

An Antidote for Despair: The Effect of Highly Active Antiretroviral Therapy (HAART) on Suicide Rates*

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Abstract

The introduction of Highly Active Antiretroviral Therapy (HAART) in 1995, transformed the prognosis of an HIV infection from a death sentence to a manageable chronic health condition. Using a difference-in-differences and triple-difference strategy, this paper exploits spatial and demographic variation in HIV incidence at the time HAART treatment was introduced and finds that, in addition to reducing HIV/AIDS deaths, the introduction of HAART led to a disproportionate decrease in suicide rates for men aged 25 to 44. Estimates suggest that HAART saved approximately 500 men aged 25 to 44 from suicide each year following its introduction.

JEL codes: D63, I12, J15, N32

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1 Introduction

The HIV/AIDS crisis represented a particular set of mental health challenges for at-risk communities in the US: heightened homophobia, increased pressure to come out, and the risk of dying from a highly infectious disease. Heightened levels of suicide risk have been found among HIV positive individuals and LGBTQ youth (who are also more likely to contract HIV). Prior to the introduction of effective combination drug therapies, testing positive for HIV was a death sentence. In 1995, the US Food and Drug Administration (FDA) approved the use of Saquinavir in combination with other nucleoside analog medications. This combination proved to be highly effective and marked a new era in combination drug therapy treatments that became known as Highly Active Antiretroviral Therapy (HAART). HAART led to substantial reductions in HIV-related morbidity and mortality and resulted in significant increases in life expectancies of HIV-positive individuals.

Using publicly available mortality data from the US Vital Statistics, this paper finds that the introduction of HAART, not only reduced HIV/AIDS mortality but also saved approximately 500 men aged 25 to 44 from suicide each year following its introduction ([NCHS, 1990-2002](#)). There are several ways the introduction of HAART can affect suicide rates for at-risk groups. First, HAART could reduce suicide rates by improving mental health outcomes for HIV positive individuals. There is a significant body of research that highlights the negative mental health effects of testing positive for HIV ([Carrico 2010](#); [Kalichman et al. 2000](#); [Schlebusch and Govender 2015](#); [Pelton et al. 2021](#)). Suicide ideation and self-harm are significantly more common among those who test positive for HIV relative to other groups. Given the improved prognosis for people living with HIV infections after the introduction of HAART, we expect lower levels of suicide risk among HIV positive individuals post-HAART. Secondly, HAART reduced the consequences of testing positive among those who fear infection

but are not yet infected with HIV. We see that more people got tested for HIV after HAART, suggesting a potential anticipated negative mental health toll a positive HIV test can accompany without treatment ([Kellerman et al. 2002](#)). Although HAART is not a cure for the infection, we expect that the treatment would ameliorate heightened fears of death among populations at increased risk of contracting infection, thus improving their mental health outcomes. Third, improved physical and mental health outcomes in the community may have spillover effects on those who are HIV negative. Some reports suggest that the HIV/AIDS crisis was accompanied by rising homophobia and stigma. Gay men routinely mourned the deaths of their friends and fellow community members.¹ Additionally, some evidence indicates that the introduction of HAART coincided with increased acceptance of same-sex couples in areas heavily affected by the HIV crisis. [Fernández et al. \(2019\)](#) uses data from the General Social Survey to demonstrate that states with higher proportions of HIV-positive individuals experienced a surge in acceptance during this period. Several studies find that decreasing homophobia decreases the risk of suicide among LGBTQ populations ([Raifman et al. 2017](#); [Tan et al. 2017](#)).

Despite the clear mechanisms through which the introduction of HAART can impact the mental health outcomes of at-risk individuals, to the best of my knowledge, no study estimates the effects of HAART on suicide rates in the United States. This paper furnishes plausibly causal estimates by exploiting spatial variations in HIV/AIDS incidence prior to the introduction of HAART and comparing suicide rates across the distribution of pre-HAART HIV/AIDS incidence, before and after the introduction of HAART.

I use a difference-in-differences event study design to estimate the effects on the group that was most affected by the HIV/AIDS crisis (men aged 25 to 44) across

¹In his 1995 book, [Odets](#) highlights the emotional and psychological impact of AIDS on the lives of HIV-negative gay men

the distribution of pre-HAART HIV/AIDS incidence. I find that counties with a standard deviation higher preexisting HIV incidence rate experiences a 0.9 per 100,00 population decrease in suicides for men aged 25 to 44. This estimate suggests that the introduction of HAART saved approximately 500 men aged 25 to 44 from suicide each year after its introduction.

Expanding my analysis to groups less affected by the introduction of HAART allows me to test for potential spillover effects and provides important control groups. My strategy finds that the introduction of HAART had little or no effect on suicide rates for groups with lower rates of HIV incidence (women, older men aged 65+, and younger men aged 19 or younger). I also employ a triple-difference strategy, where I difference out effects on the suicide rates of less affected groups and find similar estimates, which lends credence to my findings.

I conduct several robustness checks which show that the documented effects are driven by the introduction of HAART. Thereafter, I break down effects by race and find larger effects for HIV-affected white populations compared to HIV-affected non-white populations. Additionally, I examine the welfare implications of my research findings, demonstrating that suicides were not simply a substitute for HIV/AIDS deaths within the population that influenced my results. I highlight that improvements in mental health had non trivial implications for the welfare of the affected population.

My findings contribute to a growing literature exploring the specific mental health challenges experienced by the LGBTQ community and HIV-positive individuals. Sexual minorities experience elevated levels of suicide risk ([Björkenstam et al. 2016](#); [Cochran and Mays 2015](#); [Hottes et al. 2016](#); [Raifman et al. 2017](#); [Clark et al. 2020b](#)). Testing positive for HIV is also associated with heightened suicidal ideation and mortality ([Carrico 2010](#); [Kalichman et al. 2000](#); [Schlebusch and Govender 2015](#); [Pelton et al. 2021](#)).

The effect of HAART on HIV-associated morbidity and mortality is well documented (Egger et al. 2002; Keiser et al. 2004). Previous studies have also estimated the effects of HAART on health disparities, labor supply, risky sex, and health-related quality of life indicators (Hamilton et al. 2021; Chan et al. 2016; Pelton et al. 2016; Lakdawalla et al. 2006; Liu et al. 2006). More relevant to this paper, using data from a longitudinal study conducted in Australia, Judd et al. 2000 finds that the introduction of HAART was accompanied by a fall in depressive symptoms amongst HIV positive individuals. Similarly, Keiser et al. 2010 makes use of a cohort of HIV positive individuals who are part of a longitudinal study conducted in Switzerland to find that individuals who are part of the study experience a greater fall in suicides after the introduction HAART than the general population. Although the study find compelling evidence of differential trends in suicides for HIV positive individuals relative to the general population before and after the introduction of HAART, it only observes a subset of HIV positive individuals in Switzerland and does not make causal claims whereas my design makes use of the distribution of HIV incidence across the United States to estimate the causal effect of HAART on suicide. To the best of my knowledge, no current study estimates the effect of HAART on suicide rates for at-risk populations in the United States.

This study has important implications for measuring the effects of the HIV/AIDS epidemic and HAART treatment. With over 1 million people living with HIV in the US and over 40 million worldwide, understanding the mental health challenges of those living with HIV and those at risk of contracting the virus is important. It is estimated that a total of 700,000 people have died of HIV/AIDS in the United States.² This study implies that HIV/AIDS affects mortality in ways other than just HIV/AIDS mortality. Measures of total HIV/AIDS deaths may undercount the

²Estimate obtained from KKF: <https://www.kff.org/hiv aids/fact-sheet/the-hiv aids-epidemic-in-the-united-states-the-basics/> last accessed August 24th, 2022

effect of the HIV/AIDS crisis. More generally, the findings warn us about the threat of chronic medical issues on mental health outcomes and the benefits of coming up with treatments. By only focusing on the direct impacts of treatment, we miss meaningful aspects of the benefits of innovations in medical treatments.

2 Background

In 1981, a mysterious new disease emerged that seemed to affect gay men in New York and California. Similar reports of gay men suffering from a previously unknown disease emerged in other parts of the country, and by the end of the year, the New York Times published an article titled “Rare Cancer Seen in 41 Homosexuals.”³ The number of new cases grew rapidly among gay men and the disease was termed the “gay plague”. Although HIV/AIDS also affects heterosexual men and women, the high prevalence of the virus among gay men resulted in many associating the virus with the gay community. Some called the virus “God’s Judgement Against Homosexuality.”⁴ In addition to bearing the brunt of new infections from this virus, gay men also faced heightened discrimination and homophobia in this period.

In the fall of 1982, the CDC defined the disease as Acquired Immune Deficiency Syndrome (AIDS) for the first time (AIDS is the final and most severe stage of the infection). It wasn’t until 1983 that scientists identified the virus that caused the development of AIDS, Human Immunodeficiency Virus (HIV). Little was known about the virus in its early years and the care of HIV/AIDS patients was largely palliative. Scientists struggled to make progress in combating the new virus and HIV/AIDS-related deaths continued to rise. Early estimates of total HIV/AIDS cases and deaths are unreliable because misreporting was common, but by 1987, an estimated 40,849

³The New York Times, July 3rd, 1981: <https://www.nytimes.com/1981/07/03/us/rare-cancer-seen-in-41-homosexuals.html>

⁴In his book, *After the Wrath of God*, Petro (2015) documents the role of religious leaders in shaping moral debates surrounding the AIDS crisis in the US.

people had died from the virus.⁵ Figure 1 depicts the recorded rise in cumulative HIV/AIDS deaths from 1981 to 2002.

HIV is transmitted through contact with bodily fluids of an HIV-positive individual with a detectable viral load. The most common forms of transmission are through intercourse and the sharing of drug injection equipment. By 1987, men who have sex with men accounted for over 72 percent of all AIDS cases.⁶ Without treatment, HIV usually progresses to AIDS (the final stage of the virus) in 5 to 10 years, and the life expectancy of an untreated AIDS patient is approximately 2 years (Poorolajal et al., 2016). Before the introduction of combination drug therapies, testing positive for the virus was a death sentence.

In 1987, the US Food and Drug Administration (FDA) approved Zidovudine (AZT) as the first medicine to treat HIV/AIDS. Although AZT experienced some success in increasing white blood cell counts among HIV/AIDS patients, the side effects of the medication proved unbearable for patients, and it experienced only moderate success in reducing the progression of the disease. HIV/AIDS deaths continued to rise through the 1980s and early 1990s.

HIV/AIDS patients and activists grew weary and impatient about the lack of advances made to come up with an effective treatment. Many organized in protest of the government's perceived inaction in combating this crisis.⁷ In response to growing pressures to address the crisis, in 1990, Congress passed the Ryan White CARE

⁵Estimates obtained from HIV/AIDS: Snapshots of an epidemic: <https://www.amfar.org/thirty-years-of-hiv/aids-snapshots-of-an-epidemic/>

⁶Statistics obtained from November 24, 1995, CDC MMWR weekly report accessed at: <https://www.cdc.gov/mmwr/preview/mmwrhtml/00039622.htm00001361.htm>

There is some evidence that the proportion of new HIV/AIDS cases represented by men who have sex with men decreased over time and the proportion represented by drug users increased. However, even at the time HAART became available, men who have sex with men made up over 57 percent of HIV/AIDS infections.

⁷Organizations such as ACT UP mobilized the gay community and held protests bringing attention to the government's slow response to the crisis.

The crisis became a hotly contested political debate. In the early years, President Reagan refused to address the crisis altogether. In the 1992 presidential election, gay-related issues were discussed in great detail.

ACT. The Ryan White program remains the largest federally funded program in the United States aimed at improving access to healthcare for low-income, uninsured, and underinsured people living with HIV. The Act has been reauthorized in 1996, 2000, 2006, and 2009.

In 1995, the FDA approved Saquinavir, the first protease inhibitor. This ushered in a new era of combination drug therapies which became known as the “AIDS Cocktail” and later, as Highly Active Antiretroviral Therapy (HAART). HAART was a game changer in this epidemic and led to a substantial reduction in HIV-associated mortality. [Figure 2](#) shows the rapid fall in HIV/AIDS death rates after the introduction of the AIDS cocktail. From 1995 to 1997, men’s HIV/AIDS death rate fell by over 60%. Although HIV/AIDS continues to be a major health problem in the US, especially amongst Black and Latinx men, the introduction of HAART meant that testing positive for HIV was no longer a death sentence.

3 Data

3.1 Suicide & HIV/AIDS-related mortality data

I use publicly available data from the 1990-2002 US Vital Statistics National Center for Health Statistics Multiple Cause of Death Files in order to calculate Suicide and HIV/AIDS-related mortality rates [NCHS \(1990-2002\)](#). The mortality data includes all deaths that occurred in the United States and is abstracted from death certificates filed in the vital statistics departments of each state and the District of Colombia.

A large portion of the analysis in this article is based on county, sex, and age group-specific mortality rates. For the years 1989 onwards, county codes are only provided for counties with a population above 100,000. Therefore, smaller counties are dropped from my data set.⁸ To discern suicide and HIV/AIDS-related mortality, I

⁸Although I do not see a large number of smaller counties, I still observe over 60% of deaths since

use the International Classification of Disease (ICD) Code provided under the primary cause of death. Records from 1990-1998 are based on ICD-9 codes, whereas records from 1999 onwards use ICD-10 codes. Appendix [A.1](#) provides which ICD codes are associated with suicide and AIDS-related mortality. Although physicians became aware of a disease that was later classified as HIV back in 1981, HIV did not join the list of communicable diseases, and therefore is not visible in the mortality data until 1987. Although we do not have reliable estimates of HIV/AIDS-related mortality rates before 1987, this does not affect my analysis. I only use HIV/AIDS deaths before the introduction of combination drug therapy in 1995 as a proxy for HIV incidence.

I calculate the number of suicides and HIV/AIDS-related deaths per 100,000 population. Sex and age group-specific suicide deaths and population levels are used to calculate suicide rates for men aged 25-44. I chose this group for my main analysis because this group was most affected by the HIV/AIDS crisis. For example, from 1993 to 1995, HIV/AIDS was the leading cause of death for men aged 25 to 44 in the United States. I also calculate mortality rates for women aged 25 to 44, men over 65, and men under 25, in a triple-difference analysis to verify my findings. Since HIV/AIDS death rates are only used as a measure of pre-HAART HIV incidence in a county and I am exploiting differences in HIV incidence across demographic groups, I calculate overall county HIV/AIDS mortality per 100,000 population instead of sex and age-specific rates.

[Figure 2](#) shows national HIV/AIDS-related death rates in the mortality data from 1980 to 2002. As mentioned above, HIV/AIDS-related deaths were not recorded before 1987. From 1987, we see a steep rise in HIV-related mortality peaking in 1995. In 1995, the FDA approved the use of Saquinavir to be used in combination with other nucleoside analogue medications. This combination proved to be highly effective and

most Americans live in larger counties

marked a new era in combination drug therapy treatments that scientists began to call highly active antiretroviral therapy (HAART). HAART became widely available in 1996 and there was a significant fall in HIV/AIDS-related deaths thereafter.

There are important variations in HIV/AIDS-related death rates by sex and age group. As depicted in [Figure 2](#), men were significantly more likely to contract and die from HIV compared to women. Men between the ages of 25 and 44 were most likely to contract the disease followed by men aged 45 to 64. HIV/AIDS death rates were significantly lower for young (under 24) and older men (over 65).

3.2 Population and controls data

I also obtain census estimates of county population and demographics using a data set constructed by the Survey of Epidemiology and End Results [SEER \(2020\)](#). I use county unemployment data from the Bureau of Labor Statistics [BLS \(1990-2002\)](#). Population density has been identified as an important factor in determining suicide rates ([Kegler et al. 2017](#); [Ivey-Stephenson et al. 2017](#)) I calculate population density using population and land area data obtained from the 2000 census. Religious adherence may be another factor explaining some of the variation in suicide rates. I obtain county-level religious adherence data for the Church and Church Membership study conducted by the Glenmary Research Center ([Grammich et al., 2018](#)).

3.3 Summary Statistics

For my main analysis, I use a continuous treatment measure of the 1994 HIV/AIDS death rate as an indicator of HIV incidence in a county before the introduction of HAART. My analysis is based on comparing changes in suicide rates over time between high and low HIV counties. High and low HIV counties have some different observable characteristics. [Table 3](#) provides summary statistics by pre-HAART HIV/AIDS death rates. Here, I divide counties across quartiles of pre-HAART

HIV/AIDS death rates. Counties with higher pre-HAART HIV/AIDS death rates experience higher rates of unemployment, are racially more diverse, and have higher population densities than other counties. Unsurprisingly, they are also significantly more likely to be eligible for Ryan White Title 1 funding because Title 1 eligibility is determined by cumulative AIDS cases.

4 Methodology

The CDC multiple causes of death data does not provide information about an individual's HIV/AIDS status unless the individual dies because of the HIV infection. I observe county, gender, and age identifiers for all deaths. There are large geographic, age, and gender variations in HIV/AIDS incidence. I exploit geographic and demographic variations in county pre-HAART HIV/AIDS incidence to estimate the effect of HAART on suicide rates. There are several ways to measure pre-HAART HIV/AIDS incidence. We might gauge HIV prevalence by positive HIV tests, AIDS statistics (the final and most severe stage of HIV infection), and HIV/AIDS deaths. Reliable testing data is unavailable for this period and differences in testing availability across counties would result in testing data being plagued with selection issues. Although AIDS data is available through the AIDS public use data set, the data set only records AIDS cases in cities with populations that exceed 500,000 people, according to the latest available official U.S. Bureau of Census estimates. HIV/AIDS death data is available through the US vital statistics. Although county codes are only provided for counties with populations above 100,000 and I must drop counties with smaller populations, this is less restrictive than using the AIDS data.

For my main analysis, I use the HIV/AIDS death rate in 1994 as a measure of pre-HAART HIV incidence.⁹ This is an imperfect measure of HIV/AIDS incidence

⁹In appendix A.2, I repeat the analysis by changing the HIV incidence measure to the AIDS case rate in 1994 as per the AIDS public use data set and find similar effects.

because it reflects some combination of incidence, access and quality of medical care, and correct identification of cause of death. It may also underestimate HIV/AIDS incidence for younger groups who are at the earlier phases of their HIV/AIDS infection. Despite these problems, it is the best measure available during this period. I also conduct several robustness checks where I explicitly deal with some of these issues.

Although the most convincing specification estimated in this study is a triple difference where I exploit both geographic and demographic variation in HIV/AIDS death rates, I first restrict my sample to men aged 25 to 44 and estimate the effect of HAART on this group. Thereafter, I measure the effects on groups that are less affected by the HIV/AIDS crisis, and therefore less affected by the introduction of HAART. Finally, I conduct a triple difference analysis where I compare effects across demographic groups to estimate the causal effect of HAART on suicide rate.

4.1 Event Study

I choose an event study design to estimate the effects of HAART on suicide rates for several reasons:

1. The event study shows time-varying treatment effects, where suicide rates may change in response to the introduction of combination drug therapy over time. Suicide may be linked to the affected population's expectations and belief's about the effectiveness of the therapy and these are expected to change over time.
2. Examining the period leading up to the introduction of combination drug therapy tests whether suicide rates were on a pre-existing trend before the introduction of the treatment.

4.1.1 Difference-in-Differences

Formally, I estimate the following equation:

$$\text{Suicide Rate}_{ct} = \alpha + \sum_{\substack{m \neq 1994 \\ m=1990}}^{2002} \beta_m (\text{Pre-HIV/AIDS deathrate}_c \times 1[t = m]) + \mathbf{X}_{ct}\gamma + a_c + \mu_{st} + \epsilon_{ct} \quad (1)$$

where Suicide Rate_{ct} measures the number of suicides per 100,000 population in county c at year t . $\text{Pre-HIV/AIDS deathrate}_c$ is the number of HIV/AIDS deaths per 100,000 population in 1994 and serves as a measure of the level of HIV incidence before the introduction of combination drug therapy. I use the standardized form of the HIV/AIDS death rate. $1[t = m]$ is an indicator variable that equals 1 when the observations time period is m years relative to 1995.¹⁰

I control for some county characteristics \mathbf{X}_{ct} such as county unemployment rate, percentage of the population that is white, eligibility for Ryan White Title 1 funding, percentage of the population that is Evangelical Protestant, and percentage of the population that adheres to a religion.¹¹ I control for time fixed effects (η_t) and county fixed effects (a_c). I also control for the interaction of state and time fixed effects (μ_{st}) which accounts for any changes in policy at the state level.¹² I have a panel of data with complete controls from 1990 to 2002. I use all these years of data in my analysis and can observe 5 years before the introduction of combination drug therapy, and 8 years after. First, I restrict my sample to men aged 25-44. This group is the most

¹⁰I use 1995 as my first post-HAART year and 1994 as my reference year because Saquinavir was first made available on the market in 1995, and through 1995 newspapers such as the New York Times and San Francisco Chronicle published articles highlighting the promise of combination drug therapies and results from clinical trials (David Perlman, June 22, 1995; Sullivan, Nov 21, 1995; Altman, Feb 02, 1995). Over the next couple of years, the medical community made several changes in drug formulation and other combinations of drug therapies were introduced, but Saquinivir was the first of its kind.

¹¹Some recent evidence suggests that Medicolegal Systems can affect Suicide rates (Fernández et al., 2019; Klugman et al., 2013). I explore the possibility of my results being contaminated by the introduction of Medical examiner systems in Appendix A.4.3

¹²In the appendix A.5 I make certain alterations to the control variables and fixed effects used. First, I drop all control variables and find that my estimates do not change. Then, I remove my interaction of state and time fixed effects and only include county and time fixed effects. I find that my estimates are similar.

likely to be affected by HIV/AIDS. I then estimate Equation 1 for groups that are less affected by the HIV/AIDS crisis (women aged 25 to 44, men over 65, and men 19 and under). This allows me to test for any spillover effects, and these groups also serve as control groups in my analysis. Estimates are weighted by group-specific populations and standard errors are clustered at the county level.

4.1.2 Triple-Difference

To verify these findings, I also employ a triple-difference approach. In addition to spatial and temporal variation, I difference out effects on suicide rates for women aged 25 to 44, men over 65, and men 19 and under. These groups are less likely to contract HIV, and therefore less likely to be affected by the introduction of combination drug therapy.

Formally, I estimate the following equation:

$$\text{Suicide Rate}_{jct} = \alpha + \sum_{\substack{m=1990 \\ m \neq 1994}}^{2002} \beta_m (\text{Pre-HIV/AIDS deathrate}_c \times 1[t = m] \times \text{Men 25-44}_j) + \theta_{ct} + \iota_{jc} + \delta_{sjt} + \epsilon_{ctj} \quad (2)$$

where SuicideRate_{jct} measures group-specific suicide rates.¹³ Men 25-44_j is a dummy variable that measures whether the observation represents suicide rates for men aged 25 to 44. In my triple-difference analysis, I compare effects on men aged 25 to 44 to women aged 25 to 44, men over 65, and men 19 and under. $\text{Pre-HIV/AIDS deathrate}_c$ and $1[t = m]$ are the same as equation 1. Estimates are weighted by group-specific populations and standard errors are clustered at the county level.

With the triple-difference specifications, I can control for the interaction of time and county fixed effects (θ_{ct}), sex and county fixed effects (ι_{jc}), and sex and time fixed

¹³Given the differences in suicide rates across demographic groups, I also consider percentage changes of the outcome variable. In Appendix A.3, I change my outcome variable to represent the inverse hyperbolic sine of suicide rates and find similar effects.

effects (κ_{jt}). I also control for the interaction of state, sex, and time-fixed effects (δ_{sjt}). This controls for any state-level changes in policy during the period of analysis that may have affected men aged 25 to 44 differently from other groups.

4.2 Specifications without Time Variations in Treatment Effect

4.2.1 Standard Difference-in-Differences

To summarize my findings, I also consider a standard difference-in-differences specification where I focus on the impact of the introduction of combination drug therapy in a grouped post period. Formally, I estimate the following equation:

$$\text{Suicide Rate}_{ct} = \alpha + \beta (\text{Pre-HIV/AIDS deathrate}_c \times \text{Post}_t) + \mathbf{X}_{ct}\gamma + a_c + \eta_t + \mu_{st} + \epsilon_{ct} \quad (3)$$

where Post_t is a dummy variable that is equal to one after the introduction of combination drug therapy. This is a restricted version of [Equation 1](#). All other features of this equation are the same as that of [Equation 1](#). Grouping pre and post-periods also provides a convenient way of presenting results in table form.¹⁴ In [subsection A.9](#), I use the standard difference-in-differences method and make alterations to my functional form to tease out other trends which may be driving my results. In [subsection 6.2](#), I use the standard difference-in-differences specification to present a series of results exploring effects across the distribution of pre-HAART incidence.

4.2.2 Standard Triple-Difference

I also consider a triple-difference specification where I focus on the impact of the introduction of combination drug therapy on 25 to 44-year-old men's suicide rate

¹⁴I follow [Kirill and Xavier 2017](#) and do not include linear-in-unit time trends

relative to suicide rates of other groups in a grouped post period. Formally, I estimate the following equation:

$$\text{Suicide Rate}_{jct} = \alpha + \beta (\text{Pre-HIV/AIDS deathrate}_c \times \text{Post}_t \times \text{Men 25-44}_j) + \theta_{ct} + \iota_{jc} + \kappa_{jt} + \delta_{sjt} + \epsilon_{ctj} \quad (4)$$

where Post_t is a dummy variable that is equal to one after the introduction of combination drug therapy. This is a restricted version of [Equation 2](#) and all other features of this equation are the same as that of [Equation 2](#).

5 Results

[Figure 3](#) depicts the impact of the introduction of combination drug therapy on suicide rates for men aged 25 to 44. The figure implies that there is approximately a 1 per 100,000 population decrease in suicide rates for men in counties that have a 1 standard deviation higher pre-HAART HIV/AIDS death rate. The first column of [Figure 4](#) depicts effects on groups less affected by the introduction of HAART. Effects on women aged 25 to 44, men over 65, and men 19 and under appear to be close to zero. Coefficients for women and older men are negative but statistically insignificant. This may represent some spillover effects. Although other groups had significantly lower rates of HIV/AIDS, they were still affected by the virus. Rates of HIV infection were low for these groups, but they were not zero. Members of these groups may also have friends and family members who are HIV positive, and therefore it is reasonable to find some negative effects on suicidality for this group following the introduction of HAART. These results correspond with those produced in the difference-in-differences table presented in [Table 4](#). My findings are further verified by observing similar estimates in the triple-difference analysis depicted in the second

column of [Figure 4](#). I observe small immediate effects that grow over time. This may be a result of increasing trust in the efficacy of HAART treatment and increasing access to life-saving medications. This is in line with other studies which find falling suicide rates amongst HIV-positive individuals over time even after the introduction of HAART [Ruffieux et al. \(2019\)](#).

Taken together, the estimates suggest that the introduction of HAART reduced suicide rates by 0.9 per 100,000 population in counties that have a 1 standard deviation higher pre-HAART HIV/AIDS death rate. A 0.9 per 100,000 population reduction in suicide rates represents approximately 4% change since the average suicide rate for the population of interest is 24 per 100,000 population. Some back of the envelope calculations would reveal that HAART saves approximately 500 men aged 25 to 44 from suicide each year following its introduction.¹⁵

To further contextualize the magnitude of these results, I compare effects across the distribution of Pre-HAART HIV/AIDS incidence. I divide counties into deciles of Pre-HAART HIV incidence and re-estimate [Equation 1](#) where Pre-HAART HIV is now a dummy variable equal to 1 when looking at the highest decile of Pre-HAART incidence and equal to zero when looking at the lowest three deciles. Here, I am trying to consider the most extreme case so I drop the deciles in between. The first two panels of [Figure 5](#) show effects on men and women. Here, I see a 4 per 100,000 reduction in suicide rates in the top decile of HIV incidence when compared to the bottom 3 deciles for men. This represents an approximate decrease in 25-44-year-old men's suicide rate by 20%. When comparing these counties, I see small but significant negative effects on women's suicide rate. I find similar but slightly smaller effects in my triple difference analysis depicted in the third panel of [Figure 5](#).

¹⁵HIV/AIDS death rate went down by approximately 1.3 standard deviations after the introduction of HAART (HIV/AIDS death rate is 26 per 100,000 in 1994, 8 per 100,000 in 1998, and 1 standard deviation of 1994 HIV/AIDS death rate is approximately 14). Therefore, suicide rates should decrease by 1.17 per 100,000 and the population of men aged 25 to 44 is approximately 42 million.

Due to the large magnitude of estimates, I must convince the reader that these effects are reasonable. Since this is the first study of its kind, it's difficult to make direct comparisons to other studies. [Keiser et al. \(2010\)](#) estimates suicide rates among HIV-positive men and women in Switzerland before and after the introduction of HAART. They find that amongst HIV-positive men, suicide rates fell from 447.4 per 100,000 person-years to 90.1 person-years after the introduction of HAART. Although I do not observe HIV status in my suicide data, on average there were approximately 20 HIV-positive individuals in 1994 for every reported HIV/AIDS death.¹⁶ Therefore, I can infer that a 1 standard deviation increase in HIV/AIDS death rate is associated with a 240 increase in the HIV positivity rate (defined as HIV-positive individuals per 100,000 population).¹⁷ If I assume that this HIV positivity rate remained constant before and after the introduction of HAART, estimates from ([Keiser et al., 2010](#)) would suggest that the overall suicide rate for men would go down by 0.85 per 100,000 driven entirely by a fall in suicide rates amongst HIV positive individuals.¹⁸ Although we cannot discern whether our effects are driven by a reduction in suicide rates amongst HIV-positive individuals or through other mechanisms (such as reduced consequences of infection for uninfected people or reductions in homophobia), finding similar estimates to those in the Switzerland study suggests that the effects may largely be a result of falling suicide rates amongst HIV positive individuals.

¹⁶20 HIV-positive individuals for each death might be a conservative estimate. In 1994, there were a total of 41,930 HIV-related deaths in the US and an estimated 950,000 people living with HIV according to the Kaiser Family Foundation. [KFF \(2006\)](#)

¹⁷1 standard deviation increase in HIV deaths is equal to 12 HIV/AIDS deaths

¹⁸Pre-HAART: $(12 \times 20) * \frac{447.4}{100,000} = 1.07$

Post-HAART: $240 * \frac{90.1}{100,000} = 0.216$

Change: $1.07 - 0.216 = 0.85$

6 Robustness Tests

This study depicts falling suicide rates amongst men aged 25 to 44 in high HIV/AIDS counties relative to low HIV counties. It shows little to no effects on groups less affected by the HIV/AIDS virus and finds that estimates are robust after differencing out effects on low HIV/AIDS demographic groups. Back-of-the-envelope calculations reveal that the estimates suggested in this paper are reasonable when comparing them to a longitudinal study of HIV-positive populations in Switzerland.

Despite these findings, this study has several limitations and I conduct a series of robustness checks to verify the accuracy of these estimates. I can exploit geographic and demographic variations in HIV/AIDS incidence to conduct my analysis. As depicted in [Table 3](#), high HIV/AIDS counties are different from low HIV/AIDS counties. The effects mentioned above could be the result of different trends between counties with different characteristics. I also do not observe actual HIV status or sexual orientation in the mortality data. I have argued that HAART would have significantly larger effects on HIV-positive individuals and gay men but I am unable to observe if these groups are driving the observed results. The 90s are also a period of falling urban violence and crime ([Levitt, 2004](#)). Since much of this decline occurred in high HIV/AIDS areas and has larger effects on men aged 25 to 44, relative to other groups, it is possible that the same factors that are driving these changes are affecting my results. Below, I conduct several robustness tests, which provide some evidence that the estimates are driven because of the introduction of HAART.

6.1 Firearm and Non-Firearm Suicides

If my estimates are a result of the introduction of combination drug therapy, I expect my results to be driven by individuals who are more likely to be affected by the treatment. Homosexual men were significantly more likely to contract HIV, and

therefore more likely to be affected by the introduction of treatment. Although I do not observe sexual orientation in the suicide data, I do observe the method of suicide. There are significant differences in the methods of suicide employed by homosexual and heterosexual men. [Clark et al. 2020a](#) finds that sexual minority men are significantly less likely to be gun owners compared to heterosexual men. [Clark et al. 2020b](#) finds that firearms are half as likely to be used in suicide for homosexual men compared to heterosexual men. In my main specification, I collapsed all the different methods of suicide into a single suicide rate. To explore whether my results are driven by effects on homosexual men, I now categorize suicides into either firearm or non-firearm suicides and calculate rates for each category. The breakdown of ICD codes into firearm and non-firearm suicides is explained in [Appendix A.1](#).

[Figure 6](#) depicts my event studies for firearm and non-firearm suicides. I find that my effects are driven almost entirely by non-firearm suicides. This provides further evidence that my estimates are driven by effects on gay men who are more likely to contract HIV. Since my results are largely driven by non-firearm suicides, this provides some evidence that my estimates are not a result of changes in gun access, which has been found to have a significant effect on suicide rates.¹⁹

6.2 Exploiting Continuous Treatment

In order to verify that my results are driven by changes in pre-HAART HIV incidence, I also exploit the continuous nature of the treatment measure used in this study. [Meyer \(1995\)](#) argues that “differences in the intensity of the treatment across different groups allow one to examine if the changes in outcomes differ across treatment levels in the expected direction.” If my results are, in fact, driven by the introduction of HAART treatment, I expect the largest effects in counties with the highest pre-

¹⁹In [appendix A.10](#), I explicitly explore how trends in violence might affect the results by comparing trends in homicides and suicides.

HAART HIV incidence and smaller effects in counties with lower rates of pre-HAART HIV.

The first panel in [Figure 7](#) shows mean pre-HAART HIV/AIDS death rates by deciles of pre-HAART HIV/AIDS death rate in a county. The pre-HAART HIV/AIDS death rate is under 10 per 100,000 population in the first 6 deciles and we expect the treatment to have little effect on these counties. Thereafter, we see exponential increases in HIV/AIDS death rates with larger jumps when we move from the 7th to the 8th, 8th to the 9th decile, and the largest jump, when moving from the 9th to 10th decile.

I modify my treatment variable from [Equation 3](#) and [Equation 4](#) into a dummy variable which is equal to one when looking at different deciles of pre-HAART HIV incidence and zero when looking at the lowest 2 deciles. The next two panels of [Figure 7](#) represent the difference-in-differences estimates from [Equation 3](#) for men and women. I observe larger negative coefficients at higher deciles of pre-HAART HIV/AIDS death rates. In the last panel of [Figure 7](#), I present triple-difference estimates from [Equation 4](#). Again, I observe larger coefficients at higher deciles of pre-HAART HIV incidence. This verifies that changes in suicide rates differ across treatment levels in the expected direction. This provides further evidence that the results are driven by the introduction of HAART.

7 Effects By Race

There are significant differences in HIV/AIDS incidence and HAART takeup between white and non-white Americans. HIV/AIDS incidence was significantly higher among non-white Americans and some studies have documented how the introduction of HAART widened the Black-White mortality gap [Levine et al. \(2007\)](#). Although the introduction of HAART significantly reduced HIV/AIDS deaths among white men,

there remain significant concerns about the rates of HIV/AIDS mortality among non-white men. The first panel of [Figure 8](#) depicts trends in HIV/AIDS death rates for white and non-white men aged 25 to 44. We observe higher pre-HAART rates of HIV/AIDS deaths amongst non-White men. Although both groups experience a decline in death rates following the introduction of HAART, there is a starker decrease in HIV/AIDS death rates among white men.

In [Figure 8](#), I present coefficients estimates from [Equation 3](#) for white and non-white men aged 25 to 44 separately. I also present pre-HAART rates of HIV/AIDS death rates by race. I find that the effects for the two groups are similar in size. However, pre-existing rates of HIV/AIDS are greater for non-white men. This implies that effects on HIV-affected white populations are relatively larger than HIV-affected non-white populations.

These effects might be the result of different levels of access to treatment for whites and non-whites. This implies that access to treatment not only had effects on the physical but also the mental health of the affected population. This further emphasizes the importance of programs that seek to expand access to treatment. Several studies estimate the effect of Ryan White funding on the physical health of affected populations, but it may also be important to study the mental health effects of these programs.

8 Welfare Implications

Accurately measuring the welfare implications of HAART through its effects on suicide is challenging due to data limitations. Simply multiplying the measured effect with the statistical value of life would result in an incomplete and potentially misleading estimation of the overall welfare impact. This is because the population contributing to this effect differs from the general population. HIV-positive individuals

generally have a lower life expectancy compared to the general population. Consequently, preventing the death of an HIV-positive individual would result in a lower number of years of life saved in comparison to preventing the death of someone from the general population. In addition to HIV status, the number of life years saved is influenced by the age of the individual, the stage of infection, and other underlying health factors. If the men who were previously dying by suicide were in the late stages of their infection, the number of years of life saved by preventing suicide would be small. Since HAART is an intervention that improves both physical and mental health, this would imply that the welfare benefits of HAART in terms of years of life saved of those individuals who are no longer dying by suicide are driven by preventing HIV/AIDS deaths rather than mental health improvements.

To categorize the individuals driving these results, three groups can be considered: individuals who are HIV negative, individuals in the early phase of HIV infection, and individuals in the later phase of HIV (AIDS). I assume the average individual driving my results is 35 years old.²⁰ Studies find that without treatment, a person who is exposed to HIV has 5 to 10 years before their infection progresses to AIDS, after which the individual has a life expectancy of 2 years ([Poorolajal et al., 2016](#)). Therefore, I assume that the number of life years lost due to poor mental health for a person in the early phases of an HIV infection is 9.5 years whereas the years of life lost for a person in the late phase of an HIV infection is 2 years. Since HAART improves physical health in addition to mental health, I obtain life expectancies of HIV positive individuals in the years following HAART from [Harrison et al. \(2010\)](#) to determine the total years of life saved by preventing an HIV related suicide. According to [Harrison et al. \(2010\)](#), as of 1998, HIV-positive men had a life expectancy of approximately 15.9 years, while men in the late stages of their infection had a life expectancy of ap-

²⁰This is the average of ages 25, 35, and 45, weighted by coefficients presented in [Table 9](#). Men's life expectancy in 1998 is 72 years.

proximately 12.1 years. These findings provide important information for estimating the welfare implications of preventing an HIV-related suicide in terms of years of life saved, categorized by HIV status and progression. Based on these statistics, I present estimates of the average years of life saved for a person in each of these three groups through improvements in mental health and physical health in [Table 1](#).²¹

	Mental Health	Physical Health
Status	years	years
HIV -	37	0
HIV +: early stages	9.5	6.4
HIV +: late stages (AIDS)	2	10.1

Table 1. Years of life safe saved through improved mental health and physical health for those that died by suicide based on HIV status and progression.

It is reasonable to assume that my effects are being driven by some combination of these three groups. As mentioned earlier, HAART may affect the mental health of HIV positive individuals by improving the prognosis of infection but may also improve outcomes for HIV negative individuals through reducing the consequences of contracting HIV, and seeing fewer friends and family members dying from the virus. Although there is a limited literature which explores HIV/AIDS progression and suicide prior to the introduction of HAART, there is some evidence that suicide risk may be highest directly following the diagnosis of an infection or late stages of the virus when an individual might experience pain and disability ([Siegel and Meyer, 1999](#)). More recent literature offers examples of both heightened suicide risk directly following the diagnosis of an HIV infection as well as heightened suicide risk following the progression of the virus ([Pelton et al., 2021](#)).²²

²¹”Mental Health” represents how many additional years a person may have lived had they not died by suicide but there was still no treatment for HIV. ”Physical Health” represents the additional number of years a person would live due to the improved life expectancy of an HIV positive individual following HAART

²²For many individuals, the time of diagnosis and the progression of the virus to AIDS may have coincided. For example, from 1994-1999, 41% of new HIV diagnosis represented cases of AIDS.

While the stage of the virus is not observed in the dataset, the ages of individuals driving the observed effects on suicide as well as the ages of those dying from HIV/AIDS are available. In the primary analysis, the effects of HAART on suicide rates are measured using a broad age group consisting of men aged 25 to 44. While this grouping offers certain benefits, it may also be beneficial to consider effects by narrower age groups to discern who is driving these results.²³ Therefore, I reestimate [Equation 3](#) for men in their 20s, 30s and 40s.²⁴ Thereafter, I conduct a triple difference analysis where I reestimate [Equation 4](#) and measure effects on men in their 20s, 30s and 40s, relative to women in their 20s, 30s, and 40s. Estimates are provided in [Table 8](#) and [Table 9](#). My estimates are significantly larger for men in their 20s and 30s compared to men in their 40s. In [Figure 9](#), I break down pre-HAART HIV/AIDS death rates by age groups. Although the effects in this study are driven by men in their 20s, and 30s, I find higher death rates for older cohorts. Although it is possible that the kinds of people who are dying by suicide are different from those that are dying by HIV/AIDS, this provides some suggestive evidence that there is a non-trivial difference in the age of suicide and the age of HIV/AIDS death and that a significant portion of the welfare benefits from preventing an HIV related suicide are a result of improvements in mental health.

Estimates from my main specification suggest that HAART saved approximately 500 men aged 25 to 44 from suicide each year. Assuming an average value of statistical life year (VSLY) of \$302,000, I present welfare implications of HAART due to a fall in suicides for several reasonable combinations of the previously mentioned groups in [Table 2](#)²⁵.

²³Looking at a broader group allows me to estimate effects on mortality rates more precisely. Estimating rates for smaller populations are more noisy.

²⁴Although I restrict my treatment group to men aged 25 to 44 in my primary specification, men who are slightly younger and slightly older may also experience similar effects since they have similar rates of HIV/AIDS.

²⁵There is significant variation in estimates of the value of a statistical life year (VSLY), a VSLY of \$302,000 is obtained from [Aldy and Viscusi \(2008\)](#)

Scenario	Mental Health	Physical Health
	Value (USD millions)	Value (USD millions)
10% HIV-, 45% HIV+ early, 45% HIV+ late	1,340.1	1,121.2
5% HIV-, 30% HIV+ early, 65% HIV+ late	906.0	1,281.0
5% HIV-, 65% HIV+ early, 30% HIV+ late	1,302.4.7	1,085.7

Table 2. Value of statistical life years (VSLYs) saved due to HAART through a fall in suicides for different combinations of HIV status and disease progressions.

The estimates suggest that the introduction of HAART resulted in over 2 billion dollars worth of annual savings in VSLY terms through its effect on suicides.²⁶ Of the 2 billion dollars saved, a significant portion was driven by improvements in mental health. This implies that suicides were not just an alternative to an HIV/AIDS death and had non trivial implications for the welfare of the affected population. It is also important to note that suicide is an extreme mental health outcome and the estimates presented in this study are a lower bound of the benefits of HAART through improvements in mental health.²⁷

9 Conclusion

This study highlights the substantial fall in suicide rates among HIV-affected populations following the introduction of HAART. I find a 0.9-point decrease in suicide rates for men aged 25 to 44 in counties that have a 1 standard deviation higher pre-HAART HIV/AIDS death rate. In the most extreme case, where I compare effects in counties in the highest decile of pre-HAART HIV/AIDS death rate to the bottom 3 deciles, I find that suicide rates for men aged 25 to 44 fall by approximately 20% in high HIV counties relative to low HIV counties. The estimates suggest that the introduction of HAART saved approximately 500 men aged 25 to 44 from suicide each year. I

²⁶It is important to note that this figure only represents benefits through effects of suicide. There were significantly larger benefits through falling HIV/AIDS death rates.

²⁷In appendix A.11, I use data from The Behavioral Risk Factor Surveillance System to explore trends in poor mental health days for the affected population. I find suggestive evidence that HAART improved non-extreme mental health outcomes for at-risk groups.

find that suicide rates for other groups such as women aged 25-44, men over 65, and men under 20, are largely unaffected by the introduction of HAART. I also difference out effects on these groups and find that my estimates are robust to triple difference specifications.

I do not observe sexual orientation or HIV status in the suicide data so I conduct several tests to ascertain that my results are driven by the introduction of HAART. Homosexual men are half as likely as heterosexual men to use firearms as their method of suicide [Clark et al. \(2020b\)](#). After breaking down suicides into firearm and non-firearm suicides, I find that my results are driven almost entirely by non-firearm suicides. This provides some evidence that my estimates are a result of changing suicide rates among gay men.

This study finds extremely large effects. To argue that these estimates are reasonable, I extrapolate from a similar study in Switzerland and compare estimates. The Swiss study finds large reductions in suicide rates among HIV-positive populations after the introduction of HAART [Keiser et al. \(2010\)](#). Although I do not observe HIV status in my suicide data, I approximate HIV-positive populations using my HIV/AIDS death data. I use these approximations and the estimates in the Swiss study to predict the fall in suicide rates. These predictions are very similar to my estimated effects and lends credibility to my findings. Although I am unable to discern the exact mechanisms driving my result, finding similar estimates to the Swiss study provides some evidence that results are largely driven by changing patterns of suicide amongst HIV-positive individuals after the introduction of HAART.

When I break down effects by race, I find larger effects on HIV-positive white populations compared to HIV-positive non-white population suggesting that access to treatment may play an integral role in improving the mental health outcomes of HIV-affected communities. This also underscores the importance of conducting more research which explores how access to HIV/AIDS treatment might affect mental

health.

This study has important implications for understanding the effects of the HIV/AIDS crisis and the introduction of HAART treatment. By simply measuring HIV/AIDS mortality as deaths that were the direct result of HIV/AIDS progression, we are underestimating the total number of HIV/AIDS deaths. We must account for suicides among populations affected by the virus to get a more accurate figure. It also implies that if we only focus on the direct benefits of HIV treatment, we miss meaningful parts of the benefits of innovations in medical treatments. This has implications for the cost-benefit analyses of HIV treatments but may also affect how we estimate the effects of other treatments for medical conditions. Finally, this study also warns us about the potential threat of chronic health conditions to mental health outcomes. Although HIV/AIDS was a specific type of virus that affected a particularly vulnerable community, poorer mental health outcomes are observed in patients with other chronic health conditions as well. Understanding the physical and mental health challenges of those with chronic illnesses is integral to their care.

10 Figures

Figure 1 : Cumulative HIV/AIDS deaths overtime

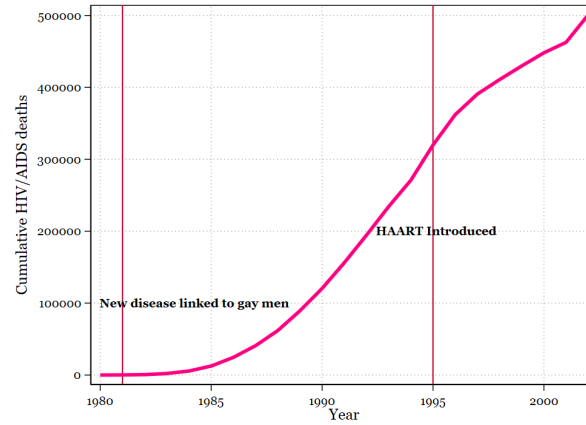


Figure 1. Source: amfAR: The Foundation for AIDS Research (<https://www.amfar.org/thirty-years-of-hiv/aids-snapshots-of-an-epidemic>)
Notes: Cumulative HIV/AIDS deaths over time.

Figure 2: HIV/AIDS Death Rates overtime

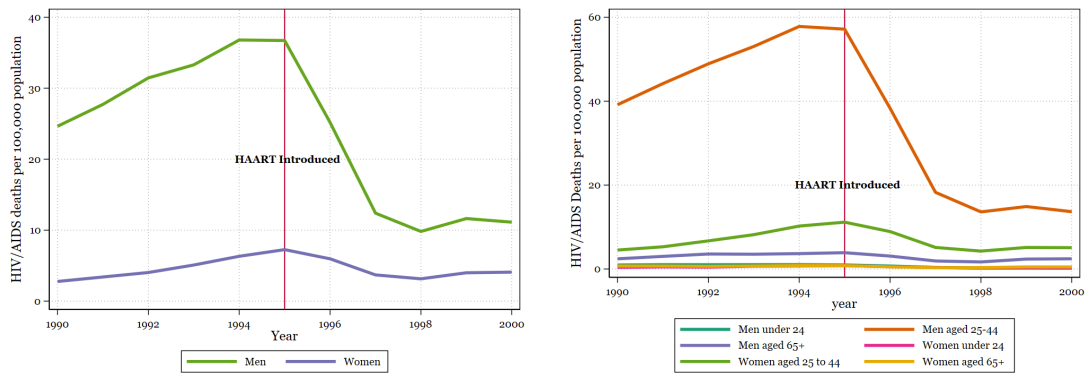


Figure 2. Source: CDC Multiple Cause of Death data NCHS (1990-2002)
Notes: HIV/AIDS death rates are calculated per 1,000,000 group population

Figure 3 : Estimates from Equation 1 for men aged 25 to 44.

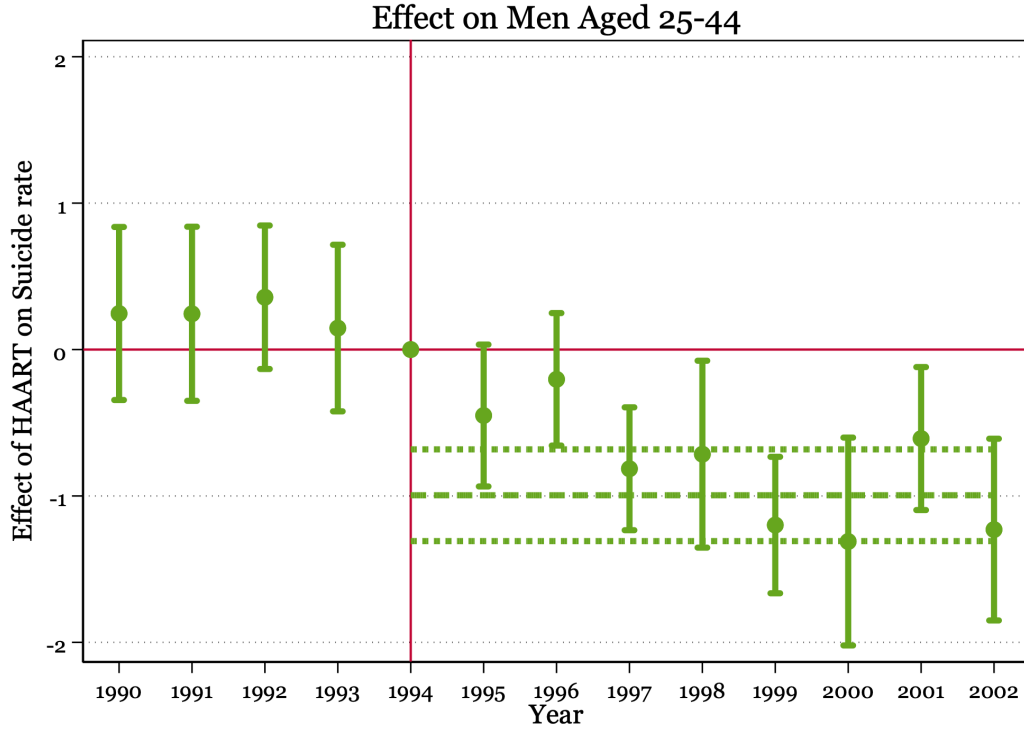


Figure 3. Source: CDC Multiple Cause of Death data NCHS (1990-2002)

Notes: Figure presents difference-in-differences estimates from Equation 1 for men aged 25 to 44. Estimates control for county fixed effects and state and time fixed effects. All estimates are weighted by group county level population and standard errors are clustered at the county level. Estimates include controls for county unemployment, percentage of population that is white, Ryan White title 1 funding eligibility, percentage of population that is evangelical protestant, percentage of population that adheres to a religion. Horizontal dashed lines present estimate and confidence intervals from specifications without time variations in treatment effect

Figure 3 : Estimates from Equation 1 & Equation 2

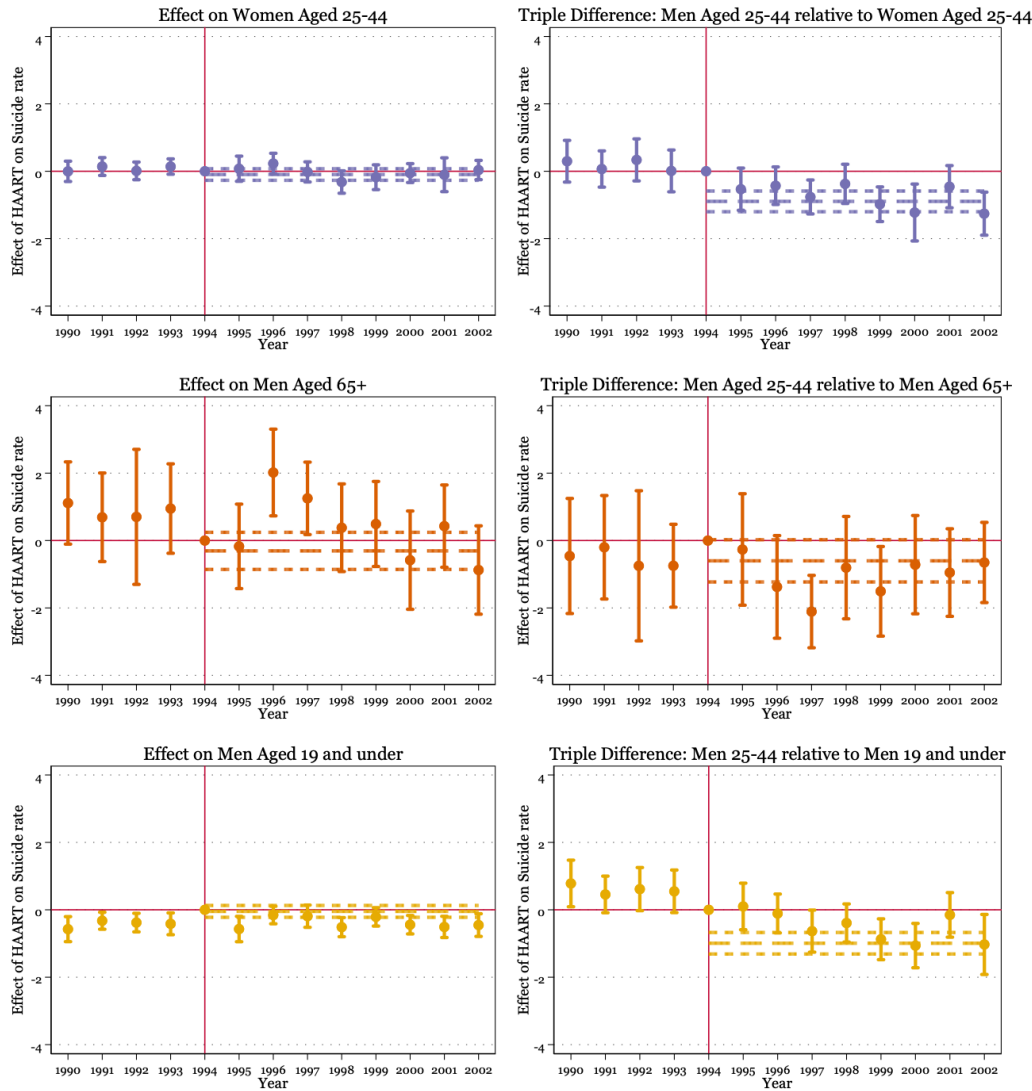


Figure 4. Source: CDC Multiple Cause of Death Data NCHS (1990-2002)

Notes: The first figure in each row presents difference-in-differences estimates from Equation 1 for women aged 25 to 44, men aged 65 plus, and men 19 and under. The second figure of each row presents triple-difference estimates from Equation 2 for men aged 25 to 44 to women aged 25 to 44, men aged 65 plus, and men aged 19 and under. The first figure in each row controls for county fixed effects and state and time fixed effects. Estimates include controls for county unemployment, percentage of population that is white, Ryan White title 1 funding eligibility, percentage of population that is evangelical protestant, percentage of population that adheres to a religion. The second figure in each row controls for the interaction sex and county fixed effects, sex and time fixed effects and state, sex and time fixed effects. All estimates are weighted by group county level population and standard errors are clustered at the county level. Horizontal dashed lines present estimate and confidence intervals from specifications without time variations in treatment effect

Figure 5 : Effects accross distribution

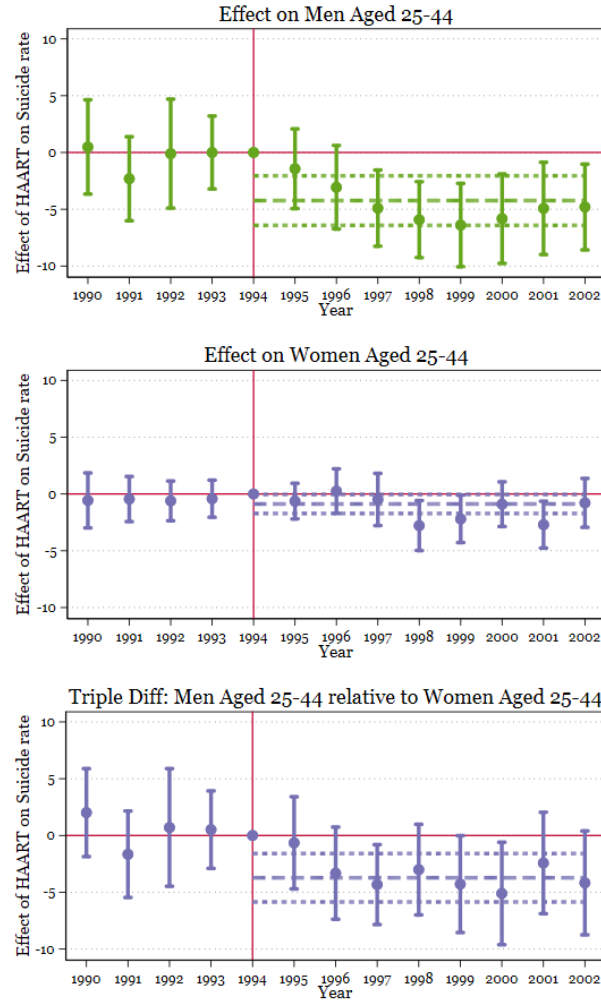


Figure 5. Source: CDC Multiple Cause of Death data [NCHS \(1990-2002\)](#)

Notes: These figures present estimates from [Equation 1](#) and [Equation 2](#), where, $HIV/AIDS\ deathrate_c$ is now a dummy variable that is equal to 1, when looking at the top decile of pre-HAART HIV/AIDS death rates and zero when looking at the bottom 3. First two figures present difference-in-differences estimates from [Equation 1](#) for men and women aged 25 to 44. First two figures control for county fixed effects and state and time fixed effects. Estimates include controls for county unemployment, percentage of population that is white, Ryan White title 1 funding eligibility, percentage of population that is evangelical protestant, percentage of population that adheres to a religion. Third figure presents triple-difference Estimates from [Equation 2](#) for individuals aged 25 to 44. Third figure controls for the interaction of sex and county fixed effects, sex and time fixed effects and state, sex and time fixed effects. All estimates are weighted by group county level population and standard errors are clustered at the county level. Horizontal dashed lines present estimate and confidence intervals from specifications without time variations in treatment effect

Figure 6: Firearm & non-firearm Suicides

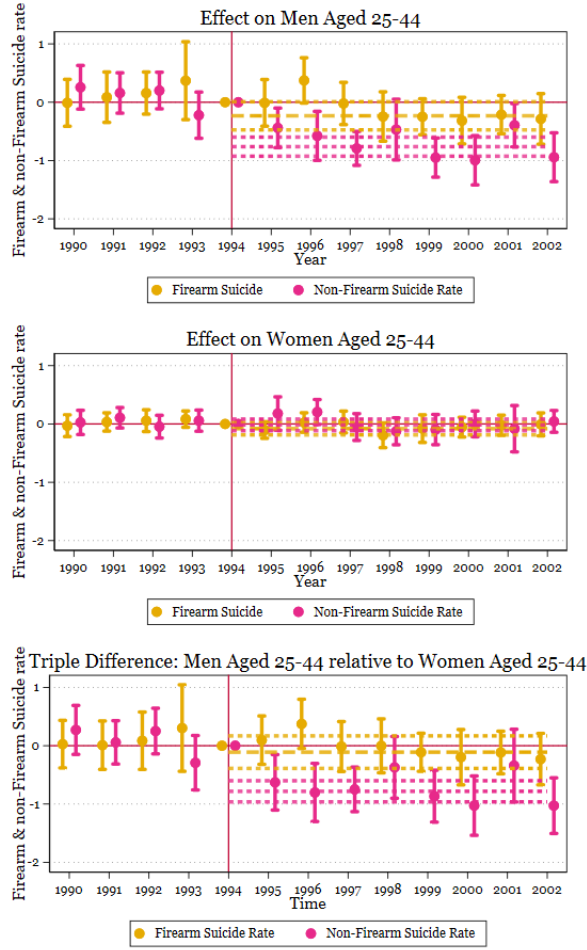


Figure 6. Source: CDC Multiple Cause of Death data [NCHS \(1990-2002\)](#)

Notes: In these figures, I change the outcome variable to represent firearm or non-firearm suicide rates instead of the overall suicide rate. First two figures present difference-in-differences estimates from [Equation 1](#) for men and women aged 25 to 44. First two figures control for county fixed effects and state and time fixed effects. Estimates include controls for county unemployment, percentage of population that is white, Ryan White title 1 funding eligibility, percentage of population that is evangelical protestant, percentage of population that adheres to a religion. Third figure presents triple-difference Estimates from [Equation 2](#) for individuals aged 25 to 44. Third figure controls for the interaction of sex and county fixed effects, sex and time fixed effects and state, sex and time fixed effects. All estimates are weighted by group county level population and standard errors are clustered at the county level. Horizontal dashed lines present estimate and confidence intervals from specifications without time variations in treatment effect

Figure 7: Exploiting Continuous Treatment

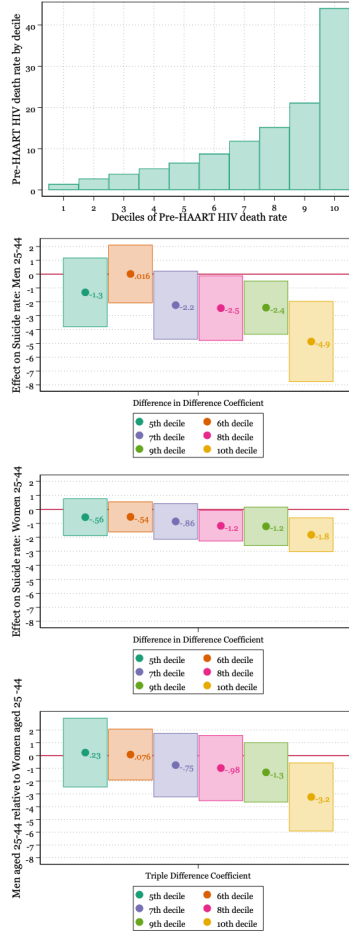


Figure 7. Source: CDC Multiple Cause of Death data NCHS (1990-2002)

Notes: The first figure presents mean pre-HAART HIV death rate at 10 deciles of pre-HAART HIV/AIDS death rate. In the next three figures, $\text{Pre-HIV/AIDS death rate}_c$ is a dummy variable that is equal to 1, when looking at a particular decile of pre-HAART HIV deaths and zero when looking at the bottom 2. The second and third figure present difference-in-differences estimates from Equation 3 for men and women aged 25 to 44. The second and third figure control for time fixed effects, county fixed effects and state and time fixed effects. Estimates include controls for county unemployment, percentage of population that is white, Ryan White title 1 funding eligibility, percentage of population that is evangelical protestant, percentage of population that adheres to a religion. The fourth figure presents triple-difference Estimates from Equation 4 for individuals aged 25 to 44. The fourth figure controls for the interaction of time and county fixed effects, sex and county fixed effects, sex and time fixed effects and state, sex and time fixed effects. All estimates are weighted by group county level population and standard errors are clustered at the county level.

Figure 8: Effects by Race

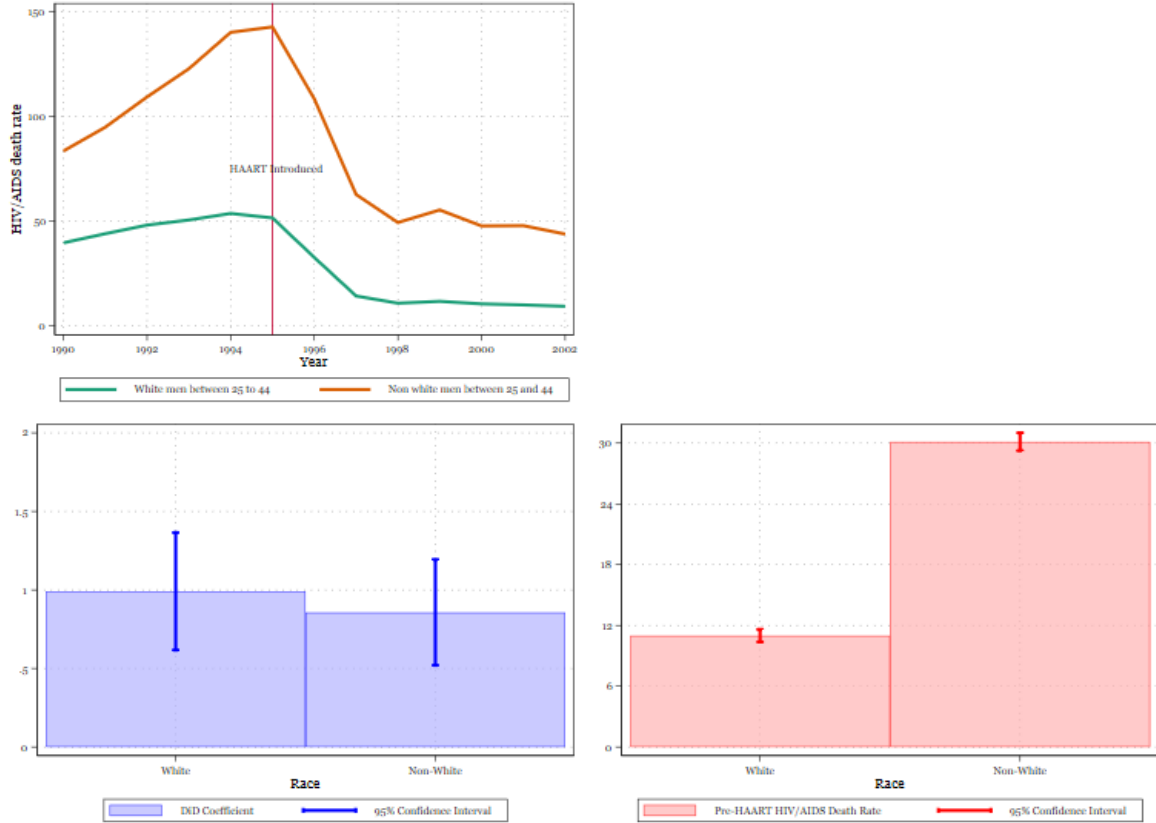


Figure 8. Source: CDC Multiple Cause of Death data [NCHS \(1990-2002\)](#)

Notes: The first figure presents estimates of HIV/AIDS-related death rates for white and non-white men aged 25 to 44. In the next figure in, I present coefficients estimates from [Equation 3](#) for white and non-white men aged 25 to 44 separately. These estimates control for county fixed effects and state and time fixed effects. Estimates include controls for county unemployment, percentage of population that is white, Ryan White title 1 funding eligibility, percentage of population that is evangelical protestant, percentage of population that adheres to a religion. In the third figure, I present 1994 HIV/AIDS-related death rates for white and non-white men aged 25-44.

Figure 9: HIV/AIDS death rates by age

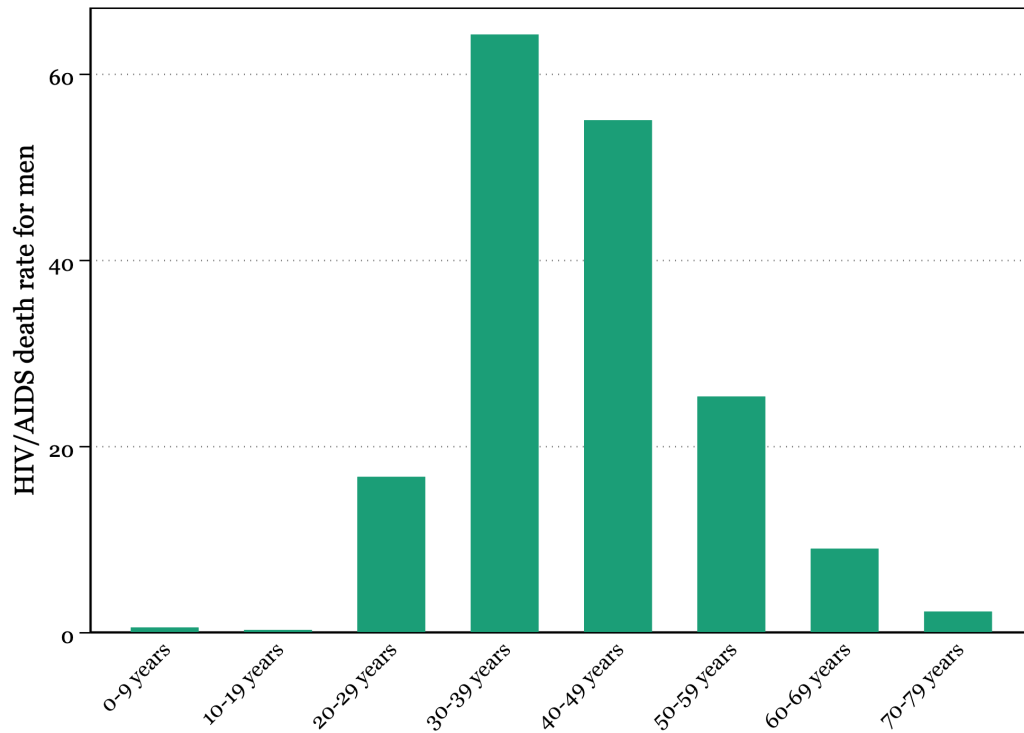


Figure 9. Source: CDC Multiple Cause of Death data [NCHS \(1990-2002\)](#)
Notes: The figure presents pre-HAART (1990-1994) HIV/AIDS-related death rates for men in different age groups.

11 Tables

Table 3: Summary Statistics

	4th Quartile	3rd Quartile	2nd Quartile	1st Quartile
Male Suicide Rate	20.39 (7.42)	18.48 (7.97)	19.03 (8.58)	17.77 (8.57)
Female Suicide Rate	5.17 (2.68)	4.72 (2.74)	4.57 (3.04)	3.94 (3.40)
Male HIV Death Rate	43.70 (55.49)	15.77 (15.95)	8.50 (9.81)	4.47 (6.04)
Female HIV Death Rate	5.07 (10.25)	1.81 (2.87)	0.78 (1.56)	0.41 (1.10)
1994 HIV Death Rate	28.85 (20.02)	11.06 (2.26)	5.34 (0.96)	2.40 (0.85)
Male Fire Arm Suicide Rate	12.26 (5.78)	11.19 (6.06)	12.07 (6.74)	11.06 (6.25)
Female Fire Arm Suicide Rate	1.94 (1.66)	1.67 (1.77)	1.57 (1.82)	1.33 (1.92)
Male Non Fire Arm Suicide Rate	8.12 (4.43)	7.29 (4.19)	6.96 (4.62)	6.71 (5.12)
Female Non Fire Arm Suicide Rate	3.23 (1.87)	3.06 (2.08)	3.00 (2.41)	2.61 (2.65)
Eligible for Title 1 funding by 1996	0.74 (0.44)	0.43 (0.49)	0.43 (0.50)	0.35 (0.48)
Unemployment Rate	6.82 (1.98)	6.25 (2.33)	6.26 (2.55)	6.53 (3.35)
Population Density	2,484.85 (2,849.46)	1,305.59 (1,160.48)	704.97 (613.32)	519.17 (539.54)
Percentage Pop Male	48.68 (1.13)	48.71 (1.13)	48.81 (0.96)	49.12 (1.21)
Percentage Pop White	74.42 (12.56)	84.81 (7.89)	89.78 (6.35)	93.77 (5.41)
Percentage Pop aged b/w 0 and 24	35.95 (3.28)	35.68 (3.63)	36.12 (3.58)	37.45 (4.72)
Percentage Pop aged b/w 25 and 44	33.95 (2.43)	32.90 (2.38)	32.91 (2.88)	32.36 (3.12)
Proportion Evangelical Protestant	0.11 (0.10)	0.11 (0.11)	0.12 (0.09)	0.10 (0.07)
Proportion Adherants to a Religion	0.57 (0.11)	0.59 (0.15)	0.56 (0.15)	0.62 (0.16)
Observations	1980	2040	1960	1820

mean coefficients; sd in parentheses

4th Quartile: counties in top 25 percentile of HIV/AIDS death rates in 1994

3rd Quartile: counties in 50th-75th percentile of HIV/AIDS death rates in 1994

2nd Quartile: counties in 25th-50th percentile of HIV/AIDS death rates in 1994

1st Quartile: counties in bottom 25 percentile HIV/AIDS death rates in 1994

Table 3. Source: CDC Multiple Cause of Death data [NCHS \(1990-2002\)](#)

Notes: Table presents summary statistics for counties across the distribution of 1994 HIV/AIDS death rates for the years before the introduction of HAART (1990-1994).

Table 4: Difference in Differences: Men aged 25 to 44 and women aged 25 to 44

	Men 25-44			Women 25-44		
	(1)	(2)	(3)	(4)	(5)	(6)
Post=1 \times Stdz 1994 HIV death rate	-1.03379*** (0.147)	-0.95096*** (0.174)	-0.99522*** (0.159)	-0.07506 (0.079)	-0.05912 (0.081)	-0.09614 (0.087)
Population Density		-0.00204 (0.002)	0.00061 (0.002)		-0.00030 (0.001)	-0.00024 (0.001)
Unemployment Rate		0.26256** (0.124)	-0.03912 (0.156)		0.01567 (0.060)	0.03712 (0.084)
Percentage Pop White		0.03613 (0.109)	0.14017 (0.120)		-0.00961 (0.053)	0.07815 (0.049)
Proportion Pop Evangelical		18.90438 (15.915)	17.47253 (17.849)		-1.89047 (7.376)	15.10848** (6.691)
Proportion Pop Religious		-2.62522 (3.177)	1.05753 (2.944)		0.11764 (1.939)	0.69799 (1.686)
Title 1 Eligible=1		-1.02496** (0.479)	-0.99244* (0.526)		-0.58575** (0.234)	-0.23886 (0.245)
Observations	5070	4970	4866	5070	4970	4866
County FE	Yes	Yes	Yes	Yes	Yes	Yes
Time FE	Yes	Yes	No	Yes	Yes	No
State X Time FE	No	No	Yes	No	No	Yes

Standard errors in parentheses

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table 4. Source: CDC Multiple Cause of Death data [NCHS \(1990-2002\)](#)

Notes: Table presents difference-in-differences estimates from [Equation 3](#) for men and women aged 25 to 44. Estimates control for county fixed effects and state and time fixed effects. All estimates are weighted by group county level population and standard errors are clustered at the county level. Estimates include controls for county unemployment, percentage of population that is white, Ryan White title 1 funding eligibility, percentage of population that is evangelical protestant, percentage of population that adheres to a religion.

Table 5: Difference in Differences: Men aged 65+ and men under 19

	Men 65+			Men under 19		
	(1)	(2)	(3)	(4)	(5)	(6)
Post=1 \times Stdz 1994 HIV death rate	-0.16355 (0.323)	0.00197 (0.263)	0.03455 (0.201)	-0.06766 (0.084)	-0.05533 (0.085)	-0.04581 (0.089)
Population Density		-0.00569*** (0.002)	-0.00477*** (0.002)		-0.00035 (0.000)	0.00040 (0.001)
Unemployment Rate		0.12260 (0.206)	0.12563 (0.300)		-0.09411 (0.057)	-0.07339 (0.067)
Percentage Pop White		-0.17883 (0.164)	-0.16314 (0.210)		-0.05438 (0.044)	-0.00933 (0.052)
Proportion Pop Evangelical		49.33260** (19.131)	0.91073 (21.295)		-7.07499 (6.799)	-15.17962** (7.705)
Proportion Pop Religious		-10.62841** (5.154)	-3.39449 (5.692)		3.88390** (1.783)	3.76248* (1.993)
Title 1 Eligible=1		-1.08891 (0.755)	-1.65386* (0.859)		-0.14091 (0.210)	0.05084 (0.250)
Observations	5070	4970	4866	5067	4967	4863
County FE	Yes	Yes	Yes	Yes	Yes	Yes
Time FE	Yes	Yes	No	Yes	Yes	No
State X Time FE	No	No	Yes	No	No	Yes

Standard errors in parentheses

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table 5. Source: CDC Multiple Cause of Death data [NCHS \(1990-2002\)](#)

Notes: Table presents difference-in-differences estimates from [Equation 3](#) for men over 65 and men under 25. Estimates control for county fixed effects and state and time fixed effects. All estimates are weighted by group county level population and standard errors are clustered at the county level. Estimates include controls for county unemployment, percentage of population that is white, Ryan White title 1 funding eligibility, percentage of population that is evangelical protestant, percentage of population that adheres to a religion.

Table 6: Triple Difference: Men aged 25-44 relative to women aged 25-44

	(1)	(2)	(3)	(4)
Post=1 \times male=1 \times Stdz 1994 HIV death rate	-0.96243*** (0.146)	-0.95850*** (0.146)	-0.95622*** (0.130)	-0.89336*** (0.156)
Population Density		-0.00117 (0.001)		
Unemployment Rate		0.14168** (0.071)		
Percentage Pop White		0.01491 (0.070)		
Proportion Pop Evangelical		8.56835 (10.315)		
Proportion Pop Religious		-1.30231 (2.137)		
Title 1 Eligible=1		-0.80935*** (0.291)		
Observations	10140	9940	10140	9958
County FE	Yes	Yes	No	No
Time FE	Yes	Yes	No	No
County X Time FE	No	No	Yes	Yes
Time X Sex FE	No	No	Yes	Yes
Sex X County FE	No	No	Yes	Yes
Time X Sex X State FE	No	No	No	Yes

Standard errors in parentheses

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table 6. Source: CDC Multiple Cause of Death data [NCHS \(1990-2002\)](#)

Notes: Table presents triple-difference estimates from [Equation 4](#) for men aged 25 to 44 relative to women aged 25 to 44. Estimates control for the interaction of sex and county fixed effects, sex and time fixed effects and state, sex and time fixed effects. All estimates are weighted by group county level population and standard errors are clustered at the county level.

Table 7: Triple Difference: Men aged 25-44 relative to men aged 65+ and Men aged 25-44 relative to men under 19

	Relative to: Men over 65		Relative to: Men under 19	
	(1)	(2)	(3)	(4)
Post=1 \times Men 25-44=1 \times Stdz 1994 HIV death rate	-0.87495** (0.358)	-0.92818*** (0.349)	-1.01339*** (0.147)	-0.97783*** (0.156)
Observations	10140	9958	10134	9952
County FE	No	No	No	No
Time FE	No	No	No	No
County X Time FE	Yes	Yes	Yes	Yes
Time X Age FE	Yes	Yes	Yes	Yes
Age X County FE	Yes	Yes	Yes	Yes
Time X Age X State FE	No	Yes	No	Yes

Standard errors in parentheses

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table 7. Source: CDC Multiple Cause of Death data [NCHS \(1990-2002\)](#)

Notes: Table presents triple-difference estimates from [Equation 4](#) for men aged 25 to 44 relative to men aged 65+ and Men aged 25-44 relative to men under 19. Estimates control for the interaction of age and county fixed effects, age and time fixed effects and state, age and time fixed effects. All estimates are weighted by group county level population and standard errors are clustered at the county level.

Table 8: Difference in Differences: Alternate age groups

	Men 20-29		Men 30-39		Men 40-49	
	(1)	(2)	(3)	(4)	(5)	(6)
Post=1 \times Stdz 1994 HIV death rate	-0.86307*** (0.251)	-0.97666*** (0.245)	-1.03428*** (0.221)	-1.03117*** (0.215)	-0.66340** (0.336)	-0.63776* (0.357)
Population Density	0.00352* (0.002)	0.00633*** (0.002)	-0.00397** (0.002)	-0.00231 (0.002)	-0.00768*** (0.002)	-0.00326 (0.002)
Unemployment Rate	0.08237 (0.169)	0.03616 (0.210)	0.17851 (0.150)	0.05419 (0.206)	0.56474*** (0.203)	-0.20805 (0.234)
Percentage Pop White	-0.26570 (0.175)	-0.08523 (0.190)	-0.09493 (0.125)	0.00131 (0.150)	0.21995 (0.170)	0.21384 (0.179)
Proportion Pop Evangelical	14.46651 (17.208)	-11.71189 (18.373)	23.59298 (20.749)	28.02732 (23.016)	3.51019 (19.802)	2.92832 (22.943)
Proportion Pop Religious	3.57914 (4.998)	5.49290 (5.629)	-1.74026 (4.375)	1.67780 (4.138)	-5.03902 (5.164)	3.14922 (5.422)
Title 1 Eligible=1	-1.49012** (0.700)	-1.28525* (0.717)	-1.36224** (0.582)	-1.23310* (0.691)	-0.20163 (0.667)	-0.27977 (0.714)
Observations	4970	4866	4970	4866	4970	4866
County FE	Yes	Yes	Yes	Yes	Yes	Yes
Time FE	Yes	No	Yes	No	Yes	No
State X Time FE	No	Yes	No	Yes	No	Yes

Standard errors in parentheses

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table 8. Source: CDC Multiple Cause of Death data [NCHS \(1990-2002\)](#)

Notes: Table presents difference-in-differences estimates from [Equation 3](#) for men aged 20-29, 30-39, and 40-49. Estimates control for county fixed effects and state and time fixed effects. All estimates are weighted by group county level population and standard errors are clustered at the county level. Estimates include controls for county unemployment, percentage of population that is white, Ryan White title 1 funding eligibility, percentage of population that is evangelical protestant, percentage of population that adheres to a religion.

Table 9: Triple Difference (Men relative to Women): Alternate age groups

	Men 20-29		Men 30-39		Men 40-49	
	(1)	(2)	(3)	(4)	(5)	(6)
Post=1 \times male=1 \times Stdz 1994 HIV death rate	-0.91218*** (0.312)	-0.99127*** (0.374)	-1.07565*** (0.250)	-0.97328*** (0.273)	-0.53483* (0.283)	-0.36952 (0.307)
Observations	10112	9930	10140	9958	10140	9958
County FE	No	No	No	No	No	No
Time FE	No	No	No	No	No	No
County X Time FE	Yes	Yes	Yes	Yes	Yes	Yes
Time X Sex FE	Yes	Yes	Yes	Yes	Yes	Yes
Sex X County FE	Yes	Yes	Yes	Yes	Yes	Yes
Time X Sex X State FE	No	Yes	No	Yes	No	Yes

Standard errors in parentheses

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table 9. Source: CDC Multiple Cause of Death data [NCHS \(1990-2002\)](#)

Notes: Table presents triple-difference estimates from [Equation 4](#) for men aged 20-29, 30-39, and 40-49 relative to women aged 20-29, 30-39, and 40-49 respectively. Estimates control for the interaction of sex and county fixed effects, sex and time fixed effects and state, sex and time fixed effects. All estimates are weighted by group county level population and standard errors are clustered at the county level.

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A Appendix

A.1 ICD Codes

Cause of Death	ICD 9 Code (1990 - 1998)	ICD 10 Code (1999 - 2002)
Suicide	E950-E959	U03, X60-X84, Y87.0
HIV/AIDS death	042	B20-B24
Firearm Suicide	E955	X72, X73, X74
Non Firearm Suicide	E950-E954, E956-E959	U03, X60-X71, X76-X84, Y87.0
Motor Vehicle Accident	E810-E825	V89.0, V89.2, V89.9

Appendix Table A1. ICD codes by cause of death

A.2 AIDS data

I obtain total annual city counts of AIDS cases from the AIDS Public Information Data set from the Department of Health and Human Services [CDC \(1990-2002a\)](#). The AIDS Public Information data set contains data through 2002 for each city that has a population of at least 500,000 people as per the 2000 census. AIDS represents the final and most severe stage of HIV infection. Before the introduction of combination drug therapy, a person diagnosed with AIDS had a life expectancy of 1-3 years.

The cumulative total of AIDS cases was an important factor in determining levels of federal funding at the city level. As per the 1990 Ryan White legislation, cities that reported a cumulative of 2000 AIDS cases to the Centers for Disease Control (CDC) by March 31st of a particular year became eligible for Title 1 funding.²⁸ [Dillender \(2021\)](#), finds that Title 1 eligibility significantly reduces HIV/AIDS deaths. We may be concerned that it has similar effects on suicide rates, and therefore I control for Title 1 eligibility. Using the AIDS case data, I impute Title 1 eligibility. I verify that my estimates match those provided by [Dillender \(2021\)](#) . There are other significant variations in Ryan White funding at the state level, however, the identification strategy employed in this paper controls for state-time fixed effects, and therefore we are less concerned with controlling for within-state differences in funding.

Due to a large number of counties with missing AIDS case data, I do not use AIDS case rates as a measure for HIV/AIDS incidence in my main analysis. However, to verify my findings, I repeat my analysis by changing the HIV incidence measure to AIDS cases instead of HIV/AIDS deaths and find similar effects.

²⁸Before the 1996 reauthorization, Title 1 eligibility required that the city experienced a cumulative total of 2000 AIDS cases but after the 1996 reauthorization, Title 1 eligibility required that the city experienced a cumulative total of 2000 AIDS cases in the past 5 years.

I repeat the analysis using AIDS case rate data as the treatment variable. As mentioned earlier, the AIDS Public Information Dataset only includes data from cities with populations over 500,000. Counties that lie outside cities are therefore dropped. [Table A2](#) presents summary statistics.

Table A2: Summary Statistics

	4th Quartile	3rd Quartile	2nd Quartile	1st Quartile
Male Suicide Rate	16.13 (6.26)	18.09 (7.71)	16.41 (7.19)	19.18 (7.66)
Female Suicide Rate	4.32 (2.32)	4.56 (2.48)	3.98 (2.35)	4.43 (2.88)
1994 AIDS Case Rate	58.89 (29.13)	30.88 (4.63)	18.32 (2.22)	11.22 (2.76)
Male HIV Death Rate	30.01 (48.68)	17.32 (22.05)	13.22 (21.07)	9.24 (14.16)
Female HIV Death Rate	5.39 (11.30)	2.82 (4.35)	2.08 (3.61)	0.96 (1.64)
1994 HIV Death Rate	28.72 (26.14)	17.19 (8.33)	13.28 (10.24)	8.37 (5.39)
Male Fire Arm Suicide Rate	9.08 (4.34)	10.36 (5.75)	9.46 (5.71)	11.28 (5.08)
Female Fire Arm Suicide Rate	1.46 (1.31)	1.54 (1.50)	1.28 (1.40)	1.42 (1.46)
Male Non Fire Arm Suicide Rate	7.05 (4.04)	7.73 (4.01)	6.95 (3.81)	7.90 (4.61)
Female Non Fire Arm Suicide Rate	2.86 (1.68)	3.02 (1.76)	2.70 (1.76)	3.01 (2.15)
Eligible for Title 1 funding by 1996	0.98 (0.14)	0.71 (0.45)	0.56 (0.50)	0.26 (0.44)
Unemployment Rate	5.58 (1.97)	5.30 (1.75)	5.12 (2.25)	5.45 (2.86)
Population Density	2,695.05 (3,037.16)	1,646.44 (1,744.34)	1,886.24 (1,911.38)	1,210.94 (1,128.67)
Percentage Pop Male	48.95 (0.99)	49.04 (1.09)	48.55 (0.86)	48.48 (1.00)
Percentage Pop White	73.73 (13.37)	79.95 (10.87)	81.77 (11.69)	86.16 (9.05)
Percentage Pop aged b/w 0 and 24	35.38 (3.53)	35.51 (3.64)	35.03 (2.28)	36.13 (3.98)
Percentage Pop aged b/w 25 and 44	33.75 (2.84)	32.53 (2.76)	32.46 (2.12)	31.01 (2.27)
Observations	3120	3016	2704	3276

mean coefficients; sd in parentheses

4th Quartile refers to counties in top 25 percentile of AIDS case rate in 1994

3rd Quartile refers to counties in 50th-75th percentile of AIDS case rate in 1994

2nd Quartile refers to counties in 25th-50th percentile of AIDS case rate in 1994

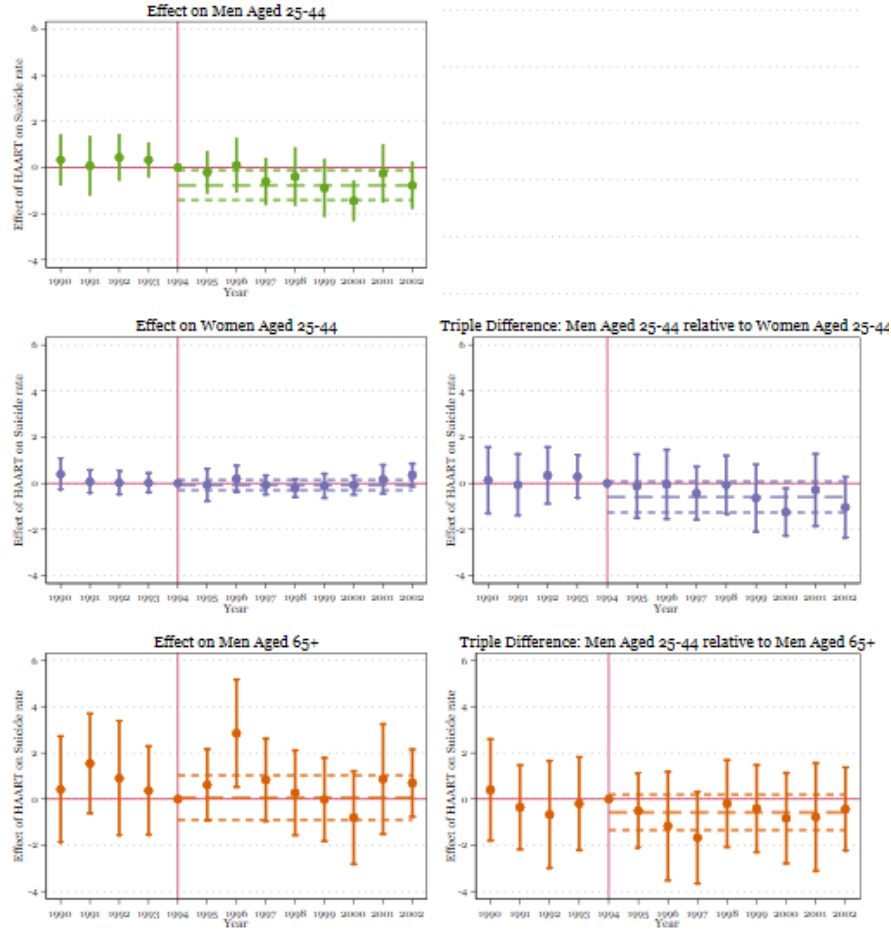
1st Quartile refers to counties in bottom 25 percentile AIDS case rate in 1994

Appendix Table A2. Source: The AIDS Public Information data set [CDC \(1990-2002a\)](#)

Notes: Table presents summary statistics for counties across the distribution of 1994 AIDS case rate for the years before the introduction of HAART (1990-1994).

Figure A1 presents Difference in Difference and Triple Difference results from Equation 1 and Equation 2 where $\text{Pre-HIV Incidence}_c$ now represents 1994 AIDS case rate (total AIDS cases per 100,000 population).

Figure A1: Effects using AIDS rate data



Appendix Figure A1. The figures on the left present difference in differences estimates from Equation 1 for men, women aged 25 to 44 and men over 65. Now, $\text{Pre-HIV/AIDS deathrate}_c$ represents the standardized form of 1994 AIDS case rate. Estimates include controls for county unemployment, percentage of population that is white, Ryan White Eligibility. I control for time fixed effects, county fixed effects and state and time fixed effects. The figures on the right presents Triple Difference Estimates from Equation 2 for men aged 25 to 44 relative to women aged 25-44 as well as men aged 25-44 relative to men over 65. I control for the interaction of time and county fixed effects, sex and county fixed effects, sex and time fixed effects and state, sex and time fixed effects. All estimates are weighted by group county level population and standard errors are clustered at the county level. Horizontal dashed lines present estimate and confidence intervals from specifications without time variations in treatment effect.

A.3 Inverse Hyperbolic Sine Transformation of Suicide Rates

The main analysis for this paper uses level suicide rates. Some researchers may argue that percentage changes are more relevant than level changes especially when comparing suicide rates across different groups (for example, women have lower rates of suicide compared to men). In order to account for this, I re-estimate [Equation 1](#) and [Equation 2](#) while applying the inverse hyperbolic sine transformation to my dependant variable. I do not use the natural logarithm because that does not retain zero valued observations.

My results are depicted in [Figure A2](#). I find larger standard errors when measuring effects on the inverse hyperbolic sin of suicide rates for several groups. Suicides by subgroup at the county level tend to be zero for many year-county-group combinations. Therefore, a unit change in the number of suicides performed would have a large effect on the percentage change of suicide rate. This explains the large standard deviations on the inverse hyperbolic sine transformation. I find that the estimates are qualitatively similar to those of my main analysis.

A.4 Accounting for Problems with data

A.4.1 Accounting for Suicide Misclassification

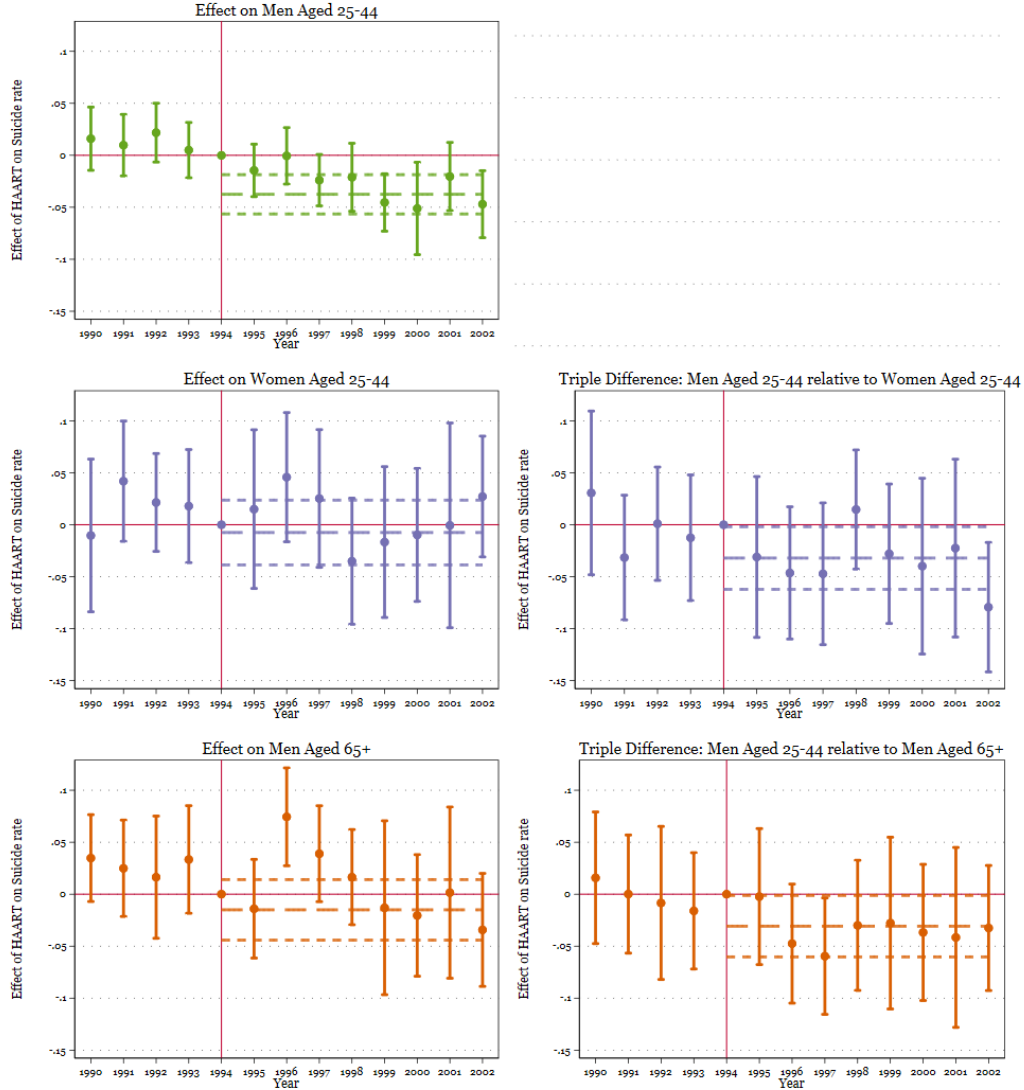
Some studies argue that a large portion of deaths classified as undetermined intent are in fact suicides [Björkenstam et al. \(2014\)](#). If there are geographic and gender differences in the proportion of suicides being classified as undetermined intent, this may affect our result. In order to account for this possibility, I include all deaths with undermined intent as suicides. Event study estimates are provided in [Figure A3](#).

A.4.2 Falsification Test

In order to ensure that my results are not being driven by problems in the way deaths are recorded in the Multiple Cause of Death data file, I conduct several falsification tests. My main analysis is based upon comparing suicide rates over time between counties and demographics with different rates of HIV/AIDS incidence. I now change my dependant variable to measure several leading causes of death for men. I only include leading causes of death that are unlikely to be impacted by the introduction of HAART. I provide triple difference estimates from [Equation 2](#) while changing my dependant variable to measure death rates from motor vehicle accidents, cerebrovascular diseases, heart diseases, and neoplasm. There is no reason for the introduction of HAART to affect these death rates.

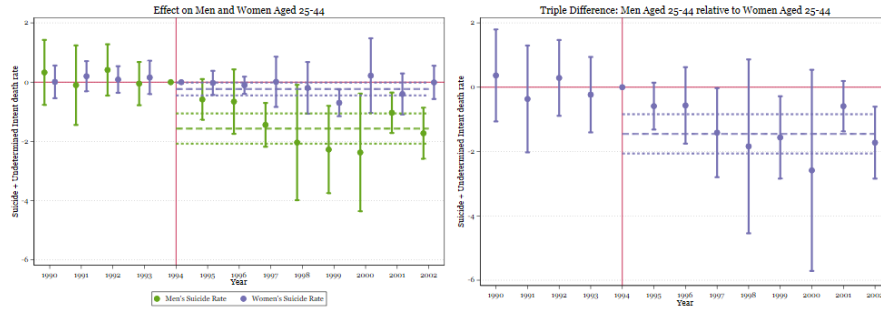
The Event Studies for this analysis are presented in [Figure A4](#). HAART appears to have little consequence on these causes of death.

Figure A2: Inverse Hyperbolic Sin Transformation



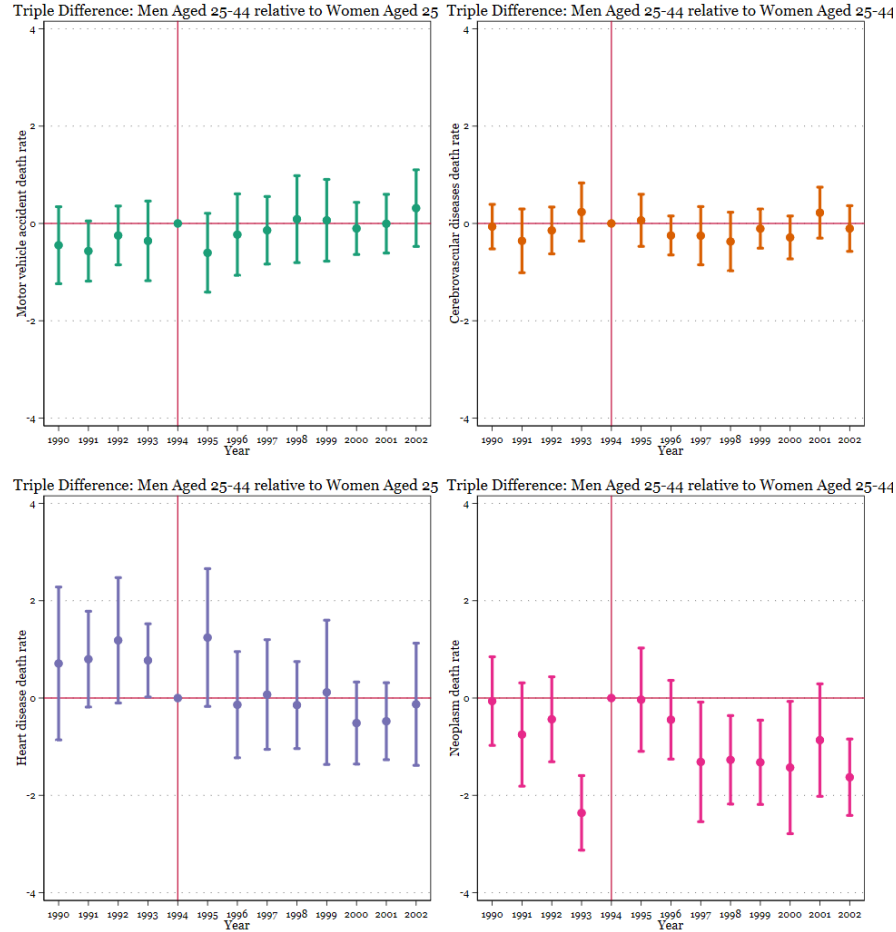
Appendix Figure A2. Figures on the left depict estimates for Equation 1 for men aged 25 to 44, women aged 25 to 44 and men over 65 after changing the dependant variable to represent the inverse hyperbolic sine transformation of suicide rates. Estimates include controls for county unemployment, percentage of population that is white, Ryan White Eligibility. I control for time fixed effects, county fixed effects and state and time fixed effects. The figure on the right presents Triple Difference Estimates from Equation 2 with women aged 25 to 44 and men over 65 as control groups. I control for the interaction of time and county fixed effects, sex and county fixed effects, sex and time fixed effects and state, sex and time fixed effects. All estimates are weighted by group county level population and standard errors are clustered at the county level. Horizontal dashed lines present estimate and confidence intervals from specifications without time variations in treatment effect.

Figure A3: Misclassified Suicide Deaths



Appendix Figure A3. The first figure presents difference in difference estimates from Equation 1 for men and women aged 25 to 44. I control for time fixed effects, county fixed effects and state and time fixed effects. Estimates include controls for county unemployment, percentage of population that is white, Ryan White Eligibility. The second figure presents Triple Difference Estimates from Equation 2 for individuals aged 25 to 44. I control for the interaction of time and county fixed effects, sex and county fixed effects, sex and time fixed effects and state, sex and time fixed effects. All estimates are weighted by group county level population and standard errors are clustered at the county level.

Figure A4: Falsification Tests



Appendix Figure A4. In these figures, I change the outcome variable represent death rates from motor vehicle accidents, cerebrovascular diseases, heart diseases, and neoplasm. I present Triple Difference Estimates from [Equation 2](#) for men aged 25 to women aged 25 to 44. I control for the interaction of time and county fixed effects, sex and county fixed effects, sex and time fixed effects and state, sex and time fixed effects. All estimates are weighted by group county level population and standard errors are clustered at the county level. Horizontal dashed lines present estimate and confidence intervals from specifications without time variations in treatment effect.

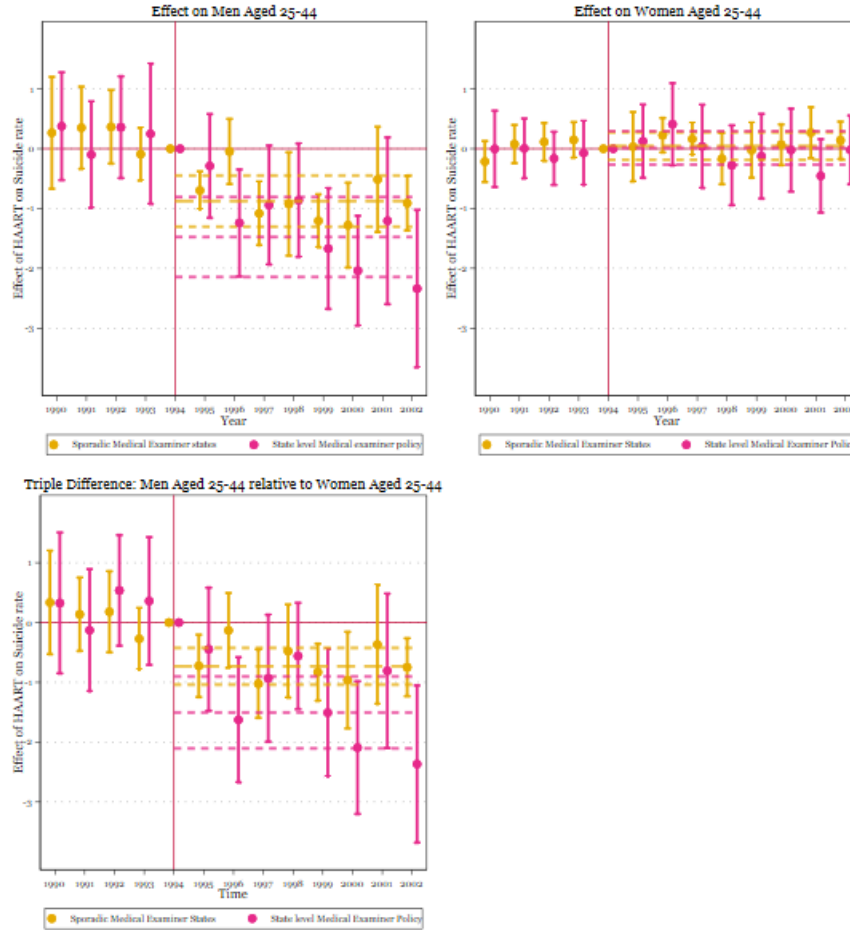
A.4.3 Coroners and Medical Examiners

Recent evidence suggests that geographic differences in Medicolegal Systems can affect suicide rates calculated using data abstracted from death certificates ([Fernández et al.2019](#) ; [Klugman et al. 2013](#)). There is spatial and temporal variation in who records the underlying cause of death on the death certificates. Medical examiners and coroners determine and report the cause of death when the death is sudden, violent, or untimely. In this section, I adjust my estimation equation and reestimate my results to rule out the possibility that effects are being driven by changes in Medicolegal system policies.

In general, there has been rise in medical examiner system overtime in the US. Many state and county coroner systems have converted to medical examiner systems. Since my analysis is at the county level and I control for state-year fixed effects, I am not concerned about changes in state level policies in Medicolegal systems. I am more concerned about states with sporadic county medical examiner systems. Therefore, I obtain the year at which counties switched from coroner systems to medical examiner systems for states with sporadic county medical examiner systems from [Hanzlick 2007](#).²⁹ Thereafter, I reestimate [Equation 1](#) and [Equation 2](#) for states with sporadic county medical examiner systems while including a control variable which equals 1 when a county has transitioned into a medical examiner system. I also provide estimates of [Equation 1](#) and [Equation 2](#) while dropping all states with sporadic medical examiner systems. Estimates for this specification are provided in [Figure A5](#). My estimates are similar to that of my main specification although my effects appear larger for states with state-level medical examiner policies.

²⁹These states include Alabama, California, Colorado, Georgia, Hawaii, Illinois, Minnesota, Missouri, New York, Ohio, Pennsylvania, Texas, Washington and Wisconsin

Figure A5: Coroners and Medical Examiners



Appendix Figure A5. In these figures, I reestimate [Equation 1](#) and [Equation 2](#) for states with sporadic county medical examiner systems (AL, CA, CO, GA, HI, IL, MN, MO, NY, OH, PA, TX, WA, and WI) while controlling for medical examiner system status and other states. In the first two figures, I present [Equation 1](#) for men and women aged 25 to 44. These figures control for county fixed effects and state and time fixed effects. Estimates include controls for county unemployment, percentage of population that is white, Ryan White title 1 funding eligibility, percentage of population that is evangelical protestant, percentage of population that adheres to a religion. In the third figure, I present Triple Difference Estimates from [Equation 2](#) for men aged 25 relative to women aged 25 to 44. I control for the interaction of time and county fixed effects, sex and county fixed effects, and state, sex and time fixed effects. All estimates are weighted by group county level population and standard errors are clustered at the county level. Horizontal dashed lines present estimate and confidence intervals from specifications without time variations in treatment effect

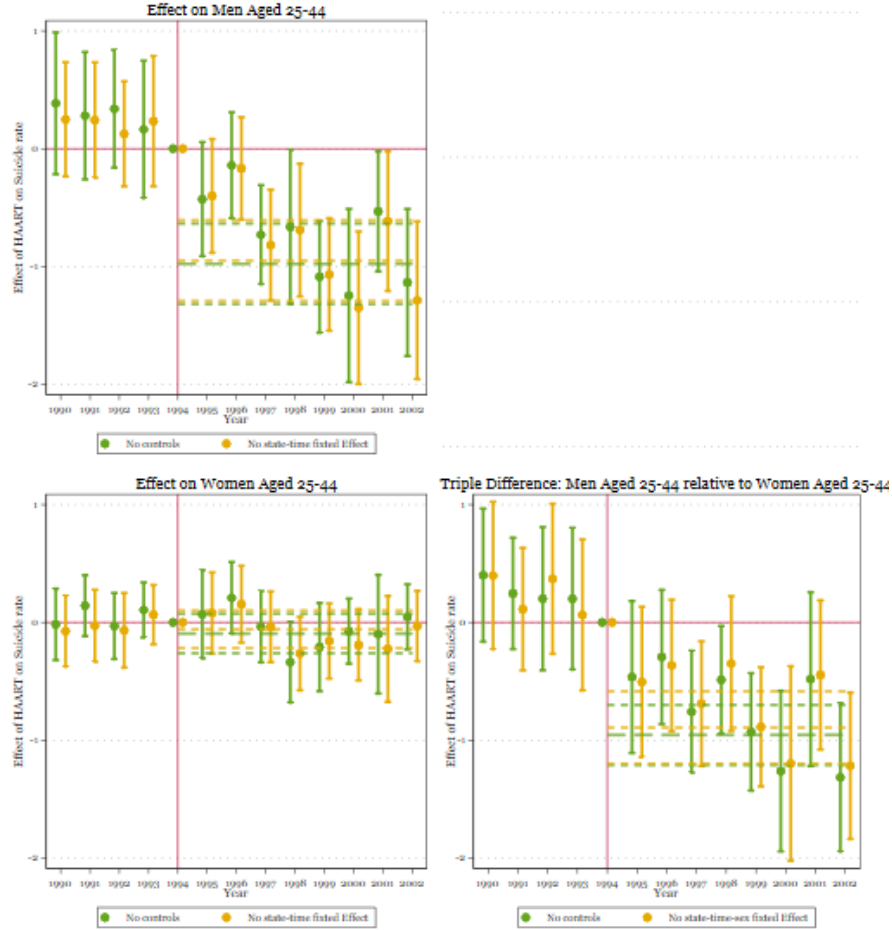
A.5 Removing Control Variables & Removing Additional Fixed Effects

Recent developments in the Difference-in-Difference literature argue that assuming parallel trends conditional on the inclusion of covariates might cause problems with the suggested estimation procedure [Sant’Anna and Zhao \(2020\)](#). Additionally, since I include state-time fixed effects in my difference in difference model and state-time-sex fixed effects in my triple difference equation, I am exploiting within state variation to conduct my analysis. Although this method accounts for changes in state policy which may be important, we might expect that our estimates are different when we consider across state variation. Therefore, I make the following changes:

1. I reestimate [Equation 1](#) without any control variables for men and women aged 25-44.
2. I reestimate [Equation 1](#) with the state-time fixed effect for men and women aged 25-44. Now, I only control for county and year fixed effects.
3. Since the fixed effects in [Equation 2](#) already account for any covariates which I control for, I present these estimates for men and women aged 25-44 once again.
4. I reestimate [Equation 2](#) while removing state-year-sex fixed effects for men and women aged 25-44. Now, I only control for county-year, county-sex, and sex sex-year fixed effects.

[Figure A6](#) shows that these changes don’t have large effects on my estimates.

Figure A6: Removing control variables



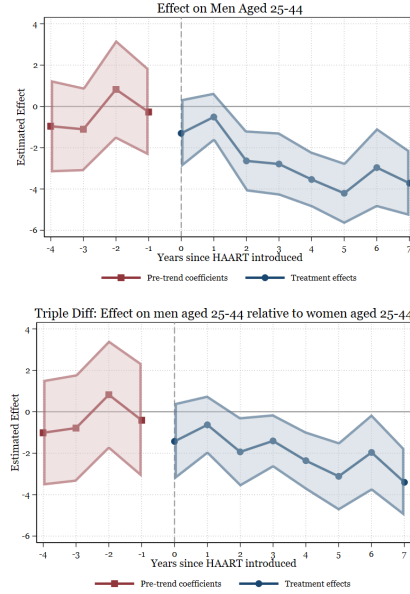
Appendix Figure A6. The figure presents difference in difference estimates from [Equation 1](#) for men and women aged 25 to 44. I control for time fixed effects, county fixed effects and state and time fixed effects. All estimates are weighted by group county level population and standard errors are clustered at the county level. Estimates do not include controls for county unemployment, percentage of population that is white, Ryan White Eligibility.

A.6 Alternative Estimator

Although my estimation strategy does not rely on the staggered timing of treatment, recent studies show that heterogeneous treatment effects can bias even non-staggered treatments (De Chaisemartin and d’Haultfoeuille 2022a; Borusyak et al. 2021). In order to account for these developments, I use the imputation estimator developed by Borusyak et al. 2021 and reestimate Equation 1 & Equation 2. Estimates are provided in Figure A7. Note that I use a binary treatment variable and considering counties in the top 10 percentile of pre-HAART HIV/AIDS death rates as treated states instead of the continuous treatment used in my main specification.³⁰

³⁰Current heterogeneity robust estimators with dynamic effects do not allow for continuous and non-staggered treatment (De Chaisemartin and d’Haultfoeuille, 2022b).

Figure A7: Alternative Estimator



Appendix Figure A7. Source: CDC Multiple Cause of Death data [NCHS \(1990-2002\)](#)
Notes: In the first figure I present estimates for [Equation 1](#) using the [Borusyak et al. \(2021\)](#) estimator. I control for county fixed effects and state-time fixed effects. Estimates include controls for county unemployment, percentage of population that is white, Ryan White title 1 funding eligibility, percentage of population that is evangelical protestant, percentage of population that adheres to a religion. The second figure presents triple-difference estimates from [Equation 2](#) for individuals aged 25 to 44. This figure controls for the interaction of sex and county fixed effects, sex and time fixed effects and state, sex and time fixed effects. All estimates are weighted by group county level population and standard errors are clustered at the county level.

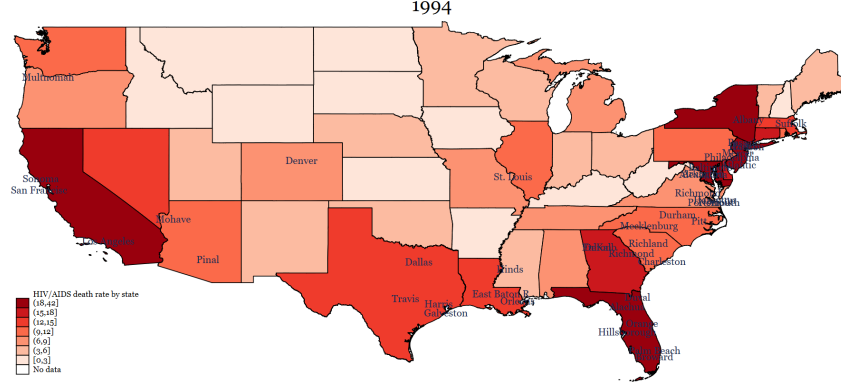
A.7 State Level Analysis

The analysis conducted in this paper exploits county level variation in pre-HAART HIV/AIDS death rates. Conducting county-level analysis rather than state-level analysis has the benefit of exploiting a greater breadth of variation in pre-HAART HIV/AIDS death rates. However, due to confidentiality rules, I only observe county codes for counties with populations exceeding 100,000. I observe state codes for all years. In order to ensure that my analysis is not affected by this selection process, I

conduct my analysis at the state-level using all the data I have at hand.

First, I present a map of the US prior to the introduction of HAART. I also list counties in the top 10 percentile of HIV/AIDS death rates.

Figure A8: Map showing 1994 HIV/AIDS death rate by state



Appendix Figure A8

In order to conduct state-level analysis, I estimate the following equation:

$$\text{Suicide Rate}_{st} = \alpha + \sum_{\substack{m \neq 1994 \\ m=1990}}^{2002} \beta_m (\text{Pre-HIV/AIDS deathrate}_t \times 1[t = m]) + a_t + \mu_s + \epsilon_{st} \quad (5)$$

Now, Suicide Rate_{st} now measures the number of suicides per 100,000 population in state s at year t for men aged 25 to 44. $\text{Pre-HIV/AIDS deathrate}_s$ is the number of HIV/AIDS deaths per 100,000 population in 1994 in state s . a_t and μ_s represent state and year fixed effects. I also estimate this equation for women aged 25-44.

Thereafter, I estimate the following triple difference equation:

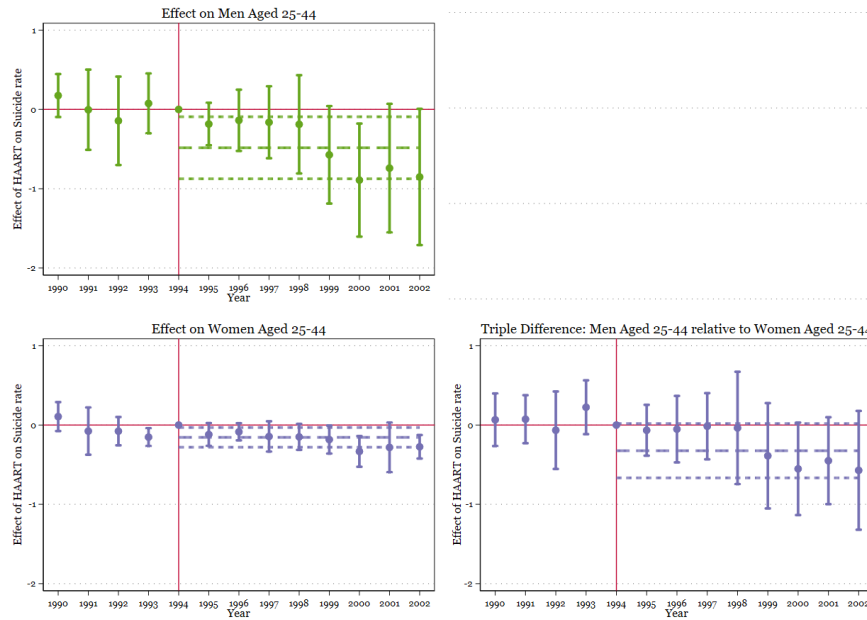
$$\text{Suicide Rate}_{jst} = \alpha + \sum_{\substack{m \neq 1994 \\ m=1990}}^{2002} \beta_m (\text{Pre-HIV/AIDS deathrate}_s \times 1[t = m] \times \text{Men 25-44}_j) + \theta_{st} + \iota_{js} + \delta_{jt} + \epsilon_{ctj} \quad (6)$$

where SuicideRate_{jst} measures state-level group-specific suicide rates.

Men 25-44_j is a dummy variable which measures whether the observation represents

suicide rates for men aged 25 to 44. In my triple difference analysis, I compare effects on men aged to 25 to 44 to women aged 25 to 44. Results presented below:

Figure A9: State level analysis



Appendix Figure A9. The figures on the left presents difference in difference estimates from Equation 5 for men and women aged 25 to 44 at the state level. I control for time fixed effects and state fixed effects. The third figure presents triple difference estimates from Equation 6. Here, I control for state-time fixed effects, sex-state fixed effects and sex-time fixed effects. All estimates are weighted by group county level population and standard errors are clustered at the state level.

The estimates presented in Figure A9 show that the introduction of HAART lead to statistically significant reductions in suicide rates for men aged 25 to 44 but not for women. Triple difference estimates suggests that state level analysis yields muted results. This highlights the importance of using disaggregated data.

Since the treatment variable in the main analysis is the standardized form of county-level pre-HAART HIV/AIDS death rates and the treatment variable for the state-level analysis is the standardized form of state-level pre-HAART HIV/AIDS death rate, we cannot compare coefficients. 1 standard deviation in pre-HAART

HIV/AIDS death rates at the state level is smaller than 1 standard deviation in pre-HAART HIV/AIDS death rates at the county level.

A.8 Deaths of Despair

Economists have documented the rise in alcohol, drug, and suicide deaths among Americans without a college degree between 1999 and 2013 [Case and Deaton \(2021\)](#). These deaths are often lumped together as “deaths of despair” and could all be the result of poor mental health. Since this study documents such large decreases in suicide rates, I also examine the effect of HAART on other deaths of despair. I reestimate [Equation 1](#) and [Equation 2](#), changing the outcome variable from suicide rates to alcohol-related deaths and drug-related deaths. The relevant ICD codes are provided in [subsection A.1](#). The first panel of [Figure A10](#) shows trends in each death of despair over time.

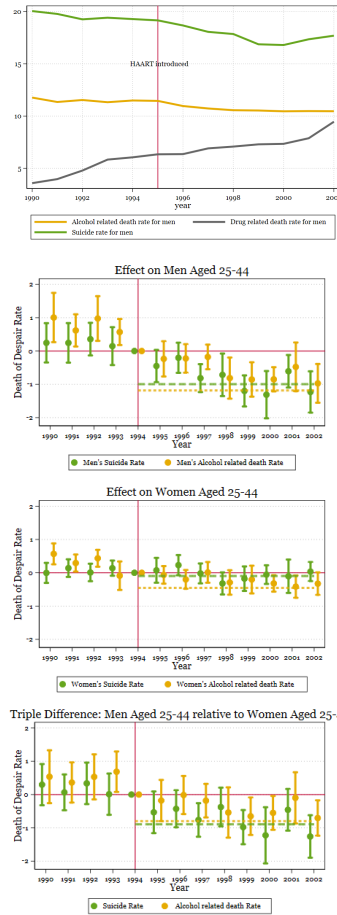
Estimating the effect on drug-related deaths proves difficult because drug-related deaths are less common, especially in the early years of our analysis. During our period of study, drug-related deaths are increasing rapidly. This results in my estimates being noisy and having large standard errors. Therefore, in my analysis, I only focus on suicide and alcohol related deaths³¹. I provide estimates for [Equation 1](#) for men aged 25 to 44 and women aged 25 to 44 for suicide rates and alcohol-related death rates in the second and third panels of [Figure A10](#). I find that men experience a large decline in alcohol-related deaths and women experience a small decline. Thereafter, I provide triple-difference estimates that show that the effect of HAART on alcohol related death rates is similar to the effect of HAART on suicide rates.

Although this estimate provides additional evidence of improved mental health outcomes in high-HIV counties for those at the highest risk of contracting HIV assuming that alcohol-related deaths are a result of poor mental health, understanding the effect of HAART on alcohol consumption and death requires

³¹Estimates for drug-related deaths are provided in the appendix.

further analysis. Unlike suicides, alcohol-related deaths need not be the result of poor mental health conditions. There is also a body of literature exploring the interaction of HAART treatments and alcohol consumption [Kumar et al. \(2012\)](#). Further research is required to better understand the effect of HAART on alcohol consumption and alcohol-related deaths.

Figure A10: Deaths of Despair



Appendix Figure A10. Source: CDC Multiple Cause of Death data [NCHS \(1990-2002\)](#)

Notes: In the first figure, I present trends in death rates by suicide, alcohol consumption, and drug related deaths. Thereafter, I present estimates for [Equation 1](#) while changing my outcome variable to alcohol related death rate in addition to providing estimates for suicide rate. The second and third figure present difference-in-differences estimates from [Equation 1](#) for men and women aged 25 to 44. These figures control for county fixed effects and state and time fixed effects. Estimates include controls for county unemployment, percentage of population that is white, Ryan White title 1 funding eligibility, percentage of population that is evangelical protestant, percentage of population that adheres to a religion. The fourth figure presents triple-difference estimates from [Equation 2](#) for individuals aged 25 to 44. This figure controls for the interaction of sex and county fixed effects, sex and time fixed effects and state, sex and time fixed effects. All estimates are weighted by group county level population and standard errors are clustered at the county level.

A.9 Altering the Functional Form

There are systematic differences between high and low HIV counties as depicted in [Table 3](#). High HIV counties tend to be more urban and have higher population densities. To ensure that my effects are not driven solely by differing trends in sex-specific suicide rates for densely and sparsely populated counties, I alter the functional form in an attempt to disentangle these effects. I also allow for non-linear relationships between Pre-HAART HIV/AIDS incidence and population densities by including squared terms. More formally, I estimate the following equation:

$$\begin{aligned}
 \text{SuicideRate}_{ct} = & \alpha + \beta_0 \text{Pre-HIV/AIDS deathrate}_c \times \text{Post}_t + \beta_1 \text{PopDense}_c \times \text{Post}_t \\
 & \beta_2 \text{PopDense}_c \times \text{Pre-HIV/AIDS deathrate}_c \times \text{Post}_t + \beta_4 \text{PopDense}_c^2 \times \text{Post}_t \\
 & + \gamma \mathbf{X}_{ct} + a_c + \eta_t + \mu_{st} + \epsilon_{ct}
 \end{aligned} \tag{7}$$

Here, PopDense_c represents the 1994 population density. I include the interaction of PopDense_c and Post_t , the interaction of the square of PopDense_c and Post_t , the interaction of PopDense_c , Pre-HAART HIV incidence, and Post_t as well as the interaction of the square of Pre-HAART HIV incidence and Post_t . All other features of this equation reflect [Equation 3](#). These additional terms allow us to assess whether the effects are driven by differential trends in more densely populated areas or whether effects are driven by underlying HIV/AIDS incidence.

The results for [Equation 7](#) are provided in [Table A3](#). Even after accounting for differential effects in more densely populated counties, I find that higher HIV counties experience a disproportionate fall in suicide rates for men aged 25 to 44. I also find no effects on women.

Although the only significant interaction term in my results table is the coefficient

for Pre-HIV/AIDS deathrate_c × Post_t, the partial effect of an increase in pre-HAART HIV before and after the introduction of HAART on suicide rates also depends on other terms. I estimate this partial effect using the following equation:

$$\frac{\partial \Delta \text{Suicide Rate}_{ct}}{\partial \text{Pre-HIV/AIDS deathrate}_c \Delta \text{Post}_t} = \beta_0 + \beta_2 \text{PopDense}_c + 2\beta_3 \text{Pre-HIV/AIDS deathrate}_c \quad (8)$$

Equation 8 measures the partial effect of HIV incidence before and after the introduction of HAART on suicide rates. I insert coefficients from my results table and values from my dataset to estimate partial effects across the distribution of pre-HAART HIV/AIDS deaths and population densities. I present these effects across percentiles of pre-HAART HIV/AIDS deaths and population densities in Figure A11. I insert pre-HAART HIV/AIDS death rate values from several points in the pre-HAART HIV /AIDS death rate distribution and estimate Equation 8 across the distribution of population densities. Here, I find some evidence that the effect of a 1 standard deviation increase in pre-HAART HIV/AIDS death rate is slightly smaller for counties with higher pre-HAART HIV/AIDS death rates.³²

According to the figure, there is approximately a 1 per 100,000 person decrease in suicides in counties with a 1 standard deviation higher pre-HAART HIV/AIDS death rate for most of the distribution of pre-HAART HIV/AIDS incidence and population densities. This estimate is similar to our main specification providing some evidence that these effects are not being driven by differential trends in more densely populated counties.

³²Note, the effect of a standard deviation increase in pre-HAART HIV/AIDS death rate implied in Figure A11 is different from the overall effects depicted in Figure 7.

Table A3: Alternative Functional Form

	Male (1)	Female (2)
Post=1 \times 1994 Population Density	-0.0002372894 (0.00028035)	-0.0000762272 (0.00015862)
Post=1 \times Standardized 1994 HIV death rate	-1.0984083416*** (0.34938719)	-0.2695155861 (0.19306975)
Post=1 \times 1994 Population Density \times Standardized 1994 HIV death rate	-0.0000417030 (0.00007358)	0.0000613873 (0.00004575)
Post=1 \times 1994 Population Density Squared	0.0000000086 (0.00000003)	-0.0000000076 (0.00000002)
Post=1 \times Standardized 1994 HIV death rate Squared	0.1013751044 (0.09800664)	-0.0263399285 (0.06070058)
1994 Population Density	0.0010606808 (0.00151360)	-0.0006765447 (0.00061887)
Unemployment Rate	-0.0408651843 (0.15431379)	0.0524218027 (0.08017326)
Percentage Pop White	0.0786624214 (0.13311908)	0.0684928191 (0.05551084)
Ryan White Title 1 eligible=1	-0.8889187543* (0.52419174)	-0.2016953969 (0.24437133)
Observations	4914	4914
County FE	Yes	Yes
Time FE	Yes	Yes
State X Time FE	Yes	Yes

Standard errors in parentheses

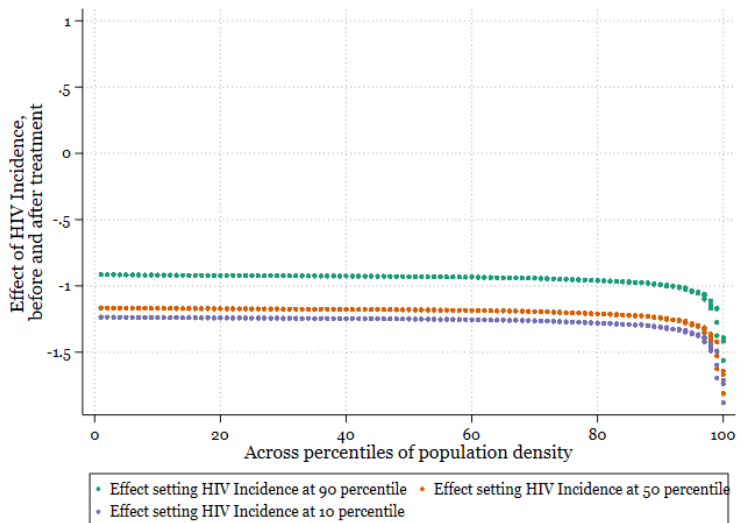
Effect of HAART on Suicide Rates

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Appendix Table A3. Source: CDC Multiple Cause of Death data [NCHS \(1990-2002\)](#)

Notes: Table presents alternative difference-in-differences estimates from [Equation 7](#) for men and women aged 25 to 44. Estimates control for county fixed effects and state and time fixed effects. All estimates are weighted by group county level population and standard errors are clustered at the county level. Estimates include controls for county unemployment, percentage of population that is white, Ryan White title 1 funding eligibility, percentage of population that is evangelical protestant, percentage of population that adheres to a religion.

Figure A11: Altering the Functional Form



Appendix Figure A11. Source: CDC Multiple Cause of Death data [NCHS \(1990-2002\)](#)

Notes: Estimates for [Equation 8](#) using coefficients from [Equation 7](#) and data from my dataset. I set pre-HAART HIV/AIDS death rate at the 10th, 50th and 90th percentile and estimate [Equation 8](#) across the distribution of Population Densities. Estimates include controls for county unemployment, percentage of population that is white, Ryan White title 1 funding eligibility, percentage of population that is evangelical protestant, percentage of population that adheres to a religion. All estimates are weighted by group county level population and standard errors are clustered at the county level.

A.10 Homicides

As mentioned earlier, the 90s was a period of falling urban violence and crime. Given the disproportionate impact of violence on young men in densely populated areas which tend to also be high HIV/AIDS areas, the results in this paper may be influenced by factors linked to trends in violence, independent of HAART. In this section, I compare trends and effects on homicides and suicides in order to convince the reader that the effects outlined in this paper are not driven by broader trends in violence.

The first panel in [Figure A12](#) depicts trends in homicide and suicide rates for men aged 25 to 44 living in the highest decile of 1994 HIV/AIDS death rate. Although we observe a fall in both suicide and homicide rates for this group, the fall in suicides occurs after the introduction of HAART in 1995 whereas homicides have been on a downwards trend since the early 90s. In the next panel of [Figure A12](#), I present trends in homicide rates for men aged 25-44 living in counties that are in the top decile of 1994 HIV/AIDS death rates and bottom 3 deciles. Similarly, the third panel of [Figure A12](#) depicts trends for women aged 25-44. Since the fall in homicide rate is driven by areas of high HIV/AIDS, it is important to ensure that the effects highlighted in this paper are driven by the introduction of HAART and not broader trends in violence.

The first panel of [Figure A13](#) presents estimates from [Equation 2](#) while changing the outcome variable to homicide rates. Given that the fall in homicides is driven by changes in high HIV/AIDS counties it is unsurprising that we observe negative effects. Unlike the estimates for suicide rate, there are very strong pre-trends and there does not appear to be a discontinuity around the time of HAART introduction. This highlights the importance of using event study models which allow us to observe pre-existing trends. These effects are likely a result of falling

homicide rates in more densely populated areas.

In order to account for this possibility, I reestimate the following equation:

$$\begin{aligned} \text{Homicide Rate}_{jct} = & \alpha + \sum_{\substack{m \neq 1994 \\ m=1990}}^{2002} \beta_m (\text{Pre-HIV/AIDS deathrate}_c \times 1[t = m] \times \text{Men 25-44}_j) + \\ & \sum_{\substack{m \neq 1994 \\ m=1990}}^{2002} \theta_m (\text{PopDence}_c \times 1[t = m] \times \text{Men 25-44}_j) + \theta_{ct} + \iota_{jc} + \delta_{sjt} + \epsilon_{ctj} \end{aligned} \quad (9)$$

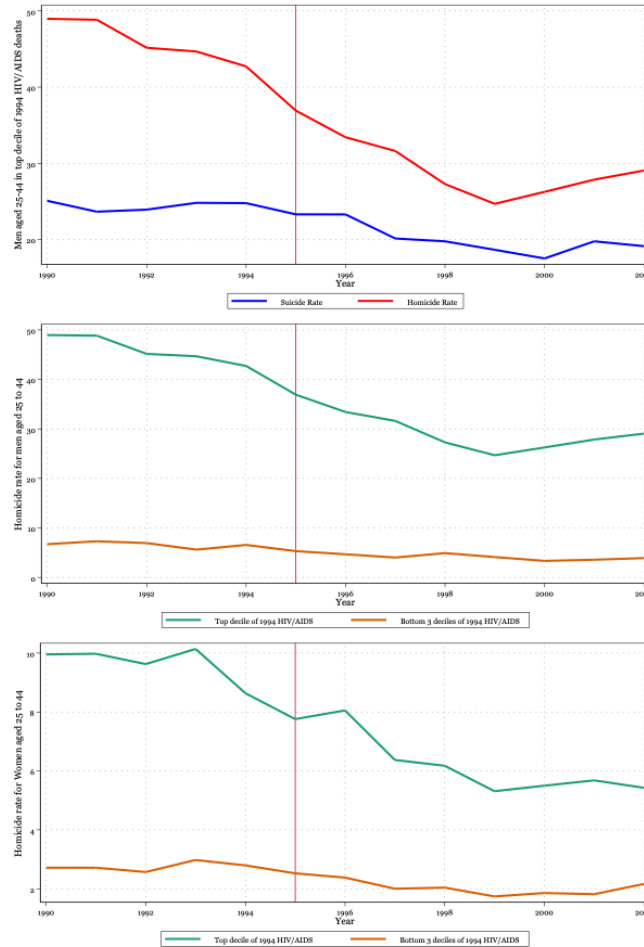
[Equation 9](#) is similar to [Equation 2](#) except it includes a term where I interact 1994 population density (PopDence_c) with the treatment variables. This should account for changing trends in homicides in more densely populated counties. Estimates for β_m are presented in the second panel of [Figure A13](#). We no longer observe pretrends and effects are much smaller and statistically not different from zero.

Since both men and women experience a fall in homicide rates in high HIV/AIDS counties relative to low HIV/AIDS counties and men have significantly higher rates of homicide, I consider percentage changes in addition to rates. I re-estimate [Equation 2](#) while changing my outcome variable to now represent the inverse hyperbolic sine transformation of homicide rates. Estimates are presented in the third panel of [Figure A13](#). The estimates are now flipped. In percentage terms, effects of women's homicide rates outweigh effects of men's homicide rate. This is in contrast to my findings in [Appendix A.3](#) where I change the outcome variable to the inverse hyperbolic sine of the suicide rate.

Taken together, we find that although homicides have been falling throughout my period of analysis, there does not appear to be a discontinuous change at the time HAART treatment was introduced. After accounting for changing trends by

population density, effects appear statistically not different from zero. When I change the outcome variable to represent percentage changes rather than rates, it appears that trends in homicide over this period are larger for women compared to men. This implies that trends in homicide rates do not match the effects on suicide highlighted in this study. Although there is no way to rule out the possibility that my effects are contaminated by broader trends in violence, it is unlikely since we do not see similar effects on homicides.

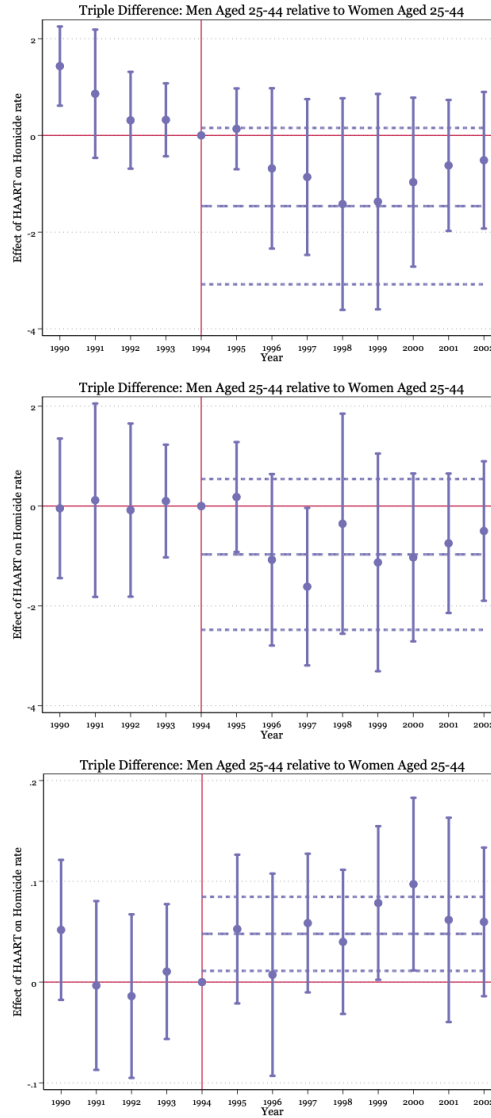
Figure A12: Trends in Homicide rates



Appendix Figure A12. Source: CDC Multiple Cause of Death data [NCHS \(1990-2002\)](#)

Notes: The above figures represent trends in homicide rates in 1990 to 2002. The first panel depicts homicide and suicide rates for men aged 25 to 44 living in high HIV/AIDS counties (counties in the top decile of per-HAART HIV/AIDS death rates). The second and third panel depict trends in homicide rates for men and women aged 25-44 living in high HIV/AIDS counties (counties in the top decile of per-HAART HIV/AIDS death rates) and low HIV/AIDS counties (counties in the bottom 3 deciles of per-HAART HIV/AIDS death rates)

Figure A13: Homicides



Appendix Figure A13. Source: CDC Multiple Cause of Death data [NCHS \(1990-2002\)](#)

Notes: The first panel depicts [Equation 2](#) while changing the outcome variable to homicide rate. The second panel presents estimates from [Equation 9](#). The third panel presents estimates from [Equation 2](#) while changing the outcome variable to represent the inverse hyperbolic sine transformation of homicide rates. I control for the interaction of time and county fixed effects, sex and county fixed effects, sex and time fixed effects and state, sex and time fixed effects. All estimates are weighted by group county level population and standard errors are clustered at the county level

A.11 Poor Mental Health Days

Thus far, this paper focuses on the effect of HAART on suicide rates. Suicides represent an extreme outcome resulting from poor mental health; however, poor mental health is often accompanied by several nonextreme outcomes. Mental health also plays an integral role in overall well-being, therefore we may also be concerned about mental health in and of itself. Due to the dearth of mental health data, this study is unable to estimate the causal effects of HAART on non-suicide mental health outcomes. Instead, I use data from the Behavioral Risk Factor Surveillance System (BRFSS) and provide suggestive evidence that the introduction of HAART reduced the average number of poor mental health days for certain at-risk groups.

The BRFSS is a national telephone survey that collects data from U.S. residents regarding their health-related risk behaviors, chronic health conditions, and use of preventive services. The BRFSS is considered to be representative at the state level but, since this paper exploits county-level variations in pre-HAART HIV/AIDS death rates, I conduct analysis at the county level and caution that this should only be considered suggestive evidence. The BRFSS only provides county codes for counties with over 50 respondents. Due to the smaller sample size, I only consider a discrete variable indicating HIV/AIDS intensity instead of the continuous variable used in my main analysis. Counties that are in the top 10 percentile of pre-HAART HIV/AIDS death rates as per the mortality data are classified as high HIV counties whereas all other counties are classified as low HIV counties.

Similar to my main sample, the BRFSS does not provide information about sexual orientation or the HIV status of respondents surveyed. I follow [Carpenter et al.2021](#) and exploit a key feature of the BRFSS to obtain a sample of the population that has a higher proportion of gay respondents. The BRFSS asks the respondent to report the number of adult males and adult females in the household. By

creating a sample that includes only households consisting of exactly two adult males, [Carpenter et al.2021](#) argues that we can construct a sample with a greater proportion of gay respondents. Since 2014, some states have added questions about sexual orientation to their survey. Carpenter uses this data and shows that when we restrict the sample to respondents who are over 25 and reside in households consisting of exactly two adult males, the sample consists of 26.1% respondents who identify as gay relative to only 1.8% of respondents who identify as gay overall.

The BRFSS also provides information about self-reported mental health from 1993 onwards. The BRFSS asks the following question: “Now thinking about your mental health, which includes stress, depression, and problems with emotions, for how many days during the past 30 days was your mental health not good?” To study the effect of HAART on self-reported mental health, I look at responses to this questions among respondents aged 25 to 44 living in households comprised of exactly two same-sex adult males in high HIV/AIDS counties (top 10 percentile). I expect this group to be at a higher risk of contracting HIV, and therefore more likely to be affected by the introduction of HAART. I compare trends for this group to trends of other groups. For some of the years in my sample, the BRFSS also provides information about the self-assessed risk of contracting HIV. Below, I provide information about the proportion of respondents reporting medium or high risk of contracting HIV by the group in question. Same-sex male households residing in high HIV counties report a higher self-assessed risk of contracting HIV. This gives further support to my assertion that this group is more affected by the introduction of HAART.

Group	High or Medium Risk of contracting HIV
Same-Sex Male HH in high HIV county	0.215
Same-Sex Male HH in low HIV county	0.148
Single Men in high HIV county	0.129
Single Men in low HIV county	0.106
Diff Sex HH Male in high HIV county	0.0850
Diff Sex HH Male in low HIV county	0.057
Diff Sex HH Women in high HIV county	0.074
Diff Sex HH Women in low HIV county	0.058

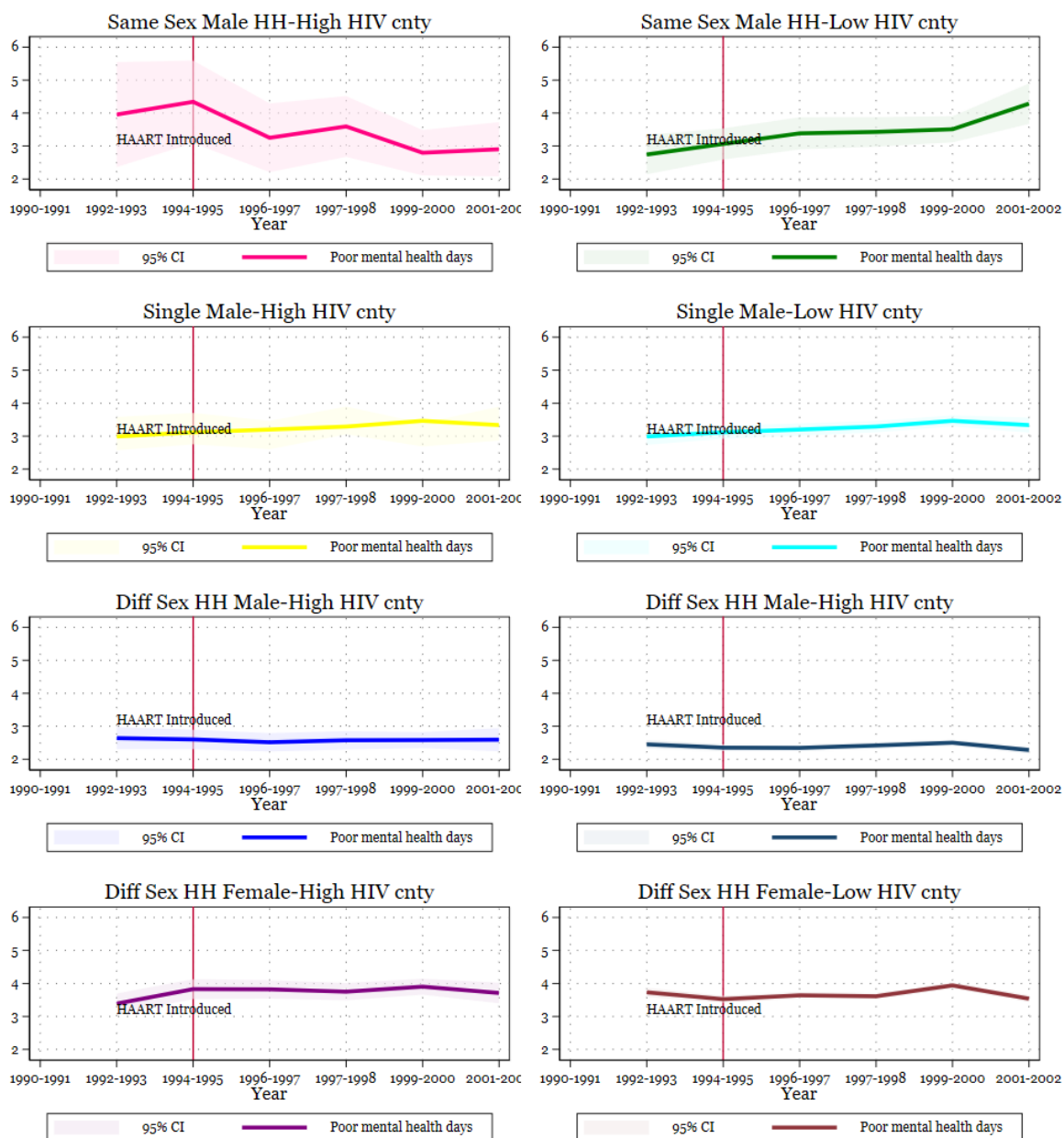
When I split my sample between the groups mentioned above and the years of the survey, the sample sizes become very small. To ensure that each group-year combination contains at least 50 respondents, I lump responses from every other year together.

I depict trends in the average number of poor mental health days for several groups in [Figure A14](#). I find that men aged 25 to 44 residing in households consisting of exactly two male adults in high HIV counties experience a steady decline in the average number of poor mental health days following the introduction of HAART. This is the only group that experiences such a reduction. No other group appears to experience a fall in the average number of days with poor mental health. This provides further evidence that these effects are a result of the introduction of HAART. Since frequent mental distress is an indicator of health-related quality of life and is characterized by 14 or more days of self-reported poor mental health in the past month, I construct an indicator variable that is equal to one when the reported number of days with poor mental health is equal to or greater than 14. Trends of this variable by group follow a similar pattern with respondents with over 14 days of poor mental health only decreasing for respondents

living in exactly two male adult households in high HIV counties.

Although using BRFSS data for this kind of analysis can be problematic because of the small sample size, the trends provide some suggestive evidence that apart from the introduction of HAART reducing suicide rates, HAART also improved non-extreme mental health outcomes for at-risk groups.

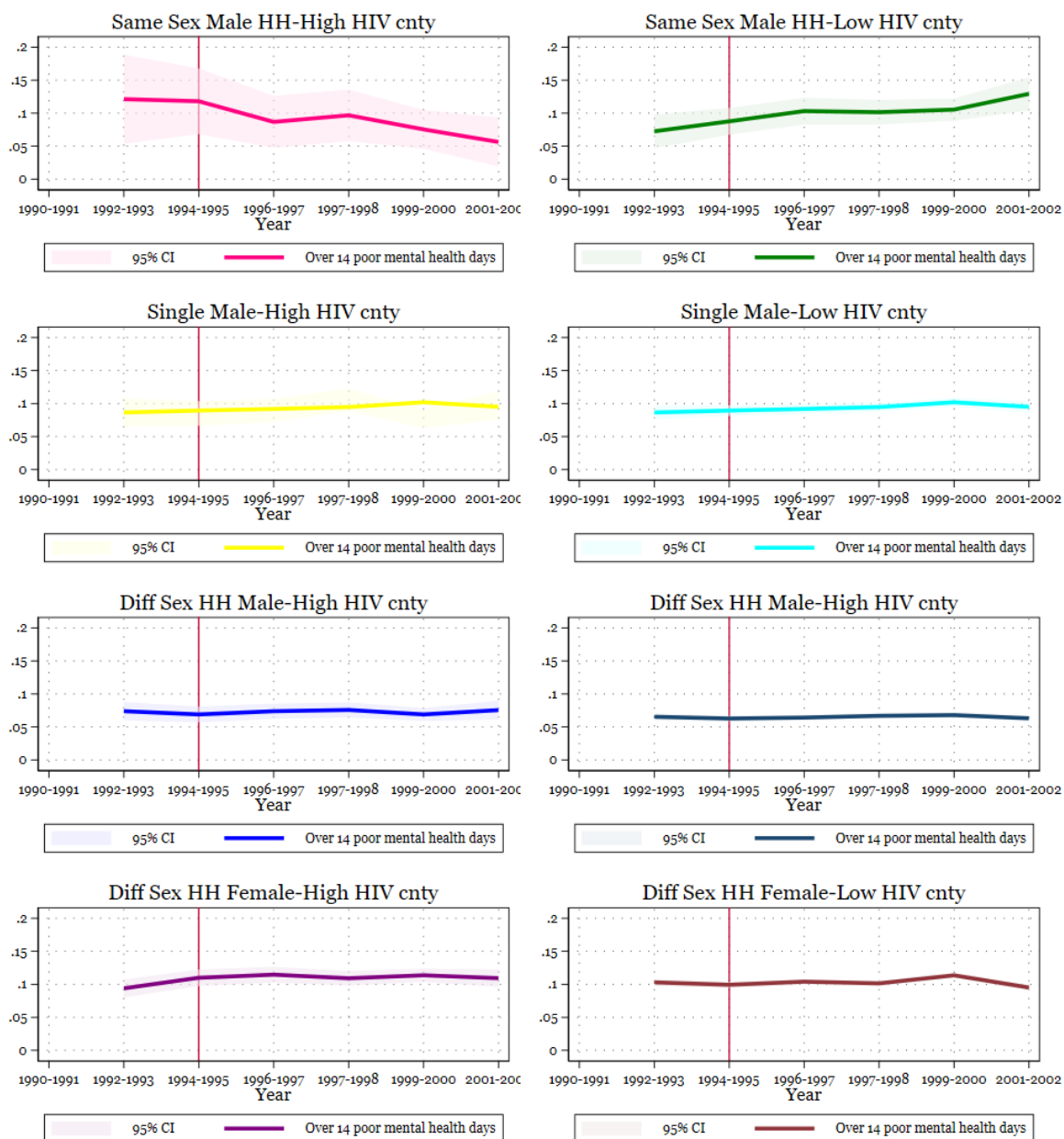
Figure A14 :Poor Mental Health Days in the last 30 days



Appendix Figure A14. Source: BRFSS 1990-2002 [CDC \(1990-2002b\)](#)

Notes: Trends for the average number of poor mental health days in the past 30 days by group.

Figure A15: Fourteen or More Poor Mental Health Days in the past 30 days



Appendix Figure A15. Source: BRFSS 1990-2002 [CDC \(1990-2002b\)](#)

Notes: Trends for the proportion of respondents reporting over 14 days of poor mental health days in the past 30 days by group.