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

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Abstract

Abstract: The HIV/AIDS crisis was associated with heightened homophobia, increased pressure to come out, and the risk of dying from a highly infectious disease. In 1995, the US Food and Drug Administration (FDA) approved Saquinavir, the first protease inhibitor. This ushered in a new era of combination drug therapies which became known as Highly Active Antiretroviral Therapy (HAART). HAART led to a substantial reduction in HIV-associated mortality and significantly altered the prognosis of HIV-positive individuals. The introduction of HAART led to a reduction in suicide risk among HIV-positive individuals (Keiser et al., 2010) and was accompanied by a reduction in homophobia in high HIV states compared to low HIV states (Fernández, Parsa, & Viarengo, 2019). Despite the clear mechanisms by which the introduction of HAART could improve mental health outcomes of at-risk groups, to the best of my knowledge, no study estimates the effects of HAART on suicide rates in the United States. 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, while controlling for other factors which might explain changes in suicide rates and finds that the introduction of HAART led to a disproportionate decrease in suicide rates for men aged 25 to 44. Estimates suggest that HAART saved approximately 1,200 men aged 25 to 44 from suicide each year following its introduction.

JEL codes: H51, I12, I18, J16, N31

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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. It was associated with heightened homophobia, an increased pressure to come out, and the risk of dying from a highly infectious disease. Heightened levels of suicide risk have been found among LGBTQ youth (who are also more likely to contract HIV) and HIV-infected patients. 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.

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, Heckman, Kochman, Sikkema, and Bergholte (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 were tested for HIV after HAART, indicating 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 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, the effects might be driven by the changing suicide rates among

HIV-negative gay men. Some reports suggest that the HIV crisis was accompanied with rising homophobia and stigma. Gay men also routinely mourned the deaths of their friends and fellow community members.[1] Additionally, some evidence suggests that, after the introduction of HAART, the crisis ultimately resulted in greater acceptance for same-sex couples in areas with greater exposure to the HIV crisis (Fernández et al., 2019). Several studies find that decreasing homophobia decreases the risk of suicide among LGBTQ populations (Raifman, Moscoe, Austin, and McConnell (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 exploits spatial variations in HIV incidence prior to the introduction of HAART and compares suicide rates across the distribution of pre-HAART HIV/AIDS incidence, in order to estimate the effects of HAART on suicide rates.

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 the distribution of pre-HAART HIV/AIDS incidence. I find that there is approximately a 0.9 per 100,000 population decrease in suicide rates for men aged 25 to 44 in counties with a preexisting HIV incidence rate that is 1 standard deviation higher. This estimate suggests that the introduction of HAART saved approximately 1,200 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 under 25). I also employ a triple-difference strategy, where I difference out effects on the suicide rates of less affected groups and find similar effects.

My findings contribute to a growing literature exploring the specific mental health

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

challenges experienced by the LGBTQ community and HIV-positive individuals. Sexual minorities experience elevated levels of suicide risk (Björkenstam, Andersson, Dalman, Cochran, and Kosidou (2016); Cochran and Mays (2015); Hottes, Bogaert, Rhodes, Brennan, and Gesink (2016); Raifman et al. (2017) ; Clark, Mays, Arah, Kheifets, and Cochran (2020)). 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, Hincapié, Kalish, and Papageorge (2021) ; Chan, Hamilton, and Papageorge (2016) ; Pelton et al. (2016); Lakdawalla, Sood, and Goldman (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) uses a longitudinal study conducted in Switzerland and finds that the introduction of HAART reduced suicide rates amongst HIV positive individuals. 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 rates in ways other than just HIV/AIDS mortality. Measures of total HIV/AIDS deaths may undercount the 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

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

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 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 viral load. The most common form of transmission is through intercourse and through the sharing of drug injection equipment. By 1987, men who have sex with

[3] Accessed at: https://www.nytimes.com/1981/07/03/us/rare-cancer-seen-in-41-homosexuals.html

[4] In his book, After the Wrath of God, Anthony Michael Petro documents the role of religious leaders in shaping moral debates surrounding the AIDS crisis in the US.

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

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 1.5 years. 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 proved 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 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 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

[6] Statistics 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 with men decreased overtime 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.

[7] Organizations 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.

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. To discern suicide and HIV/AIDS-related mortality, I 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, 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 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 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 1-2 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.[8] 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.

3.3 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. I calculate population density using population and land area data obtained from the 2000 census. Religious adherance 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).

3.4 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 in high and low HIV

[8]Before 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.

counties. High and low HIV counties have some different observable characteristics. Table 11 provides summary statistics by pre-HAART HIV/AIDS death rates. Here, I divide counties across quartiles of pre-HAART HIV/AIDS death rates. I find that 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 variation 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 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. In the appendix, 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. 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.

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:

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

where Suicide Rate_{ct} measures the number of suicides per 100,000 population in county c at year t for men aged 25 to 44. 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. $\mathbb{1}[t=m]$ is an indicator variable that equals 1 when the observations time period is m years relative to 1995.[9]

[9] I 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(Nov 21, 1995)). In 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.

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. 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.[10] 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 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 under 25). 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 under 25. 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:

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

where $SuicideRate_{jct}$ measures group-specific suicide rates. 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 under 25. Pre-HIV/AIDS deathrate$_c$ and $\mathbb{1}[t = m]$

[10] In the appendix 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.

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 specification, 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 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 and women differently.

4.2 Standard Specification

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:

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

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.[11] In subsection 6.2, 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 6.3, 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 relative

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

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

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

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-point 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 under 25 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 weren't.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 11. 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). For my main analysis, I provide results for men aged 25 to 44 and all three of my control groups. After showing that the three groups produce similar estimates, the remainder of this paper will only depict estimates for men aged 25 to 44, women aged 25 to 44, and triple-difference estimates. Although all groups might have different sets of reasons for dying by suicide, since these two groups are the same ages and have widely differing HIV/AIDS incidence, I believe this is the most intuitive control group.

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 a large change considering that the average suicide rate for the population of interest is 18 per 100,000 population. To further explore 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. Expectedly, I find similar but slightly smaller effects in my triple difference analysis depicted in the third panel of Figure 5.

Due to the large magnitude of estimates, I must argue 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.[12] 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). [13] 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.[14] 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.

6 Robustness Tests

Thus far, this study depicts falling suicide rates amongst men aged 25 to 44 in high HIV/AIDS counties relative to low HIV counties, 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 then 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 11, 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

[12] 20 HIV-positive individuals for each death is 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)

[13] 1 standard deviation increase in HIV deaths is equal to 12 HIV deaths

[14] $240 * \frac{447.4}{100{,}000} = 1.07$
$240 * \frac{90.1}{100{,}000} = 0.216$
$1.07 - 0.216 = 0.85$

different characteristics. I also do not observe actual HIV status or sexual orientation in the mortality data. I have argued that the effects of HAART would have significantly larger effects on HIV-positive and gay men but I am unable to observe if these groups are driving the observed 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, Blosnich, et al. (2020) finds that sexual minority men are significantly less likely to be gun owners compared to heterosexual men. Clark, Mays, et al. (2020) 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.

6.2 Altering the Functional Form

There are systematic differences between high and low HIV counties as depicted in Table 11. 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:

$$\text{Suicide Rate}_{ct} = \alpha + \beta_0 \text{ Pre-HIV/AIDS deathrate}_c \times \text{Post}_t + \beta_1 \text{PopDense}_c \times \text{Post}_t + \beta_2 \text{PopDense}_c \times \text{Pre-HIV/AIDS deathrate}_c \times \text{Post}_t + \beta_3 \text{Pre-HIV/AIDS deathrate}_c^2 \times \text{Post}_t + \beta_4 \text{PopDense}_c^2 \times \text{Post}_t \gamma \mathbf{X}_{ct} + a_c + \eta_t + \mu_{st} + \epsilon_{ct} \tag{5}$$

Here, PopDense_c represents the 1994 population density. I include the interaction of this term and Post_t, the interaction of the square of this term and Post_t, the interaction of this term, Pre-HAART HIV incidence, and Post_t and the interaction of the square of Pre-HAART HIV incidence and Post_t. All other features of this equation reflect Equation 3. I use the standard difference-in-differences specification because it allows me to conveniently present many coefficients in table form.

The results for Equation 6.2 are provided in Table 11. 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 $\text{deathrate}_c \times \text{Post}_t$, the partial effect of an increase in pre-HAART HIV before and after the introduction of HAART on suicide rates will include other terms. I estimate this partial effect using the following equation:

$$\frac{\partial \triangle \text{Suicide Rate}_{ct}}{\partial \text{Pre-HIV/AIDS deathrate}_c \triangle Post_t} = \beta_0 + \beta_2 \text{PopDense}_c + 2\beta_3 \text{Pre-HIV/AIDS deathrate}_c \tag{6}$$

The above equation 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 7. I insert pre-HAART HIV death rate values from several points in the pre-HAART HIV death rate distribution and estimate Equation 6 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.[15]

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.

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

The first panel in Figure 8 shows mean pre-HAART HIV/AIDS death rates by deciles of pre-HAART HIV/AIDS death rate in a county. As we move from one decile to the next, we see only small increases in pre-HAART HIV death rates in the first 6 deciles.

[15] Note, the effect of a standard deviation increase in pre-HAART HIV/AIDS death rate implied in Figure 7 is different from the overall effects depicted in Figure 8.

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. Results for effects on the 10th decile represent a reduced form of the results presented in Figure 5. The next two panels of Figure 8 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 8, 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 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, Eppink, Gonzales, and McKay(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 9. 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.

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 & 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 Appendix A.1. The first panel of Figure 11 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 deaths[16]. I provide estimates for equation 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 11. 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

[16]Estimates for drug-related deaths are provided in the appendix.

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 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.

9 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 12 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 12, 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 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.

10 Conclusion

This study highlights the substantial fall in suicide rates amongst 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, I find that suicide rates for men aged 25 to 44 fall by approximately 20% in high HIV counties relative to low HIV counties. My estimates suggest that the introduction of HAART saved approximately 1,200 men aged 25 to 44 from suicide each year. I 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 to use firearms as their method of suicide (Clark, Mays, et al., 2020). After breaking down suicides into firearm and non-firearm suicides, I find that my results are driven almost entirely by non-firearm suicides providing some evidence that my estimates are a result of changing suicide rates among gay men. I also change my functional form to disentangle any differential trends that occur in more urban counties relative to rural counties and find that my estimates are robust to alternative functional forms.

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 amongst 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 stud provides some evidence that results are largely driven by changing patterns of suicide amongst HIV-positive individuals after the introduction of HAART.

Since suicide is an outcome of poor mental health, I also explore the effect of HAART on self-reported mental health and other outcomes of poor mental health. I utilize BRFSS data and find suggestive evidence that the introduction of HAART reduced the reported number of poor mental health days for groups at the highest risk of contracting the virus. I also find some evidence that the introduction of HAART was accompanied by a fall in alcohol-related deaths for high-risk groups relative to other groups. 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 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 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 HAART 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.

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11 Figures & Tables

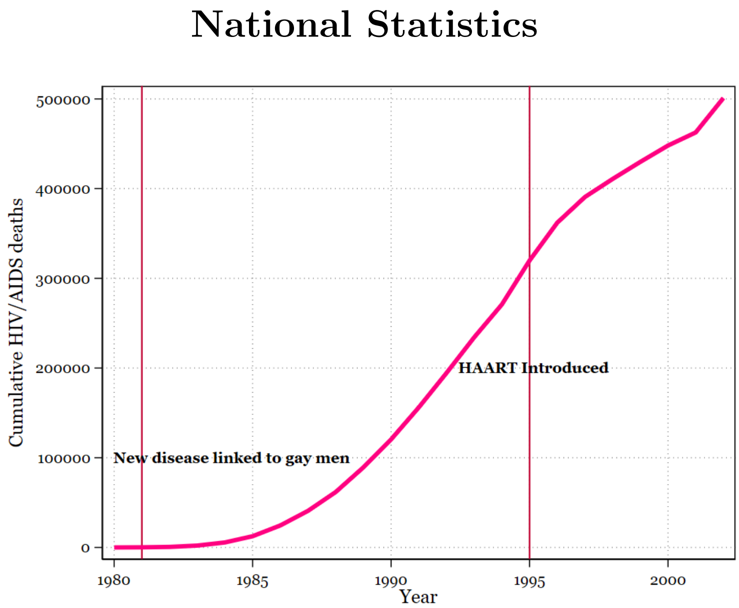


Figure 1: Source: amfAR: The Foundation for AIDS Research (https://www.amfar.org/thirty-years-of-hiv/aids-snapshots-of-an-epidemic)
Notes: Cumulative HIV/AIDS deaths over time.

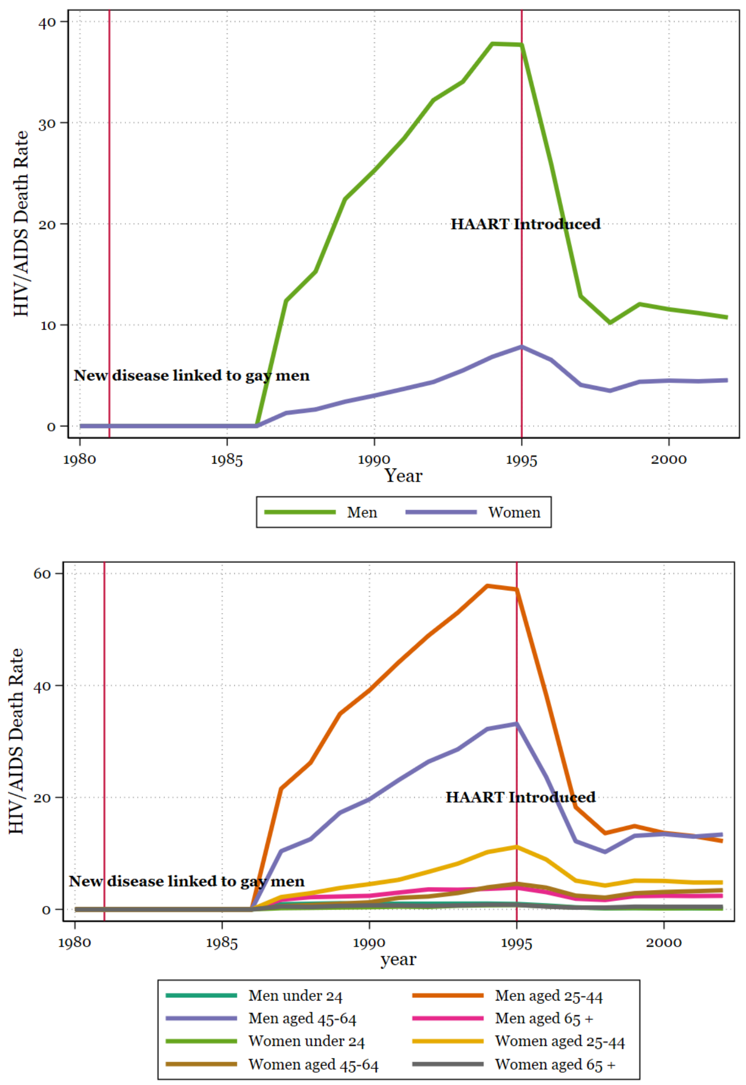


Figure 2: Source: CDC Multiple Cause of Death data (NCHS, 1990-2002)
Notes: HIV/AIDS death rates are calculated per 1,000,000 group population

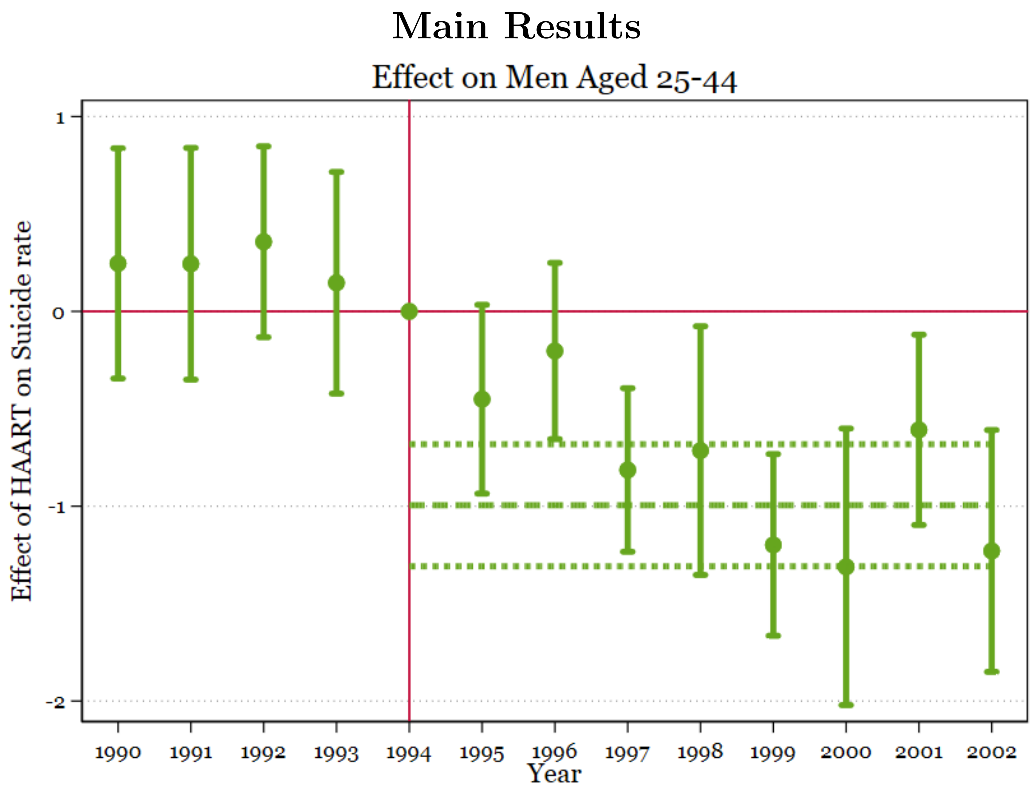


Figure 3: Source: CDC Multiple Cause of Death data (NCHS, 1990-2002)
Notes: Figure presents difference-in-differences estimates from Equation 1 for men aged 25 to 44. Estimates control for county fixed effects and state and time fixed effects. All estimates are weighted by group county level population 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.

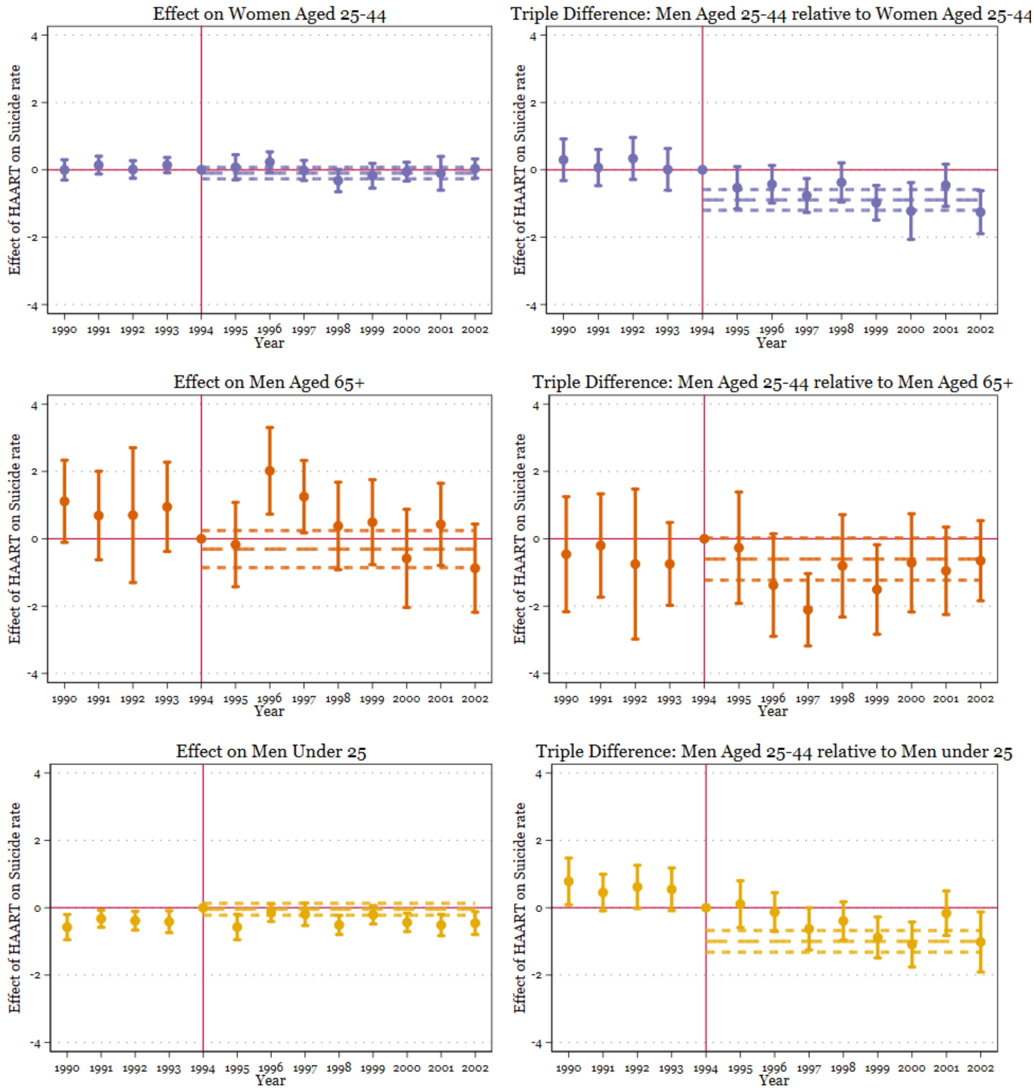


Figure 4: Source: CDC Multiple Cause of Death Data (NCHS, 1990-2002)
Notes: The first figure in each row presents difference-in-differences estimates from Equation 1 for women aged 25 to 44, men aged 65 plus, and men under 25. The second figure of each row presents triple-difference estimates from Equation 2 for men aged 25 to 44 to women aged 25 t0 44, men aged 65 plus, and men under 25. 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.

Effects Across the Distribution

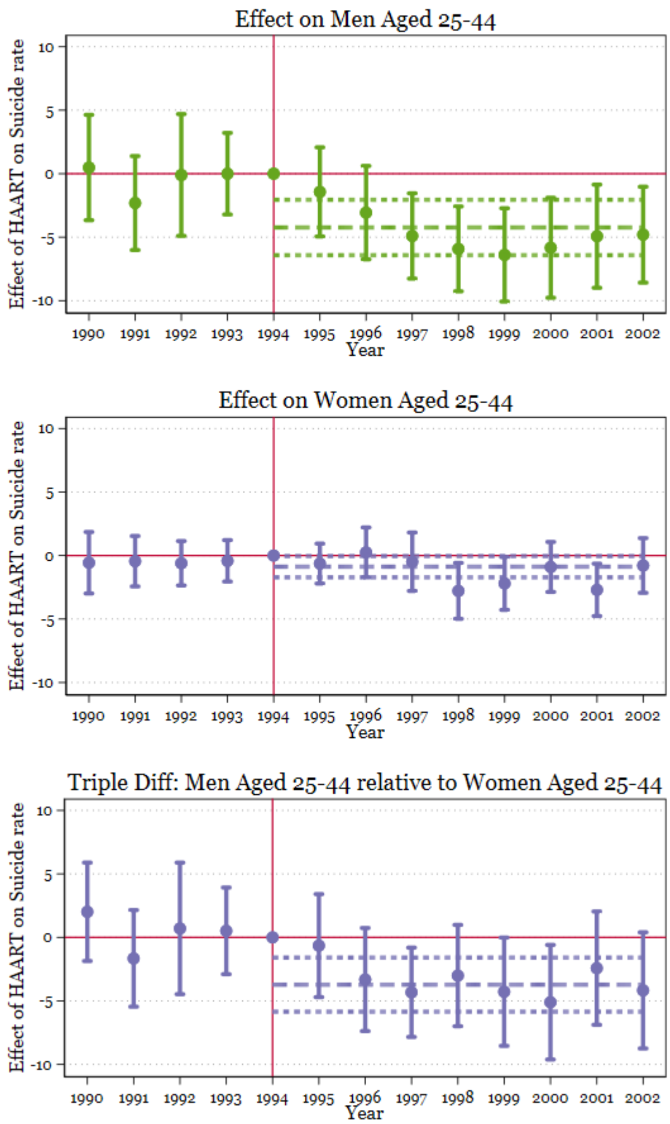


Figure 5: Source: CDC Multiple Cause of Death data (NCHS, 1990-2002)
Notes: These figures present estimates from Equation 1 and Equation 2, where, HIV Inc_c is a dummy variable that is equal to 1, when looking at the top decile of pre-HAART HIV/AIDS death rates and zero when looking at the bottom 3. 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.

Firearm & non-firearm Suicides

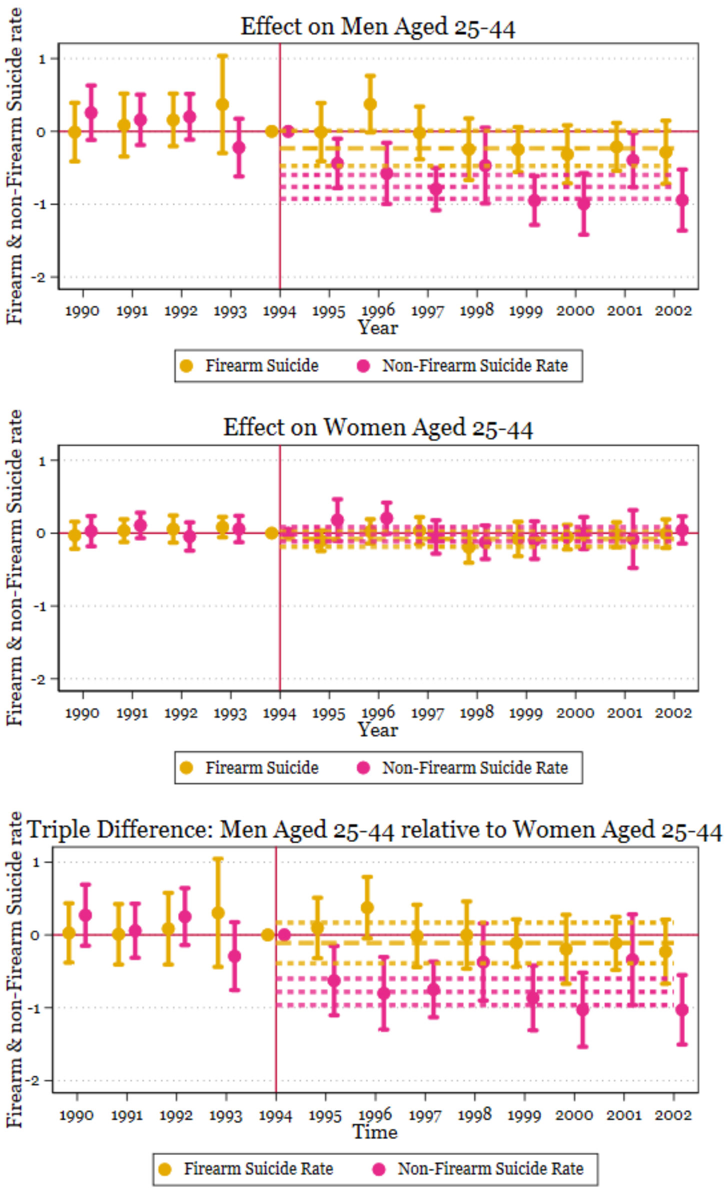


Figure 6: Source: CDC Multiple Cause of Death data (NCHS, 1990-2002)
Notes: In these figures, I change the outcome variable to represent firearm or non-firearm suicide rates instead of the overall suicide rate. First two figures present difference-in-differences estimates from Equation 1 for men and 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.

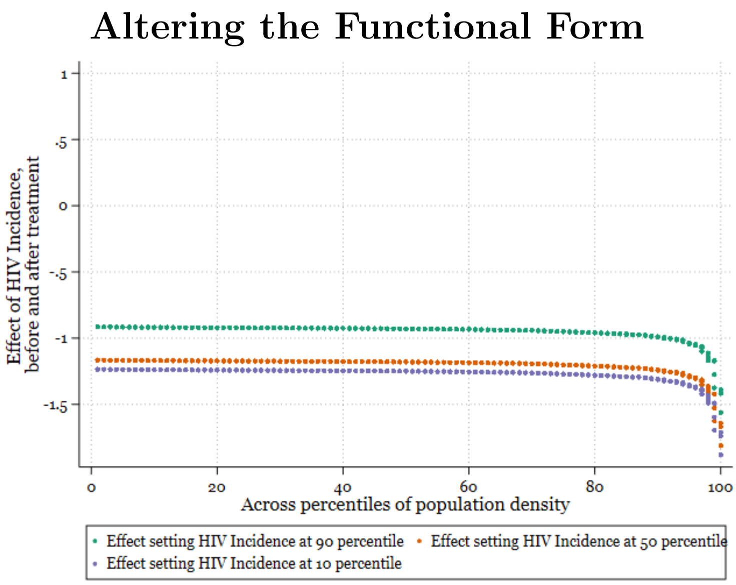


Figure 7: Source: CDC Multiple Cause of Death data (NCHS, 1990-2002)
Notes: Estimates for Equation 6 using coefficients from Equation 6.2 and data from my dataset. I set pre-HAART HIV/AIDS death rate at the 10th, 50th and 90th percentile and estimate Equation 6 across the distribution of Population Densities. 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.

Exploiting Continuous Treatment

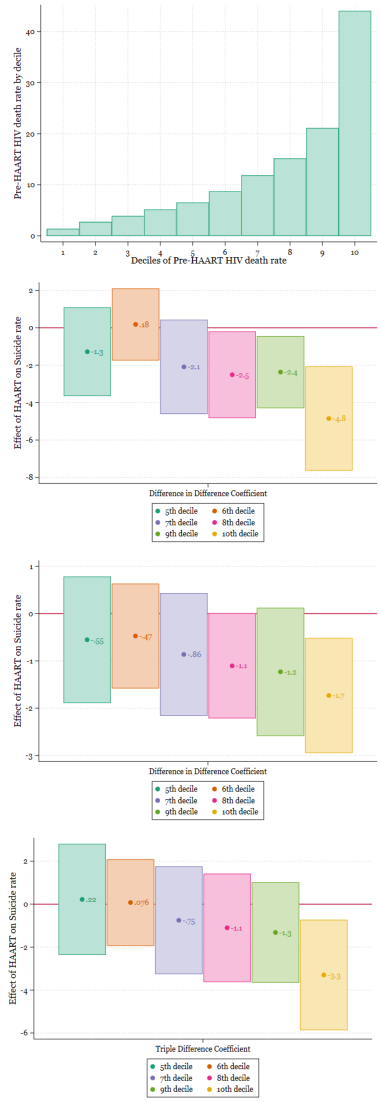


Figure 8: Source: CDC Multiple Cause of Death data (NCHS, 1990-2002)
Notes: The first figure presents mean pre-HAART HIV death rate at 10 deciles of pre-HAART HIV incidence. In the next three figures, HIV Inc_c is a dummy variable that is equal to 1, when looking at a particular decile of pre-HAART HIV 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.

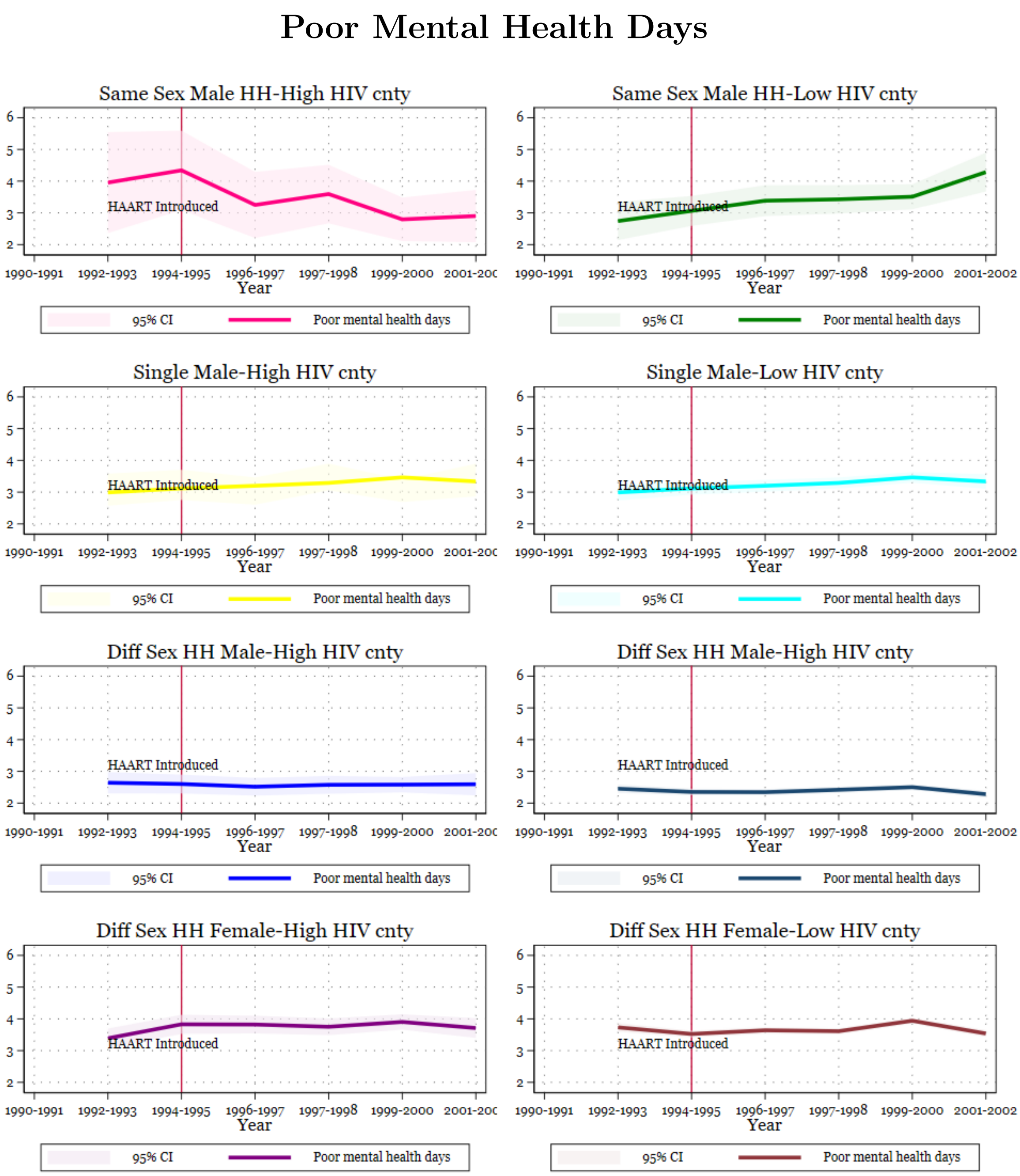


Figure 9: Source: BRFSS 1990-2002 (CDC, 1990-2002b)
Notes: Trends for the average number of poor mental health days in the past 30 days by group.

Fourteen or More Poor Mental Health Days

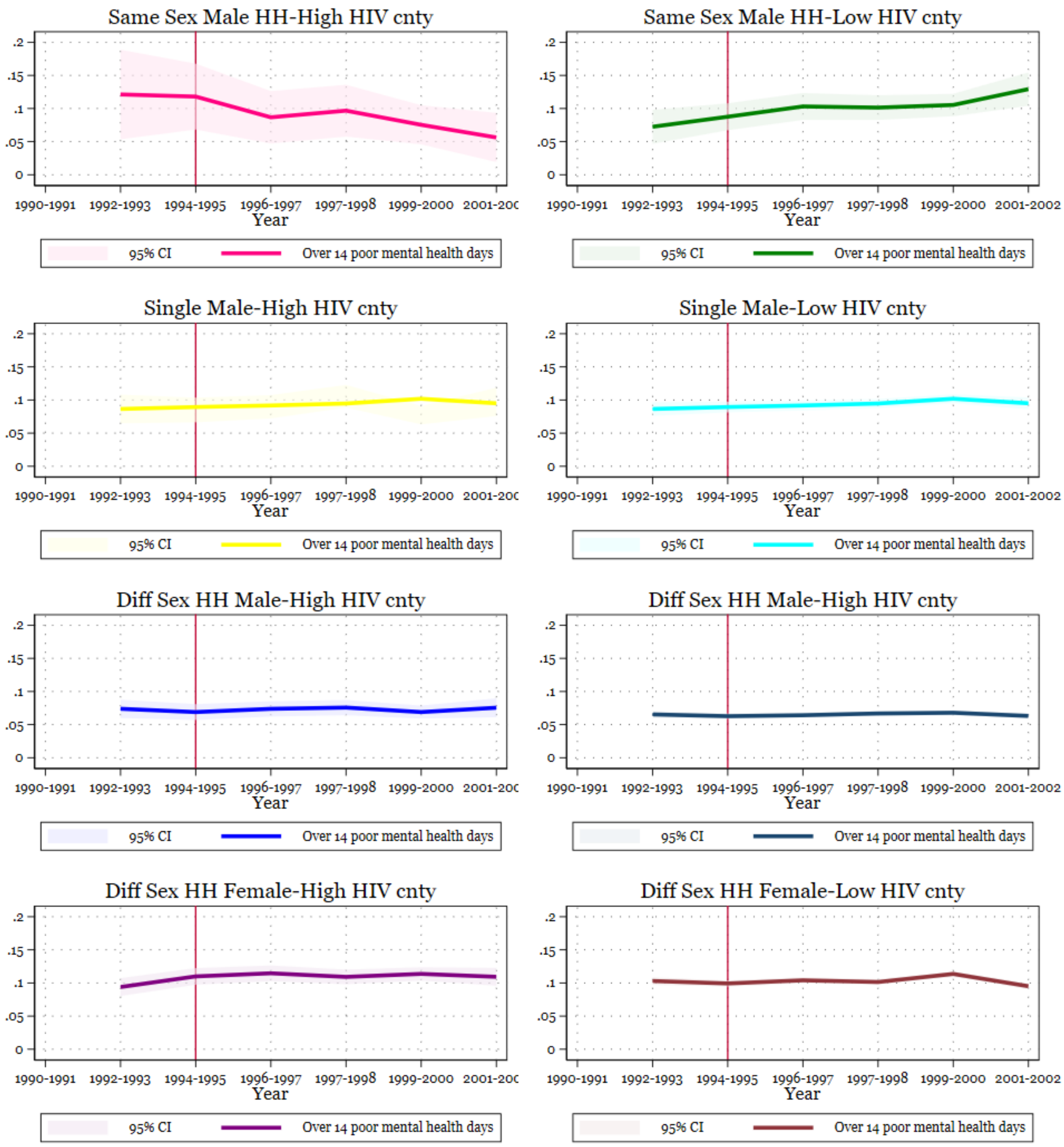


Figure 10: Source: BRFSS 1990-2002 (CDC, 1990-2002b)
Notes: Trends for the proportion of respondents reporting over 14 days of poor mental health days in the past 30 days by group.

Deaths of Despair

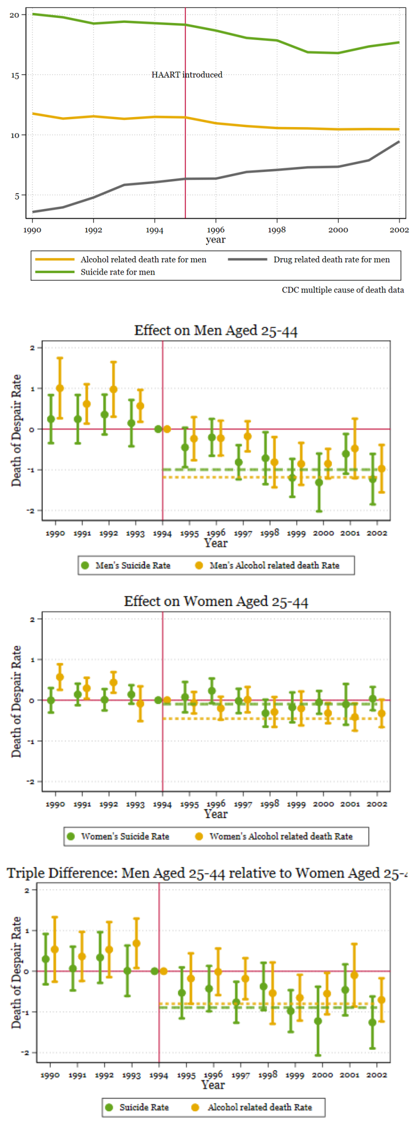


Figure 11: Source: CDC Multiple Cause of Death data (NCHS, 1990-2002)
Notes: In the first figure, I present trends in death rates by suicide, alcohol consumption, and drug related deaths. Thereafter, I present estimates for Equation 1 while changing my outcome variable to alcohol related death rate in 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.

Effects by Race

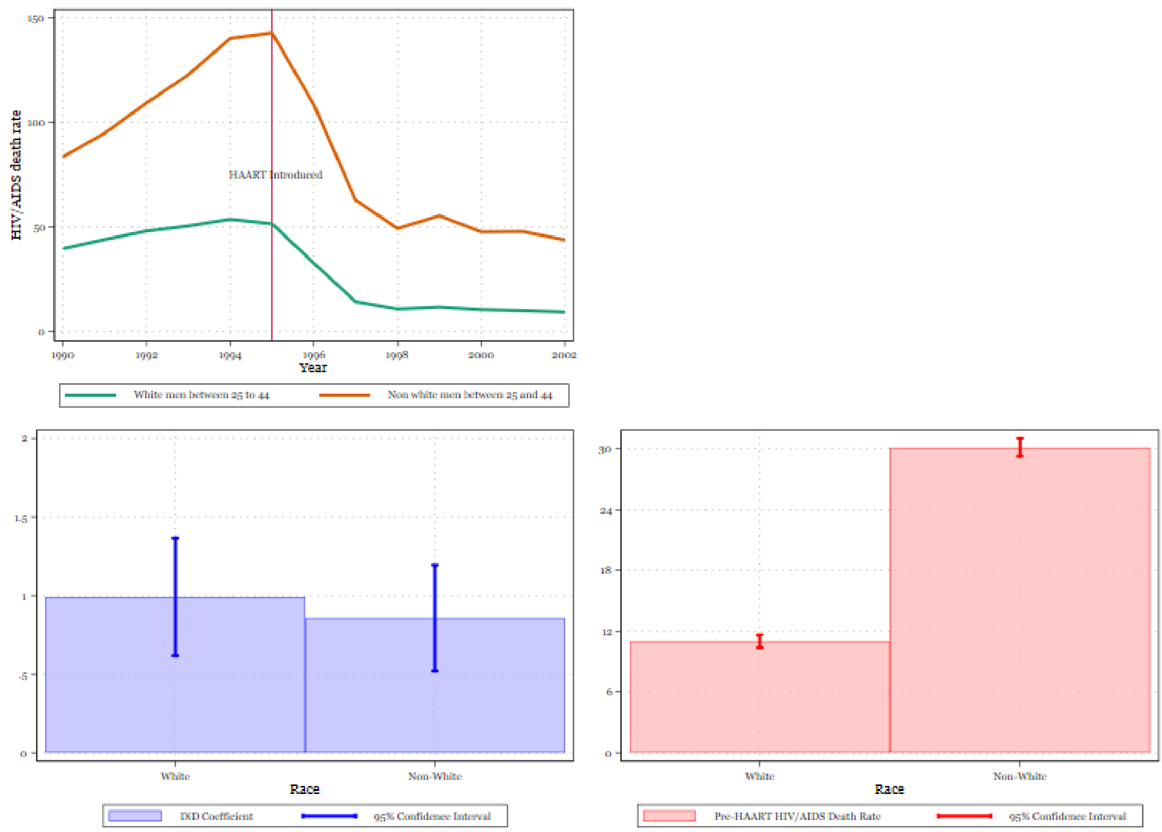


Figure 12: Source: CDC Multiple Cause of Death data (NCHS, 1990-2002)
Notes: The first figure presents estimates of HIV/AIDS-related death rates for white and non-white men aged 25 to 44. In the next figure in, I present coefficients estimates from Equation 3 for white and non-white men aged 25 to 44 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.

Summary Statistics

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

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).

Standard Difference-in-Differences

	Men 25-44			Women 25-44		
	(1)	(2)	(3)	(4)	(5)	(6)
Post=1 × Stdz 1994 HIV death rate	-1.03368***	-0.95086***	-0.99512***	-0.07505	-0.05911	-0.09613
	(0.147)	(0.174)	(0.159)	(0.079)	(0.081)	(0.087)
Population Density		-0.00204	0.00061		-0.00030	-0.00024
		(0.002)	(0.002)		(0.001)	(0.001)
Unemployment Rate		0.26256**	-0.03912		0.01567	0.03712
		(0.124)	(0.156)		(0.060)	(0.084)
Percentage Pop White		0.03613	0.14017		-0.00961	0.07815
		(0.109)	(0.120)		(0.053)	(0.049)
Proportion Evangelical Protestant		18.90438	17.47253		-1.89047	15.10848**
		(15.915)	(17.849)		(7.376)	(6.691)
Proportion Adherants to a Religion		-2.62522	1.05753		0.11764	0.69799
		(3.177)	(2.944)		(1.939)	(1.686)
Ryan White Title 1 eligible=1		-1.02496**	-0.99244*		-0.58575**	-0.23886
		(0.479)	(0.526)		(0.234)	(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

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

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.

	Men 65 plus			Men under 25		
	(1)	(2)	(3)	(4)	(5)	(6)
Post=1 × Stdz 1994 HIV death rate	-0.32657***	-0.29265**	-0.28613**	-0.06523	-0.05305	-0.04439
	(0.113)	(0.123)	(0.121)	(0.083)	(0.085)	(0.089)
Population Density		-0.00117	-0.00093		-0.00034	0.00040
		(0.001)	(0.001)		(0.000)	(0.001)
Unemployment Rate		0.02495	0.08143		-0.09543*	-0.07221
		(0.080)	(0.108)		(0.057)	(0.067)
Percentage Pop White		-0.04411	0.01517		-0.05491	-0.00961
		(0.066)	(0.085)		(0.044)	(0.052)
Proportion Evangelical Protestant		-4.24829	-14.71042		-6.89793	-14.95471*
		(9.056)	(10.435)		(6.824)	(7.708)
Proportion Adherants to a Religion		0.02737	0.34268		3.89130**	3.76222*
		(2.499)	(2.482)		(1.783)	(1.995)
Ryan White Title 1 eligible=1		-0.41692	-0.48077		-0.13877	0.05061
		(0.271)	(0.319)		(0.210)	(0.251)
Observations	10101	9901	9901	5031	4931	4827
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

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

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.

Standard Triple-Difference

	(1)	(2)	(3)	(4)
Post=1 × male=1 × Stdz 1994 HIV death rate	-0.96234***	-0.95841***	-0.95612***	-0.89327***
	(0.146)	(0.146)	(0.130)	(0.156)
Population Density		-0.00117		
		(0.001)		
Unemployment Rate		0.14168**		
		(0.071)		
Percentage Pop White		0.01491		
		(0.070)		
Proportion Evangelical Protestant		8.56835		
		(10.315)		
Proportion Adherants to a Religion		-1.30231		
		(2.137)		
Ryan White 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

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

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.

Alternative Functional Form

	Male (1)	Female (2)
Post=1 × 1994 Population Density	-0.0002372894	-0.0000762272
	(0.00028035)	(0.00015862)
Post=1 × Standardized 1994 HIV death rate	-1.2641352717***	-0.2264553574
	(0.46660544)	(0.26711010)
Post=1 × 1994 Population Density × Standardized 1994 HIV death rate	-0.0000417030	0.0000613873
	(0.00007358)	(0.00004575)
Post=1 × 1994 Population Density Squared	0.0000000086	-0.0000000076
	(0.00000003)	(0.00000002)
Post=1 × 1994 HIV death rate squared	0.0004662589	-0.0001211464
	(0.00045077)	(0.00027918)
1994 Population Density	0.0010606807	-0.0006765447
	(0.00151360)	(0.00061887)
Unemployment Rate	-0.0408651832	0.0524218024
	(0.15431379)	(0.08017326)
Percentage Pop White	0.0786624222	0.0684928187
	(0.13311907)	(0.05551084)
Ryan White Title 1 eligible=1	-0.8889187538*	-0.2016953974
	(0.52419174)	(0.24437133)
Observations	4914	4914
County FE	Yes	Yes
Time FE	Yes	Yes
State X Time FE	Yes	Yes

Standard errors in parentheses

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

Source: CDC Multiple Cause of Death data (NCHS, 1990-2002)
Notes: Table presents alternative difference-in-differences estimates from Equation 6.2 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.

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 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

A.2 Using AIDS Public Information Dataset

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 A.2 presents summary statistics.

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

2rd 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

Figure 13 presents Difference in Difference and Triple Difference results from Equation 1 and Equation 2 where Pre-HIV Incidence$_c$ now represents 1994 AIDS case rate (total AIDS cases per 100,000 population).

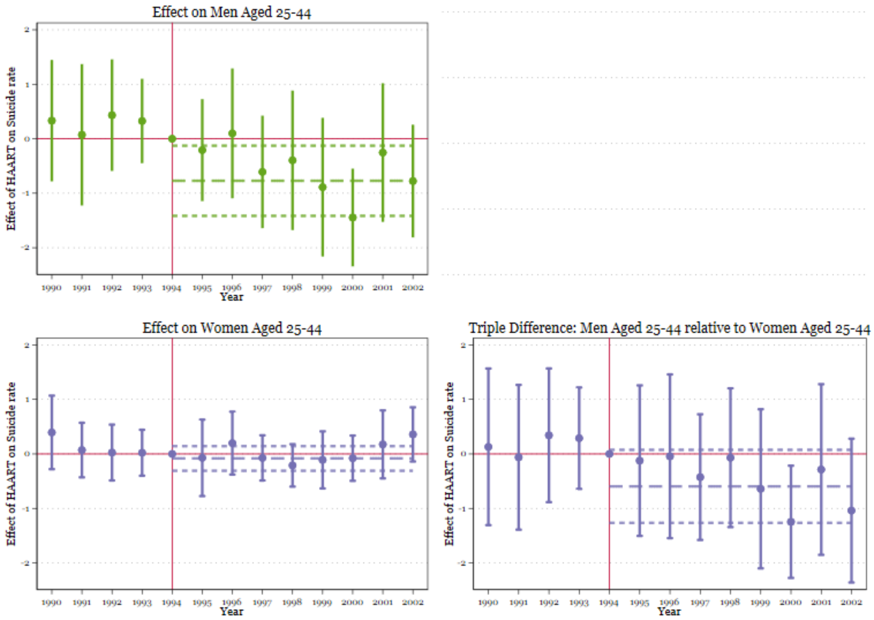


Figure 13: The figure on the left present difference in difference estimates from Equation 1 for men and women aged 25 to 44. Now, Pre-HIV Incidence$_c$ represents the standardized form of 1994 AIDS case rate. I control for time fixed effects, county fixed effects and state and time fixed effects. The 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. Estimates include controls for county unemployment, percentage of population that is white, Ryan White Eligibility.

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 14. 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.

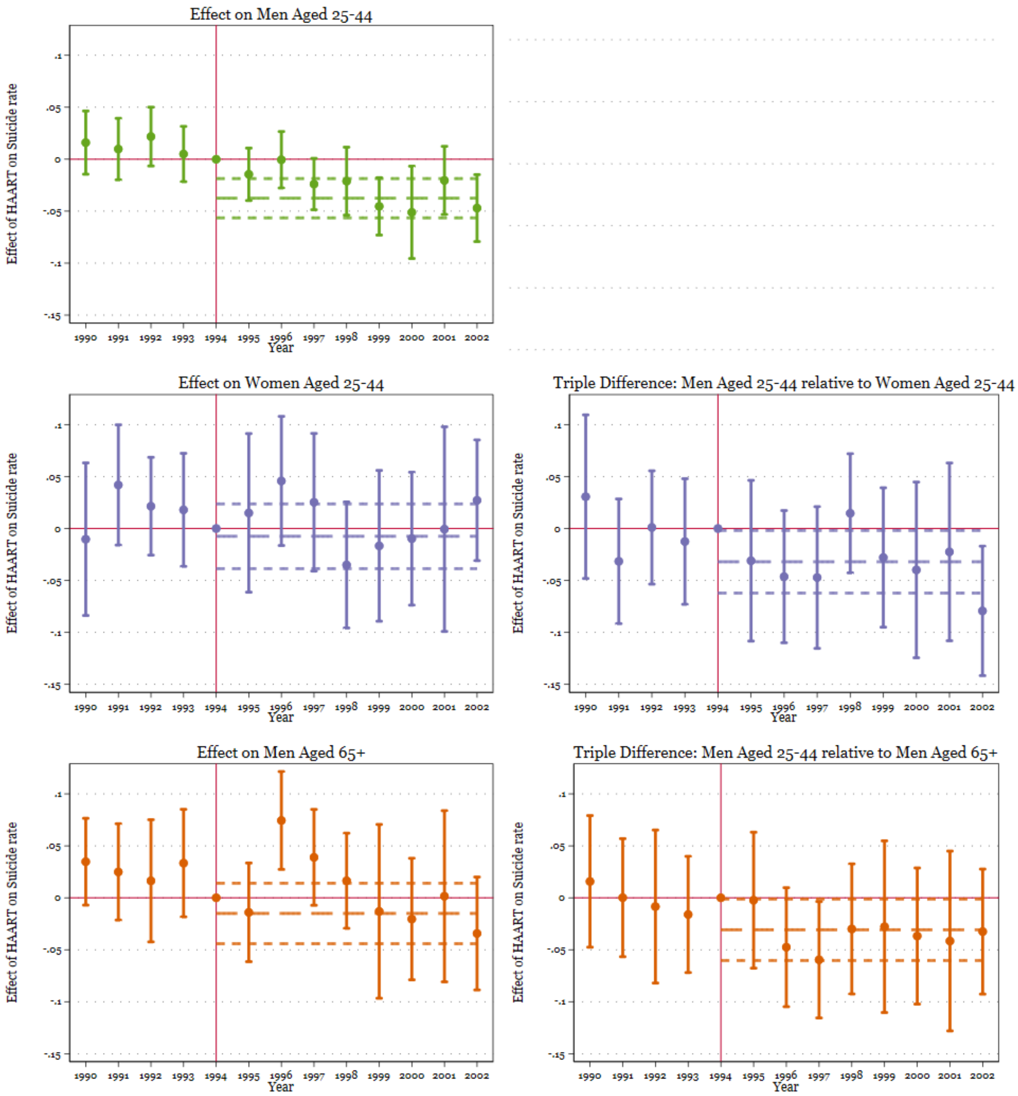


Figure 14: Figures on the left depict estimates for Equation 1 for men aged 25 to 44, women aged 25 to 44 and men over 65 after changing the dependant variable to represent the inerse hyyperbolic sine of sucide rates. I control for time fixed effects, county fixed effects and state and time fixed effects. 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. Estimates include controls for county unemployment, percentage of population that is white, Ryan White Eligibility.

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 15.

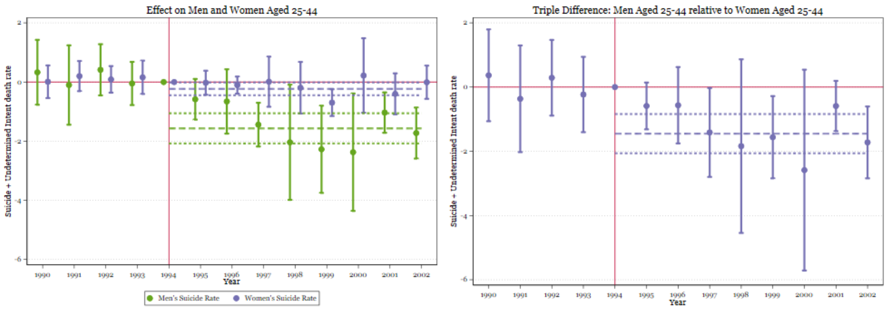


Figure 15: The first figure presents difference in difference estimates from Equation 1 for men and women aged 25 to 44. I control for time fixed effects, county fixed effects and state and time fixed effects. The second figure presents Triple Difference Estimates from Equation 2 for individuals aged 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. Estimates include controls for county unemployment, percentage of population that is white, Ryan White Eligibility.

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 16. HAART appears to have little consequence on these causes of death.

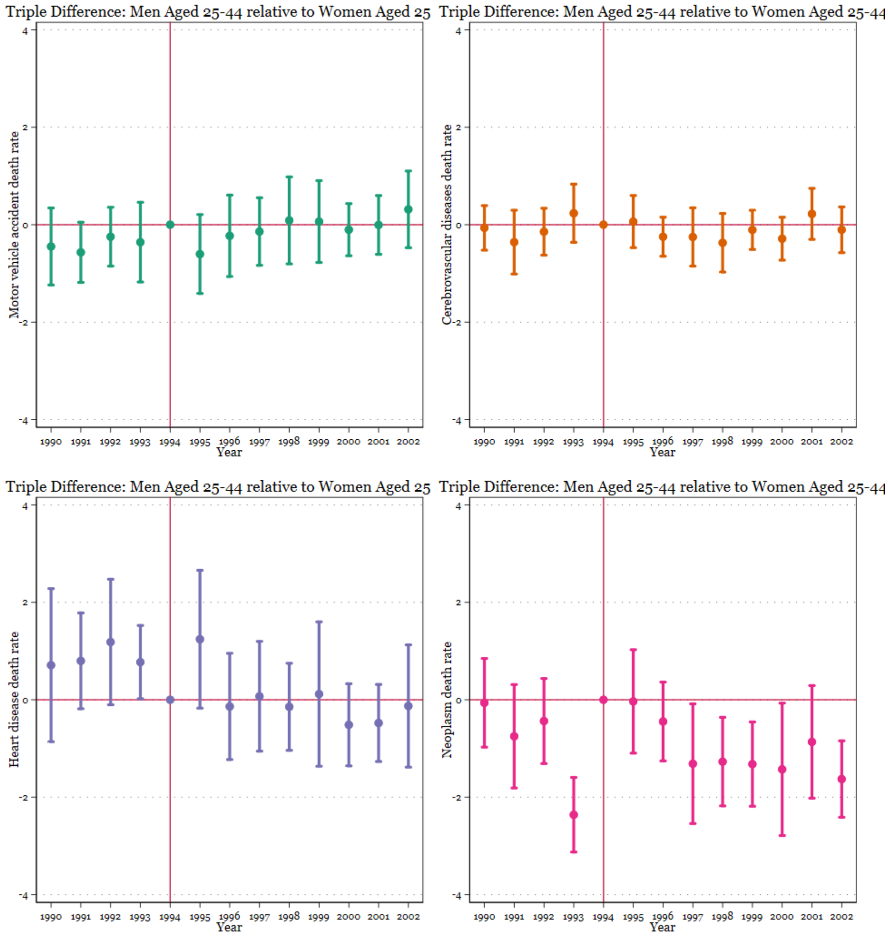


Figure 16: In these figures, I change the outcome variable represent death rates from motor vehicle accidents, cerebrovascular diseases, heart diseases, and neoplasm. I present Triple Difference Estimates from Equation 2 for men aged 25 to women aged 25 to 44. I control for the interaction of time and 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.

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 & Zhao, 2020). Aditionally, 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 reeestimate 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 covariated which I control for, I present the 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 17 shows that these changes don't have large affects on my estimates.

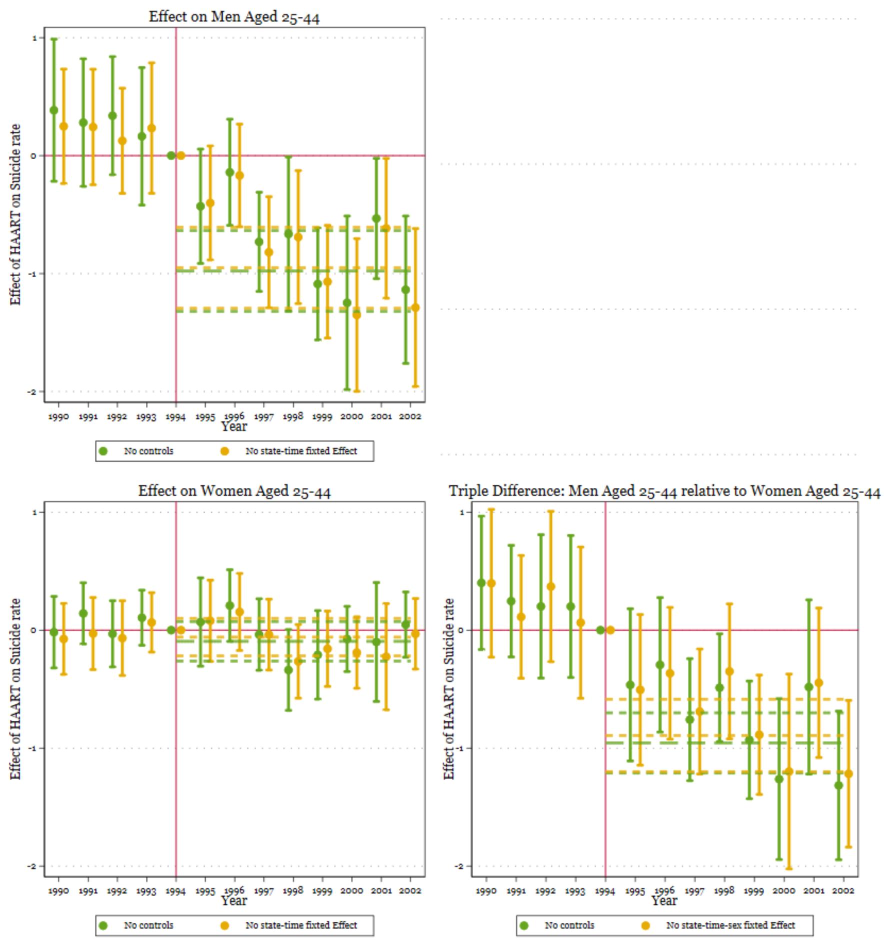
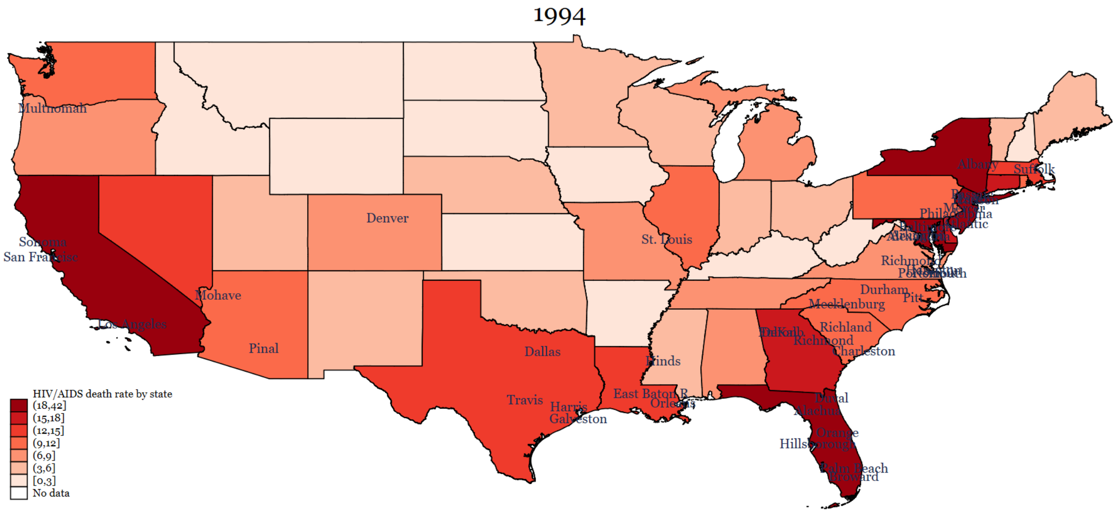


Figure 17: The figure presents difference in difference estimates from Equation 1 for men and women aged 25 to 44. I control for time fixed effects, county fixed effects and state and time fixed effects. All estimates are weighted by group county level population and standard errors are clustered at the county level. Estimates do not include controls for county unemployment, percentage of population that is white, Ryan White Eligibility.

A.6 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 bredth 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.



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

$$\text{Suicide Rate}_{st} = \alpha + \sum_{\substack{m \neq 1994 \\ m=1990}}^{2002} \beta_m \left(\text{Pre-HIV/AIDS deathrate}_t \times \mathbb{1}[t=m]\right) \;\; + a_t + \mu_s + \epsilon_{st} \tag{7}$$

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. 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:

$$\text{Suicide Rate}_{jst} = \alpha + \sum_{\substack{m \neq 1994 \\ m=1990}}^{2002} \beta_m \left(\text{Pre-HIV/AIDS deathrate}_s \times \mathbb{1}[t=m] \times \text{Men 25-44}_j \right) + \theta_{st} + \iota_{js} + \delta_{jt} + \epsilon_{ctj} \tag{8}$$

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:

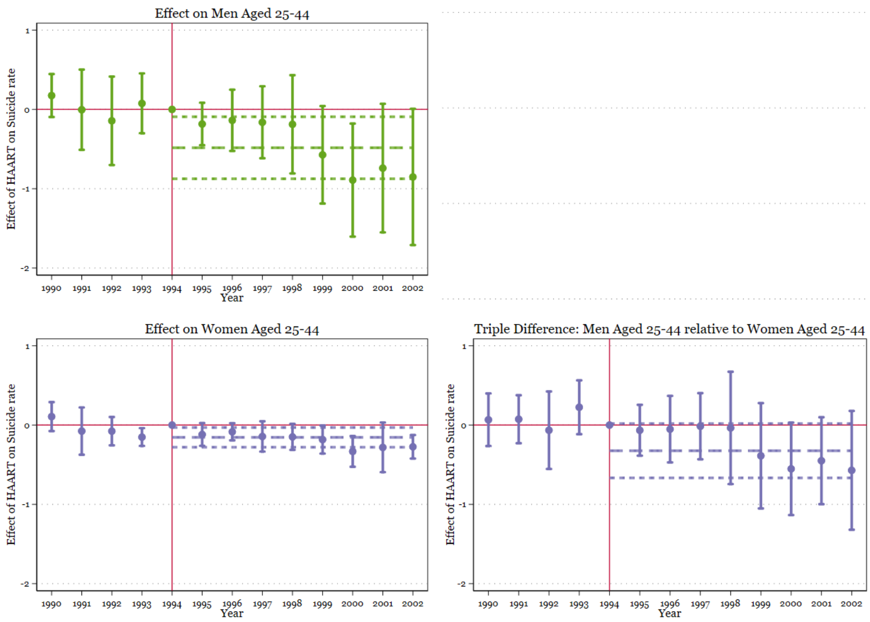
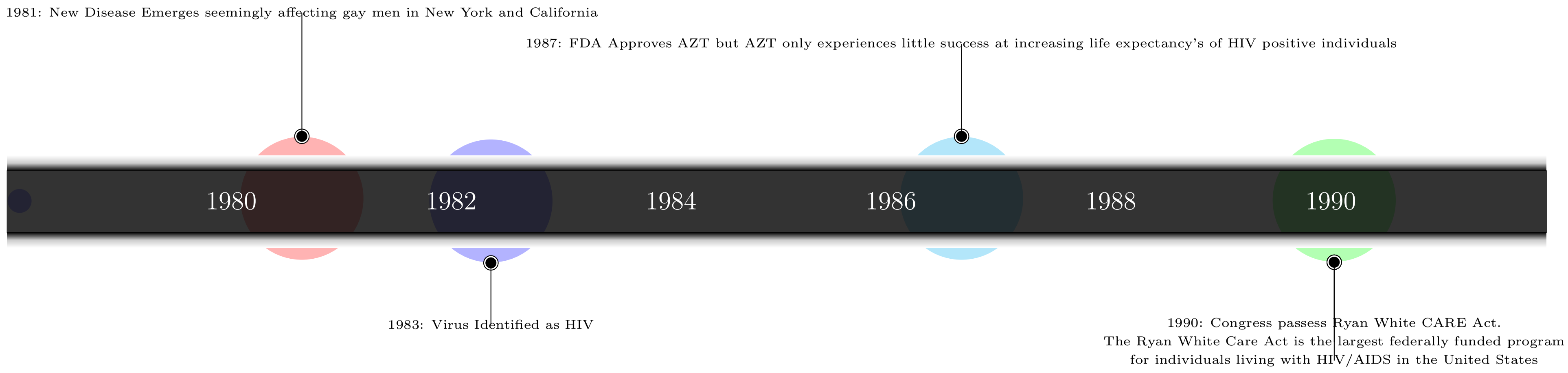


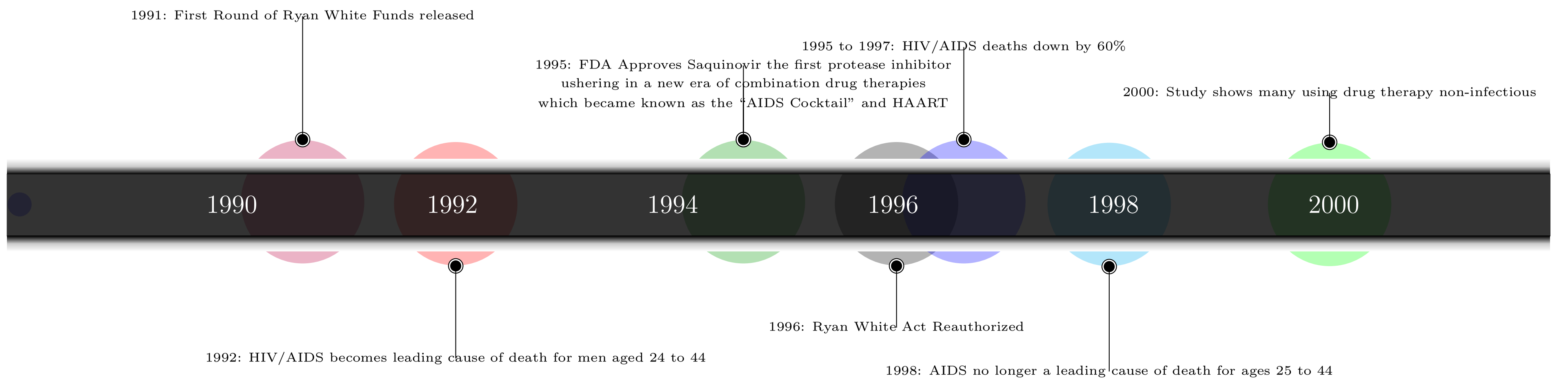
Figure 18: The figures on the left presents difference in difference estimates from Equation 7 for men and women aged 25 to 44 at the state level. I control for time fixed effects and state fixed effects. The third figure presents triple difference estimates from Equation 8. Here, I control for state-time 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 18 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 suggest robustness. 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. Once we account for these differences, the estimates suggest similar magnitudes.

A.7 Timeline


1981: New Disease Emerges seemingly affecting gay men in New York and California
1987: FDA Approves AZT but AZT only experiences little success at increasing life expectancy's of HIV positive individuals
1980
1982
1984
1986
1988
1990
1983: Virus Identified as HIV
1990: Congress passess Ryan White CARE Act.
The Ryan White Care Act is the largest federally funded program
for individuals living with HIV/AIDS in the United States


1991: First Round of Ryan White Funds released
1995 to 1997: HIV/AIDS deaths down by 60%
1995: FDA Approves Saquinovir the first protease inhibitor
ushering in a new era of combination drug therapies
which became known as the "AIDS Cocktail" and HAART
2000: Study shows many using drug therapy non-infectious
1990
1992
1994
1996
1998
2000
1996: Ryan White Act Reauthorized
1992: HIV/AIDS becomes leading cause of death for men aged 24 to 44
1998: AIDS no longer a leading cause of death for ages 25 to 44