar increases in Panel D (1.8 p.p. for those born in 1978-80, or 2.2% of pre-treatment cohorts' mean), which again suggests that unlike the extensive margin impacts, the share living in states of birth does little to explain the reduction in racial income inequality. Importantly, the event study plots in Figure 9 show that pre-treatment cohorts had their outcomes move in parallel across treatment and control municipalities of birth.

Taken together, the individual-level evidence reinforces the municipality-level results: vector control appears to have made treated areas more attractive to existing residents, and then over time as chronic morbidity was likely averted, more non-white Brazilians were able to (re)enter the labor market. These results are robust to controlling for exposure to the introduction of GE crops in Column (4), although Appendices F1 and F2 show that only the non-zero income effect can survive a linear violation of the parallel trends assumption.²⁵

5.3. Mediation Analyses

To quantify how important the increases in schooling and earning a positive income were for the long-run and intergenerational results in Section 4, we conduct municipality- and individual-level mediation analyses. In the respective first steps, we calculate the impact of treatment on the mediator of interest M by repeating the estimation of equation (1) or (2) with this variable as the outcome,

$$M_{m,t} = \alpha_m + \gamma_t + \sum_{k \neq 1980} \tau_k \cdot \mathbb{1}[m \in \text{Treat}] \cdot \mathbb{1}[t = k] + (\delta_{s(m)} + \mathbf{X}_{m,1980}) \times \gamma_t + \epsilon_{m,t}, \quad (3)$$

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

and compute the average of the post-treatment τ_k^{Med} , or $\bar{\tau}^{\text{Med}}$. In the second step, we modify equation (1) or (2) by adding the mediator to the right-hand side,

$$y_{m,t} = \alpha_m + \gamma_t + \sum_{k \neq 1980} \tau_k \cdot \mathbb{1}[m \in \text{Treat}] \cdot \mathbb{1}[t = k] + \mu M_{m,t} + (\delta_{s(m)} + \mathbf{X}_{m,1980}) \times \gamma_t + \epsilon_{m,t}, \quad (5)$$

$$\begin{aligned} y_{i,m,c} = \alpha_m + \gamma_c + \sum_{k \neq 1967} \tau_k \cdot \mathbb{1}[\mathbb{P}_i(m \in \text{Treat}) > 0] \cdot \mathbb{1}[c = k] + \mu M_{i,m,c} + (\delta_{s(m)} + \mathbf{Z}_m) \times \gamma_c \\ + \mathbf{X}_i \beta + \epsilon_{i,m,c}. \end{aligned} \quad (6)$$

We then estimate the average of the post-treatment τ_k ($\bar{\tau}$), or the direct impact of treatment on the outcome, and $\mu \bar{\tau}^{\text{Med}}$, the effect of the mediator obtained by substituting equation (3) into equation (5) for municipality-level outcomes or (4) into equation (6) for individual-level outcomes. In this way, we quantify the share of the total impact $\bar{\tau} + \mu \bar{\tau}^{\text{Med}}$ due to the second term in this sum.

The estimated effects for municipality-level outcomes presented in Table 7 suggest that both the extensive margin and schooling effects were important for increasing incomes and decreasing inequality in treated municipalities. But the contribution of more adults earning non-zero incomes (10% to 113%) was considerably greater than that of increased schooling (5% to 22%).

²⁵ It remains at the margin of statistical significance if there is a deviation from linearity of up to 0.4 standard errors of the average estimate for the 1960-66 cohorts.

Table 7: Mediation Analysis for Municipality-Level Results

	Mediating Variables			
	Income Above Zero (1)	Years of Schooling (2)	Living in Birth State (3)	Male (4)
<i>Panel A. Log GDP per Capita</i>				
Mediation Effect	0.016	0.007	-0.004	0.001
Total Effect	0.156	0.156	0.156	0.156
Amount Mediated	10%	5%	-3%	1%
<i>Panel B. Log Gini Coefficient</i>				
Mediation Effect	-0.012	-0.001	-0.000	-0.002
Total Effect	-0.011	-0.011	-0.011	-0.011
Amount Mediated	109%	10%	1%	23%
<i>Panel C. Income Percentile</i>				
Mediation Effect	0.73	0.15	-0.02	0.14
Total Effect	0.65	0.65	0.65	0.65
Amount Mediated	113%	22%	-3%	22%

Notes: Effects are calculated using the procedure described in the text. GDP per capita is calculated using data from Ipeadata for all available census years and all other data are from IPUMS.

Indeed, the impact of the increase in the male share of the population (1% to 23%) was essentially equivalent to that of greater educational attainment. The individual-level mediation analysis for non-white Brazilians in Table 8 Panels A and B shows similar results: earning a positive income accounted for 67-90% of the increases in incomes, whereas years of schooling accounted for only 16-23%. In Panel C, however, the two mediators contributed roughly equal amounts to the inter-generational literacy effect (7% due to non-zero incomes and 10% due to schooling), which makes intuitive sense because literacy is an educational outcome. Taken together, these mediation effects suggest that the extensive margin labor market channel—made possible by avoiding both the acute and chronic phase of Chagas disease—was the principal mechanism through which the campaign raised incomes and reduced (racial) inequality in the long run and into the next generation. Below, we test for direct evidence of reductions in circulatory system disease.

5.4. Effects on Health

If cardiovascular morbidity from chronic Chagas disease was severe enough to reduce labor force participation, the costs it imposed on society could have extended into hospitalizations covered by Brazil's *Sistema Único de Saúde* (SUS), the world's largest government-run health care system,

Table 8: Mediation Analysis for Individual-Level Non-White Results

	Mediating Variables		
	Income Above Zero (1)	Years of Schooling (2)	Living in Birth State (3)
<i>Panel A. Log Income</i>			
Mediation Effect	0.091	0.016	-0.002
Total Effect	0.102	0.102	0.0045
Amount Mediated	90%	16%	-2%
<i>Panel B. Income Percentile</i>			
Mediation Effect	0.69	0.24	-0.05
Total Effect	1.03	1.03	1.03
Amount Mediated	67%	23%	-5%
<i>Panel C. Child 9-18 Literate</i>			
Mediation Effect	0.0003	0.0004	-0.0002
Total Effect	0.0045	0.0045	0.0045
Amount Mediated	7%	10%	-4%

Notes: Effects are calculated using the procedure described in the text. Panel C uses fathers' values of the mediating variables. Data are from the IPUMS 2010 census sample.

which consumes about 4% of GDP each year and 70% of the population relies on.²⁶ Specifically, according to SUS data, circulatory system diseases caused one-tenth of the hospitalizations it paid for between 2010 and 2019 (over 850,000 per year), accounting 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, we now examine the effects of Chagas disease control on circulatory system-related hospital care covered by the SUS and deaths.

5.4.1. Data and Empirical Strategy

Our outcomes of interest are the logs of hospitalizations, spending on hospital care, and deaths, each of which is categorized by International Classification of Diseases (ICD) codes. The first two of these measures are from the SUS's Hospital Information System (SIH/SUS), and we deflate the spending data so that figures are in 2019 R\$. The data on deaths are from the SUS's Mortality Information System (SIM). Because Chagas disease is highly under-diagnosed and its chronic effects manifest primarily as cardiovascular problems 10 or more years after infection (see Section

²⁶ 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>.

²), we focus on all diseases of the circulatory system.²⁷ We set our omitted year to 1994, or a decade after IRS began.²⁸ However, as the SUS does not consistently provide municipality-level data for years prior to 1995, we restrict our focus to state-level data from 1991 to 2010.²⁹

Along the lines of Section 4.2, our measure of each state's exposure to Chagas disease vector control is whether any of its population was living in treatment municipalities in 1980. However, because the SUS is a heavily decentralized system with transfers of responsibilities and funds to states and municipalities (Castro et al., 2018), there are likely confounders that vary across both state and year (e.g., public health priorities and non-hospital care spending) in violation of the difference-in-differences common trends assumption. To address this complication, we use a triple-differences strategy using all other non-circulatory system disease categories as the additional control group, under the assumption that they are subject to the same state-specific, time-varying factors.³⁰ The specification that we estimate is thus

$$y_{s,t,d} = \alpha_{s,t} + \gamma_{t,d} + \delta_{s,d} + \sum_{k \neq 1994} \tau_k \cdot \mathbb{1}[\mathbb{P}_{s,1980}(m \in \text{Treat}) > 0] \cdot \mathbb{1}[t = k] \cdot \mathbb{1}[d = \text{Circ}] \\ + \eta_{r(s)} \times \gamma_{t,d} + \epsilon_{s,t,d}, \quad (7)$$

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.³¹ We also include the interaction of $\eta_{r(s)}$ and $\gamma_{t,d}$ to control for region-disease category-specific trends.

This strategy first estimates the differences in outcomes due to circulatory system diseases in a given year between states with any individuals living in treatment municipalities in 1980 and those in the same region that did not, relative to the size of that difference in 1994. Then we compare this double-difference to the analogous one for non-circulatory diseases. Because 85% of the 1980 population outside of São Paulo lived in states with treatment municipalities, we frame our results as moving from 85% to 0% of a state's population with vector exposure. To be parsimonious and minimize the impact of year-specific noise, we report average effects for multi-year bins (1995-99, 2000-04, and 2005-10) in the table and show point estimates and confidence

²⁷ For hospital care outcomes in 1991-97, 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.

²⁸ 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.

²⁹ While SUS does provide some hospitalization data at the municipality level, coverage in the mid 1990s is incomplete and inconsistent, making it unreliable for assessing pre-treatment trends. For this reason, we focus on state-level data from this period, which are more consistently and fully reported.

³⁰ If it holds, the triple-difference approach is a valid strategy even when difference-in-differences rejects the absence of differential pre-treatment trends for each disease group (Olden and Møen, 2022)

³¹ Brazilian states are grouped into five regions: North, Northeast, Southeast, South, and Center-West.

Table 9: Effects of Chagas Disease Vector Control on Circulatory Disease Outcomes

	Log Hospitalizations		Log Hospital Care Spending		Log Deaths	
	(1)	(2)	(3)	(4)	(5)	(6)
Treat × Circulatory × 1995-99	-0.046 {0.116}	-0.060 {0.089}	-0.044 {0.620}	-0.094 {0.353}	-0.023 {0.397}	-0.065 {0.400}
Treat × Circulatory × 2000-04	-0.132 {0.016}	-0.152 {0.047}	-0.070 {0.391}	-0.158 {0.130}	0.001 {0.989}	-0.081 {0.207}
Treat × Circulatory × 2005-10	-0.261 {0.001}	-0.207 {0.005}	-0.263 {0.134}	-0.180 {0.092}	0.030 {0.780}	-0.088 {0.183}
Average Annual Change 1995-2010 (%/year)	-1.7	-1.4	-1.7	-1.2	0.2	-0.6
State-Year FE, Year-Disease Category FE, and State-Disease Category FE	x	x	x	x	x	x
Region FE × Disease Category FE × Year FE		x		x		x
Observations	1,392	1,392	1,392	1,392	1,392	1,392
Consistent States	24	24	24	24	24	24

Notes: Observations are state-year-disease categories. Data are from DATASUS. Wild cluster bootstrap *p*-values in curly braces are calculated after clustering standard errors by consistent states.

intervals for each year in event study plots. We cluster standard errors by the 24 consistent states in the sample and use the wild cluster bootstrap (Cameron, Gelbach and Miller, 2008).

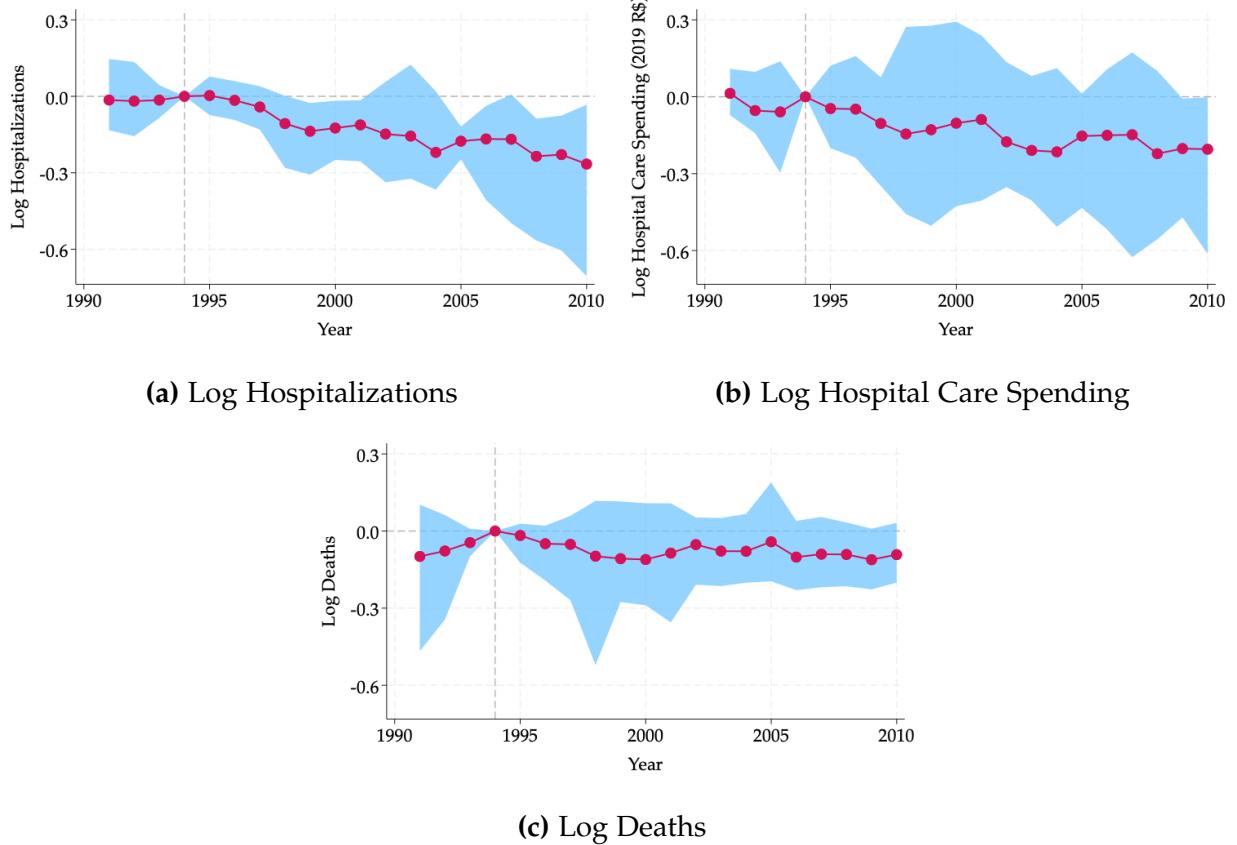
5.4.2. Circulatory Disease Hospital Care, Spending, and Deaths

Table 9 Column (2) shows that by 2005-10—i.e., 10-15 years after reductions in chronic Chagas disease symptoms should have begun—SUS-covered hospitalizations due to circulatory diseases had fallen by what converts to about 19% more in states where the population was formerly exposed to *T. cruzi* transmission, and this estimate is distinguishable from zero at the 1% level. Importantly, the impact was smaller but still detectable in 1995-99 (6%) and it grew over time (about 14% in 2000-04), and Figure 10a shows that outcomes evolved in parallel prior to 1994.

Consistent with this result, the estimate for hospital care spending in Column (4) converts to a 16% larger decline in treated states by 2005-10. This effect is distinguishable from zero at the 10% level and the point estimates for the previous multi-year bins are similar in magnitude to those in Column (2), albeit without statistical significance. The event study plot in Figure 10b also shows a similar pattern in the point estimates but with much wider confidence intervals, possibly reflecting the effects of Brazil's (hyper)inflation and currency changes in this period.

In contrast, Column (6) shows no statistically detectable effect on circulatory disease mortality by 2005-10. Across all bins, estimates are much smaller than in Columns (2) and (4) and do not

Figure 10: Event Study Plots for Circulatory Disease Effects



Notes: Graphs show dynamic estimates with 95% wild cluster bootstrap (WCB) confidence intervals. Data are from DATASUS. Regressions include fixed effects for state-year, year-disease category, state-disease category, and region-year-disease category, as in Table 9 Columns (2), (4), and (6). WCB confidence intervals are calculated after clustering standard errors by the 24 consistent states in the sample.

reach conventional significance levels. Figure 10c shows that the pattern throughout this period is a flat trajectory that remains close to zero. We view the absence of an effect on mortality as reassuring given that the length of the lag between the manifestation of chronic Chagas disease symptoms and death is uncertain (see Section 2.1 and Appendix A2).

Overall, these findings indicate that vector control delivered substantial long-run reductions in circulatory disease hospitalizations and associated public spending, and that effects on mortality had not yet materialized by the end of our study period. In Appendices G1 through G3, we show that these conclusions do not change when using new estimators or as a result of linear violations of the parallel trends assumption or a small deviation from linearity (no more than 0.1 standard errors of the estimated coefficient for 1991-93).

6. Cost-Benefit Analyses and Extrapolation

Given the substantial benefits of Chagas disease vector control for individuals' incomes (Section 4.2) and states' public health care systems (Section 5.4), we provide two simple cost-benefit analyses to understand the 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 about 20% and an infinite marginal value of public funds (MVPF) arising from the health spending savings. 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.

6.1. Internal Rate of Return

In our first cost-benefit analysis, we calculate the IRR, or the discount rate required for a net present value of zero. We report all values below in 2019 US dollars. On the cost side, [Piola \(1981\)](#) reported what the Public Health Campaigns Superintendency (SUCAM) spent on Chagas disease control for 1975-80. We could not find spending data for 1981-83, so we assume SUCAM spent the 1975-80 average (\$38 million) in these three years. Similarly, the only figure from the first three IRS years we found was from [Dias \(1987\)](#) for 1986 (\$74 million), so we assign that value to 1984-85 because [Dias \(1986\)](#) noted that public health authorities intended to spend a constant amount each year in real terms. After the 1986-89 pause in IRS—which we interpret as nothing being spent on Chagas disease control—we take from [SUCAM \(1994\)](#) the amount it was appropriated for this purpose in 1990 (\$72 million) and assume that its 1991-94 appropriations decreased by 10% per year, as forecasted in its budget requests given the elimination of *T. infestans* from progressively more territory and the shift to surveillance.

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.³² For incomes, we assume that an additional 1.4% of each non-white cohort from treatment municipalities—the average of the effects for the multi-cohort bins in Table 6 Panel A Column (2)—were shifted from earning no income to the median income in the 2010 sample, which was \$239/month. We also assume that this amount was constant in each year from 1984 to 2010, the size of each non-white

³² 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 (for acute Chagas disease and for controlled, medically managed heart failure).

cohort from treatment municipalities was 750,000, and they entered the labor force at age 16.³³ To calculate averted circulatory disease hospital care spending, we use the fact that the SUS spent approximately 0.1% of GDP on it each year in 2010-19 and multiply those amounts by the estimated percentage effects in the multi-year bins in Table 9 Column (4).³⁴

As a result, we estimate an IRR of 19.9%. For comparison, Hamory et al. (2021) calculated an IRR of 37% in a 20-year follow-up of deworming in western Kenya by comparing consumption gains to the costs of administering the drugs and hiring additional teachers resulting from the increase in schooling, and Bütkofer and Salvanes (2020) estimated an IRR of 3.2-8.5% for Norway's mid-20th century anti-tuberculosis campaign. These differences likely emerge from the differences in upfront costs across these interventions. Specifically, deworming pills are extremely cheap and relatively easy to distribute in schools, whereas IRS against *T. infestans* requires spraying many homes in many municipalities with insecticides, and tuberculosis testing and vaccination were even more expensive.³⁵ Thus, years of discounting offset much of the savings from controlling Chagas disease even though they comprised a non-trivial fraction of GDP each year. To illustrate this point, note that the IRR would be 18.9% if we only considered the income gains. Nonetheless, failing to consider the chronic health effects of eliminating Chagas disease transmission means understating its return by 1.0 p.p. (over 5%), so this omission could still result in suboptimal allocations of scarce funds to promote economic development.

6.2. Marginal Value of Public Funds

We also use the assumptions above to calculate the 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).³⁶ Because the IRR when considering only the hospital care savings is 7.6%—more than 50% above the 5% discount rate used in studies of developing economies (Haacker, Hallett and Atun, 2020)—the implication is that this intervention's MVPF is also infinity. This result notably arises without raising additional revenue, as the marginal tax

³³ 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. Our cohort size comes from averaging the number of non-white individuals from the 1968-80 cohorts in the treatment group in the 2010 census sample.

³⁴ Note that these years are well into the post-treatment period, so the actual share of GDP spent on circulatory disease hospital in 1984-2010 was likely higher.

³⁵ The cost of mass deworming (semi-)annually is often around \$1/pupil. In contrast, Oliveira Filho (1989) estimated that IRS to protect a house with five inhabitants from *T. infestans* for a year in 1985 cost \$8-30/person, and Bütkofer and Salvanes (2020) calculated that the tuberculosis control program cost \$122/person in municipalities where at least 1% of residents had active infections.

³⁶ For example, these authors estimated an infinite MVPF arising from the long-run health care savings generated by the childhood Medicaid eligibility expansion studied by Wherry et al. (2018).

rate at the median income in the 2010 census data (2019 R\$ 908) was 0%.³⁷ Therefore, from the perspective of the governments of developing economies, 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.

6.3. Extrapolating Effects across Latin America

For a simple assessment of the implications of controlling Chagas disease across Latin America, we extrapolate the estimated effects on Brazilian municipalities' output per person and inequality to all countries in the region. We use data from the [World Health Organization \(2015\)](#) on the share of each country's population still exposed to transmission (see Appendix H1 for a map), the region-wide average of which is 13%. Following [Bleakley \(2007, 2010\)](#), we perform a back-of-the-envelope calculation on the potential gains over 26 years by multiplying World Bank data on 2019 GDP per capita and Gini coefficients by this share and our regression estimates in Section 4.1.³⁸

With the appropriate caveats in mind, Table 10 presents these extrapolated impacts. The analysis suggests that, if Chagas disease transmission were eliminated across Latin America, after 26 years the region's GDP per capita could be about 2.6% higher and its Gini coefficient could be around 0.2% lower. Naturally, there is significant variation across countries depending on the shares exposed, implying that countries like Ecuador (29%), Guyana and Suriname (25%), and Mexico (21%) would experience large effects while unexposed wealthier ones (Chile and Uruguay) would not.³⁹ 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.

7. 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

³⁷ For marginal tax rates in Brazil in 2010, see: <https://www.gov.br/receitafederal/pt-br/assuntos/meu-imposto-de-renda/tabelas/2010>.

³⁸ 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.

³⁹ Along the lines of Figure 2, 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 10: Extrapolated 26-Year Impacts of Chagas Disease Control across Latin America

Country	Population Exposed (%)	GDP per Capita (\$)	Extrapolated Change (\$)	Gini Coefficient	Extrapolated Change
Argentina	5	10,076	99	42.9	-0.04
Belize	22	4,983	216		
Bolivia	6	3,552	42	41.6	-0.04
Brazil	13	8,876	228	53.5	-0.12
Chile	0	14,699	0	44.4	0.00
Colombia	11	6,419	139	51.3	-0.10
Costa Rica	5	12,762	126	48.2	-0.04
Ecuador	29	6,223	356	45.7	-0.24
El Salvador	15	4,168	123	38.8	-0.10
Guatemala	10	4,639	91	48.3	-0.09
Guyana	25	6,610	326		
Honduras	15	2,574	76	48.2	-0.13
Mexico	21	9,950	412	46.7	-0.17
Nicaragua	11	1,924	42	46.2	-0.09
Panama	13	15,774	404	49.8	-0.12
Paraguay	20	5,384	212	45.7	-0.16
Peru	4	7,023	55	41.6	-0.03
Suriname	25	6,854	338		
Uruguay	0	17,688	0	39.7	0.00
Average	13	7,904	205	45.8	-0.11

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 2010 regression estimates for Brazilian municipalities in Table 2 Column (2).

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 that Brazil's campaign to control the main vector of Chagas disease, which has both acute and chronic phases like many other NTDs, had many important short- and long-run benefits beyond individuals' labor market returns. In particular, we found that within a decade, it led to substantive increases in GDP per capita and reductions in inequality in treated municipalities, and these effects continued to grow over time. Furthermore, exposure to vector control in childhood raised adult incomes for non-white Brazilians, helping to increase the speed of racial convergence in a country where such disparities are extremely wide. We also found evidence that improved long-run health (as captured by earning non-zero incomes) played a much larger role than educational attainment in driving these results. In addition, we showed

that controlling this NTD may help to interrupt the intergenerational transmission of poverty by increasing the literacy of the children of these non-white adults.

Because circulatory system disease causes a substantial share of hospitalizations (10%) and hospital care 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 system causes than all others 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 20% 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.⁴⁰

Thus, these results present a more complete picture of the economic consequences of NTD control for developing countries, the vast majority of which can be eliminated via environmental management programs like the IRS campaign studied in this paper. Whether our results generalize beyond this malady that exclusively afflicts the Americas is an open question we leave to future research. Nonetheless, we believe that this paper has identified novel areas through which health improvements can generate inclusive growth in developing countries, helping to strengthen justifications for controlling transmission of not just this NTD—which an estimated 8 million people throughout the Western Hemisphere suffer from and another 75 million are exposed to—but also of all the others that cause chronic health problems among the poorest billion people on the planet.

⁴⁰ However, because these benefits generally materialize many years later, the results raise important questions for the political economy of disease control given potentially short electoral time horizons. We view this political economy margin as an important area of 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

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University of Chicago

August 21, 2025

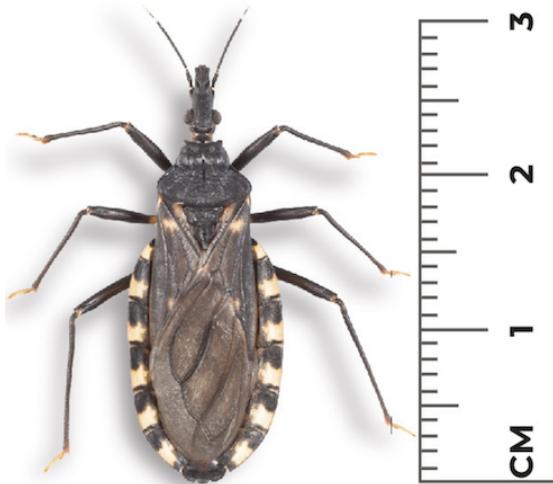
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Appendix A. Additional Figures: Chagas Disease and Its Control in Brazil

A1. Image of *Triatoma infestans*

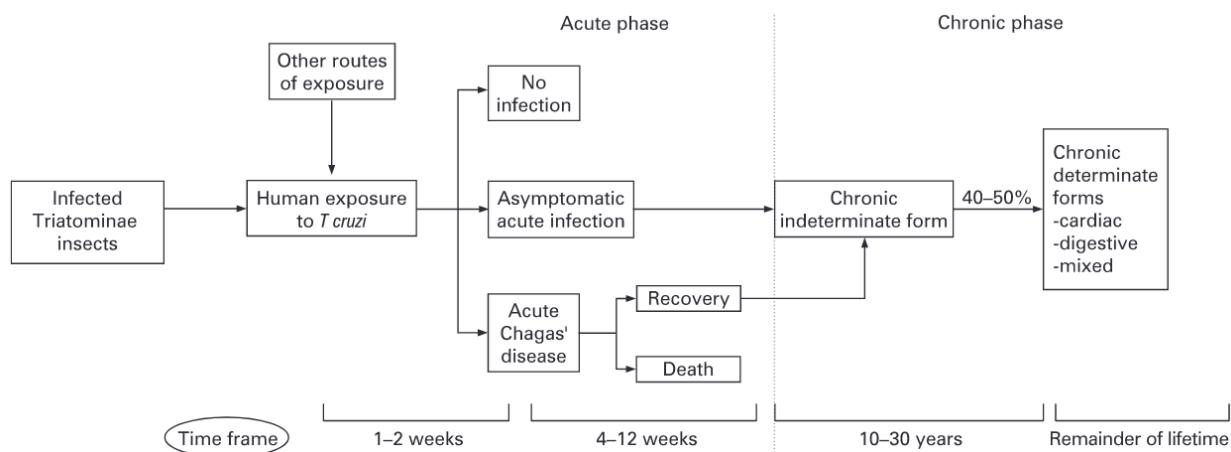
Figure A1: Image of *Triatoma infestans* [6]



Notes: Image from CeNDIE and CEPAVE (2023, p. 15) shows *T. infestans*, Brazil's main vector before 1984. Triatomine bugs are known as kissing bugs (US and Canada), vinchucas (Argentina, Bolivia, Chile, Ecuador, and Uruguay), chinches (Central America), barbeiros (Brazil), chipos (Venezuela), and pitos (Colombia), among other names.

A2. Phases of Chagas Disease

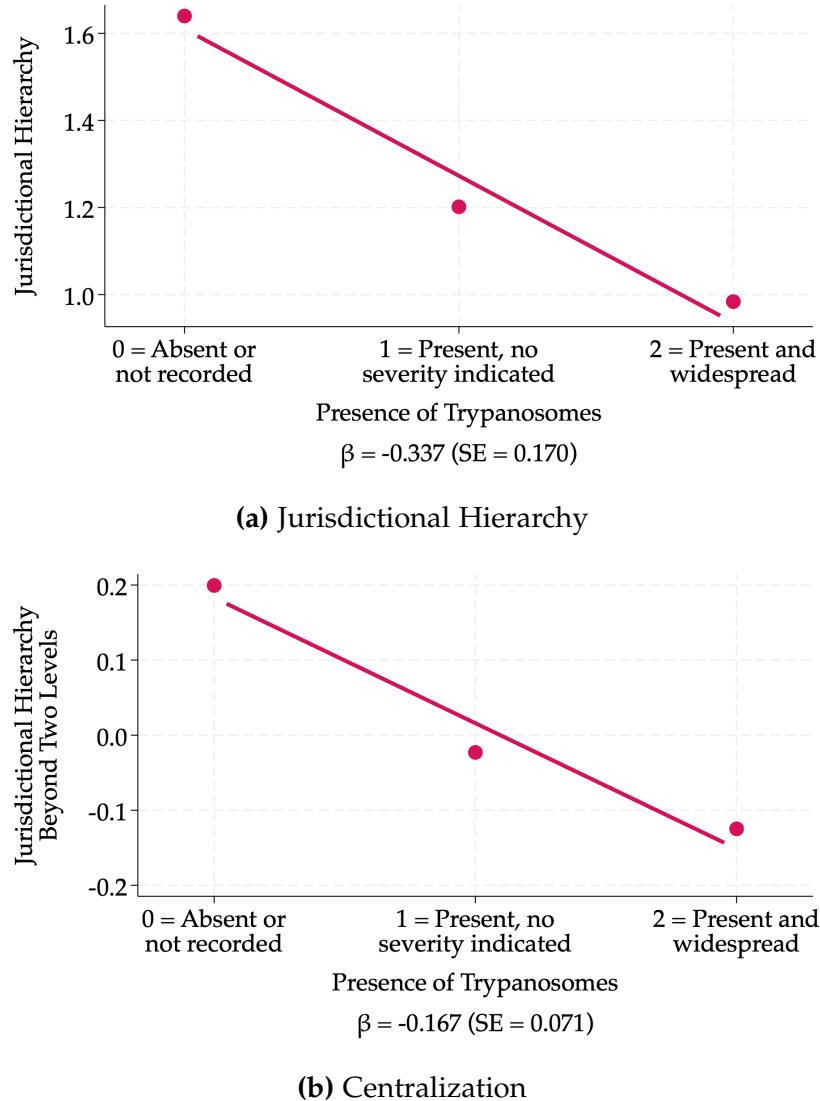
Figure A2: Phases of Chagas Disease [6, 34]



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

A3. Precolonial Presence of Trypanosomes and Jurisdictional Hierarchy

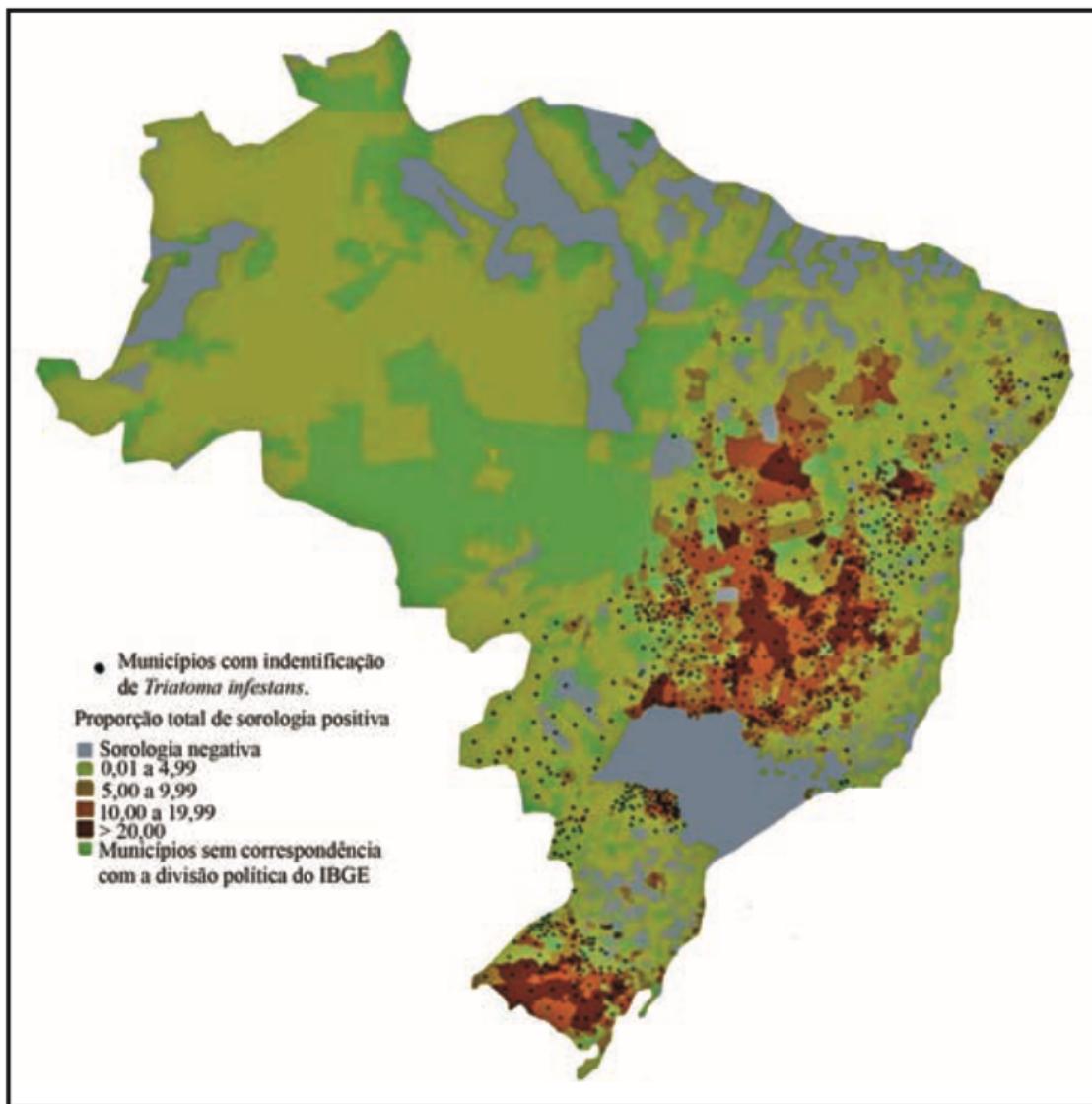
Figure A3: Precolonial Presence of Trypanosomes and Jurisdictional Hierarchy [7]



Notes: Figure presents binned scatterplots. The outcome in the top panel is jurisdictional hierarchy beyond the local community, and the outcome in the bottom panel is an indicator variable for a jurisdictional hierarchy above two. The categorical explanatory variable takes one of the three listed values. Observations are 62 societies in the Standard Cross-Cultural Survey in the Americas. All plots include controls for a quadratic in latitude and longitude, average rainfall, average temperature, elevation, agricultural suitability, and malaria ecology. Estimated slopes and robust standard errors correspond to the fitted lines.

A4. *T. cruzi* Seroprevalence, 1975-83

Figure A4: *T. cruzi* Seroprevalence, 1975-83 [8]



Notes: Figure reproduces a map of *T. cruzi* Seroprevalence prior to the Chagas control campaign across municipalities from Passos and Silveira (2011, p. 48). Black dots represent municipalities in which *T. infestans* were present, gray shading indicates 0% *T. cruzi* prevalence, darker colors from yellow to brown indicate higher prevalence (see the ranges given in the legend), and green indicates municipalities whose boundaries changed over time.

A5. Images of Indoor Residual Spraying

Figure A5: 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):

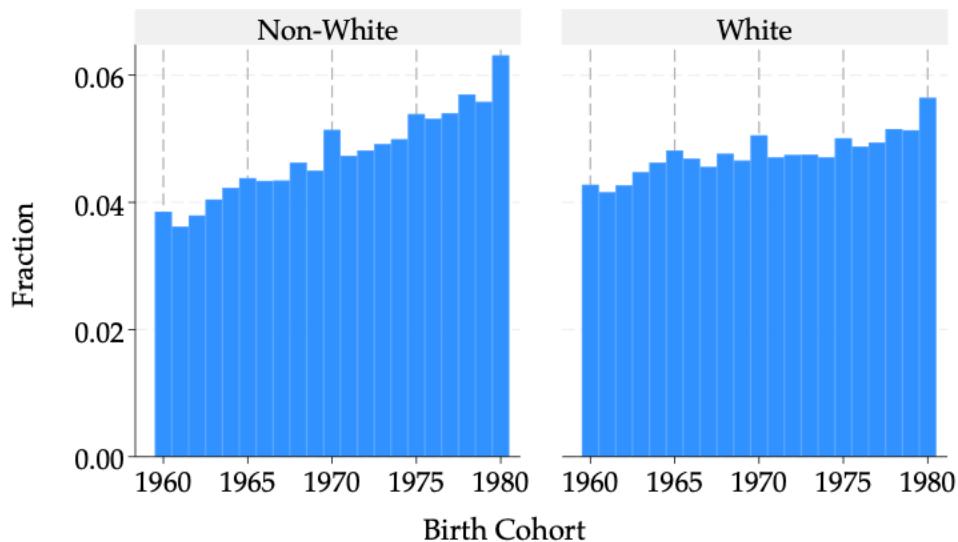
- 1 *The Preparatory Phase.* This includes mapping the area to be treated, and manual sampling of bugs from each house and peri-domestic habitats in the region. From this information, the costs and activities in terms of personnel, insecticides, equipment and transport can be programmed.
- 2 *The Attack Phase.* The initial spraying of an area is known as 'attack one' or massive attack, during which all houses and out-buildings 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 \(1987, p. 338\)](#).

Appendix B. Additional Figures: Data and Empirical Strategy

B1. Heaping of Birth Years in Individual-Level Data

Figure B1: Histogram of Birth Years [14, 21]

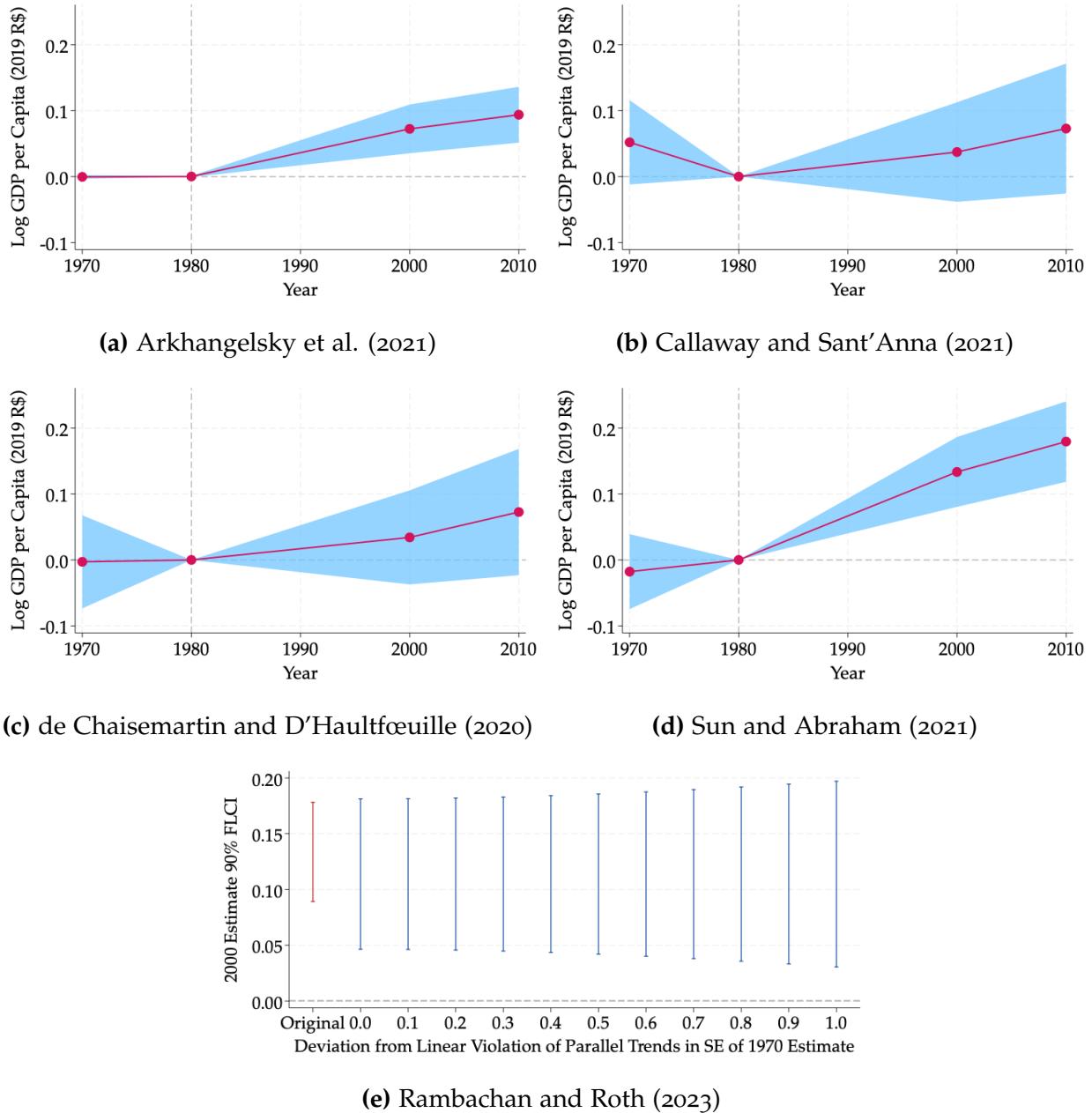


Notes: Data are from the IPUMS 2010 census sample.

Appendix C. Additional Figures: Main Results for Municipalities

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

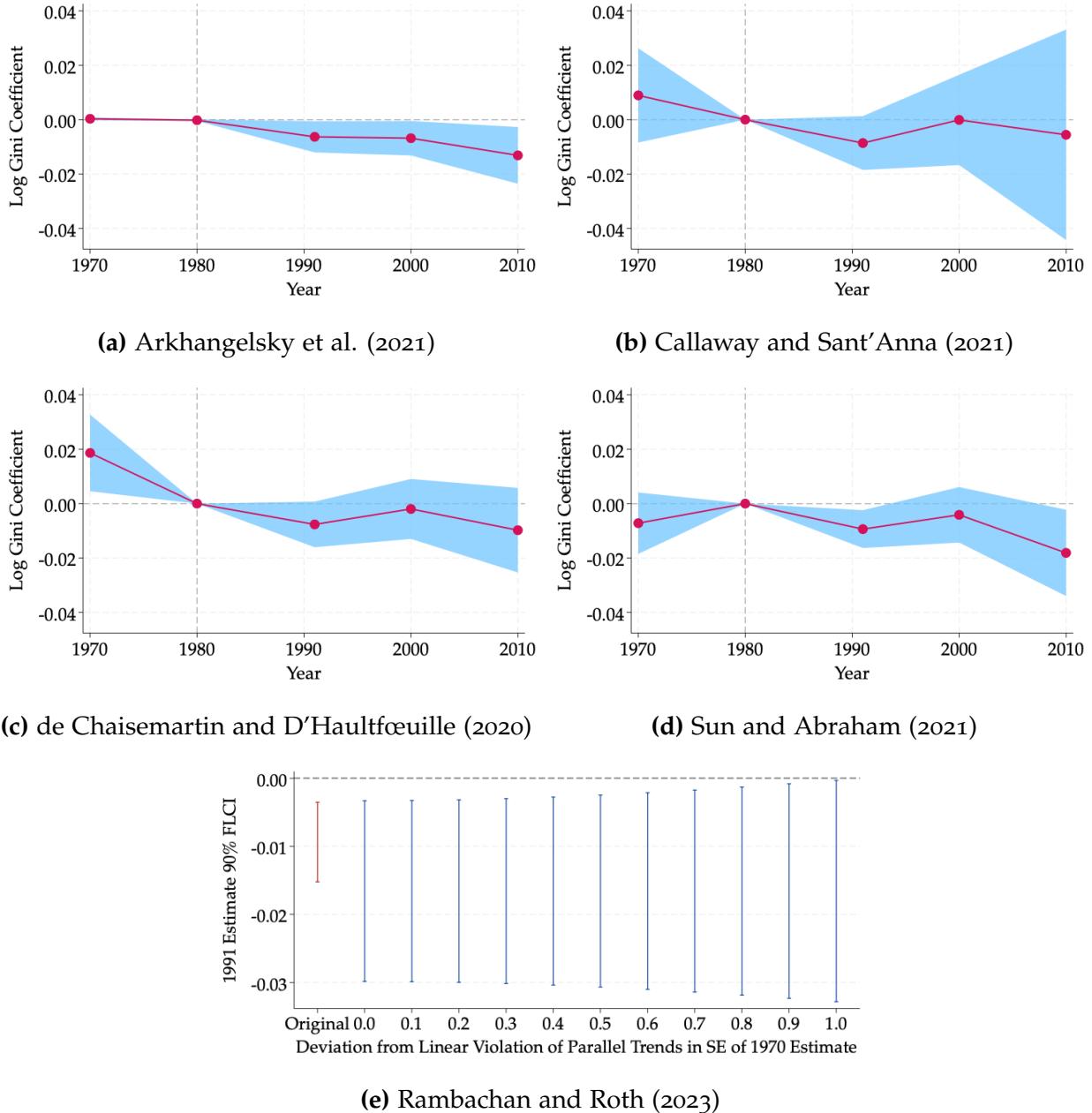
Figure C1: GDP per Capita Results Using New Estimators [19]



Notes: Observations are municipality-years. Data are from Ipeadata. The top four panels show estimates when using a given procedure with 95% confidence intervals. The bottom panel shows 90% fixed length confidence intervals for the 2010 estimate given deviations from linear violations of parallel trends. Regressions include year, municipality, and state-year fixed effects. Standard errors are clustered by municipality.

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

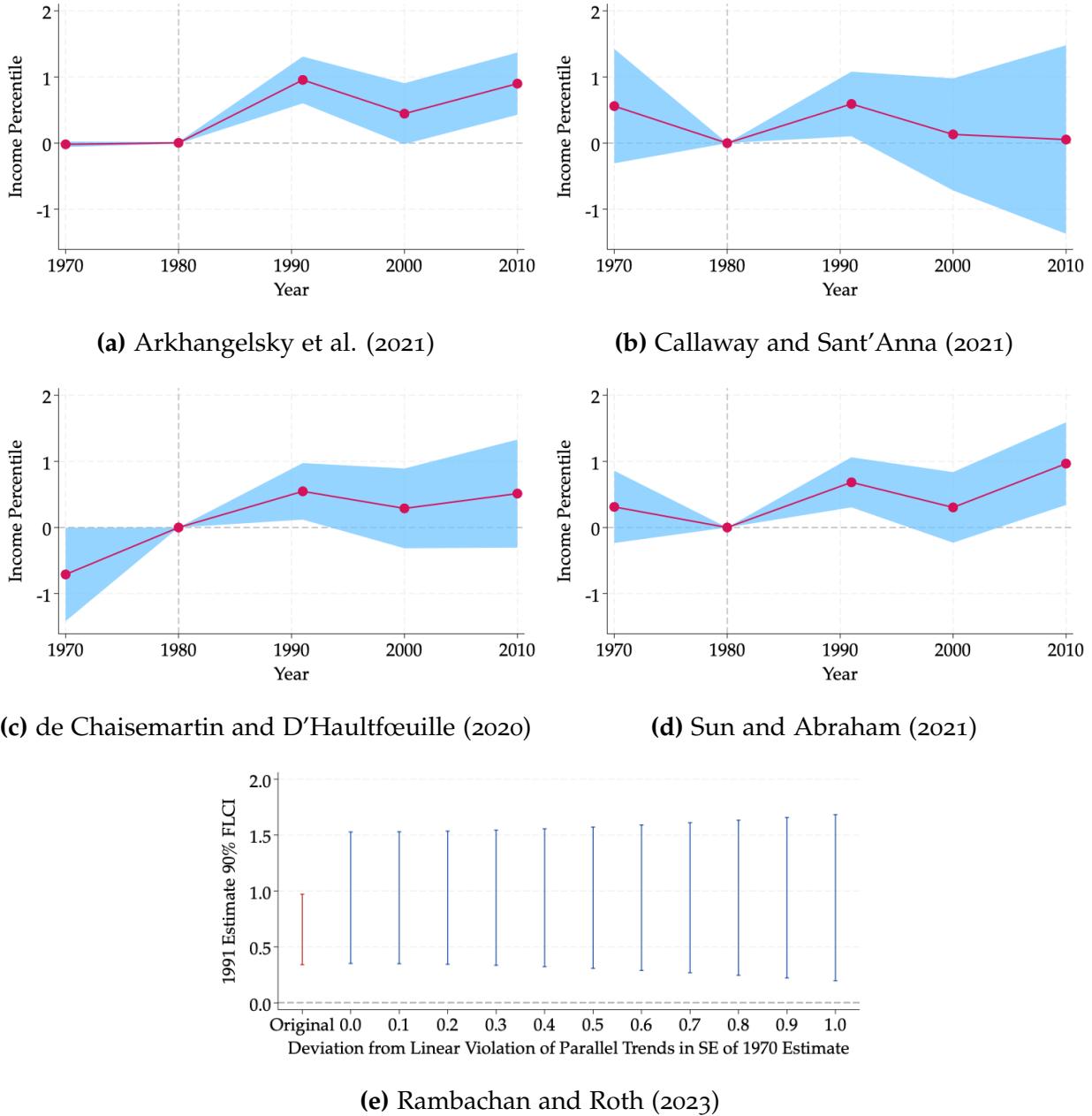
Figure C2: Gini Coefficient Results Using New Estimators [19]



Notes: Observations are municipality-years. Data are from Ipeadata. The top four panels show estimates when using a given procedure with 95% confidence intervals. The bottom panel shows 90% fixed length confidence intervals for the 2010 estimate given deviations from linear violations of parallel trends. Regressions include year, municipality, and state-year fixed effects. Standard errors are clustered by municipality.

C3. Income Percentile Results Using New Difference-in-Differences Techniques

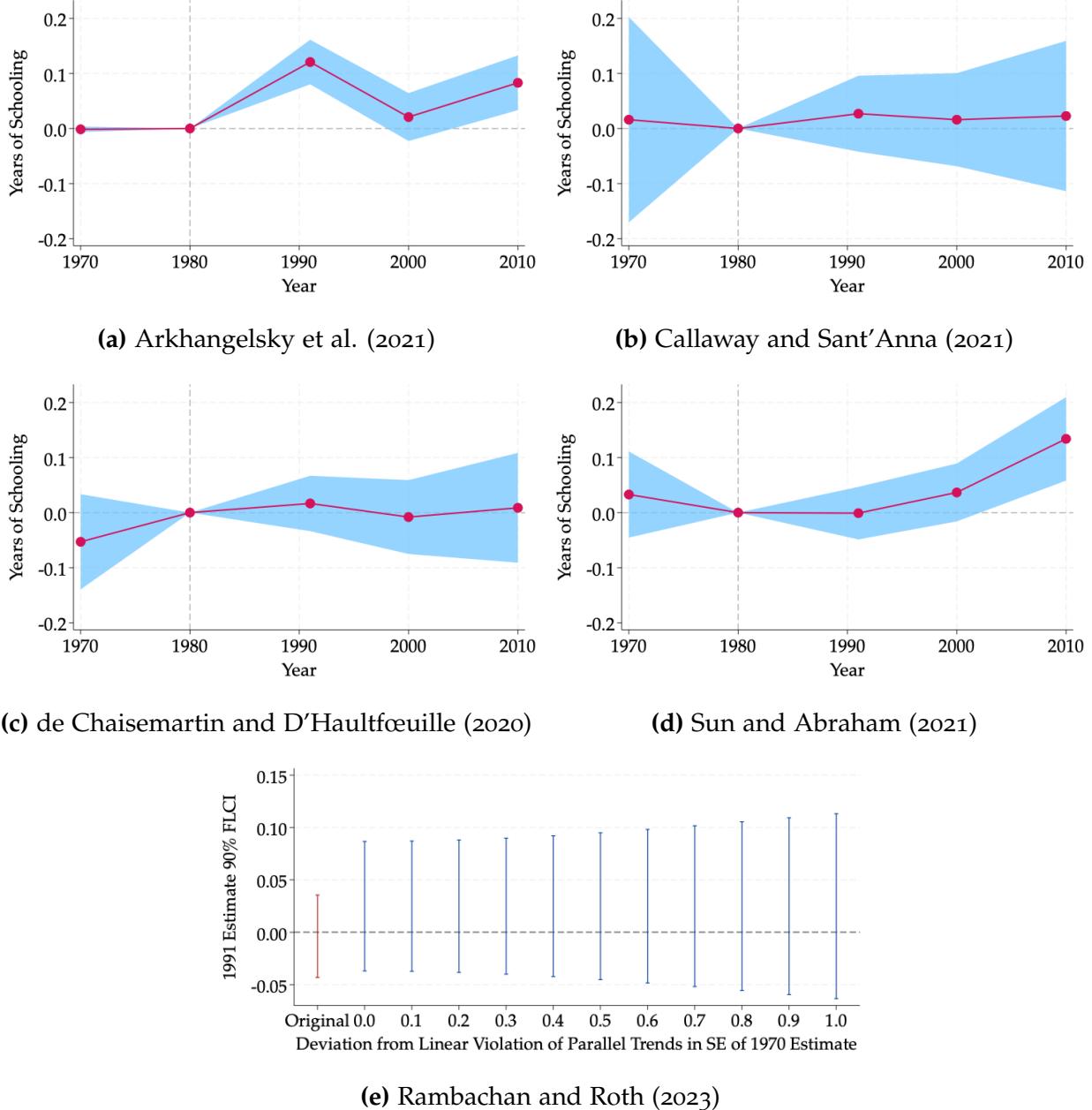
Figure C3: Income Percentile Results Using New Estimators [19]



Notes: Observations are municipality-years. Data are from Ipeadata. The top four panels show estimates when using a given procedure with 95% confidence intervals. The bottom panel shows 90% fixed length confidence intervals for the 2010 estimate given deviations from linear violations of parallel trends. Regressions include year, municipality, and state-year fixed effects. Standard errors are clustered by municipality.

C4. Years of Schooling Results Using New Difference-in-Differences Techniques

Figure C4: Years of Schooling Results Using New Estimators [19]

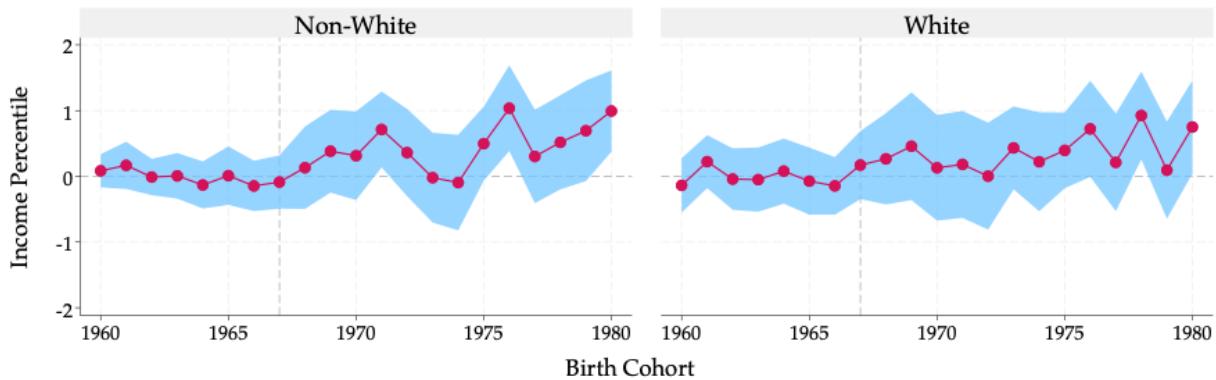


Notes: Observations are municipality-years. Data are from Ipeadata. The top four panels show estimates when using a given procedure with 95% confidence intervals. The bottom panel shows 90% fixed length confidence intervals for the 2010 estimate given deviations from linear violations of parallel trends. Regressions include year, municipality, and state-year fixed effects. Standard errors are clustered by municipality.

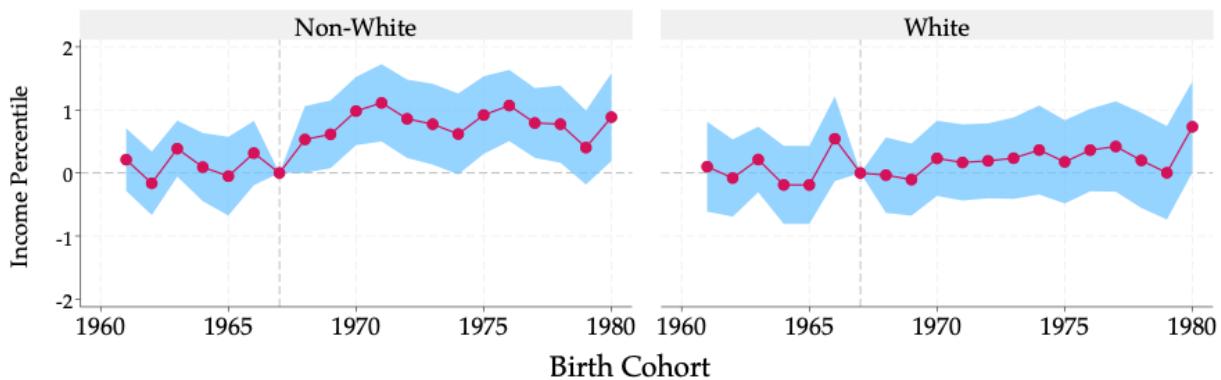
Appendix D. Additional Figures: Main Results for Individuals

D1. Income Percentile Results Using New Difference-in-Differences Techniques

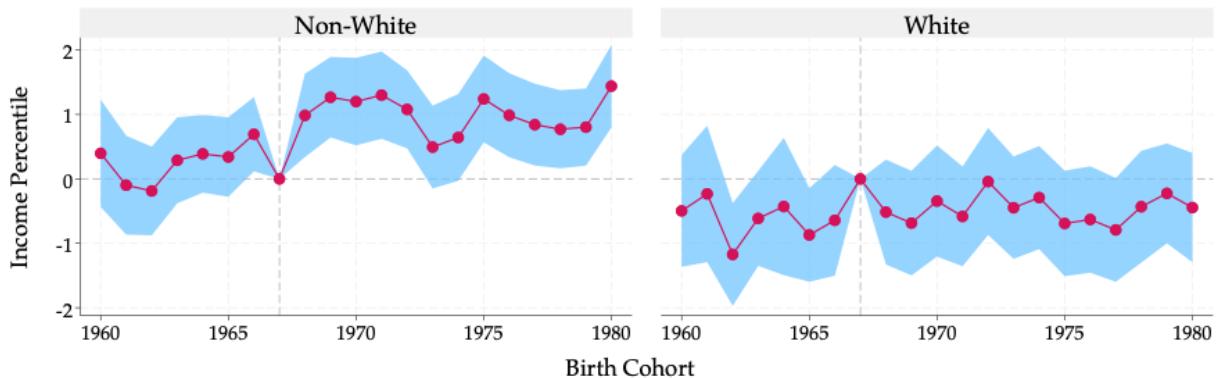
Figure D1: Income Percentile Results Using New Estimators [23]



(a) Arkhangelsky et al. (2021)

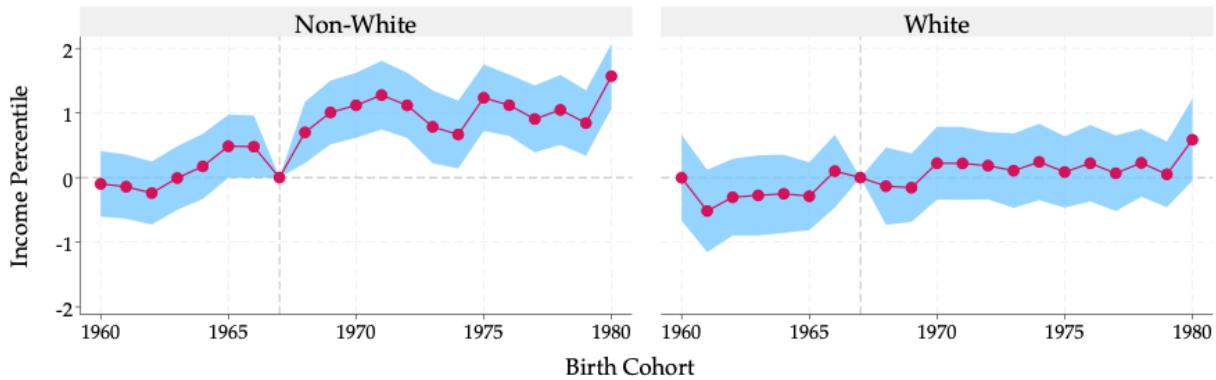


(b) Callaway and Sant'Anna (2021)

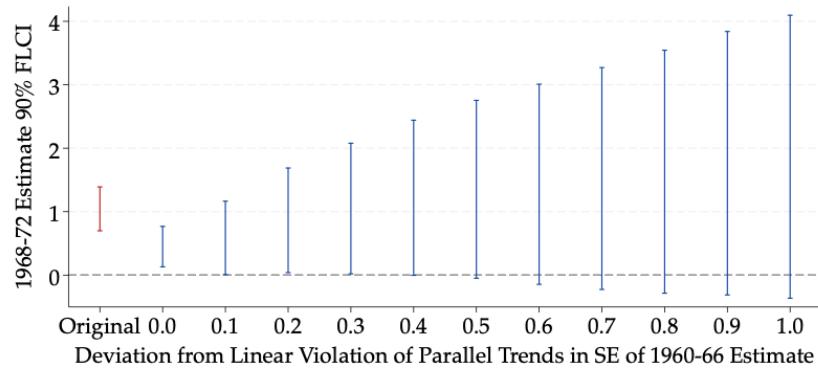


(c) de Chaisemartin and D'Haultfœuille (2020)

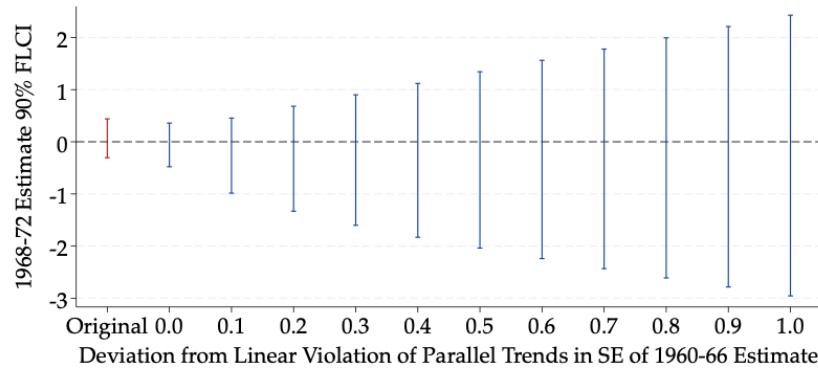
Figure D1: Continued



(d) Sun and Abraham (2021)



(e) Rambachan and Roth (2023): Non-White

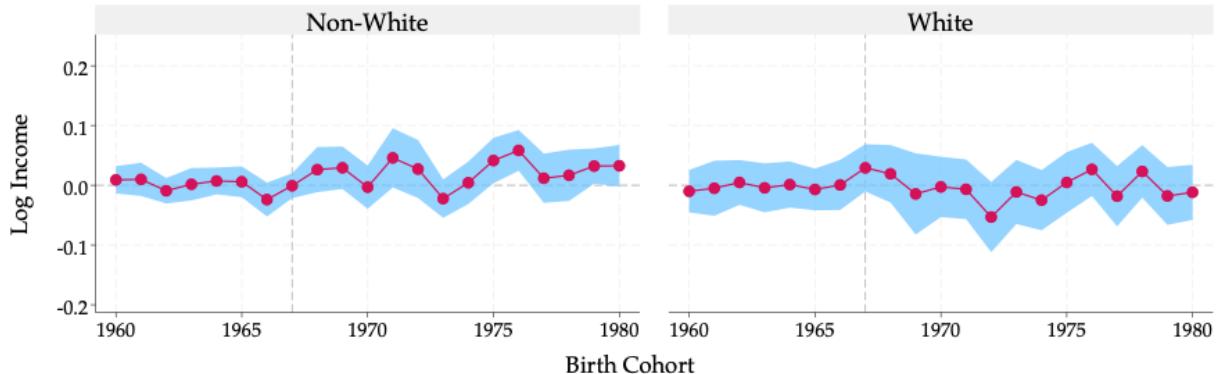


(f) Rambachan and Roth (2023): White

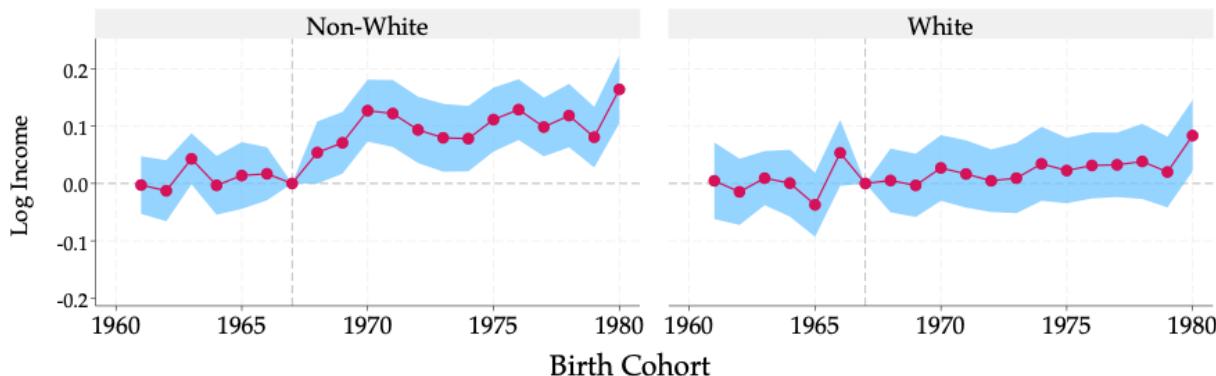
Notes: The top four panels show dynamic estimates for the respective subgroups when using a given estimator with 95% confidence intervals. The bottom two panels show 90% fixed length confidence intervals for the 1978-80 estimate given deviations from linear violations of parallel trends. Data are from the IPUMS 2010 census sample. In the top panel, the data are collapsed to the birth cohort-birth municipality level. All regressions include fixed effects for birth cohort and birth municipality, and those in the three bottom panels include state-by-year, sex, and racial category fixed effects. Standard errors are clustered by birth municipality.

D2. Log Income Results Using New Difference-in-Differences Techniques

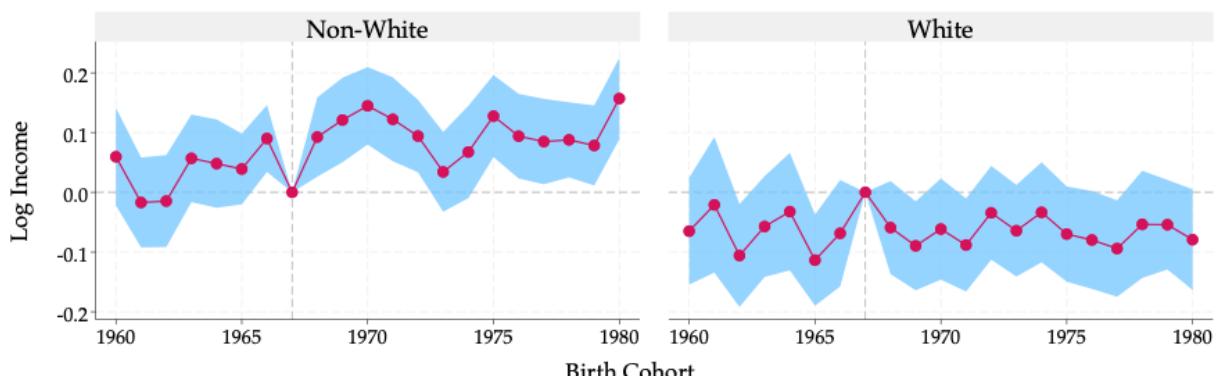
Figure D2: Log Income Results Using New Estimators [23]



(a) Arkhangelsky et al. (2021)

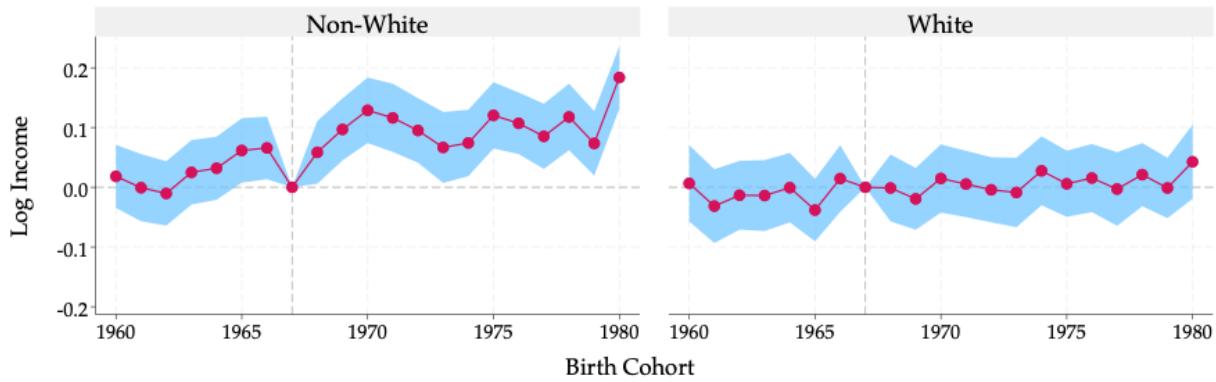


(b) Callaway and Sant'Anna (2021)

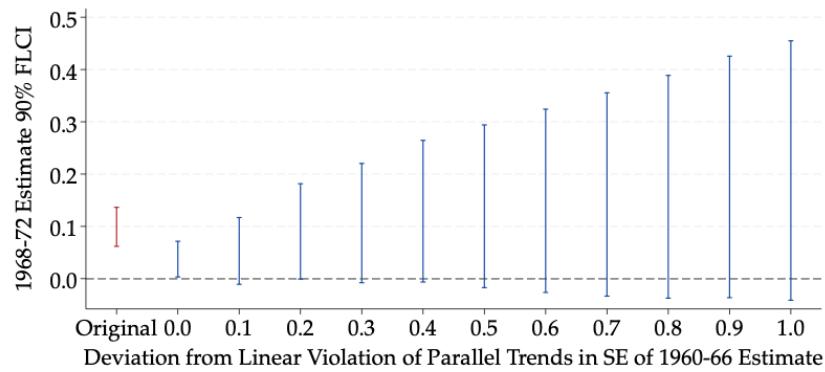


(c) de Chaisemartin and D'Haultfœuille (2020)

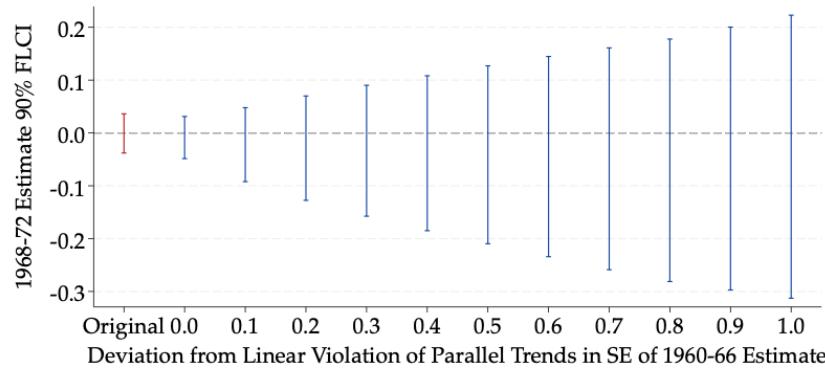
Figure D2: Continued



(d) Sun and Abraham (2021)



(e) Rambachan and Roth (2023): Non-White

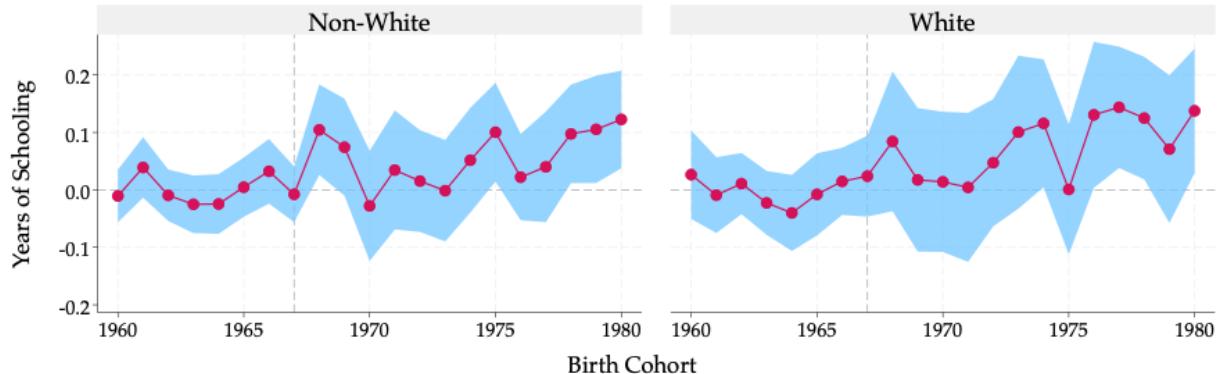


(f) Rambachan and Roth (2023): White

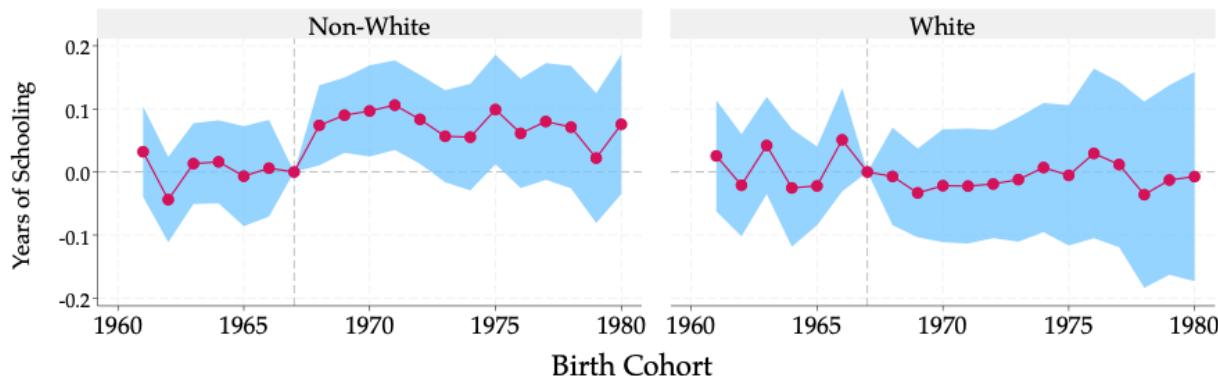
Notes: The top four panels show dynamic estimates for the respective subgroups when using a given estimator with 95% confidence intervals. The bottom two panels show 90% fixed length confidence intervals for the 1978-80 estimate given deviations from linear violations of parallel trends. Data are from the IPUMS 2010 census sample. In the top panel, the data are collapsed to the birth cohort-birth municipality level. All regressions include fixed effects for birth cohort and birth municipality, and those in the three bottom panels include state-by-year, sex, and racial category fixed effects. Standard errors are clustered by birth municipality.

D3. Years of Schooling Results Using New Difference-in-Differences Techniques

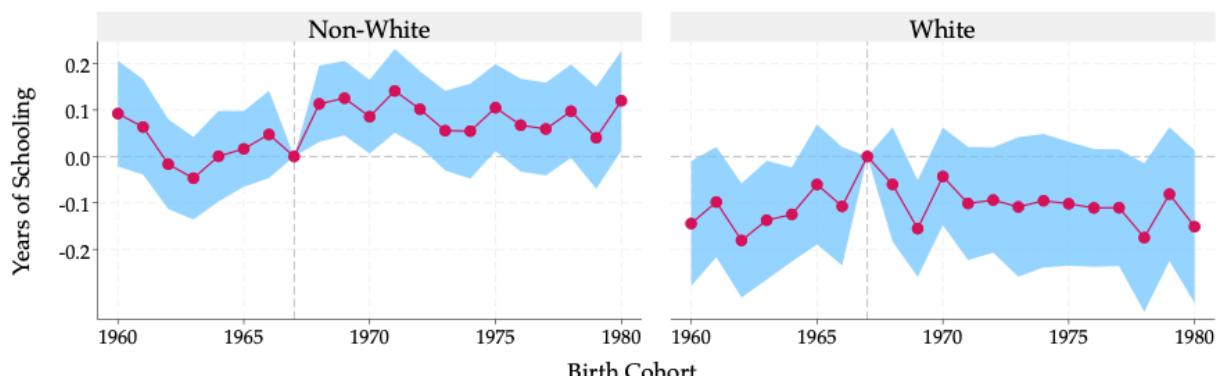
Figure D3: Years of Schooling Results Using New Estimators [23]



(a) Arkhangelsky et al. (2021)

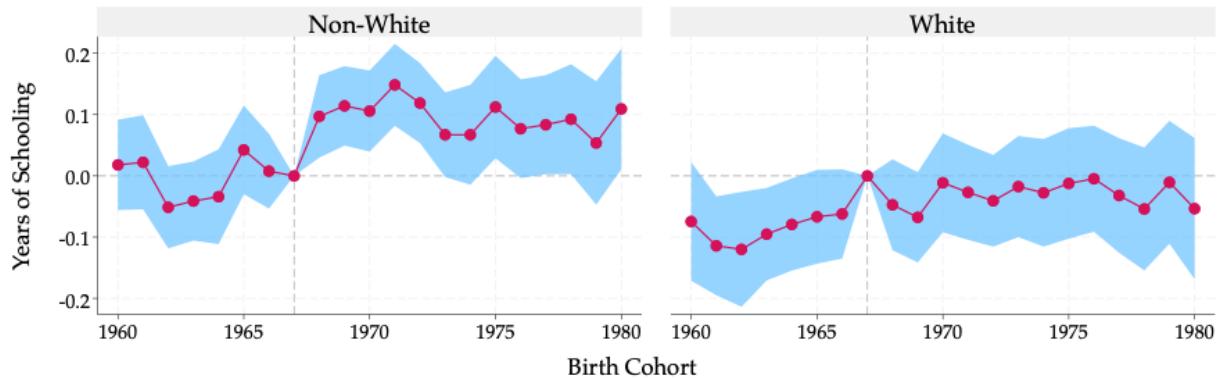


(b) Callaway and Sant'Anna (2021)

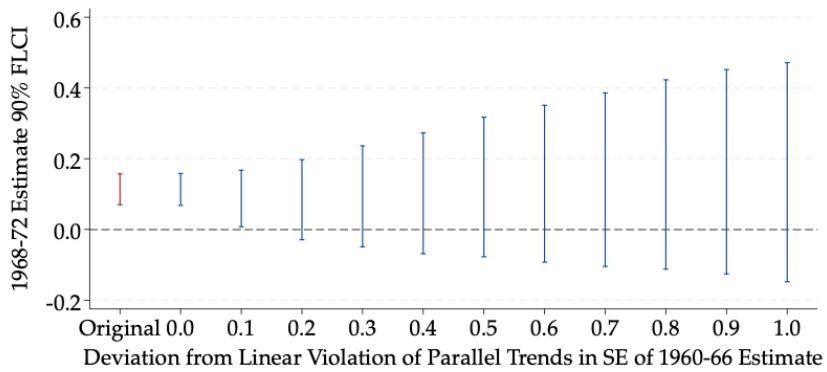


(c) de Chaisemartin and D'Haultfœuille (2020)

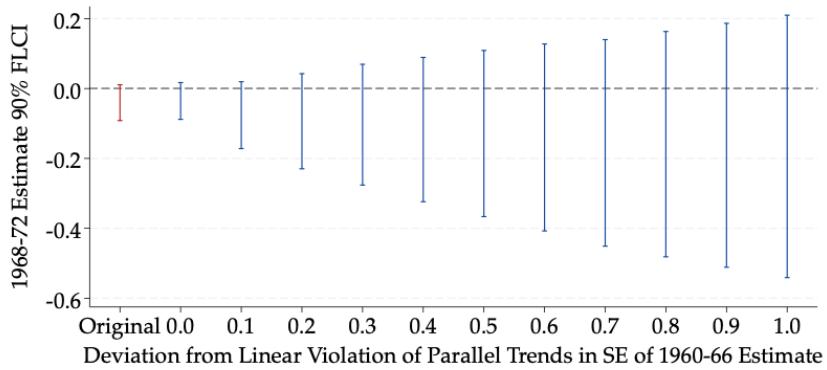
Figure D3: Continued



(d) Sun and Abraham (2021)



(e) Rambachan and Roth (2023): Non-White

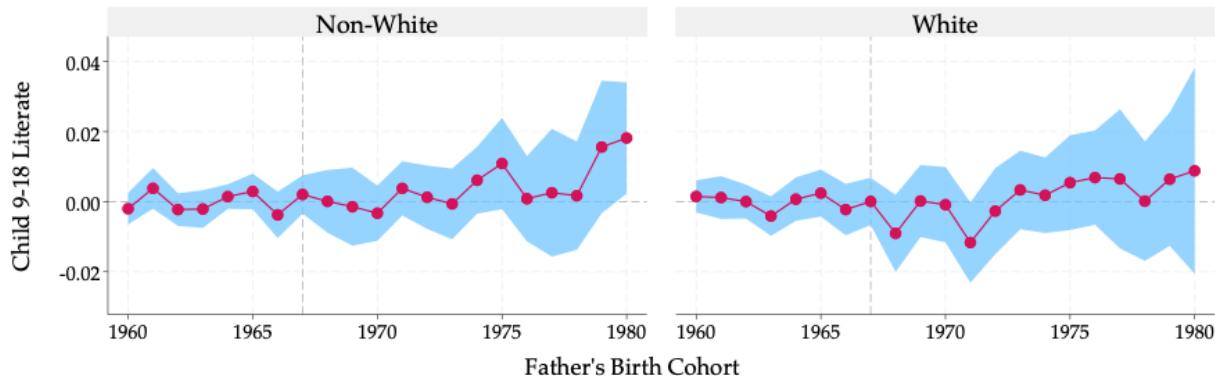


(f) Rambachan and Roth (2023): White

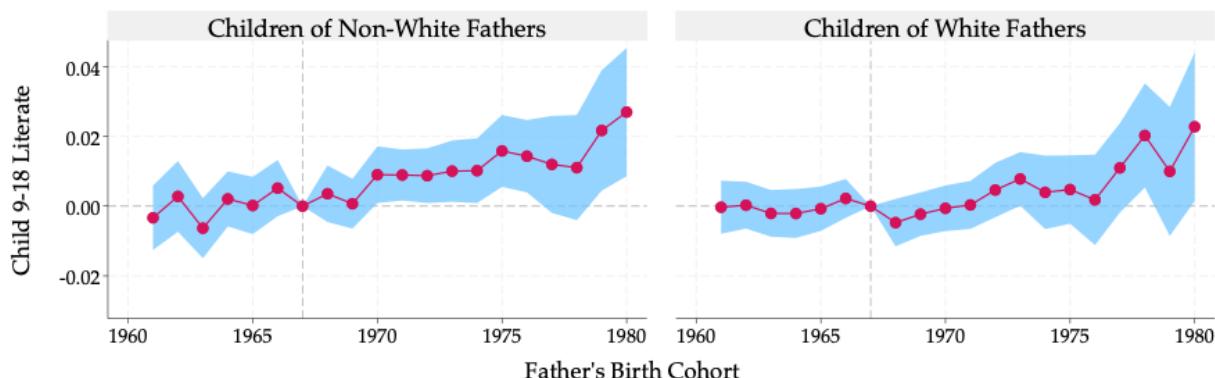
Notes: The top four panels show dynamic estimates for the respective subgroups when using a given estimator with 95% confidence intervals. The bottom two panels show 90% fixed length confidence intervals for the 1978-80 estimate given deviations from linear violations of parallel trends. Data are from the IPUMS 2010 census sample. In the top panel, the data are collapsed to the birth cohort-birth municipality level. All regressions include fixed effects for birth cohort and birth municipality, and those in the three bottom panels include state-by-year, sex, and racial category fixed effects. Standard errors are clustered by birth municipality.

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

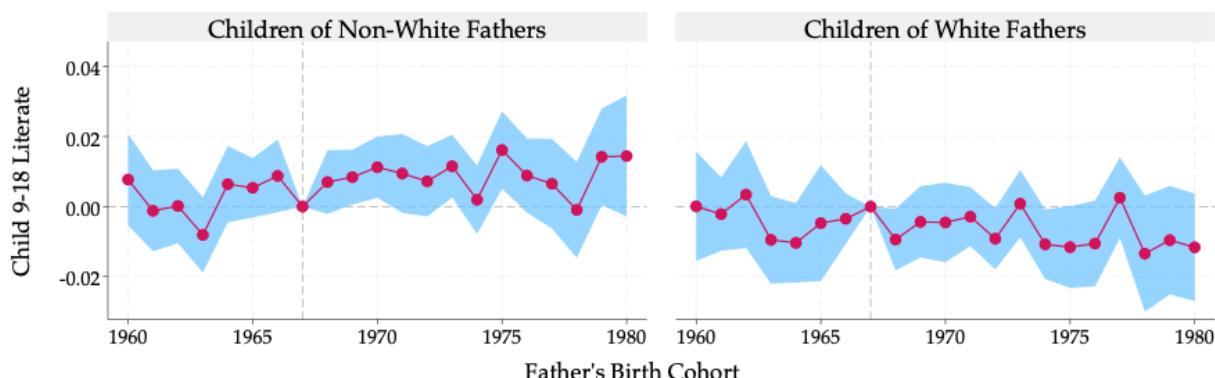
Figure D4: Next Generation Literacy Results Using New Estimators [23]



(a) Arkhangelsky et al. (2021)

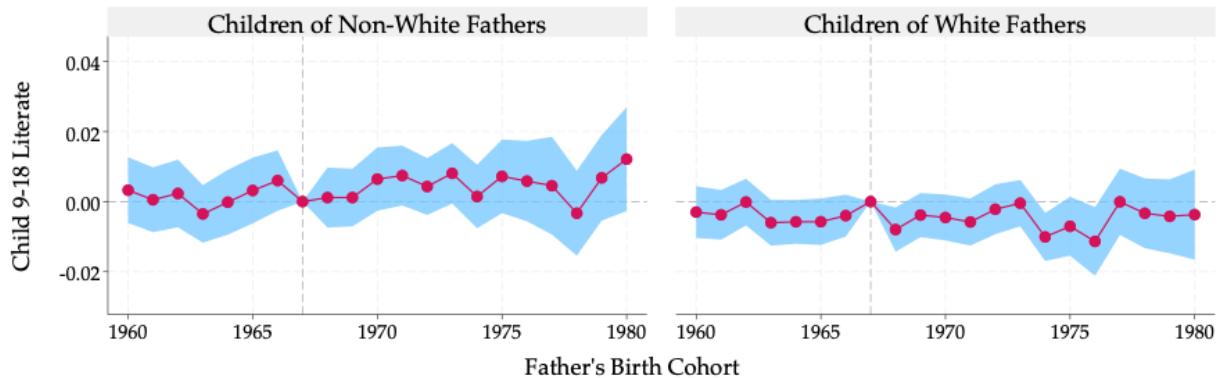


(b) Callaway and Sant'Anna (2021)

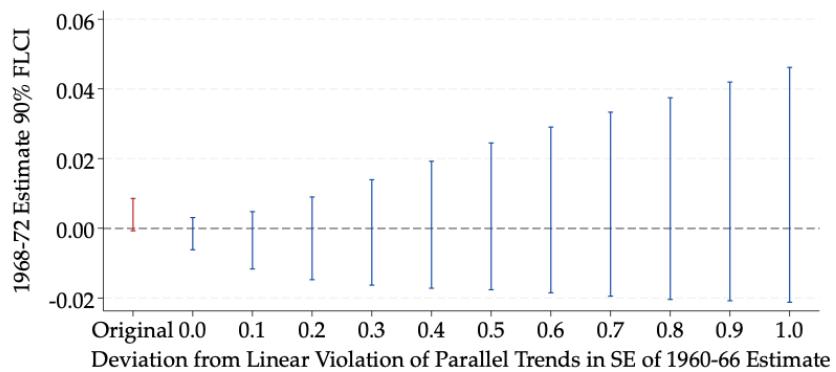


(c) de Chaisemartin and D'Haultfœuille (2020)

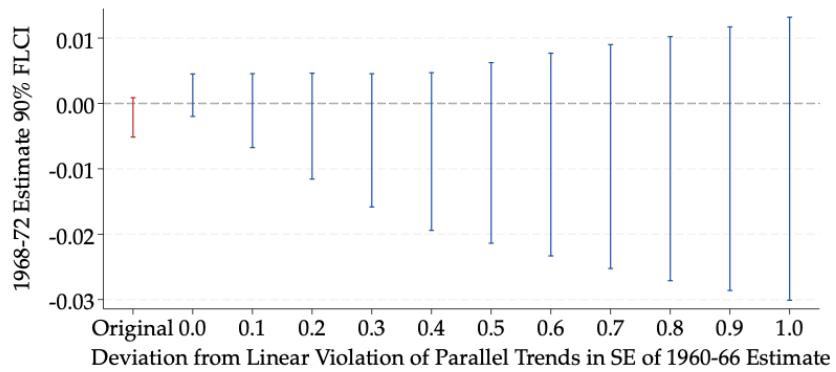
Figure D4: Continued



(d) Sun and Abraham (2021)



(e) Rambachan and Roth (2023): Non-White



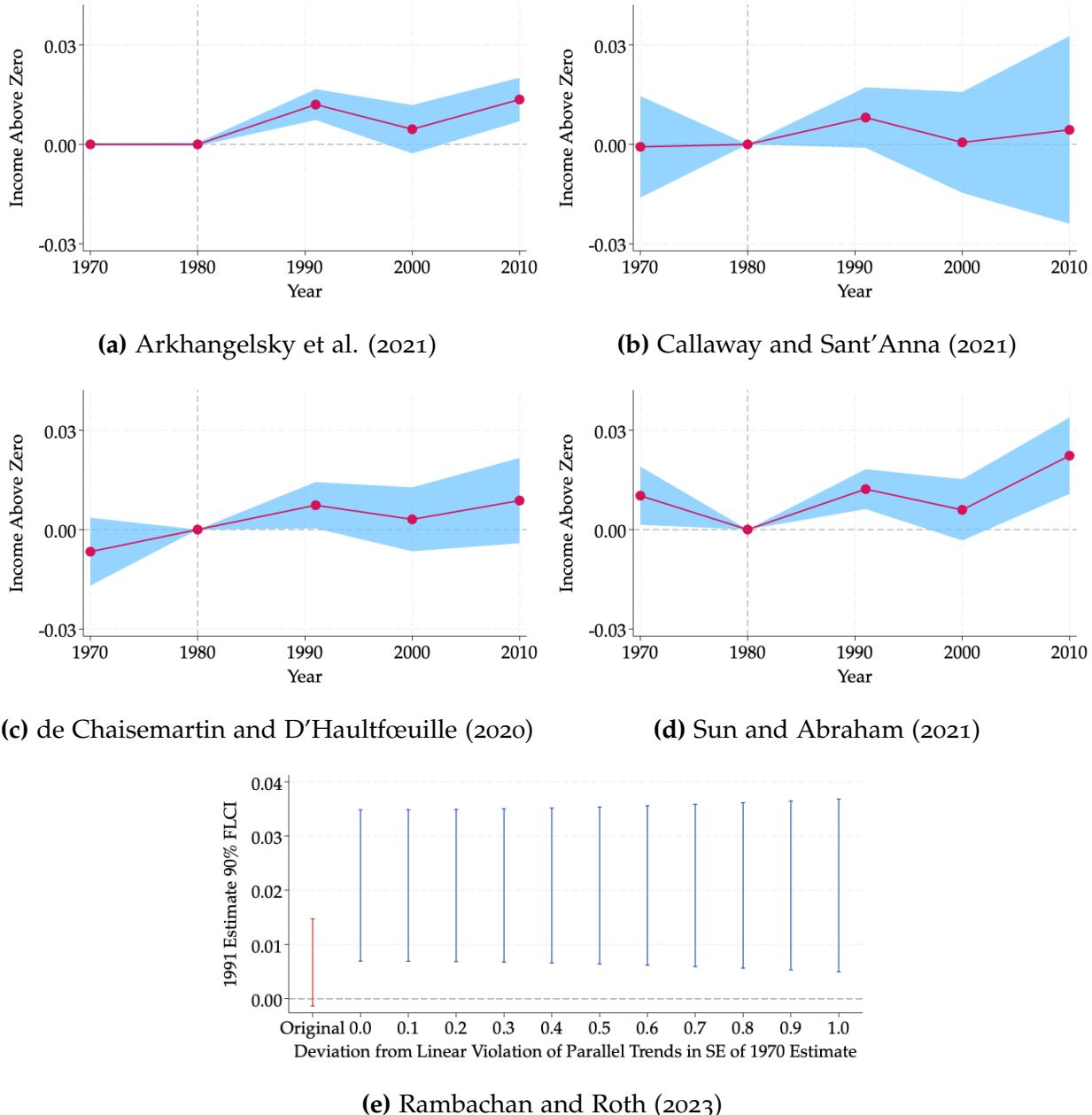
(f) Rambachan and Roth (2023): White

Notes: The top four panels show dynamic estimates for the respective subgroups when using a given estimator with 95% confidence intervals. The bottom two panels show 90% fixed length confidence intervals for the 1978-80 estimate given deviations from linear violations of parallel trends. Data are from the IPUMS 2010 census sample. In the top panel, the data are collapsed to the birth cohort-birth municipality level. All regressions include fixed effects for birth cohort and birth municipality, and those in the three bottom panels include state-by-year, sex, and racial category fixed effects. Standard errors are clustered by birth municipality.

Appendix E. Additional Figures: Municipality-Level Mechanisms

E1. Income Above Zero Results Using New Difference-in-Differences Techniques

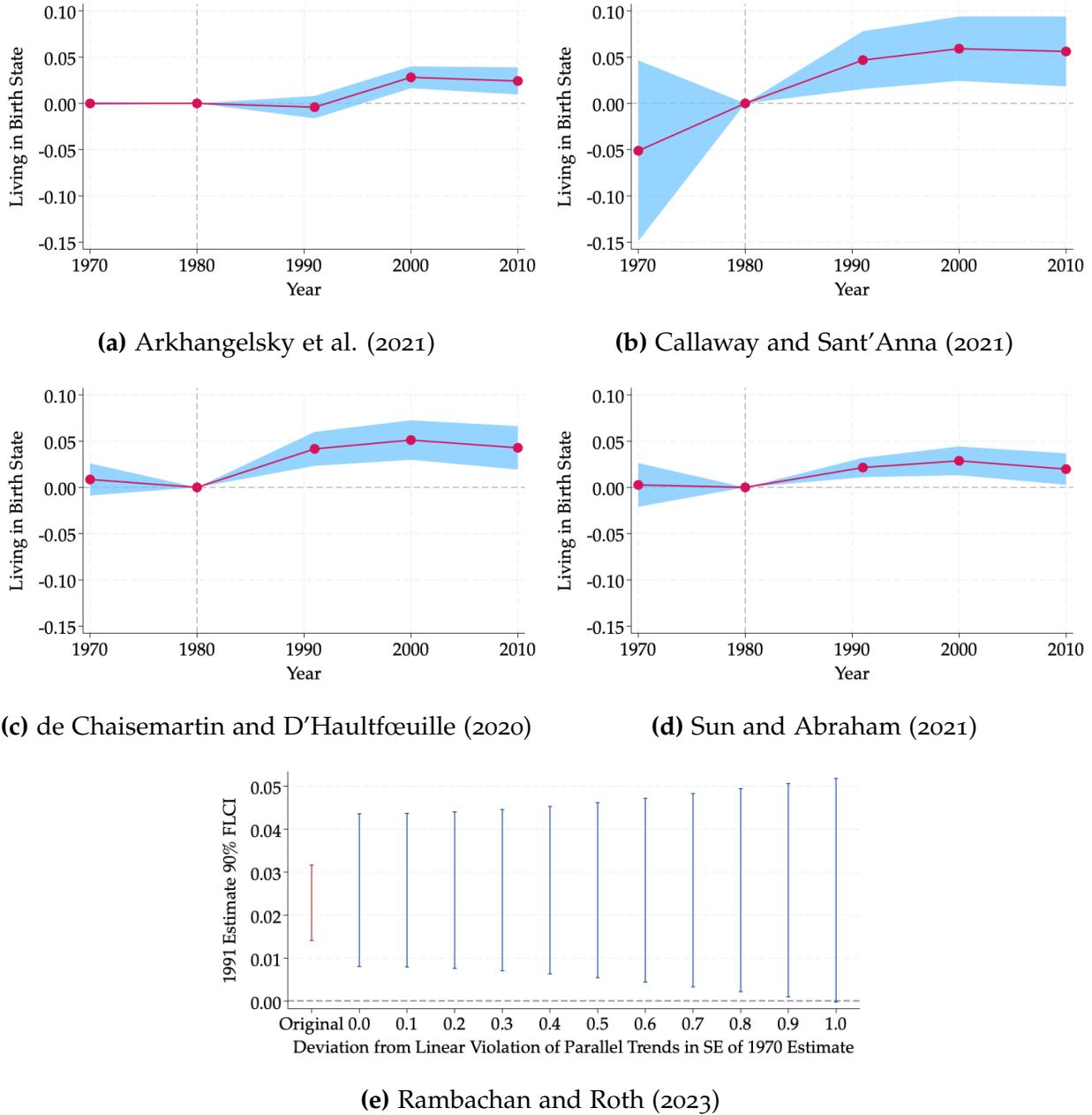
Figure E1: Income Above Zero Results Using New Estimators [25]



Notes: Observations are municipality-years. Data are from Ipeadata. The top four panels show estimates when using a given procedure with 95% confidence intervals. The bottom panel shows 90% fixed length confidence intervals for the 2010 estimate given deviations from linear violations of parallel trends. Regressions include year, municipality, and state-year fixed effects. Standard errors are clustered by municipality.

E2. Living in Birth State Results Using New Difference-in-Differences Techniques

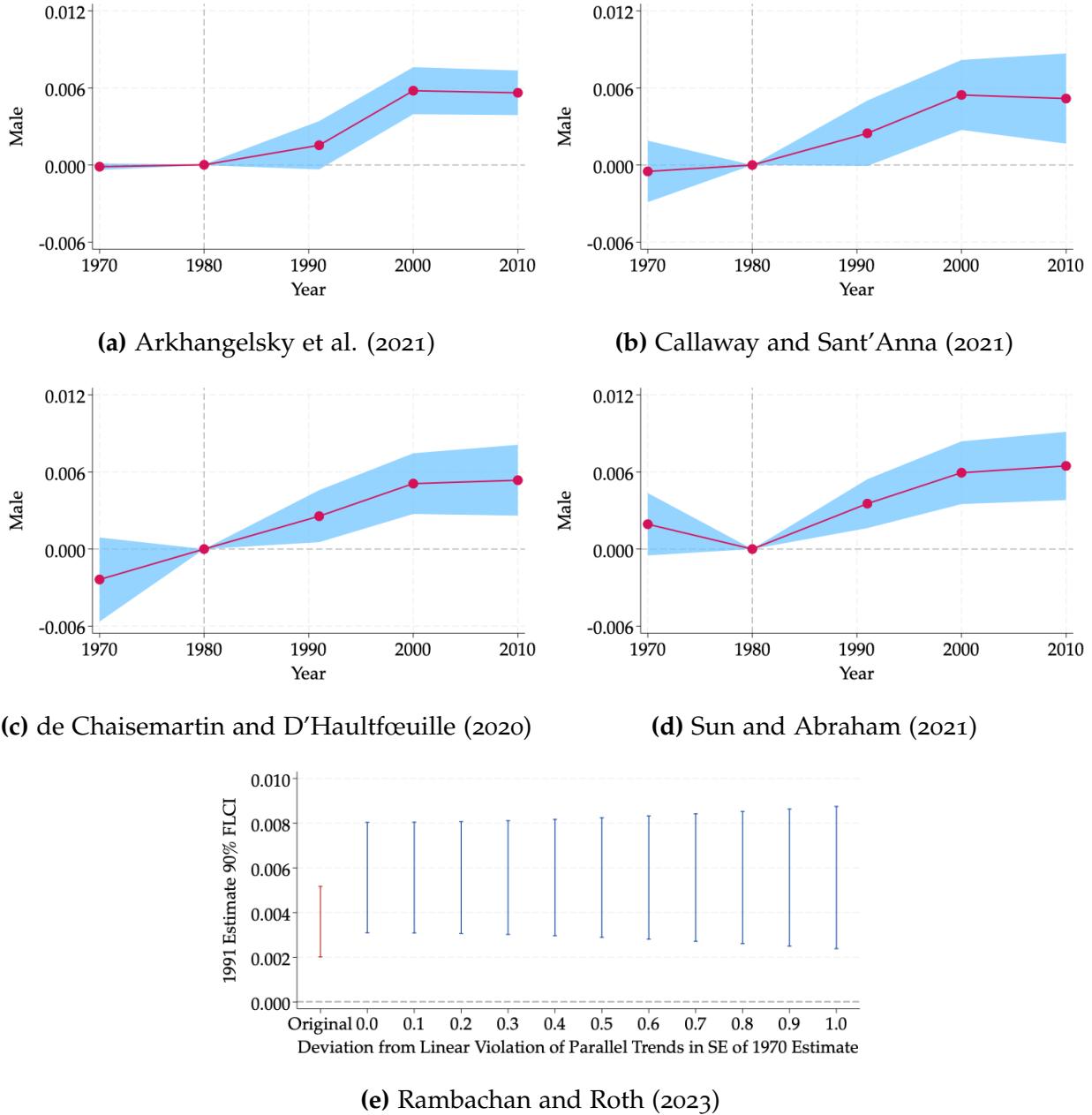
Figure E2: Living in Birth State Results Using New Estimators [25]



Notes: Observations are municipality-years. Data are from Ipeadata. The top four panels show estimates when using a given procedure with 95% confidence intervals. The bottom panel shows 90% fixed length confidence intervals for the 2010 estimate given deviations from linear violations of parallel trends. Regressions include year, municipality, and state-year fixed effects. Standard errors are clustered by municipality.

E3. Male Share Results Using New Difference-in-Differences Techniques

Figure E3: Male Share Results Using New Estimators [25]

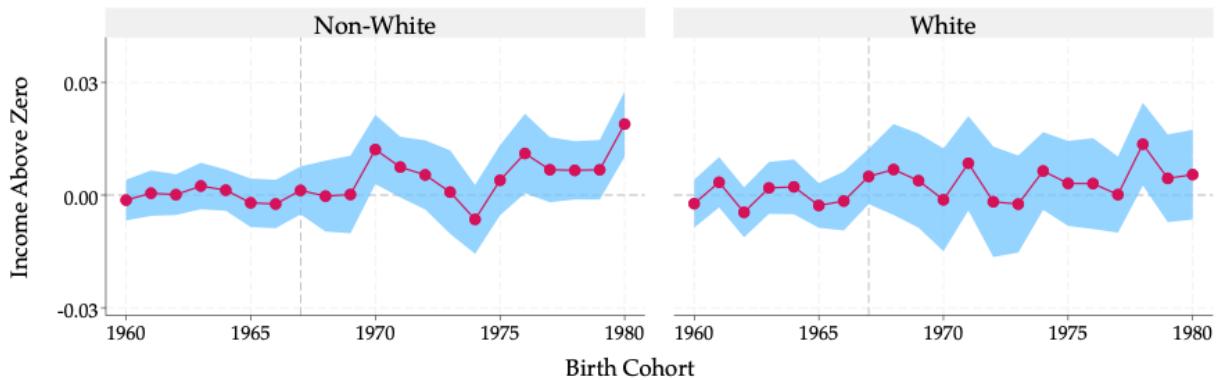


Notes: Observations are municipality-years. Data are from Ipeadata. The top four panels show estimates when using a given procedure with 95% confidence intervals. The bottom panel shows 90% fixed length confidence intervals for the 2010 estimate given deviations from linear violations of parallel trends. Regressions include year, municipality, and state-year fixed effects. Standard errors are clustered by municipality.

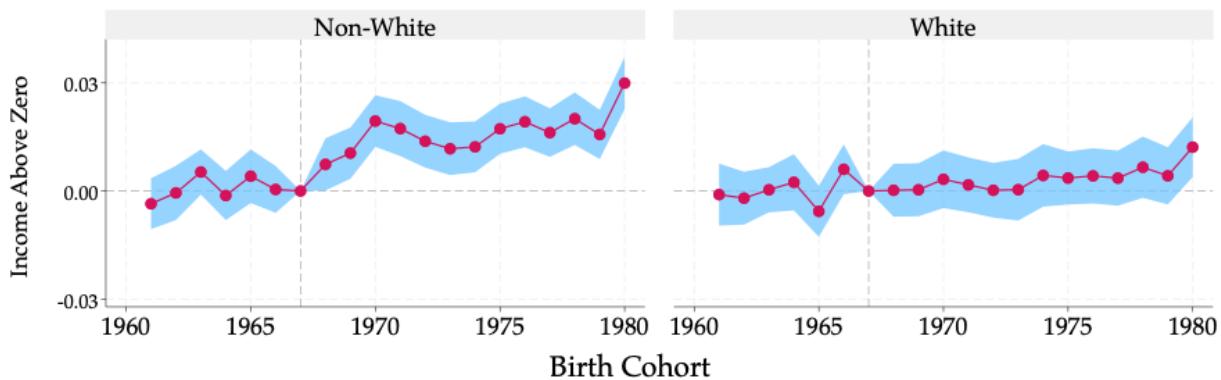
Appendix F. Additional Figures: Individual-Level Mechanisms

F1. Income Above Zero Results Using New Difference-in-Differences Techniques

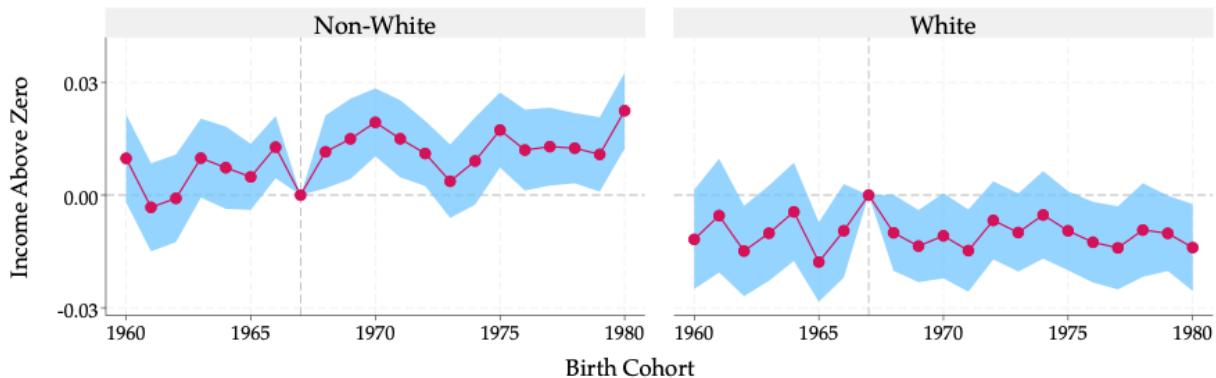
Figure F1: Income Above Zero Results Using New Estimators [29]



(a) Arkhangelsky et al. (2021)

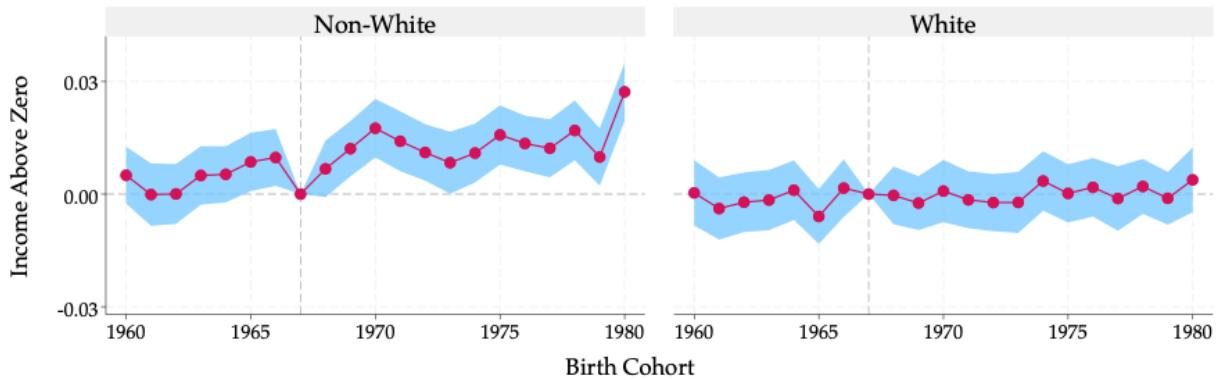


(b) Callaway and Sant'Anna (2021)

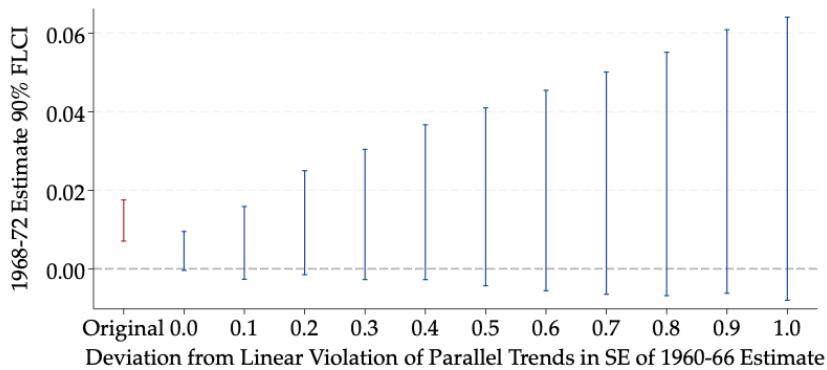


(c) de Chaisemartin and D'Haultfœuille (2020)

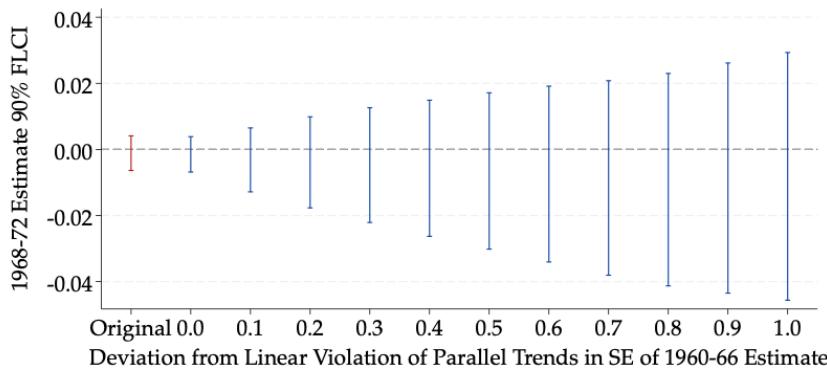
Figure F1: Continued



(d) Sun and Abraham (2021)



(e) Rambachan and Roth (2023): Non-White

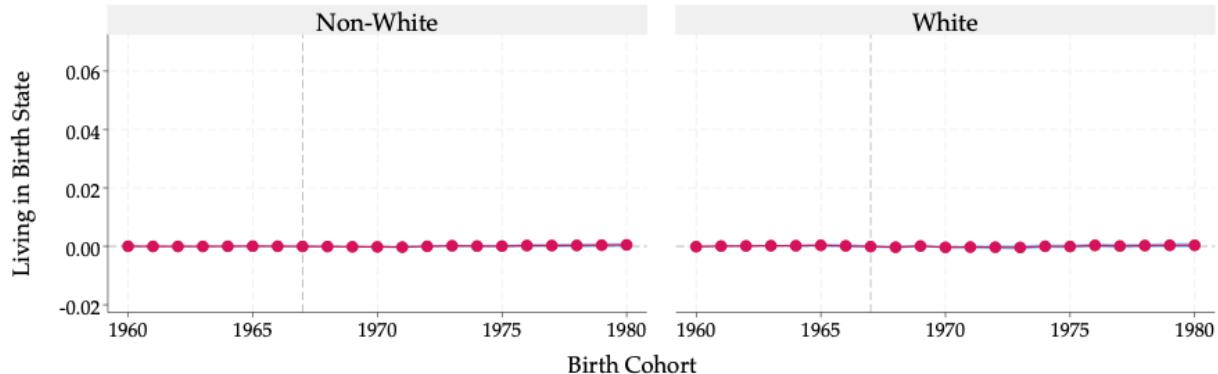


(f) Rambachan and Roth (2023): White

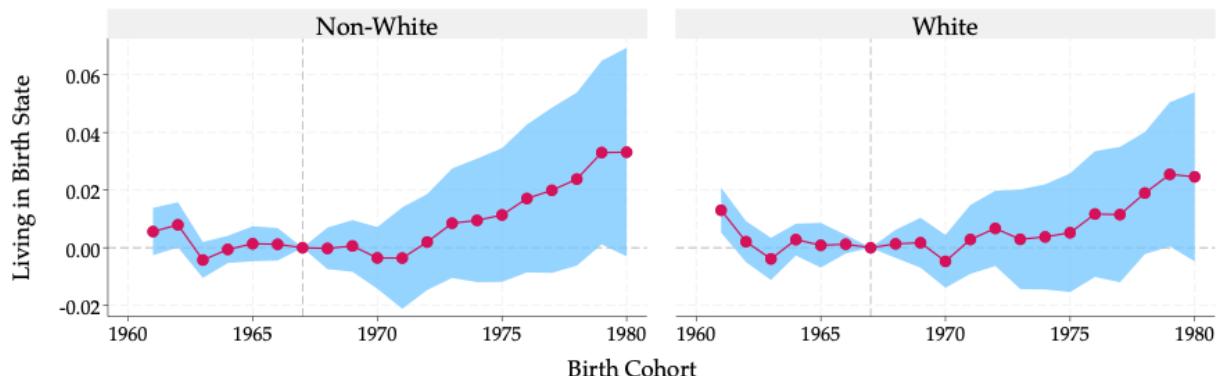
Notes: The top four panels show dynamic estimates for the respective subgroups when using a given estimator with 95% confidence intervals. The bottom two panels show 90% fixed length confidence intervals for the 1978-80 estimate given deviations from linear violations of parallel trends. Data are from the IPUMS 2010 census sample. In the top panel, the data are collapsed to the birth cohort-birth municipality level. All regressions include fixed effects for birth cohort and birth municipality, and those in the three bottom panels include state-by-year, sex, and racial category fixed effects. Standard errors are clustered by birth municipality.

F2. Living in Birth State Results Using New Difference-in-Differences Techniques

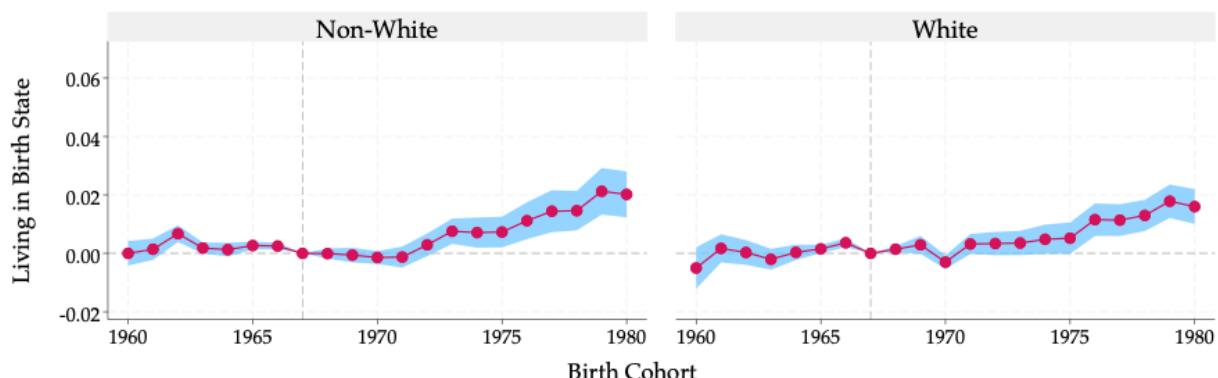
Figure F2: Living in Birth State Results Using New Estimators [29]



(a) Arkhangelsky et al. (2021)

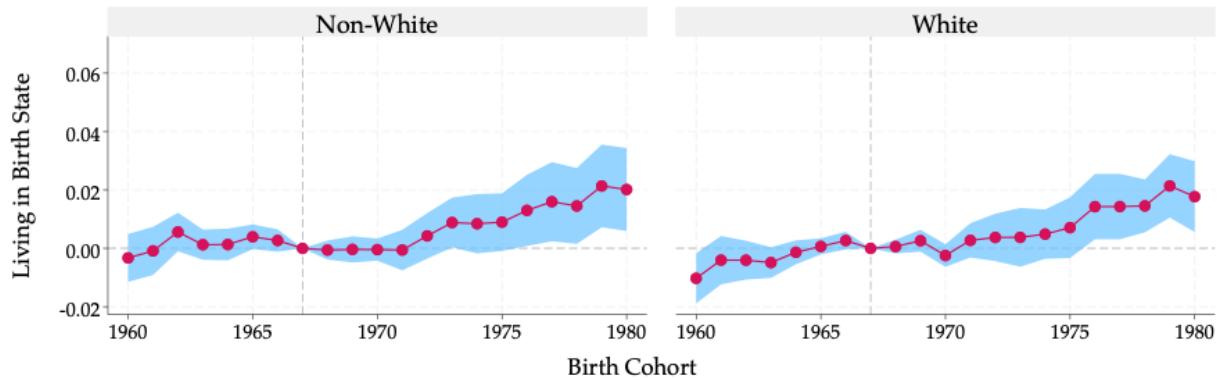


(b) Callaway and Sant'Anna (2021)

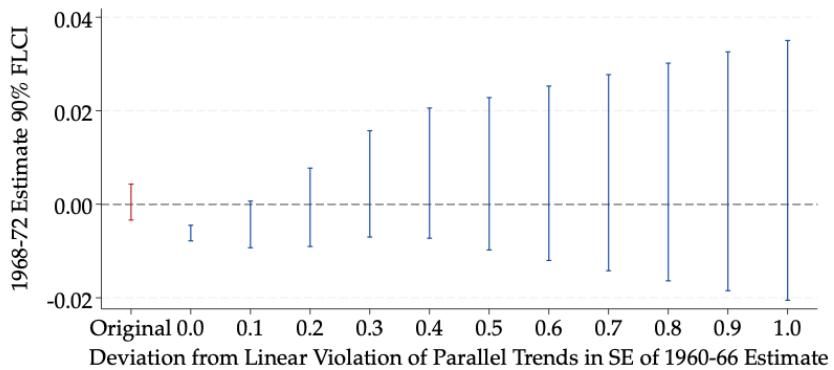


(c) de Chaisemartin and D'Haultfœuille (2020)

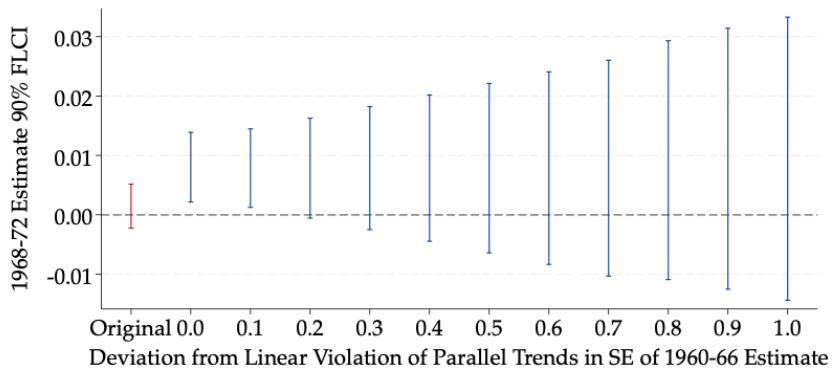
Figure F2: Continued



(d) Sun and Abraham (2021)



(e) Rambachan and Roth (2023): Non-White



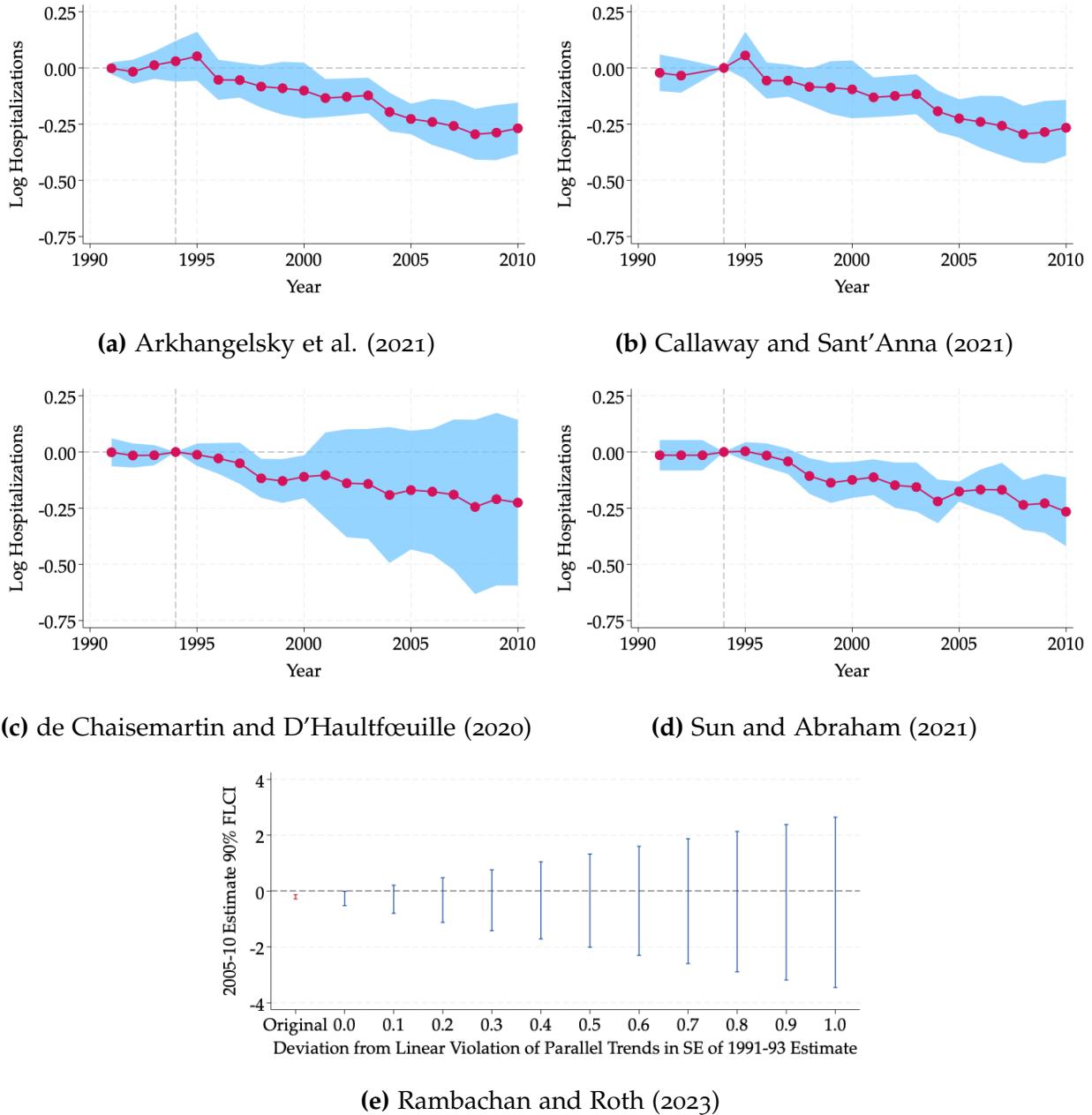
(f) Rambachan and Roth (2023): White

Notes: The top four panels show dynamic estimates for the respective subgroups when using a given estimator with 95% confidence intervals. The bottom two panels show 90% fixed length confidence intervals for the 1978-80 estimate given deviations from linear violations of parallel trends. Data are from the IPUMS 2010 census sample. In the top panel, the data are collapsed to the birth cohort-birth municipality level. All regressions include fixed effects for birth cohort and birth municipality, and those in the three bottom panels include state-by-year, sex, and racial category fixed effects. Standard errors are clustered by birth municipality.

Appendix G. Additional Figures: Health Mechanisms

G1. Hospitalization Results Using New Difference-in-Differences Techniques

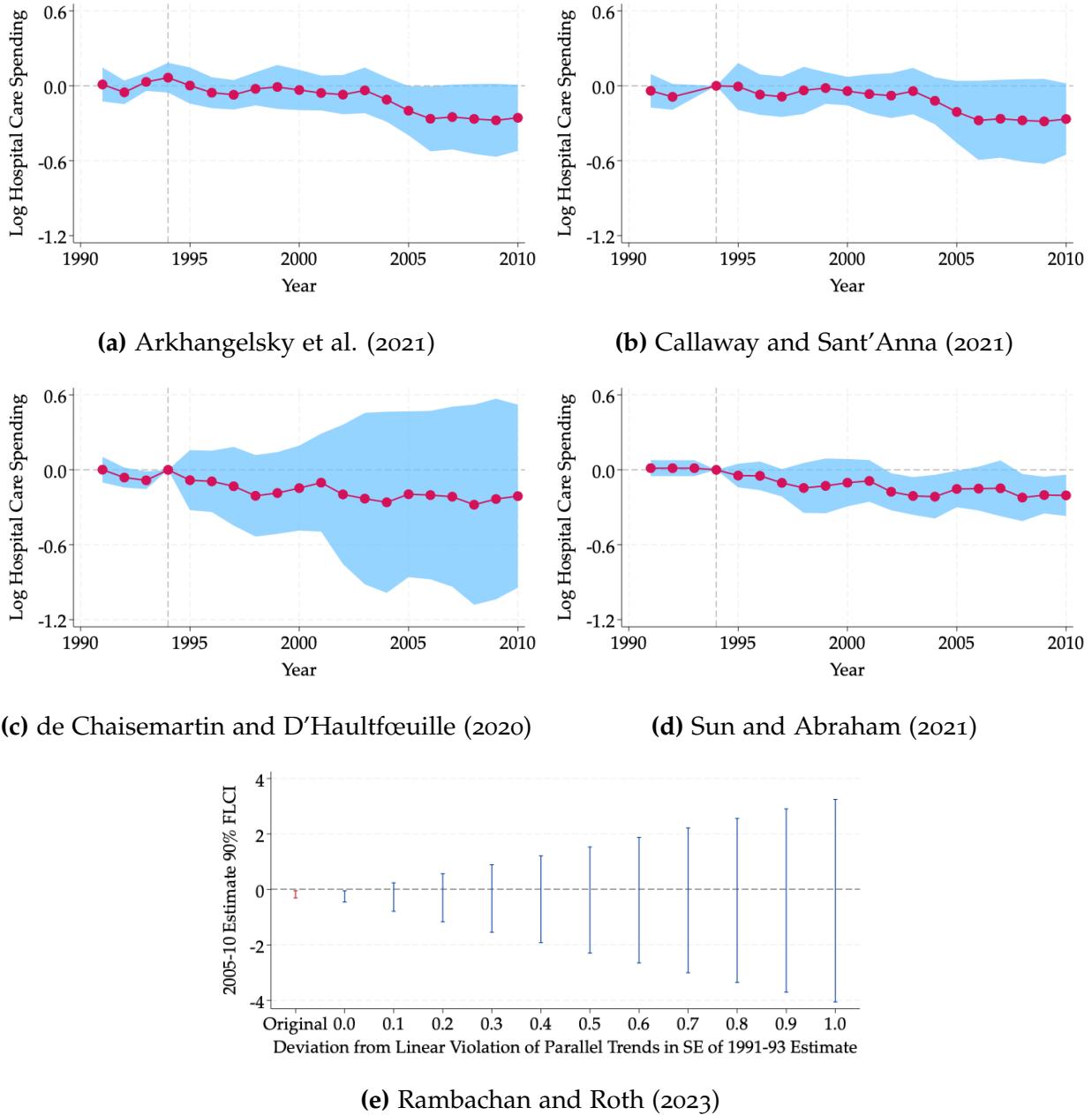
Figure G1: Hospitalization Results Using New Estimators [34]



Notes: Observations are year-state-disease categories. Graphs show dynamic estimates with 95% confidence intervals. Data are from DATASUS. All regressions include fixed effects for state-year, year-disease category, and state-disease category, and those in the bottom three panels include region-year-disease category fixed effects. Standard errors are clustered by state.

G2. Hospital Care Spending Results Using New Difference-in-Differences Techniques

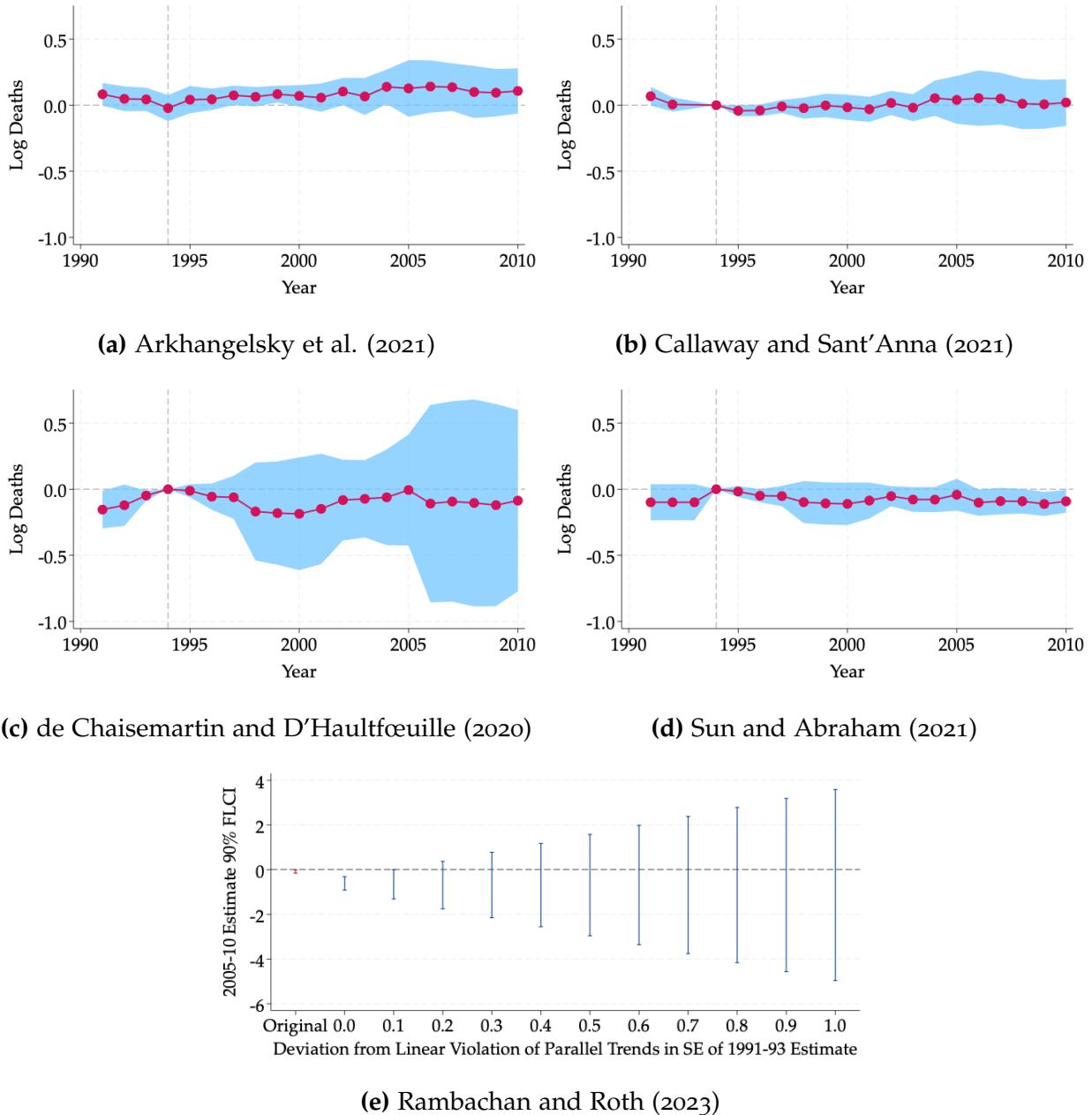
Figure G2: Hospital Care Spending Results Using New Estimators [34]



Notes: Observations are year-state-disease categories. Graphs show dynamic estimates with 95% confidence intervals. Data are from DATASUS. All regressions include fixed effects for state-year, year-disease category, and state-disease category, and those in the bottom three panels include region-year-disease category fixed effects. Standard errors are clustered by state.

G3. Death Results Using New Difference-in-Differences Techniques

Figure G3: Death Results Using New Estimators [34]

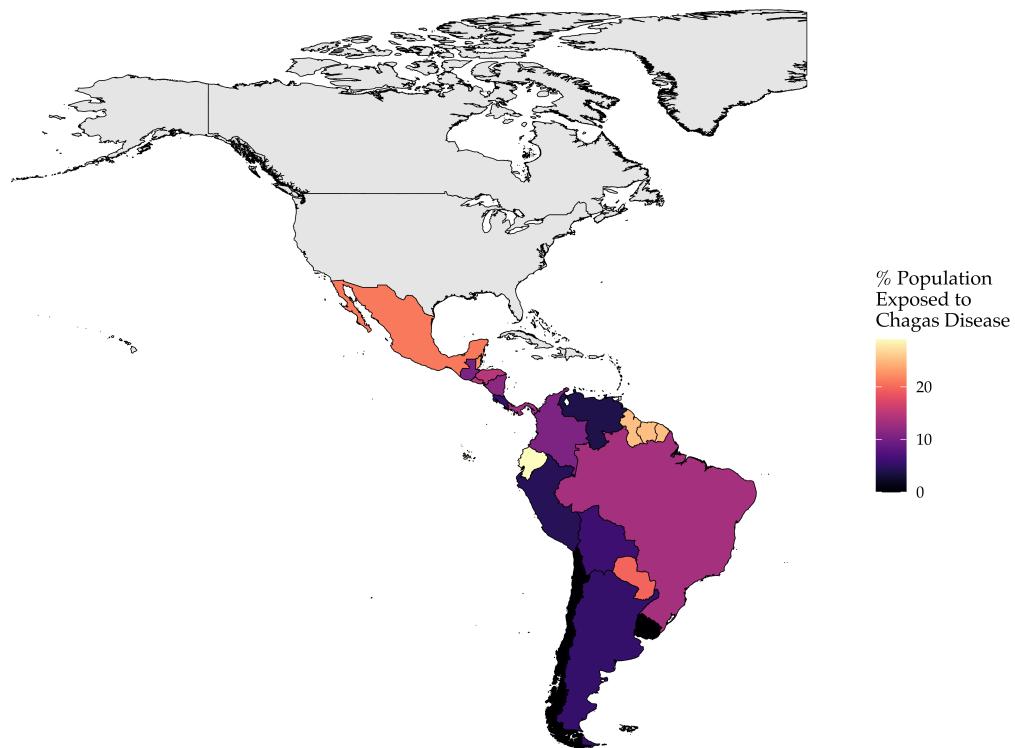


Notes: Observations are year-state-disease categories. Graphs show dynamic estimates with 95% confidence intervals. Data are from DATASUS. All regressions include fixed effects for state-year, year-disease category, and state-disease category, and those in the bottom three panels include region-year-disease category fixed effects. Standard errors are clustered by state.

Appendix H. Additional Figures: Extrapolation

H1. Estimated Population Exposure to Chagas Disease

Figure H1: Estimated Population Exposure to Chagas Disease [38]



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