WHEN DEATH WAS POSTPONED: THE EFFECT OF HIV MEDICATION ON WORK AND MARRIAGE

Mette Ejrnæs

Esteban Garcia-Miralles

Mette Gørtz

Petter Lundborg

C ENTER FOR
E CONOMIC
B EHAVIOR &
I NEQUALITY

When Death was Postponed: The Effect of HIV Medication on Work and Marriage [*]

Mette Ejrnæs[†]   Esteban García-Miralles[‡]

Mette Gørtz[§]   Petter Lundborg[¶]

July 13, 2022

Abstract

Over the last century, global life expectancy has increased tremendously. A longer planning horizon may change individuals' incentives to work, save, and marry but it has proven challenging to disentangle such incentive effects from those of improved health. In this paper, we study how individuals diagnosed with HIV reacted to the introduction of HIV medicine in 1995, which dramatically increased their life expectancy. To isolate the incentive effect, we use Danish register data on HIV-infected individuals and compare how outcomes evolved for individuals who were diagnosed before and after the medicine was introduced, but whose health had not yet been affected by their HIV diagnosis. Our results show that increases in the life expectancy of HIV-infected individuals greatly reduced the negative effect of receiving a HIV diagnosis on labor supply and earnings but did not affect important financial decisions, despite a much longer investment horizon. An increased life expectancy also affected marital behavior, where those facing a longer life expectancy where less likely to marry or cohabit after receiving a HIV diagnosis. Our results highlight that life expectancy gains from medical innovations impact individuals' incentives to work and marry, even when their underlying health is unchanged.

Keywords: Life Expectancy, Labor Supply, Marriage, HIV
JEL Classification: D84, I12, J12, J21

[†]University of Copenhagen and CEBI. (email: mette.ejrnes@econ.ku.dk)
[‡]Bank of Spain. (email: esteban.garcia.miralles@bde.es)
[§]University of Copenhagen and CEBI. (email: mette.gortz@econ.ku.dk)
[¶]IZA and Department of Economics, Lund University. (email: petter.lundborg@nek.lu.se)

People who had been planning to die sooner rather than later – quitting their jobs, cashing in their insurance policies, running their credit cards to the limit, avoiding fresh romances or clinging to old relationships – began finding themselves back in the business of living, with all its complications.

"From the AIDS Conference, Talk of Life, not Death" by David W. Dunlap. Published in New York Times, July 15, 1996.

1 Introduction

Over the last century, populations around the world gained substantially in life expectancy. Candidate explanations are generally increasing incomes, a more fine social security net, including widespread – public or private – health insurance, and new or improved treatments as a consequence of medical research and innovations in health technologies. Human capital theory predicts that a longer life expectancy increases the incentive to invest in skill acquisition (Becker, 1964), and it has therefore been argued that life expectancy affects economic growth through a human capital channel.

New treatments potentially alter the life expectancy of patients, affecting their long-run economic decisions on savings and investments in e.g. education through a change in how they discount the future (Hamermesh, 1985; Bloom et al., 2003). Improvements in health can thus improve individual productivity and employment options and facilitate a longer work life. Changes in life expectancy may also affect the incentives to work, save, and marry, net of any human capital investments. A longer planning horizon could affect individuals' trade-offs between labor and leisure and their demand for long-term financial investments. Moreover, a longer life expectancy could affect marriage market behavior, as it can affect the value of marriage as insurance mechanism, the value of leisure, the planning of bequests, or the attractiveness as a spouse in the marital market. It has proven challenging to empirically disentangle such pure incentive effects of longer life expectancy from those of improved health, however. Healthier workers are more productive and improvements

1

in health can also lead to better employment options and facilitate a longer work life. While a few studies have attempted to isolate the incentive effect of a longer lifespan on human capital acquisition by using variation in aggregate mortality over time and space (Fortson, 2011; Jayachandran and Lleras-Muney, 2009) or in individuals' genetic information about expected life expectancy (Oster et al., 2013b), there is little evidence to date on the incentive effects on other outcomes such as employment, earnings, savings, and marital behavior.

In this paper, we focus on a medical breakthrough that dramatically increased life expectancy for HIV positive individuals. The treatment, known as Highly Active Antiretroviral Therapy (HAART), was gradually introduced through 1995 and formally approved in 1996, and it significantly improved survival probabilities among HIV positive individuals (Legarth et al., 2014; Mocroft and Lundgren, 2004). Its appearance has meant that HIV today is viewed as a chronic illness that can be alleviated with the correct treatment. Without treatment, a newly infected individual faces a life expectancy of 11 years after being infected (Papageorge, 2021). Thus, before the introduction of HAART in 1996, a HIV diagnosis was associated with a substantially shortened life expectancy and many patients died shortly after being diagnosed.

To illustrate the impact on HIV infected individuals from the information of the new and more effective treatment, we refer to the quote at the beginning of the paper. The article was published in the New York Times on July 15 1996, just a few days after the medical breakthrough had been finally confirmed at the XI International AIDS conference. The article described how infected individuals before the HAART treatment had been quitting their jobs, stopped saving for retirement, and clung to their partners as they thought they were going to die soon, while after the treatment could resume to an almost normal life. The article underlines how the positive news of the increased life expectancy that had gradually been documented through clinical trials through 1995-96 impacted HIV-infected individuals' work, financial and marital decisions.

From a theoretical point of view, we would expect that a longer time horizon would increase human capital accumulation and hence lead to increased labor supply

as there is now more time to benefit from the returns to human capital accumulation (see Ben-Porath (1967)). We would also expect more long-term financial investments, although the empirical evidence is mixed (see (Hurwitz and Sade, 2020)). In terms of savings, the literature is, however, ambiguous, depending on the role of bequest motives. In the absence of a bequest motive, we would expect agents with short time horizon to run down their wealth faster (De Nardi et al., 2009), whereas the bequest motive may moderate such behavior (Dynan et al., 2002).

Regarding marital decisions, models from family economics mention five broad sources of potential material gain from marriage, namely enjoying the benefits of specialization, sharing of collective goods, extending credit within the household, coordinating child care, and risk pooling – for example when one partner works while the other partner is sick or unemployed (Browning et al., 2014; Weiss, 1997). When studying the implications of poor health and shorter life expectancy for partnering decisions, however, evidence points in several directions. On the one hand, facing a shorter time horizon may accelerate the marriage decision due to an increased need for insurance against health shocks (see also Anderberg (2007) and Persson (2020) and Potoms and Rosenberg (2021)), to secure formal heirs and a need for informal care. Also, experiencing a shorter time period to find a partner and enjoying the benefits of partnership implies that the "option value of waiting" for a high-quality partner declines (Strobel, 2003). On the other hand, if the quality of marriage depends on the expected time horizon, individuals with a shorter life expectancy might be less attractive in the marital market, leading to less matches.

We use the advent of the information of the new and more effective treatment (the HAART treatment) through 1995 to study the effects of a drastic change in life expectancy on HIV-infected individuals' work, savings, and marital decisions. For this purpose, we use unique and high-quality longitudinal register data from Denmark on HIV-infected individuals, observed before and after the appearance of HAART. Since the data includes information on the stage of the disease, we are able to select and study HIV-positive (HIV+) individuals who were still in good health and who did not face any symptoms yet. Yet, those diagnosed before and after 1995 differed radically in their life expectations. Before 1995, otherwise healthy HIV

patients could expect a low life expectancy, whereas those diagnosed after 1995 could expect an almost normal life expectancy. By comparing changes in outcomes before and after the HIV diagnosis across these groups, we can estimate the effect of longer life expectancy, net of changes in underlying health.

In our empirical analyses, we rely on a triple-difference design. Since we are comparing changes in the outcomes of HIV-infected individuals before and after 1995, we need to ensure that the differences in the changes are not driven by calendar time, e.g. effects due to business cycle effects or structural changes or reforms. To account for such changes, we separately match a control group of non-HIV-infected (HIV-) individuals to HIV-infected individuals before and after 1995.

Our first set of results shows that the increase in life expectancy following the introduction of HAART dramatically reduced the negative effect of receiving a HIV diagnosis on labor supply and earnings. After receiving the diagnosis, those with access to HAART had a 12 percent higher employment rate and 17 percent higher earnings compared to those without access. The effects are to a large extent driven by sharp reductions in employment and earnings among those diagnosed before 1995, suggesting a substitution towards leisure as life expectancy is reduced.

Our second set of results shows that life expectancy only had small effects on financial decisions. Bank account savings, stock market participation, and home ownership were essentially unaffected. When we zoom in on HIV positive individuals in poor health, who are closer to the end of their life, we find that spending down is greater among those without access to HAART, however.

Our third set of results suggest that changes in life expectancy have large effects on marriage and cohabitation rates. In the group of HIV+ individuals diagnosed before 1995, who faced a much reduced life expectancy, marriage rates went up. This finding can be interpreted in a family economics framework, where cohabitation and marriage provides an important source of private insurance against health shocks (Anderberg, 2007; Persson, 2020; Potoms and Rosenberg, 2021). The arrival of the HAART treatment shares common features with a change in health insurance - as insurance improves (better treatments arrive), the need for intra-marriage insurance is reduced. In the absence of HAART, and with a large negative shock to life ex-

pectancy, the insurance value of having a partner taking care of you, financially as well as practically through informal caregiving, increases. Moreover, if relative preferences for consumption and leisure change such that more weight is put on leisure relative to consumption, as the results above suggested, the utility of being in a couple could also increase if leisure complementarities are positive (Johnsen et al., 2022; Lalive and Parrotta, 2017). For HIV positive patients diagnosed after 1995, the insurance motive for having a partner is weaker due to the arrival of HAART.

Our paper relates to the small literature that estimates the effect of longer life expectancy on human capital investments (Fortson, 2011; Jayachandran and Lleras-Muney, 2009; Oster et al., 2013b). Using data from sub-Saharan Africa, Fortson (2011) find that regions with higher HIV (Human Immunodeficiency Virus) prevalence experienced relatively larger declines in schooling. Jayachandran and Lleras-Muney (2009) find that a sudden drop in maternal mortality in Sri Lanka in the 1950s sharply increased the life expectancy of girls, leading to an increase in girls' education relative to boys' in areas with larger maternal mortality declines. Oster et al. (2013a,b) estimate the impact of life expectancy on human capital investment using data on individuals at risk for Huntington's disease. Our paper contributes to this literature by studying the effect of longer life expectancy on post-education outcomes (i.e. employment, savings, and marital behavior). Moreover, the source of variation in life expectancy that we study originates from a medical breakthrough, rather than stemming from a multitude of sources or from naturally occurring mortality variation for a particular condition like Huntington's disease.

Our paper also relates to Chan et al. (2015), who developed a dynamic framework for studying how the innovation of HAART medicine lowered both the expected cost and likelihood of HIV infection by raising the implicit price of risky sex. Our paper also speaks to papers by Keiser et al. (2010) and Shahid (2022), which show that suicide rates of HIV patients declined with the introduction of HAART in Switzerland and the US, respectively. Moreover, recent research suggests that the introduction of HAART was associated with a reduction in homophobia in high-HIV states versus low-HIV states in the US (Fernandez et al., 2021). Our paper furthermore relates to Papageorge et al. (2021), who investigate the effect of the introduction of HAART

treatment on the prevalence of domestic violence and drug use among low-income women in the U.S. They find a reduction in domestic violence and drug use, which they interpret as an indicator of increased human capital investment in response to increased life expectancy. Furthermore, they document an increase in employment. We differ in that we study a representative sample of HIV-positive individuals (basically the universe of HIV+ individuals). Moreover, by linking our sample of HIV patients to administrative registers, we can study a number of important socio-economic outcomes, such as employment, earnings, wealth, bank holdings, disability, and marital status, thus painting a rich picture of the consequences of increased life expectancy. In a broader sense, our paper contributes to the literature that addresses the connection between HIV and economic activity (Oster, 2012).

The paper unfolds as follows. Section 2 presents the institutional context of HIV and HIV treatment in Denmark and how HAART radically changed the situation for HIV positive individuals. Section 3 presents the data we used in our analyses. Section 5 introduces our empirical strategy. Section 6 presents our main set of results. Section 7 provides a set of robustness analyses. Section 8 concludes.

2 Background and institutional context

2.1 HIV and AIDS: medical facts

HIV (Human Immunodeficiency Virus) is a chronic virus that reduces the immune system's ability to protect against ordinary infections, so called immunodeficiency. It is predominantly transmitted through sexual contact and through blood. The HIV virus attacks the immune system and especially the CD4 cells (T-cells). Before treatment became available, the immunodeficiency that followed led HIV-infected individuals to develop infections (AIDS) and ultimately to die.

To monitor the progress of the HIV disease, CD4 counts, defined as the number of white blood cells per mm^3 of blood, are measured with a blood test conducted by a health professional. Without HIV, a healthy immune system has a CD4 count between 500 to 1,600 cells per cubic millimeter of blood (cells/mm3). When the

6

CD4 count is below 200 cells/mm3, a person will receive a diagnosis of AIDS. With a cell count above 350, an HIV-positive individual has yet to experience any physical symptoms. As explained in the introduction, focusing on this group of individuals is an important part of our empirical strategy, as physical symptoms cannot explain any differences in the behavioral responses to new HIV treatments within this group.

2.2   HIV in society

The first scientific account of HIV was in 1981 and soon the number of cases increased dramatically through the 1980's worldwide. In many countries, the HIV/AIDS epidemic represented a significant demographic and economic shock (Karlsson and Pichler, 2015). As of today, AIDS has killed more than 32 million people worldwide. Prevalence is higher among homosexuals, people with haemophilia, drug addicts and people from Africa South of Sahara.

In Denmark, the first accounts of what is today known as HIV/AIDS appeared in Danish newspapers in late 1981, where the disease was thought to be a form of cancer that predominantly hit homosexual men. HIV testing was rolled out in 1985. Individuals could receive a blood test (free of charge) at their general practitioner (GP), at a hospital, or a clinic for sexual diseases. Anonymous tests could be taken at some hospitals and clinics. It quickly became known that AIDS was highly lethal. Until the mid-1990'es, 175-240 (or 3 in 1000) people died from AIDS annually. However, this number dropped dramatically to 1/3 of its previous level around 1996 when new treatments were implemented.

2.3   A medical breakthrough: HAART medication

The HAART medication, introduced in 1996, rapidly and effectively reduced mortality and the number of people sick from HIV/AIDS. The medical breakthrough came from combining three antiretroviral drugs including an at that time new type of drugs: protease inhibitors. In June 1995, the Food and Drug Administration approved the first protease inhibitors for treatment of HIV patients and in December

1995 FDA approved the combination treatment[1]. About six months later in July 1996, the promising results of the new combined treatment were confirmed at the XI AIDS conference.

These medical innovations were rapidly conveyed by the media to the public. Anecdotal evidence and news articles from Denmark indicate that already from September 1995 there were growing optimism and a sense that it was the beginning of a new era with an effective treatment of HIV (see Appendix A.1)

Antiretroviral treatment inhibits some of HIV's enzymes, reduces HIV in the body, and increases CD4 counts. While the treatment does not cure HIV entirely, it will halter its progression, leading to a significantly reduced risk of developing and dying from AIDS. Most treatment guidelines, including those in Denmark, recommend that HAART treatment is initiated when CD4 counts fall below 350, although in recent years, earlier initiation has shown positive results (INSIGHT START Study Group et al., 2015).

Due to the introduction of the HAART treatment, HIV is today seen as a chronic infection in Western world, with a survival probability close to that of the general population. Figure 1 illustrates survival rates by year following HIV diagnosis in Denmark. People diagnosed from 1990-1993 experienced similar survival rates - five years after the diagnoses, only 20% had survived. For patient groups diagnosed from 1994 and later, survival curves are much less steep, and 5-year survival was more than 50%. For cohorts diagnosed after 1996, 4 in 5 patients were still alive 5 years after diagnosis.

A study of living conditions of HIV infected in Denmark points to remarkable changes in expectations and hopes for the future for patients diagnosed before and after the arrival of the new medicine (Carstensen and Dahl, 2007). Patients diagnosed in the early period were more likely to stop educating themselves further or to report HIV infection as an important cause for retirement than patients diagnosed in the more recent years.

---

[1]see https://hivinfo.nih.gov/understanding-hiv/fact-sheets/fda-approved-hiv-medicines

3 Data

Our data are drawn from a unique data set that combines high-quality longitudinal register data for the entire population from Statistics Denmark with a medical database that includes clinical health information on individuals diagnosed with HIV. This section describes the data and key variables.

3.1 Danish Registers

Register data from Statistics Denmark allows us to follow the entire population of HIV positive individuals from the 1980'es until 2000. The register data provides background information as well as socioeconomic outcomes and information on health care use.[2] The socioeconomic outcomes studied in this paper include employment, income, social transfers, wealth and housing. As these outcomes are reported in registers for taxation and social security, the outcomes studied so not suffer from under-reporting or recall bias. Information on cohabitation and marriage patterns is also reported in the registers with great precision. For cohabitation, we rely on Statistics Denmark's definition in which two adults, who are not family related, are considered to be cohabiting if they are living on the same address, of opposite sex and with less than 15 years of age difference.[3] We furthermore rely on the rich information in Statistics Denmark's health care registers to establish the timing of the first HIV test taken for each individual and to study general health status.

---

[2]All residents in Denmark get their own personal identifier just minutes after they are born. These personal identifiers are used in all contacts with doctors, hospitals, schools, tax authorities etc. Statistics Denmark provides access to these data to researchers in anonymized form. Importantly, the population registers also contain family links. This allows us to follow individual's cohabitation and marital status.

[3]As a large part of our sample are homosexual men, we are also interested in partnering of men living with men. To this end, we use information on registered partnership (equivalent to marriage for homosexuals). In order to identify cohabiting men, we define a cohabitation identifier in the same way as Statistics Denmark's definition of cohabiting couples of opposite sex.

3.2 HIV Medical Database

In order to supplement our rich register data on socioeconomic characteristics and use of health care, we gained access to a rich clinical database, DANHIV, which has been assembled by all public and private hospitals in Denmark since the 1990'es.[4] Data in DANHIV goes back to early 1995 and has information on all patients diagnosed with HIV (ICD-10 codes B20-24) who were alive in 1995 when the database was started. It should be noted, however, that the database includes retrospective information on the date of HIV diagnosis and the source of infection also for those diagnosed before 1995. Each individual in the database has been linked to an individual in Statistics Denmark's (anonymized) register data. This implies that register information has been augmented with detailed information on the patients and their disease, three of these variables are crucial for our analysis:

CD4 Counts. This variable is the leading indicator of immune system health as it indicates how advanced the HIV disease is. It is a key variable for defining our sample, which we restrict to healthy HIV-positive individuals whose immune system has not yet deteriorated.

In our sample, each individual is tested on average 2.5 times a year. Because our outcomes of interest are measured with annual frequency, we construct for each individual an annual measure of CD4 counts defined as the mean value of all measurements from that individual on a given year. Importantly, HIV patients are informed of their CD4 counts in their visits to the doctor, as this is a crucial indicator of their current health.

Because the HIV medical database starts in 1995, CD4 counts prior to 1995 are not observed in this database. We must therefore impute the pre-1995 CD4 counts for individuals diagnosed prior to 1995, based on their CD4 counts observed since 1995 onward. It is important to emphasize that these imputed CD4 counts will not be used as a variable in any regression analyses, but only to identify individuals with good enough health at the time of diagnosis.

Our preferred imputation method estimates a quadratic model with individual

---

[4]More information on DANHIV (in Danish): https://www.rkkp.dk/kvalitetsdatabaser/databaser/Dansk-HIV-database-/.

fixed effects, with CD4 counts as a function of time from diagnosis. We estimate this model only using observations between the time of diagnosis and the beginning of HAART treatment, as the treatment itself will affect CD4 counts. Specifically, we estimate the following regression:

$$CD4_{it} = \phi_i + \beta_1 time + \beta_2 time^2 + \epsilon_{it}$$

where *time* is years from diagnosis and ϕ_i is an individual fixed effect captures differences in levels across the different individuals. The slope parameters β_1 and β_2 capture the yearly change in CD4 counts that occurs to individuals, on average, since they are diagnosed with HIV and until the start of HAART treatment. We then use this specification to impute CD4 count values that are missing between the time of diagnosis and the start of HAART treatment.[5]

Source of Infection. The medical database includes information about source of infection as reported by the patient. The patient may have been infected by sexual transmission, distinguishing between heterosexual transmission and transmission by men having sex with men (MSM). Source of infection may also be related to drug abuse, transmission to children during pregnancy, blood transfusions, or unknown. This information is crucial to define our sample of analysis, where we focus on individuals having been infected through one of the two dominating sources of infection, namely transmission through heterosexual contact and men having sex with men.

Time of HAART treatment. The DANHIV database includes information on the date of diagnosis as well as the data of commencing HAART treatment. In the first year, the latter date was decided based on CD4 counts. Recently, HAART treatment has been initiated immediately after being diagnosed with HIV.

---

[5]Our imputation equation estimates a common slope parameter of the change in CD4 from the time of diagnosis until starting HAART treatment. As an illustration of the heterogeneity in CD4 changes among our sample of analysis, which is composed by healthy HIV+ individuals, we report in Appendix Figure A.1 the distribution of individual changes in CD4 counts estimated for each individual who is ultimately included in our estimation sample. The distributions are similar in the treatment and control groups.

3.3 Sample selection

To define our sample used in the analyses, we impose the following four restrictions. First, we select the 2,153 individuals that were diagnosed with HIV either in the five years preceding the introduction of HAART (1990 to 1994) or in the five years following its introduction (1995-1999). Second, we exclude individuals who are drug addicts, according to their source of HIV infection, as we expect the behavior of these individuals to differ markedly from the rest of the sample. This leaves us with 1,932 individuals. Third, we restrict the sample to individuals with a healthy immune system at the time of diagnosis, who are thus not expected to suffer from any HIV-related physical symptoms in the years following the diagnosis. Specifically, we keep individuals whose CD4 count levels are equal to or above 400. The limits the sample to 596 individuals, where 289 are diagnosed before 1995 (the "control group") and 307 are diagnosed after 1995 (the "treatment group").

Fourth, we balance the sample by keeping individuals who are observed every year since 4 years before they are diagnosed until 4 years after their diagnosis, reducing our final sample to 439 individuals, of which 229 are in the control group and 210 are in the treated group. In the robustness section, we show that our results are robust to alternative ways of selecting the sample.

3.4 Descriptive Statistics and Balancing Tests

Columns 1 and 2 of Table 1 show summary statistics for key background and outcome variables in sample. The background variables are measured one year before the diagnosis, while the CD4 count is measured in the year of diagnosis. The treatment group consists of individuals diagnosed with HIV between 1995 and 1999, when the HAART treatment was available. The control group consists of those diagnosed between 1990 and 1994, before HAART was available. Some notable features of the sample are that males are heavily over represented (about 80 percent) and that the average age is about 34. Heterosexuals constitute 43 percent of the sample.

Column 3 shows the difference in means between the control and treatment groups and column 4 reports p-values for tests for equal means. The tests reveal that the

treatment and control groups are similar on most of the observable characteristics. Only two of the 20 differences are significant; the larger share of stock holding in the control group and the higher fraction of individuals with partners in the treatment group. Hamilton et al. (2021) develop a dynamic structural model and show that drugs used for HIV vary by efficiency and side-effects and argue that subsidies to medicine in its experimental phase could have improved social welfare and reduced inequalities in health due to differential access to the medicine.[6]

3.5 Balancing on Health

A key feature of our empirical design is our focus on HIV positive individuals who are in good health, with high CD4 counts and who have yet to experience physical health symptoms. In this sample, we can be certain that any sharp differences in life expectancy arising from differential access to HAART treatment do not coincide with differences in physical symptoms. We provide four pieces of evidence to support this claim.

First, Table 1 shows that the CD4 counts are high in our sample and hardly differ across the treatment and control groups (618 vs. 621). This suggests that both groups have CD4 counts well above the thresholds for which physical symptoms starts to arise. Second, Table 1 shows that the treatment and controls groups have similarly low rates of infections and low scores on the Charlson Index. Third, the treatment and control groups face high and similar survival rates during the first years after diagnosis, which we illustrate in Appendix Figure A.2. Note that, by construction, individuals in the control group must survive at least one year to be included in the medical data and in our sample. If we impose the same restriction on the treatment group, the survival curves align even better, as seen when comparing the dotted line and the red line.

---

[6]Hamilton et al. (2022) argue that there may be side-effects associated with taking HAART medicine. Based on US data, they find that HIV-infected men often forego medication to avoid side effects, in part to remain employed; this effect is stronger for people with less education. Goldman and Smith (2002) similarly argue that SES-gaps in patient self-management may lead to differential access to new treatments.

Fourth, we can compare the share of individuals below certain CD4 thresholds in the treatment and control groups. Importantly, in Figure 2 we show that the share of individuals below CD4 thresholds where individuals might start experiencing physical symptoms remains small and similar in both treatment and control groups. Overall, these results establish that (1) the sample is in good health and (2) well balanced on both socio-economic, demographic, and health-related characteristics.

4   Methodology

Our empirical design is based on comparing the evolution of outcomes over time for individuals diagnosed with HIV before and after the introduction of HAART in 1995. Specifically, our treatment group includes individuals who were diagnosed between 1995 and 1999, whereas our control group includes individuals diagnosed between 1990 and 1994. By restricting our sample to HIV positive individuals with high CD4 counts, we ensure that the groups are in good health and have yet to experience a deterioration of their immune system.

Since the treatment and control groups are observed in different years, a simple comparison would risk confounding the effects of increased life expectancy with other factors that change over time. To control for such calendar time effects, we construct and match additional synthetic control groups of individuals not infected with HIV (HIV–). We construct these synthetic control groups by matching 1,000 HIV– individuals of the same cohort, age, gender and education for each HIV+ individual in our sample. The matches are based on characteristics of the HIV+ individuals observed four years before diagnosis, and the matched HIV– individuals are then followed over time, preserving the panel structure of the data. In the robustness section we show that matching year by year leads to very similar results.

With our matched synthetic controls, we estimate the following standard dynamic

triple difference specification:

$$Y_{it} = \alpha_0 + \sum_{t \neq -1} \beta_t \cdot Treat \cdot Inf \cdot Time_{j=t} + \sum_{t \neq -1} \gamma_t \cdot Inf \cdot Time_{j=t} +$$

$$\sum_{t \neq -1} \eta_t \cdot Treat \cdot Time_{j=t} + \sum_{t \neq -1} \theta_t \cdot Time_{j=t} + \phi_1 \cdot Treat \cdot Inf + \phi_2 \cdot Inf + \quad (1)$$

$$\phi_3 \cdot Treat + \phi_4 \cdot Age \cdot Sex + \phi_5 \cdot Dane + \epsilon_{it}$$

where Y_{it} is the outcome variable of interest for individual i in time t, $Treat$ is a dummy variable that takes value one if an individual is diagnosed with HIV in the period 1995–1999 when HAART was available, and zero if the individual is diagnosed with HIV in the period 1990–1994 when HAART was not yet available, $Time_{j=t}$ is a dummy variable equal to one if the year since the diagnosis is equal to t, and Inf is an indicator that takes one if an individual is ever infected with HIV and zero if it is not, that is if it belongs to the synthetic sample of individuals who are not diagnosed with HIV.[7]

The β_t coefficients identify the causal effect of the introduction of HAART medication that increased the life expectancy of HIV patients. By plotting β_t over time t we are able to evaluate the identifying assumption that both treatment and control groups move in parallel before the HIV diagnosis that occurs in $t = 0$ and we observe the effect of the diagnosis under different availability of HAART. We present and discuss these graphical results in Section 5.

To quantify the average effect of the introduction of HAART we estimate a static version of the previous equation, which differs only in that the dummy variables for time since diagnosis $Time_{j=t}$ are now replaced by a single dummy variable $Post$ that takes the value one for all years after diagnosis, including $t = 0$.

$$Y_{it} = \beta_0 + \beta_1 \cdot Treat \cdot Inf \cdot Post + \beta_2 \cdot Inf \cdot Post + \beta_3 \cdot Treat \cdot Post + \beta_4 \cdot Post +$$

$$\beta_5 \cdot Treat \cdot Inf + \beta_6 \cdot Inf + \beta_7 \cdot Treat + \beta_8 \cdot Age \cdot Sex + \beta_9 \cdot Dane + \epsilon_{it}$$

$$(2)$$

---

[7]Each individual of the synthetic sample of HIV– individuals is assigned the same value of $Treat$ as the HIV+ individual to whom they were matched as well as a relative time to diagnosis t.

Impact of Mental Health Symptoms on Behavior. In Section 3.4, we showed that our treatment and control groups consist of HIV positive individuals in good physical health. A remaining concern is that drastic changes in life expectancy can have effects on mental health and the mood of individuals and that such effects can affect behavior. In particular, it appears reasonable that a positive shock to life expectancy may improve mental health. While changes in mental health can be part of the causal chain through which life expectancy affects behavior, it would be a mechanism distinct from that of the pure incentive effect of changes in life expectancy (Oster et al., 2013b).

The descriptive statistics in Table 1 give little reason for concern, however. In both the treatment and control groups, only 2 percent have seen a psychiatrist and less then 1 percent have visited a psychologist for psychotherapy. When we run Equation (2) above on these outcomes, the estimates are tiny and insignificant.[8]

5   Results

In this section, we present our main findings of the effect of having access to HAART medication around the time of the HIV diagnosis. For our main outcomes, we illustrate the results in two ways. First, we present the dynamic effects graphically, where we for each outcome report a four-field figure, illustrating the different contrasts used in the Triple Difference analysis model in Equation (1). Second, we present static regression results, based on Equation (2).

In each four-field figures, Graph (a) plots an event study for the control group (those diagnosed with HIV between 1990 and 1994, before the introduction of HAART) against the synthetic control of HIV– individuals. Graph (b) plots an event study for the treatment group (those diagnosed with HIV between 1996 and 1999, after the introduction of HAART) against the synthetic control of HIV– individuals. Graph (c) plots the control and treatment groups de-meaned by their respective synthetic controls. Graph (d) plots the β_t estimates of the triple difference-in-differences model

---

[8] The coefficients for vising a psychiatrist and a psychologist are are 0.002 (se=0.010) and -0.001 (se=0.007), respectively.

16

estimated in Equation (1), which identify dynamic causal effects of the introduction of HAART medical innovation.

5.1   Labor Market Outcomes

We begin by studying whether having access to HAART around the time of the HIV diagnosis, and thus facing a higher life expectancy, affects labor market outcomes. Figure 3 reports the results for employment, where we see that the treatment and control groups faced similar employment trends in the years before the diagnosis (Graphs (a) and (b)). In both cases, they also depart from the employment trends among the synthetic controls, showing the importance of bringing in this additional control group to account for calendar time effects. At the time of diagnosis, however, the trend diverges between the treatment and control groups, where the treatment group exhibits a less negative employment trend in the years that follow.

The divergence in employment trends between the treatment and control group at the time of diagnosis becomes even clearer in Graph (c), where we plot the outcomes of the groups, demeaned by their respective synthetic control group. The treatment and control groups closely follow each other until the year of the diagnosis, where they depart. Recall that the divergence cannot be explained by any divergence in health between the groups, as the share of individuals with CD4 counts below critical thresholds for symptoms is similar and low in both groups for the follow-up period considered, as shown in Figure 2. Rather, the divergence likely reflects the sharp differences in life expectancy between the groups, which may affect labor market behavior.

Graph (d) plots the event study estimates from the triple difference specification, which paint a similar picture. The figure confirms that the groups face parallel employment trends before the diagnosis, but that the trends depart thereafter. The employment rate in the treatment group is about 10 percentage points higher in the treatment group in the third to fourth year after diagnosis. If we take the average of the employment rate in the years following the diagnosis, the effect is to increase employment by 7.5 percentage points, corresponding to a 11 percent difference (Table

2).

We next consider labor earnings. Sharp changes in life expectancy may affect labor supply at both the extensive and intensive margin, as well as affecting productivity. Figure 4 reveals large effects on labor earnings. Before the diagnosis, the treatment and control groups follows similar trends, which are also largely in line with the trends in the synthetic control groups (Graphs (a) and (b)). After receiving the diagnosis, Graph (c) shows that the groups sharply diverges, with a sharp decline in earnings in the control group. The triple difference estimates in Graph (d) shows that the effects are large; in the year following the diagnosis, earnings are 20,000 to 30,000 DKK greater in the treatment group, whereas no such difference can be seen in the years prior to the diagnosis. Table 2 shows that the average effect, taken across all post-treatment years, amounts to 23,897 DKK. This corresponds to a 18 percent difference in earnings.

5.2   Savings, Housing, and Stock-holding

The introduction of HAART radically changed the life expectancy of HIV positive individuals and thereby their financial investment horizon. We continue by examining how the increased life expectancy affected bank account savings, home ownership, and stock-holding. Figure 5 shows the triple difference estimates for these outcomes, while Figure A.5, A.6 and A.7 in the appendix shows how the treatment and control groups evolved in comparison with their matched synthetic controls. Graph (a) does not provide any strong evidence that bank account savings among HIV positive individuals were much affected by the increased life expectancy. If anything, savings went down more in the group gaining access to HAART. Table 2 puts a number to this decrease; 6,415 DKK, which corresponds to a 24 percent decrease. This effect is not significant, however. In Figure A.6 in the appendix, we see that the effect reflects a decline in the treatment group, whereas the savings in the control group appears surprisingly unaffected by receiving a HIV diagnosis.

Did the increase in life expectancy encourage the treatment group to invest more in risky assets? Graph (b) provides no strong evidence for such an effect, as the

triple difference estimates show stock ownership does not evolve very differently in the treatment and control groups. This is further illustrated in Figure A.5 Graphs (a) and (b) in the appendix, where stock ownership in both the treatment and control groups largely follow the trends in their synthetic control groups. Table 2 shows that the average effect over the post-treatment years amounts to a 3 percentage points, insignificant, decline in stock ownership.

Do changes in life expectancy among HIV positive individuals affect another important long-run investment; home ownership? The answer is no, as illustrated in Graph (c). Figure A.7 in the appendix shows that the treatment and control groups follow similar trends, both before and after receiving an HIV diagnosis.

Overall, the results show that the dramatically increased life expectancy following from HAART treatment had surprisingly small effects on economic decisions about bank account savings, home ownership, and stock-holding. What can explain these results? First, recall that we study a relatively healthy group of HIV-positive individuals where death is not imminent. Perhaps spending down savings and assets mainly takes place closer to the point of death. This is suggested by the results in Figure A.8 in the appendix, which focuses on HIV positive individuals with low cell counts, who are closer to death. Graphs (a)-(c) suggest that stock-holding, bank account savings, and home ownership decline greatly in the control group as time progresses, whereas no such patterns can be observed in the control group.

Moreover, if the individuals in our sample have strong bequest motives, this could also explain why less spending down is observed. In the next section, we turn to decisions about partner formation and how they were affected by the extended life expectancy following from HAART.

Taken together, the results for earnings and the wealth components studied above have implications for how consumption is affected in the treatment and control groups. Since earnings declined sharply in the control group at the time of the HIV diagnosis, while important wealth components were unaffected, consumption may have been reduced. This could reflect a greater emphasis on leisure as the individuals in the control group learn about their limited life expectancy. Since we do not observe all components of wealth, we cannot rule out, however, that other

components of wealth, such as the value of financial assets, were spent down, in order to maintain the same consumption level.

5.3   Marriage market outcomes

Drastic changes in life expectancy can change the incentives for when and whom to marry or partner up with. In Figure 6, we illustrate the effects on partner formation, defined as marriage or cohabitation. Graph (a) shows an upward jump in the likelihood of having a partner in the control group, just after they receive their HIV diagnosis. No such jump is observed in the synthetic control group. In the treatment group, illustrated in Graph (b), there is instead a jump downward at the point of diagnosis. The different patterns in the treatment and control groups are further illustrated in Graph (c) and Graph (d) show the resulting triple difference estimates. These estimates shows a negative effect on partner formation, which reflects the differential patterns observed in the treatment and control groups. Table 3 shows that the average effect over the post-treatment years is about 9 percentage point.

How should we interpret the different effects observed in the treatment and control groups? We may interpret the increase in partner formation in the control group, who faced a negative shock to life expectancy, in a family economics framework. Cohabitation and marriage can provide an important source of intra-marriage insurance against health shocks (Anderberg, 2007; Persson, 2020; Potoms and Rosenberg, 2021). With declining health, and increasing care demands, the value of having a partner taking care of you increases. Another possible interpretation is that, with few years left, HIV positive individuals attach a greater weight to leisure relative to consumption, where the utility of being in a couple increases. This interpretation is supported by the results in the previous section, where a HIV diagnosis was associated with decreased consumption in the control group.

In contrast, the group of individuals diagnosed after 1995 did not face the same insurance motive for partner formation and "the option value of waiting" for the right match was higher, due to the arrival of HAART. Instead, the decline in marriage and

20

cohabitation, we see for this group may reflect social stigma and marriage turmoil.[9]

Table 3 summarizes the results for the marriage effects and shows that the effect is driven by marriage and cohabitation rather than divorces. The triple difference estimates in Figure A.9, graph (d), do not suggest that the results in this group reflect increased divorce rates, however.[10] Instead, we observe a small decrease in the divorce rate in the treatment group in the years following the diagnosis, although these effects are not statistically significant. The decrease in partner formation observed in the treatment group is thus not driven by increased divorces but rather occurs despite the small decrease in divorces. This is consistent with our interpretation of the main result being driven by the control individuals partnering and marrying more when they learn about their HIV diagnosis and their reduced life expectancy. Interestingly, the effect is driven both by marriage and cohabitation and the size of the effect is also the same. This indicates that the bequest motive may not be the dominating motive as we then would expect a strong impact on marriage where the spouse automatically inherits. Although, we do not find significant differences when the sample is split according to sexual orientation, we find that effect seems to be mainly driven by heterosexual individuals, see Table 3. While the HIV and AIDS crises and the introduction of HAART treatment may have lead to a change in public opinion towards gay people and an increase in approval of same-sex relationships in the 1990s and on, as shown in Fernandez et al. (2021), this gradual change works in the opposite direction of our results as this trend would lead to an increase in cohabitation or registered partnerships in the homosexual part of the population.

---

[9]A potential additional mechanism could relate to the observation that the arrival of HAART also reduced the risk of spreading HIV infection through sexual activity. However, reports from this period do not suggest that the stigma and sense of fear surrounding HIV went down immediately after the introduction of HAART, but only gradually happened years later (Danish AIDS foundation, 2021). Derksen et al. (2021) discusses a recent experiment in Malawi that informed about the positive externality of antiretrovirals in preventing HIV transmission.

[10]We define divorce as a flow variable that takes the value one if an individual transitions from being married with a specific partner to being either non-partnered or married with a different person.

6 Robustness

We next show that our main results are robust to alternative specifications and sample definitions. For each of the main outcomes we considered, Appendix Figures A.15 to A.16 show our baseline triple difference event study estimates in panel (a), while panels (b)-(e) show the corresponding estimates from the alternative specifications and sample definitions.

Stricter definition of the control group. An implication of our research design is that individuals in the control group, which are diagnosed with HIV between 1990 and 1994, before the introduction of HAART innovation, eventually become treated. The reason is that we restrict our sample to those alive in 1995, when HAART is introduced and when CD4 counts starts to be measured in our data. The control group thus also gets access to HAART at some point, which could potentially bias our results downwards, as the behavior of the control group would become similar to that of the treatment group.

To address this concern, we replicate the analysis on a more restrictive sample, where we drop observations of individuals in the control group beyond 1995. Individuals diagnosed in 1993, for instance, are then kept up to two years from the diagnosis, while individuals diagnosed in 1990 are kept up to 5 years after their diagnosis. As shown in Figures A.15 and A.16 our results for earnings and partner formation are robust to imposing a stricter sample restriction.

Unbalanced sample. Our main analyses are based on a balanced sample, observed 4 years before they are diagnosed until 4 years after. Figure A.2 showed that both the treatment and control groups experienced similar survival rates in the years following their diagnosis, suggesting that the balancing of the sample does not induce any differential sample selection between the treatment and control groups. Nevertheless, Graphs (c) replicates the analysis on an unbalanced sample, with similar results.

Matching period by period. In our main specification, we match each HIV positive individual with 1000 HIV negative individuals, based on their characteristics 4 years before the HIV diagnosis, and then follow both groups over time. An alternative approach is to match individuals on a year by year basis, using the same characteristics

as before. Graphs (d) show that this alternative matching strategy leads to virtually identical results.

7 Conclusion

Populations in the Western world have experienced dramatic gains in life expectancy during the last century (Case and Deaton, 2020). A longer planning horizon changes individual incentives to work, save, and marry. Our paper studies a sudden and dramatic improvement in life expectancy due to an important medical innovation. When assessing the effects of gains in life expectancy due to medical innovations, it has proven challenging to disentangle the pure incentive effect from that of improved health, since the two typically coincide. We overcome this challenge by focusing on the introduction of HAART treatment for HIV. By focusing on a sample of HIV-positive individuals who were still in good health, but who faced different access to HAART, we can observe how otherwise healthy individuals react to sharp differences in life expectancy and thus isolate the pure incentive effect due to increased life expectancy.

With inspiration from a standard life cycle model, we form hypotheses on the effects of increased life expectancy on individual choices of work/leisure, consumption/savings, and partnering/marriage. Theory informs us that individuals who receive a positive shock to life expectancy will work and save more. Moreover, given that they will be facing a longer planning horizon, they will be less inclined to select into marriage or cohabitation due to higher demands on the match quality.

We use a unique high-quality longitudinal register data set on the entire population of HIV-positive individuals in Denmark over the period 1990-2000. For this sample, we have access to administrative records on employment, income, wealth, and marriage and cohabitation status. Moreover, we use health records to follow individuals from before the onset of the HIV diagnosis until 5 and even 10 years after the diagnosis. A particularly valuable feature of our data is that we are able to link these to clinical data with detailed information on the severity of each individual's progression of HIV, measured by the CD4 (cell) count. This allows us to distinguish

23

between those who are HIV-positive, but not yet ill from AIDS, and those who suffer from full-blown AIDS. This additional information on severity of the condition allows us to separate out effects on socioeconomic choices that are mainly due to changes in life expectancy after the arrival of the new medication.

Our empirical results show that a positive shock to life expectancy due to the introduction of HAART had substantial effects on the behaviors of HIV positive individuals. First, a positive shock to life expectancy implied higher labor supply and earnings for patients diagnosed after the introduction of the new treatment. Second, higher life expectancy resulted in fewer, but better, relationships. Thus after the introduction of HAART, HIV-positive individuals no longer married and cohabited at the same rates as HIV-positive individuals diagnosed before HAART was introduced. And third, we find no significant differences in savings behavior for the treatment and control group (i.e. those diagnosed after and before the introduction of HAART, respectively). Our results highlight that life expectancy gains have important implications for individual incentives to work and marry, even when underlying health is unchanged.

References

Anderberg, D. (2007). Marriage, divorce and reciprocity-based cooperation. *The Scandinavian Journal of Economics 109*(1), 25–47.

Becker, G. (1964). *Human Capital.* University of Chicago Press.

Ben-Porath, Y. (1967). The production of human capital and the life cycle of earnings. *Journal of political economy 75*(4, Part 1), 352–365.

Bloom, D. E., D. Canning, and B. Graham (2003). Longevity and life-cycle savings. *The Scandinavian Journal of Economics 105*(3), 319–338.

Browning, M., P.-A. Chiappori, and Y. Weiss (2014). *Economics of the Family.* Cambridge Surveys of Economic Literature. Cambridge University Press.

Carstensen, M. and A. Dahl (2007). HIV og levekår - en undersøgelse af HIV-smittedes levekår i Danmark.

Case, A. and A. Deaton (2020). *Deaths of Despair and the Future of Capitalism.* Princeton University Press.

Chan, T. Y., B. H. Hamilton, and N. W. Papageorge (2015). Health, risky behaviour and the value of medical innovation for infectious disease. *The Review of Economic Studies 83*(4), 1465–1510.

Danish AIDS foundation (2021). *Da døden blev aflyst - 40 år med AIDS.*

De Nardi, M., E. French, and J. B. Jones (2009, May). Life expectancy and old age savings. *American Economic Review 99*(2), 110–15.

Derksen, L., A. Muula, and J. van Oosterhout (2021). Love in the Time of HIV: How Beliefs about Externalities Impact Health Behavior. Unpublished manuscript.

Dynan, K. E., J. Skinner, and S. P. Zeldes (2002, May). The importance of bequests and life-cycle saving in capital accumulation: A new answer. *American Economic Review 92*(2), 274–278.

Fernandez, R., S. Parsa, and M. Viarengo (2021). Coming Out in America: Thirty Years of Cultural Change. Unpublished paper.

Fortson, J. G. (2011). Mortality Risk and Human Capital Investment: The Impact of HIV/AIDS in Sub-Saharan Africa. *The Review of Economics and Statistics 93*(1), 1–15.

Goldman, D. P. and J. P. Smith (2002). Can patient self-management help explain the SES health gradient? *Proceedings of the National Academy of Sciences 99*(16), 10929–10934.

Hamermesh, D. S. (1985). Expectations, Life Expectancy, and Economic Behavior. *The Quarterly Journal of Economics 100*(2), 389–408.

Hamilton, B. H., A. Hincapié, E. C. Kalish, and N. W. Papageorge (2022). Medical innovation and health disparities. Unpublished manuscript.

Hamilton, B. H., A. Hincapié, R. A. Miller, and N. W. Papageorge (2021). Innovation and diffusion of medical treatment. *International Economic Review 62*(3), 953–1009.

Hurwitz, A. and O. Sade (2020). An investigation of time preferences, life expectancy, and annuity versus lump sum choices: Can smoking harm long-term saving decisions? *Journal of Economic Behavior & Organization 180*, 812–825.

INSIGHT START Study Group, J. D. Lundgren, A. G. Babiker, F. Gordin, S. Emery, B. Grund, S. Sharma, A. Avihingsanon, D. A. Cooper, G. Fätkenheuer, J. M. Llibre, J. M. Molina, P. Munderi, M. Schechter, R. Wood, K. L. Klingman, S. Collins, H. C. Lane, A. N. Phillips, and J. D. Neaton (2015). Initiation of antiretroviral therapy in early asymptomatic hiv infection. *The New England journal of medicine 373*, 795–807.

Jayachandran, S. and A. Lleras-Muney (2009). Life Expectancy and Human Capital Investments: Evidence from Maternal Mortality Declines. *The Quarterly Journal of Economics 124*(1), 349–397.

Johnsen, J. V., K. Vaage, and A. Willén (2022). Interactions in public policies: Spousal responses and program spillovers of welfare reforms. *The Economic Journal 132*(642), 834–864.

Karlsson, M. and S. Pichler (2015). Demographic consequences of HIV. *Journal of Population Economics 28*, 1097–1135.

26

Keiser, O., A. Spoerri, M. W. G. Brinkhof, B. Hasse, A. Gayet-Ageron, F. Tissot, A. Christen, M. Battegay, P. Schmid, E. Bernasconi, M. Egger, S. H. C. Study, and S. N. Cohort (2010). Suicide in hiv-infected individuals and the general population in switzerland, 1988-2008. *Suicide in HIV-infected individuals and the general population in Switzerland 167*, 143–50.

Lalive, R. and P. Parrotta (2017). How does pension eligibility affect labor supply in couples? *Labour Economics 46*, 177–188.

Legarth, R., L. H. Omland, G. Kronborg, C. S. Larsen, C. Pedersen, G. Pedersen, U. B. Dragsted, J. Gerstoft, and N. Obel (2014). Employment status in persons with and without HIV infection in Denmark: 1996–2011. *AIDS 28*.

Mocroft, A. and J. D. Lundgren (2004). Starting highly active antiretroviral therapy: why, when and response to HAART. *Journal of Antimicrobial Chemotherapy 54*(1), 10–13.

Oster, E. (2012). Routes of Infection: Exports and HIV Incidence in Sub-Saharan Africa. *Journal of the European Economic Association 10*(5), 1025–1058.

Oster, E., I. Shoulson, and E. Dorsey (2013a). Optimal expectations and limited medical testing: evidence from Huntington disease. *American Economic Review 103*(2), 804–30.

Oster, E., I. Shoulson, and E. R. Dorsey (2013b). Limited Life Expectancy, Human Capital and Health Investments. *American Economic Review 103*(5), 1977–2002.

Papageorge, N. W. (2021). Modeling behavior during a pandemic: Using hiv as an historical analogy. Working Paper 28898, National Bureau of Economic Research.

Papageorge, N. W., G. C. Pauley, M. Cohen, T. E. Wilson, B. H. Hamilton, and R. A. Pollak (2021). Health, human capital and domestic violence. *Journal of Human Resources*.

Persson, P. (2020). Social Insurance and the Marriage Market. *Journal of Political Economy 128*(1), 252–300.

Potoms, T. and S. Rosenberg (2021). Public insurance and marital outcomes: Evidence from the Affordable Care Act's Medicaid expansions.

Shahid, H. (2022). The Effect of Highly Active Antiretroviral Therapy (HAART) on Suicide Rates. Unpublished paper.

Strobel, F. (2003). Marriage and the value of waiting. *Journal of Population Economics 16*(3), 423–430.

Weiss, Y. (1997). Chapter 3 the formation and dissolution of families: Why marry? who marries whom? and what happens upon divorce. Volume 1 of *Handbook of Population and Family Economics*, pp. 81–123. Elsevier.

**Figure 1:** Survival by Year of HIV Diagnosis
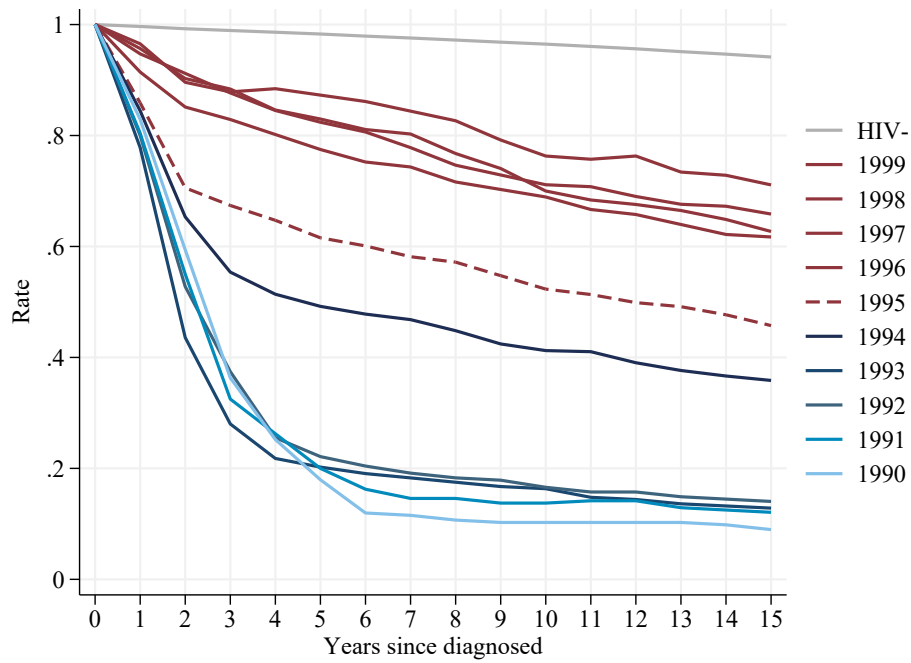
Notes: This figure plots the survival rate of individuals diagnosed with HIV distinguishing by year of diagnosis. The Figure illustrates that individuals diagnosed during earlier calendar years face sharp drops in their survival rates in the years following the diagnosis, while individuals diagnosed later, after the introduction of HAART medical innovation in 1995, face much improved survival rates. The gray line plots, for reference, the survival rates of a sample of individuals not diagnosed with HIV. The Figure is constructed using Danish hospital records on all HIV diagnoses (*landspatientregisteret*) which is not affected by any break or change of definitions during the period considered.

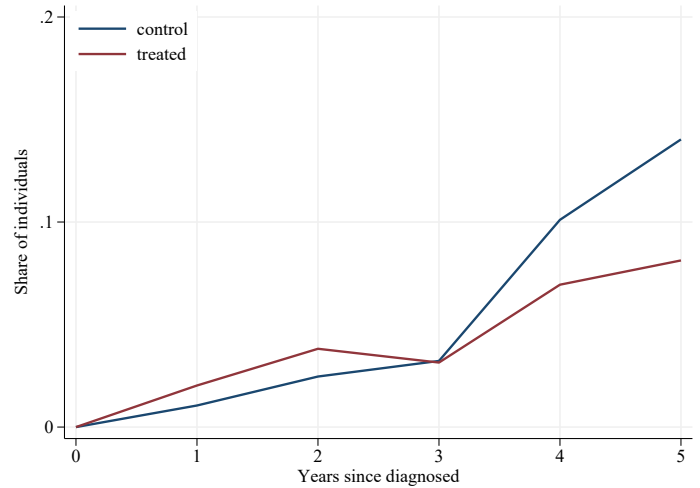**Figure 2:** Share of Treated and Control Individuals Below CD4 Count Thresholds

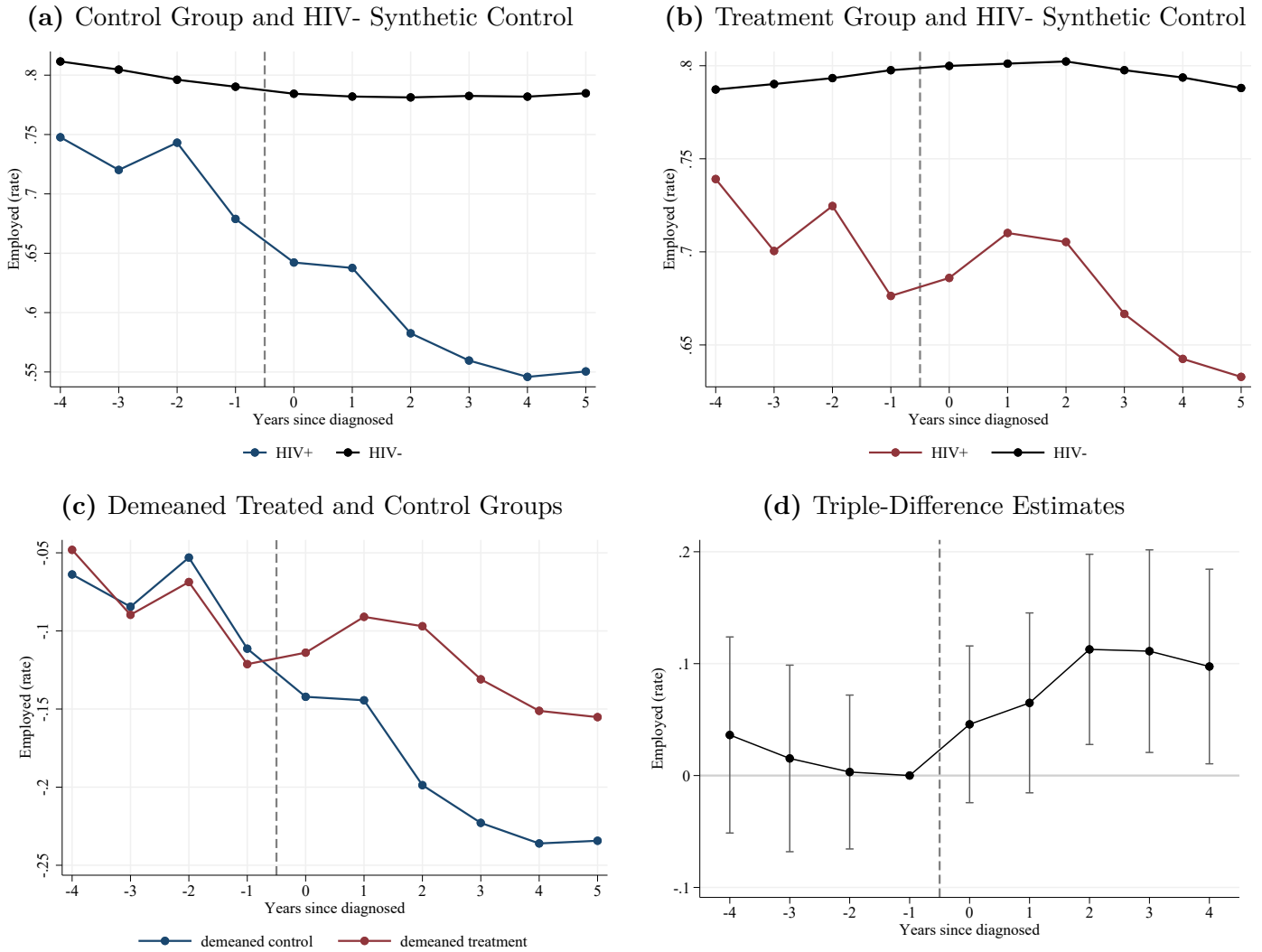**(a)** CD4 Count Under 200
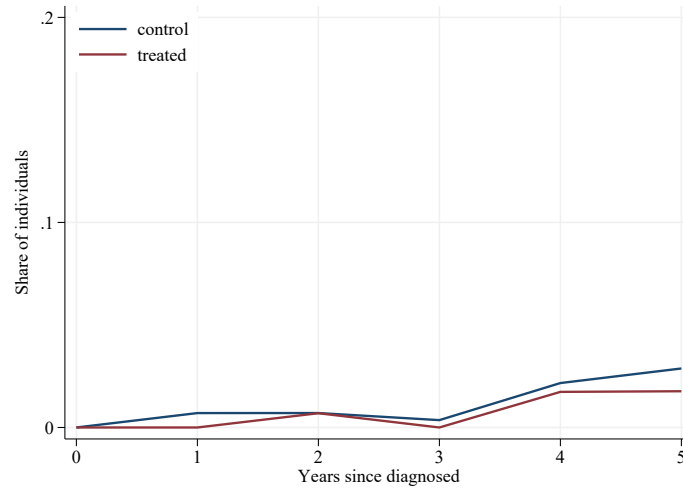
(b) CD4 Count Under 250
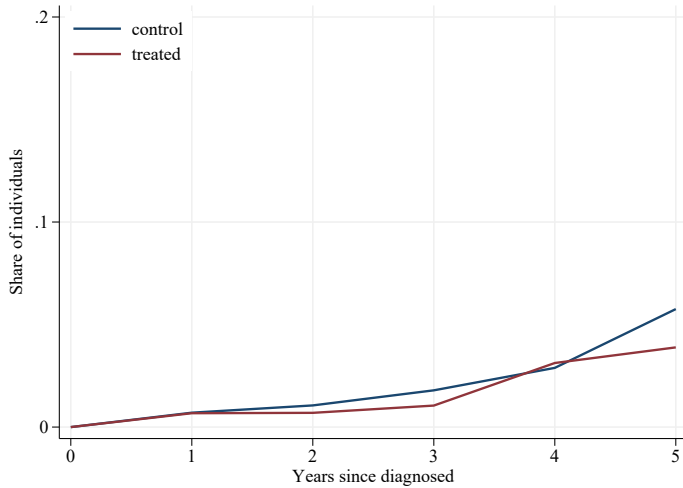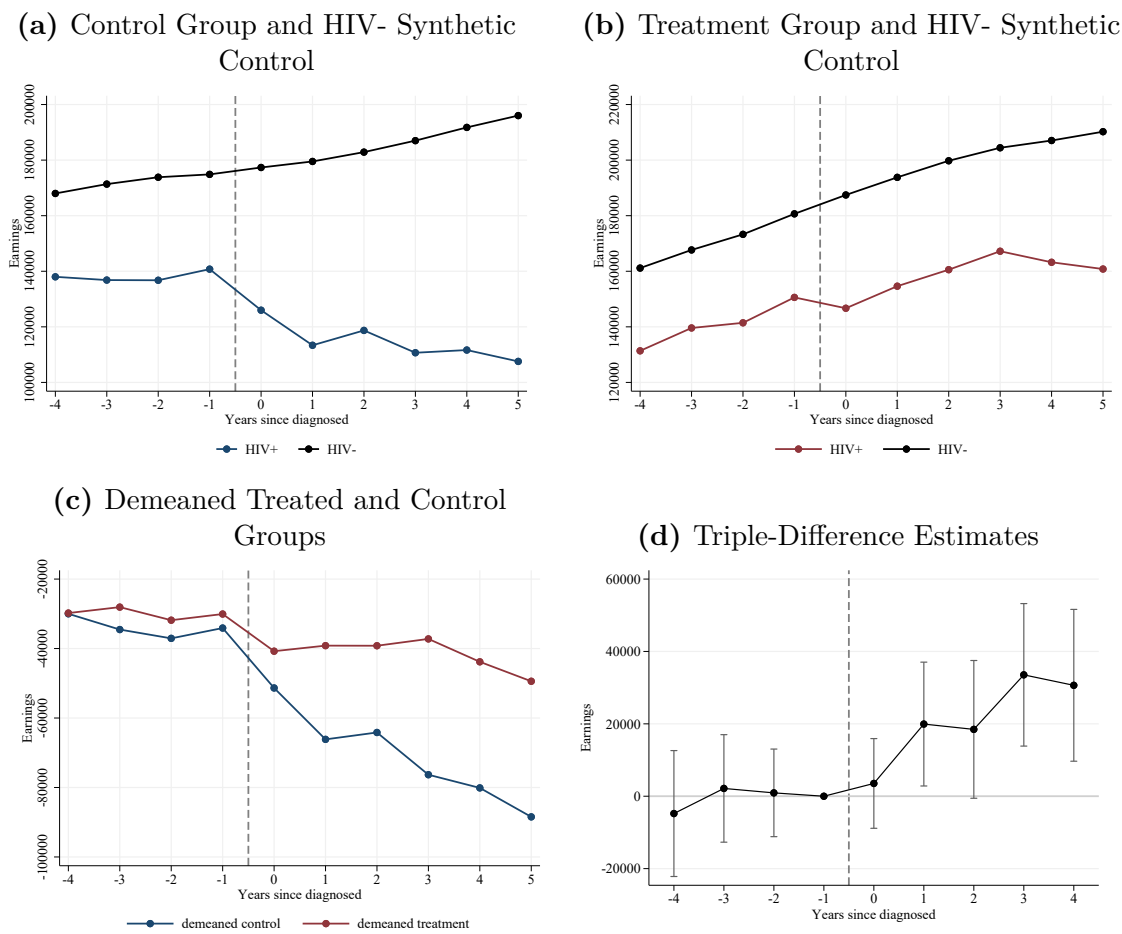

(c) CD4 Count Under 300

Notes: These figures plot the share of individuals in the treatment and control groups whose CD4 counts are below a certain threshold. In panel (a) the threshold is 200, which is considered the level where AIDS can begin. In panel (b) the threshold is 250. In panel (c) the threshold is 300.

Figure 3: Effects on Employment from Medical Innovation around the Time of HIV Diagnosis

(a) Control Group and HIV- Synthetic Control

(b) Treatment Group and HIV- Synthetic Control

(c) Demeaned Treated and Control Groups

(d) Triple-Difference Estimates

Notes: These graphs plot the effects on employment of the introduction of HAART medical innovation on individuals around the time when they are diagnosed with HIV. Graph (a) plots an event study for the control group (those diagnosed with HIV between 1990 and 1994, before the introduction of HAART) against a synthetic control of HIV– individuals matched in year -4 from the same cohort, age, gender, and education. Graph (b) plots an event study for the treatment group (those diagnosed with HIV between 1996 and 1999, after the introduction of HAART) against a synthetic control of HIV– individuals matched in year -4 from the same cohort, age, gender, and education. Graph (c) plots the control and treatment groups demeaned by their respective synthetic controls. Graph (d) plots the β_t estimates of the triple difference-in-differences model estimated in Equation (1), which identify dynamic causal effects of the introduction of HAART medical innovation. We report 95% confidence intervals calculated from clustered standard errors.

31

Figure 4: Effects on Earnings from Medical Innovation around the Time of HIV Diagnosis

(a) Control Group and HIV- Synthetic Control

(b) Treatment Group and HIV- Synthetic Control

(c) Demeaned Treated and Control Groups
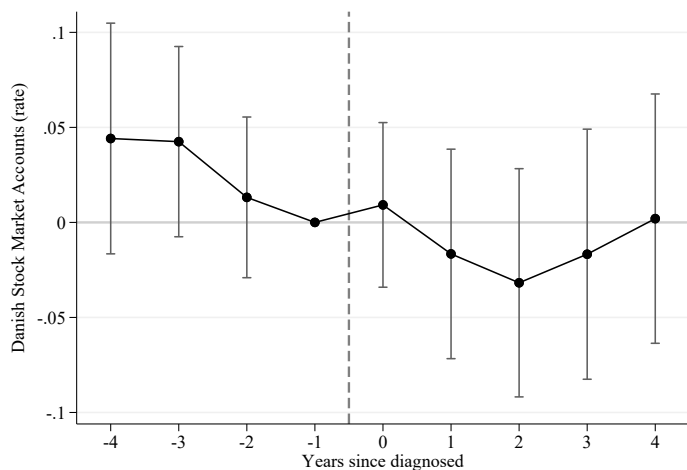

(d) Triple-Difference Estimates

Notes: These graphs plot the effects on earnings of the introduction of HAART medical innovation on individuals around the time when they are diagnosed with HIV. Graph (a) plots an event study for the control group (those diagnosed with HIV between 1990 and 1994, before the introduction of HAART) against a synthetic control of HIV– individuals matched in year -4 from the same cohort, age, gender, and education. Graph (b) plots an event study for the treatment group (those diagnosed with HIV between 1996 and 1999, after the introduction of HAART) against a synthetic control of HIV– individuals matched in year -4 from the same cohort, age, gender, and education. Graph (c) plots the control and treatment groups demeaned by their respective synthetic controls. Graph (d) plots the β_t estimates of the triple difference-in-differences model estimated in Equation (1), which identify dynamic causal effects of the introduction of HAART medical innovation. We report 95% confidence intervals calculated from clustered standard errors.

Figure 5: Triple-Difference Estimates of the Effects on Savings, Stocks, and Housing from Medical Innovation around the Time of HIV Diagnosis
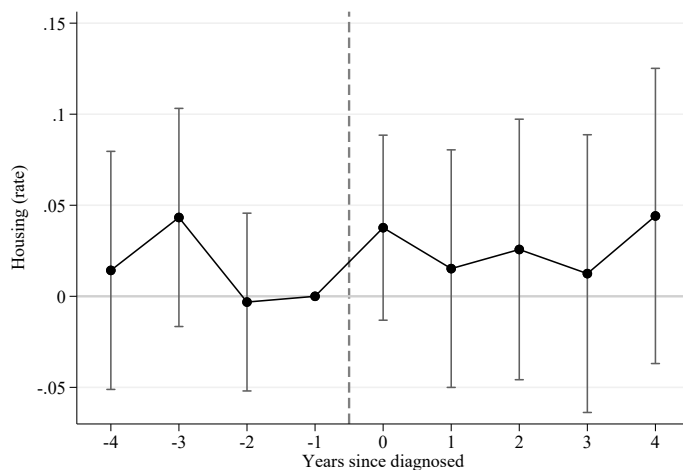
(a) Bank Deposits
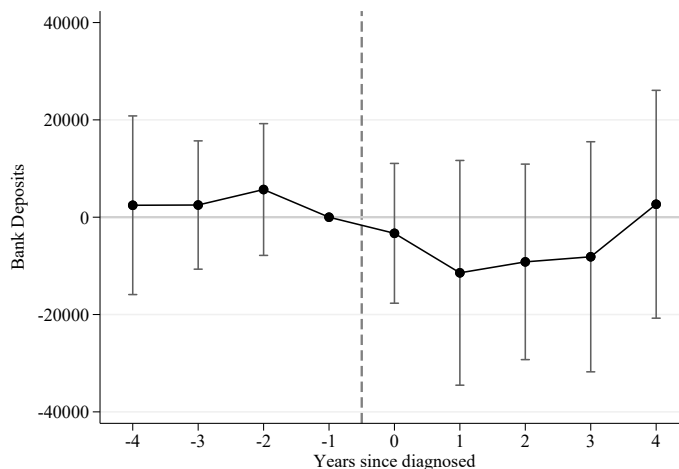
(b) Stock Ownsership

(c) Housing Ownsership

Notes: These graphs plot the effects on Savings, Stocks, and Housing of the introduction of HAART medical innovation on individuals around the time when they are diagnosed with HIV. Each graph plots the β_t estimates of the triple difference model estimated in Equation (1), which identify dynamic causal effects of the introduction of HAART medical innovation. We report 95% confidence intervals calculated from clustered standard errors.

Figure 6: Effects on Partnership from Medical Innovation around the Time of HIV Diagnosis

(a) Control Group and HIV- Synthetic Control

(b) Treatment Group and HIV- Synthetic Control

(c) Demeaned Treated and Control Groups

(d) Triple-Difference Estimates
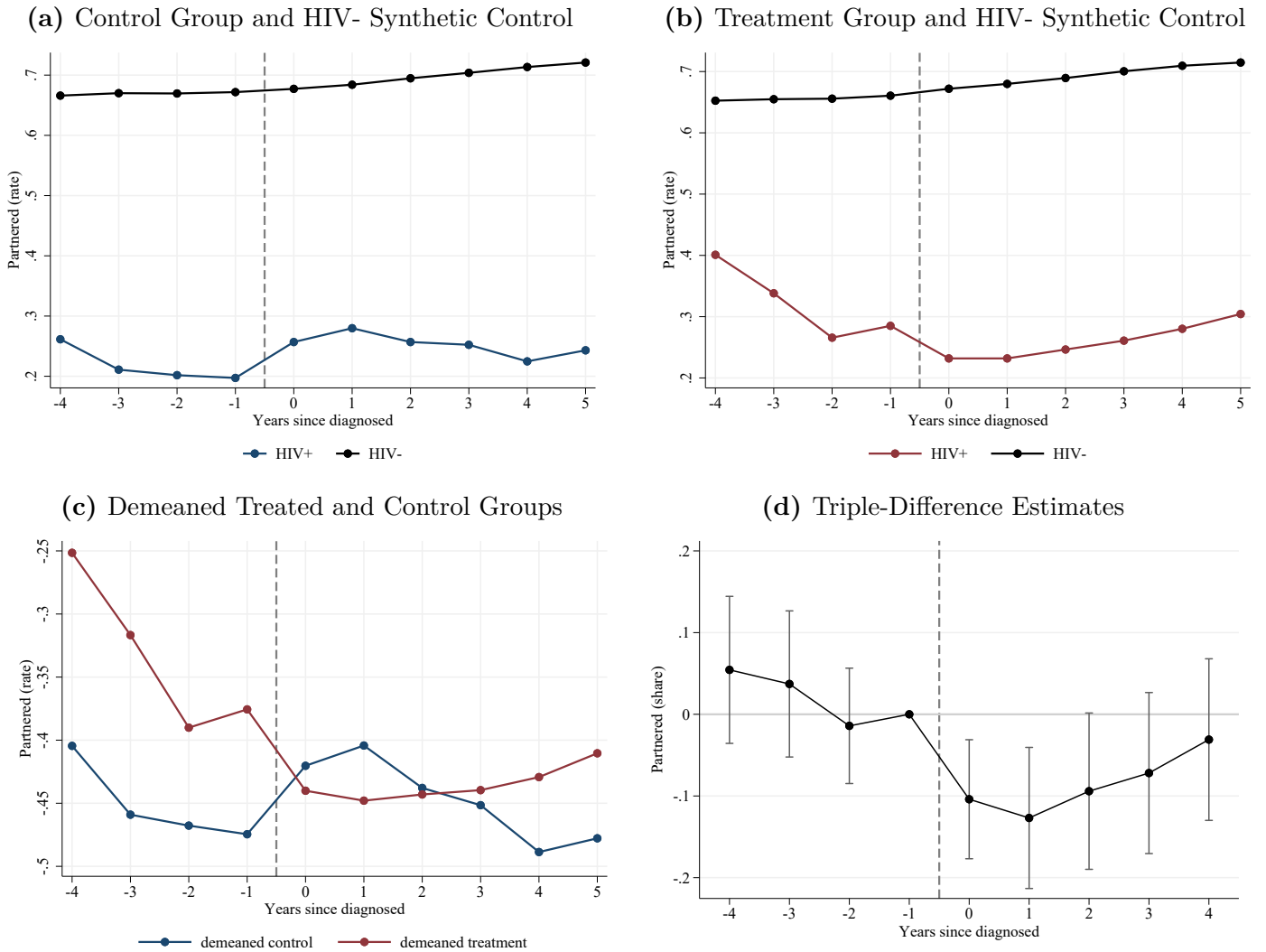
Notes: These graphs plot the effects on marital status (being married or in cohabitation) of the introduction of HAART medical innovation on individuals around the time when they are diagnosed with HIV. Graph (a) plots an event study for the control group (those diagnosed with HIV between 1990 and 1994, before the introduction of HAART) against a synthetic control of HIV– individuals matched in year -4 from the same cohort, age, gender, and education. Graph (b) plots an event study for the treatment group (those diagnosed with HIV between 1996 and 1999, after the introduction of HAART) against a synthetic control of HIV– individuals matched in year -4 from the same cohort, age, gender, and education. Graph (c) plots the control and treatment groups demeaned by their respective synthetic controls. Graph (d) plots the β_t estimates of the triple difference-in-differences model estimated in Equation (1), which identify dynamic causal effects of the introduction of HAART medical innovation. We report 95% confidence intervals calculated from clustered standard errors.

34

Figure 7: Effects on Marriage from Medical Innovation around the Time of HIV Diagnosis

(a) Control Group and HIV- Synthetic Control

(b) Treatment Group and HIV- Synthetic Control

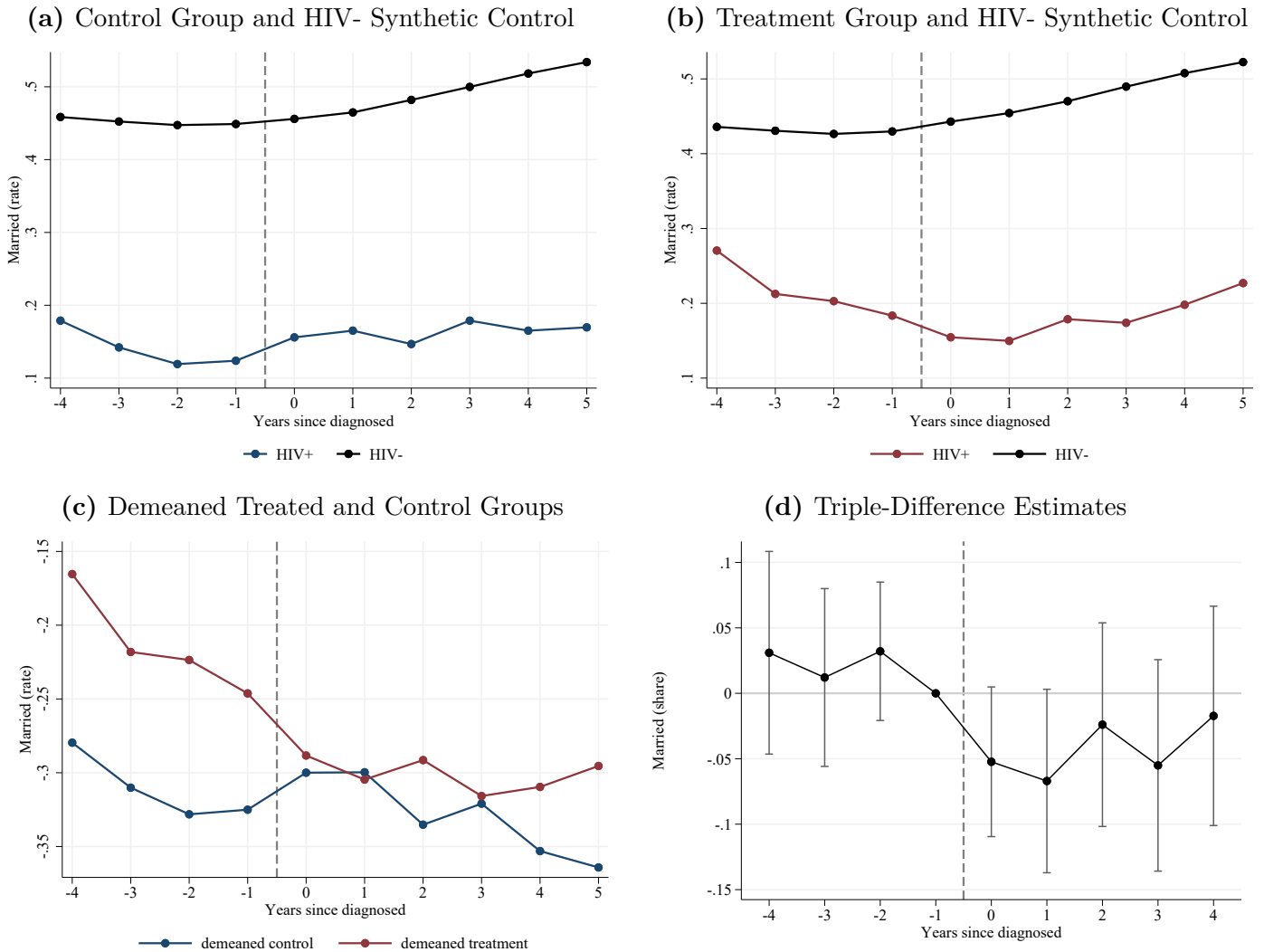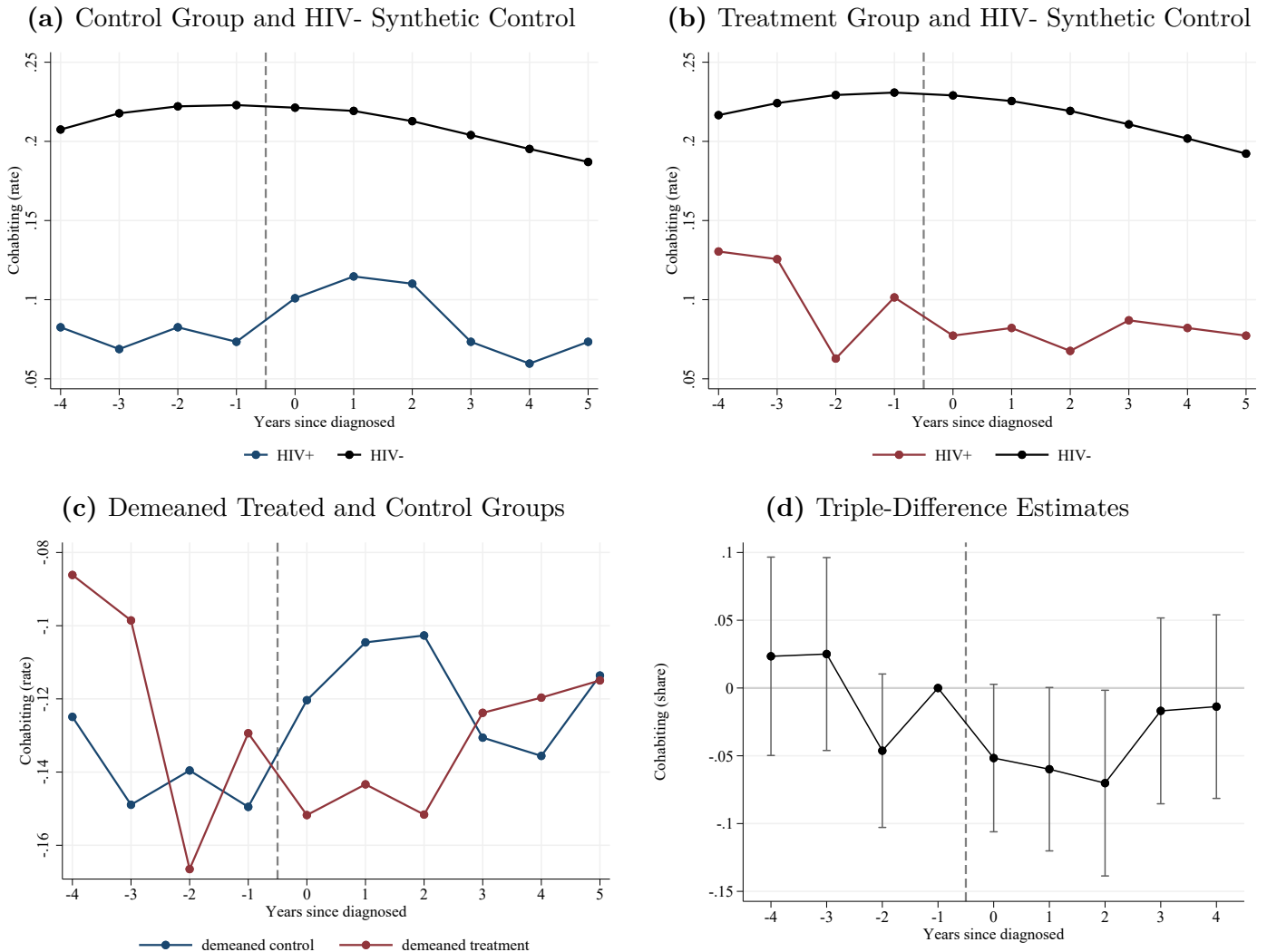**(c)** Demeaned Treated and Control Groups

(d) Triple-Difference Estimates

Notes: These graphs plot the effects on marriage of the introduction of HAART medical innovation on individuals around the time when they are diagnosed with HIV. Graph (a) plots an event study for the control group (those diagnosed with HIV between 1990 and 1994, before the introduction of HAART) against a synthetic control of HIV− individuals matched in year -4 from the same cohort, age, gender, and education. Graph (b) plots an event study for the treatment group (those diagnosed with HIV between 1996 and 1999, after the introduction of HAART) against a synthetic control of HIV− individuals matched in year -4 from the same cohort, age, gender, and education. Graph (c) plots the control and treatment groups demeaned by their respective synthetic controls. Graph (d) plots the β_t estimates of the triple difference-in-differences model estimated in Equation (1), which identify dynamic causal effects of the introduction of HAART medical innovation. We report 95% confidence intervals calculated from clustered standard errors.

Figure 8: Effects on Cohabitation from Medical Innovation around the Time of HIV Diagnosis

(a) Control Group and HIV- Synthetic Control

(b) Treatment Group and HIV- Synthetic Control

(c) Demeaned Treated and Control Groups

(d) Triple-Difference Estimates

Notes: These graphs plot the effects on marriage of the introduction of HAART medical innovation on individuals around the time when they are diagnosed with HIV. Graph (a) plots an event study for the control group (those diagnosed with HIV between 1990 and 1994, before the introduction of HAART) against a synthetic control of HIV– individuals matched in year -4 from the same cohort, age, gender, and education. Graph (b) plots an event study for the treatment group (those diagnosed with HIV between 1996 and 1999, after the introduction of HAART) against a synthetic control of HIV– individuals matched in year -4 from the same cohort, age, gender, and education. Graph (c) plots the control and treatment groups demeaned by their respective synthetic controls. Graph (d) plots the β_t estimates of the triple difference-in-differences model estimated in Equation (1), which identify dynamic causal effects of the introduction of HAART medical innovation. We report 95% confidence intervals calculated from clustered standard errors.

Table 1: Descriptive Statistics

	Control	Treated	Difference	P-value	HIV−
	(1)	(2)	(3)	(4)	(5)
Demographics					
Age	33.64	34.49	-0.85	0.39	34.04
Male	0.82	0.80	0.03	0.61	0.81
Months of education	138.73	141.39	-2.67	0.34	162.84
Dane	0.93	0.90	0.03	0.25	0.96
Economics					
Employed	0.68	0.68	0.00	1.00	0.81
Earnings (percentile)	41.58	44.07	-2.49	0.37	50.5
Disability Recipient	0.07	0.07	0.01	0.76	0.04
Home Owner	0.24	0.27	-0.03	0.52	0.49
Housing (percentile)	37.06	39.23	-2.17	0.28	50.5
Net wealth (percentile)	35.93	35.43	0.50	0.87	50.5
Stocks Ownership	0.14	0.07	0.07	0.01	0.16
Marital					
Partnered	0.20	0.28	-0.08	0.04	0.67
Married	0.13	0.18	-0.05	0.11	0.44
Cohabiting	0.07	0.10	-0.03	0.26	0.23
Health					
Psychotherapy	<0.01	<0.01	0.00	1.00	0.002
Psychiatry	0.02	0.03	-0.01	0.65	0.008
Charlson Index	0.00	0.02	-0.02	0.08	0.006
Infections	<0.01	<0.01	-0.01	0.14	0.001
DANHIV					
CD4 Count	617.99	620.89	-2.90	0.87	–
Heterosexual	0.43	0.44	-0.01	0.82	–
Observations	229	210			439,000

Notes: This table presents summary statistics of key variables for different samples, measured one year before diagnosis, except CD4 counts that are measured on the year of diagnosis. Column (1) corresponds to the control group of the analysis sample: individuals diagnosed with HIV between 1990 and 1994. Column (2) corresponds to the treatment group of the analysis sample: individuals diagnosed with HIV between 1995 and 1999. Column (3) shows the difference in means between columns (1) and (2). Column (4) reports the p-value for a test of equal means. Column (5) corresponds to a sample of individuals who are not diagnosed with HIV. This sample is constructed by matching 1,000 individuals of the same cohort, age and gender, to each of the individuals in the analysis sample.

Table 2: Effects of Medical Innovation after HIV Diagnosis. Triple-Difference Estimates

	Estimate (1)	Mean (2)
A: Labor Supply		
Employment	0.0753*** (0.028)	0.663
Earnings	23,897*** (7,706)	130,918
Earnings (cond. part.)	23,909*** (8,879)	180,733
B: Public Benefits		
Any Disability Benefits	-0.0654*** (0.0210)	0.075
Disability Benefits	-4,068** (1,987)	6,168
C: Assets		
Any Stocks	-0.0330 (0.0245)	0.119
Bank Accounts	-6,415 (7,537)	26,882
Home Ownership	0.0213 (0.0307)	0.267
Obs.	4,394,390	4,394,390
N. Clusters	439,439	–

Notes: Column (1) in this table reports the coefficient of interest β_1 estimated in Equation (2) that captures the causal effect of the introduction of HAART medical innovation that extended life expectancy on different outcomes, up to 5 years following diagnosis. Column (2) reports the average value of a given outcome measured the year before HIV diagnosis for the sample of analysis.

Table 3: Marital Effects of Medical Innovation after HIV Diagnosis. Triple-Difference Estimates.

| | 5 years post-period | | | 3 years post-period | | | |
| | Full Sample | Hetero. | Homo. | Full Sample | Hetero. | Homo. | Mean |
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Partnered	-0.0944***	-0.0965*	-0.0952**	-0.119***	-0.146**	-0.0958**	0.24
	(0.0360)	(0.0576)	(0.0440)	(0.0356)	(0.0572)	(0.0433)	
Married	-0.0540*	-0.0431	-0.0650	-0.0684**	-0.0782	-0.0604	0.15
	(0.0323)	(0.0505)	(0.0411)	(0.0313)	(0.0490)	(0.0397)	
Cohabiting	-0.0404*	-0.0534	-0.0302	-0.0502**	-0.0680*	-0.0353	0.09
	(0.0217)	(0.0383)	(0.0217)	(0.0224)	(0.0393)	(0.0232)	
Divorce	-0.0106	-0.0180	-0.00328	-0.00642	-0.0144	-0.00152	0.03
	(0.00983)	(0.0164)	(0.0113)	(0.0110)	(0.0175)	(0.0135)	
Obs.	4,394,390	2,092,090	2,302,300	3,515,512	1,673,672	1,841,840	4,394,390
N. Clusters	439,439	209,209	230,230	439,439	209,209	230,230	439,439

Notes: Columns (1) to (6) in this table report the coefficient of interest β_1 estimated in Equation (2) that captures the causal effect of the introduction of HAART medical innovation that extended life expectancy on different outcomes, for different subsamples. Column (7) reports the average value of a given outcome measured the year before HIV diagnosis for the full sample of analysis. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$

Table 4: Health Effects of Medical Innovation after HIV Diagnosis. Triple-Difference Estimates

	Estimate (1)	Mean (2)
A: Physical Health		
Charlson Index	0.0021 (0.010)	0.021
Infections	0.0009 (0.005)	0.007
B: Mental Health		
Psychologist	-0.0010 (0.007)	0.023
Psychiatrist	0.0092 (0.010)	0.023
Obs.	4,394,390	4,394,390
N. Clusters	439,439	–

Notes: Column (1) in this table reports the coefficient of interest β_1 estimated in Equation (2) that captures the causal effect of the introduction of HAART medical innovation that extended life expectancy on different outcomes, up to 5 years following diagnosis. Column (2) reports the average value of a given outcome measured the year before HIV diagnosis for the sample of analysis.

Appendix A

A.1   The development of the HAART treatment

The HAART medication was introduced in 1996 in Denmark, but already in 1995 the first positive indications of the new treatment were presented. In the mid-90'ties there was a lot of attention on the medical innovations on HIV treatment and the media were frequently reporting from scientific conferences and events. In the Table A.1, we show the important date of the medical breakthrough and examples of how the information was disseminated to a wide audience. Anecdotal evidence suggests that the optimism among Danish researchers started after the "Fifth European Conference on Clinical Aspects and Treatment of HIV Infection" held in September 26-29, 1995 in Copenhagen. Already the day after the conference, the positive news were disseminated to the Danish population. A article published on September 30, 1995 in the Danish newspaper Politiken had the headline "Great confidence in new HIV medicine" by Kaare Skovmand. A quote from the article *"The AIDS conference in Copenhagen gave international researchers a rare opportunity to bring out the smile. For the first time in many years, definite positive results could be presented, as several studies independently showed that many HIV-positive people can look forward to a longer life by being treated with a combination of the old drug AZT and the two newer drugs ddI and ddC."*[11] illustrates the growing optimism and the beginning of a new era with an effective treatment of HIV.

A.2   Tables and Figures

---

[11]"Stor tiltro til ny HIV-medicin", Politiken September 30, 1995. Our translation

Table A.1: Development of HAART Treatment

Date	Event	Example on news coverage
June,1995	FDA approves the saquinavir, which is the first protease inhibitor. The protease inhibitor is an import component of the HAART treatment	
September,1995	Fifth European Conference on Clinical Aspects and Treatment of HIV Infection, Copenhagen, Denmark Positive results on the treatment with the protease inhibitor are presented.	Article in Politiken, September 30: *Great confidence in new HIV medicine* by Kaare Skovmand.
December, 1995	FDA approves the use of saquinavir in combination with other drugs.	Article in NYT, December 8: *FDA backs a new drug to fight AIDS*
July, 1996	11th AIDS conference Vancouver, Canada confirms the positive effect of HAART	Article in NYT, July 15: *From the AIDS conference: Talk about life Not Death*

Figure A.1: Distribution of individual's Observed CD4 Count Changes
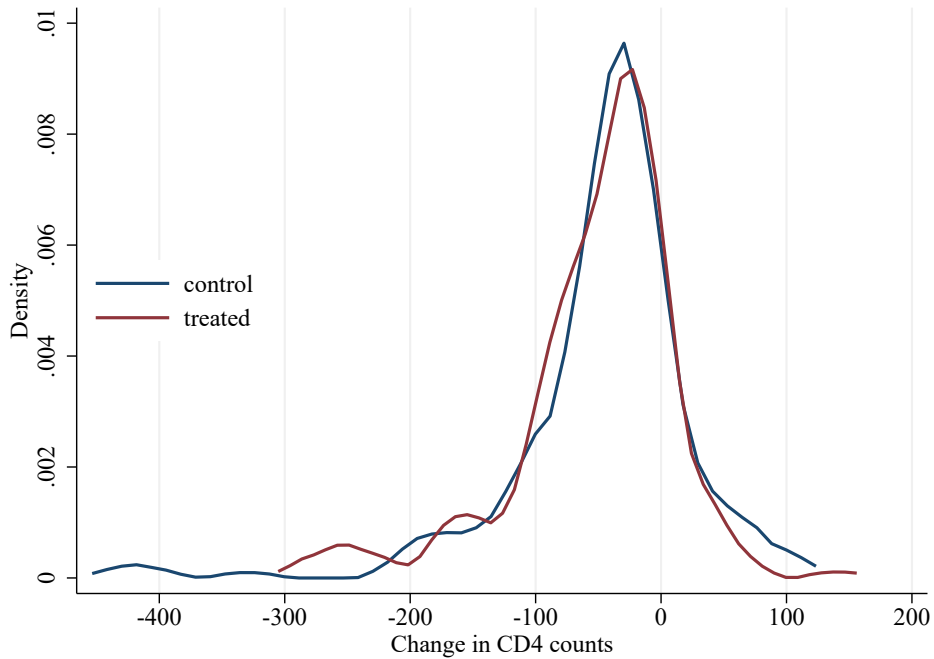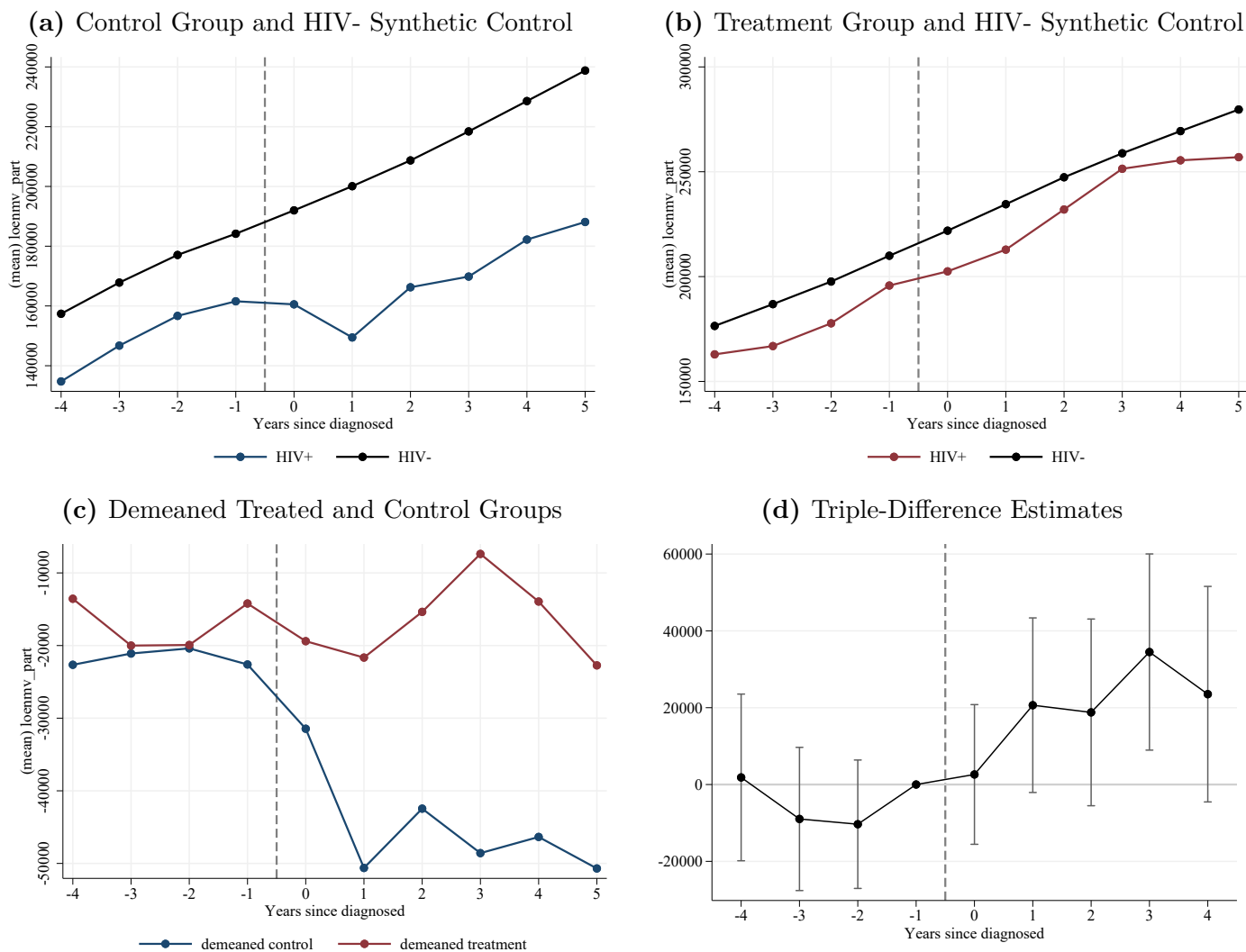

Notes: This figure shows the distribution of the slope parameters resulting from estimating the following linear equation: $CD4_{it} = \alpha + \beta * time_i + \epsilon_{it}$ for each individual observed at least two times between being diagnosed with HIV and receiving HAART treatment. $CD4_{it}$ refers to observed CD4 counts, and $time$ are years from diagnosis. Note that in our main imputation equation we also use a quadratic term for $time$ and we use a flat imputation for individuals observed for less than 2 periods between diagnosis and starting HAART medication.
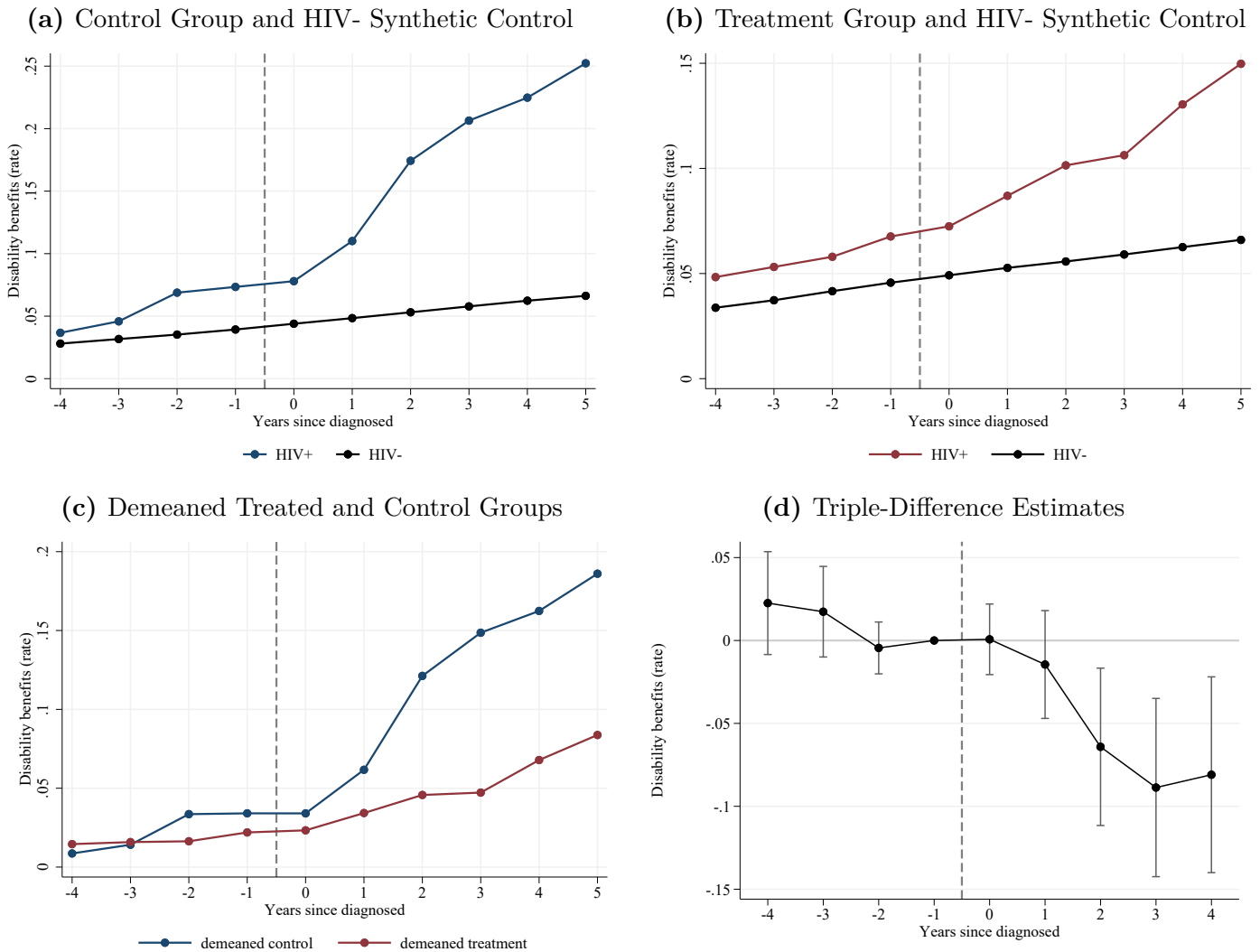
Figure A.2: Survival by Treatment Group
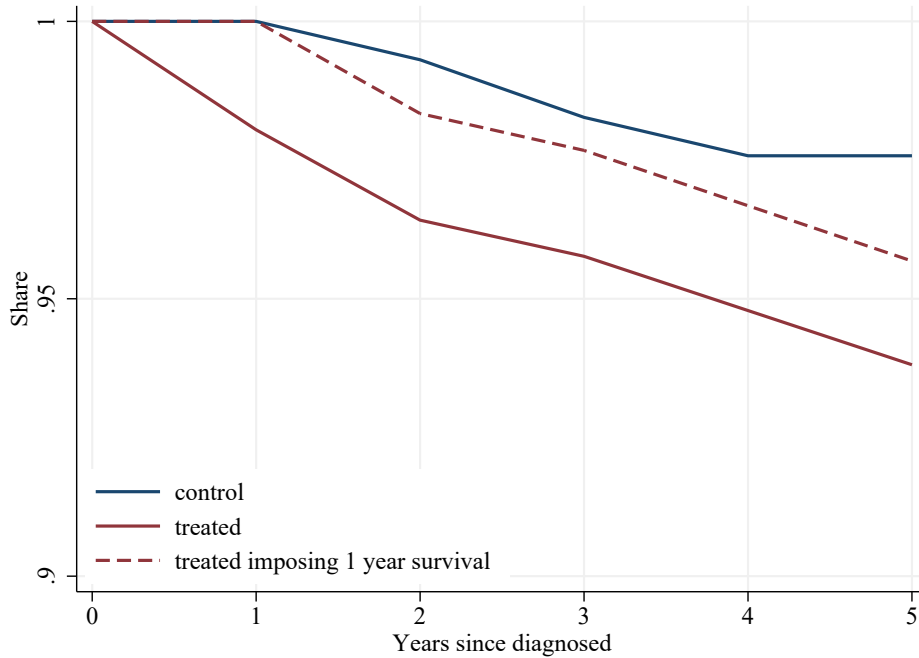
Notes: This figure shows the share of individuals alive distinguishing by treatment and control group
before we impose a balancing of the sample (that is, before we keep only those individuals who are
alive and observed every year since four years before diagnosis until five years after diagnosis). All
these individuals are diagnosed with high levels of CD4 (above 400). The solid blue line corresponds
to individuals diagnosed before 1995. The solid blue line corresponds to individuals diagnosed after
1995. The dashed red line also corresponds to individuals diagnosed after 1995, but imposing that
they survive for at least one year, so that they are more comparable to individuals in the blue line
(diagnosed before 1995) because by construction all individuals diagnosed before 1995 must survive
at least one year to be included in the dataset, which was created starting in 1995.

Figure A.3: Effects on Earnings Conditional on Participation from Medical Innovation around the Time of HIV Diagnosis

(a) Control Group and HIV- Synthetic Control

(b) Treatment Group and HIV- Synthetic Control

(c) Demeaned Treated and Control Groups
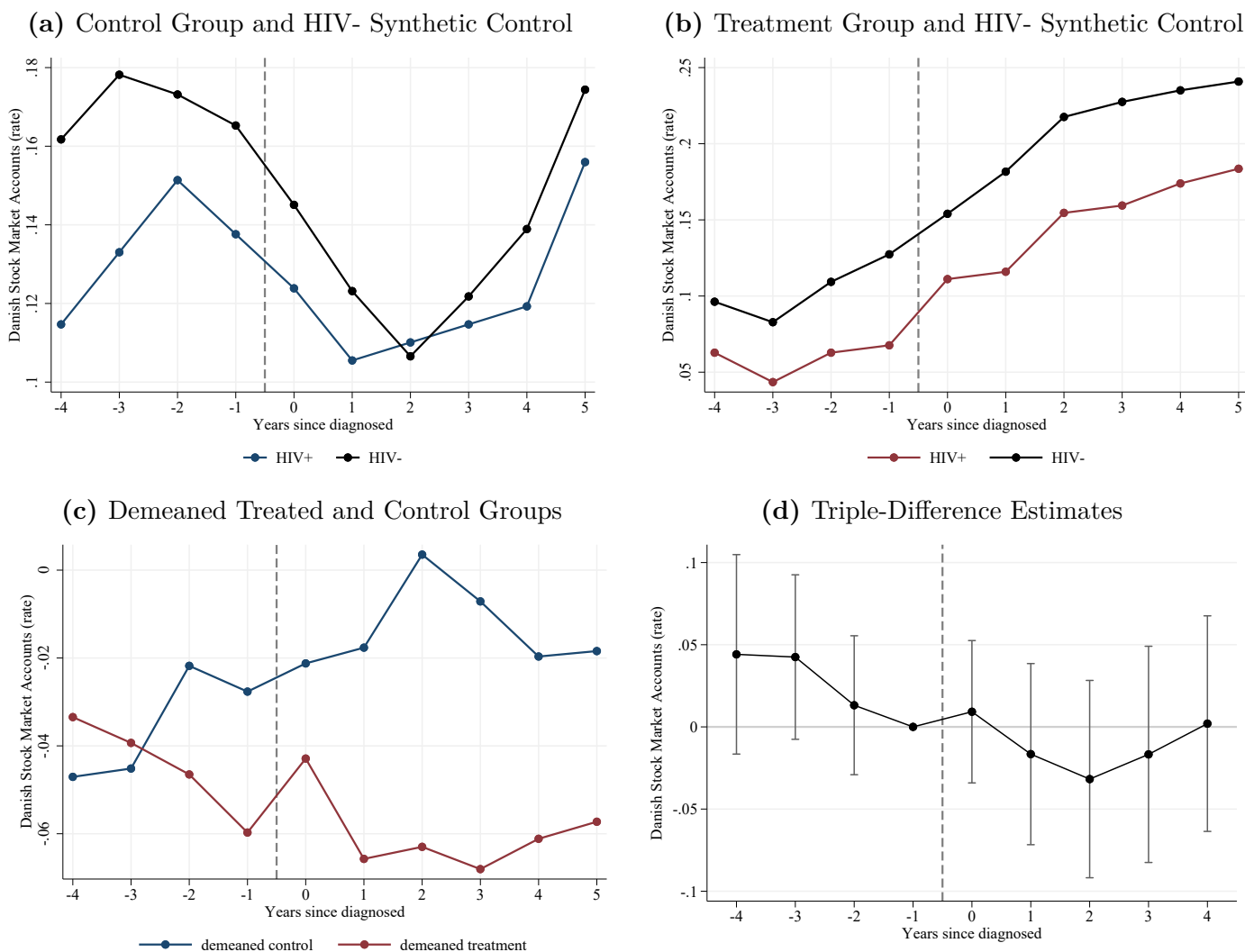

(d) Triple-Difference Estimates

Notes: These graphs plot the effects on earnings conditional on participation of the introduction of HAART medical innovation on individuals around the time when they are diagnosed with HIV. Graph (a) plots an event study for the control group (those diagnosed with HIV between 1990 and 1994, before the introduction of HAART) against a synthetic control of HIV– individuals matched in year -4 from the same cohort, age, gender, and education. Graph (b) plots an event study for the treatment group (those diagnosed with HIV between 1996 and 1999, after the introduction of HAART) against a synthetic control of HIV– individuals matched in year -4 from the same cohort, age, gender, and education. Graph (c) plots the control and treatment groups demeaned by their respective synthetic controls. Graph (d) plots the β_t estimates of the triple difference-in-differences model estimated in Equation (1), which identify dynamic causal effects of the introduction of HAART medical innovation. We report 95% confidence intervals calculated from clustered standard errors.

Figure A.4: Effects on Disability Benefits from Medical Innovation around the Time of HIV Diagnosis

(a) Control Group and HIV- Synthetic Control

(b) Treatment Group and HIV- Synthetic Control

(c) Demeaned Treated and Control Groups
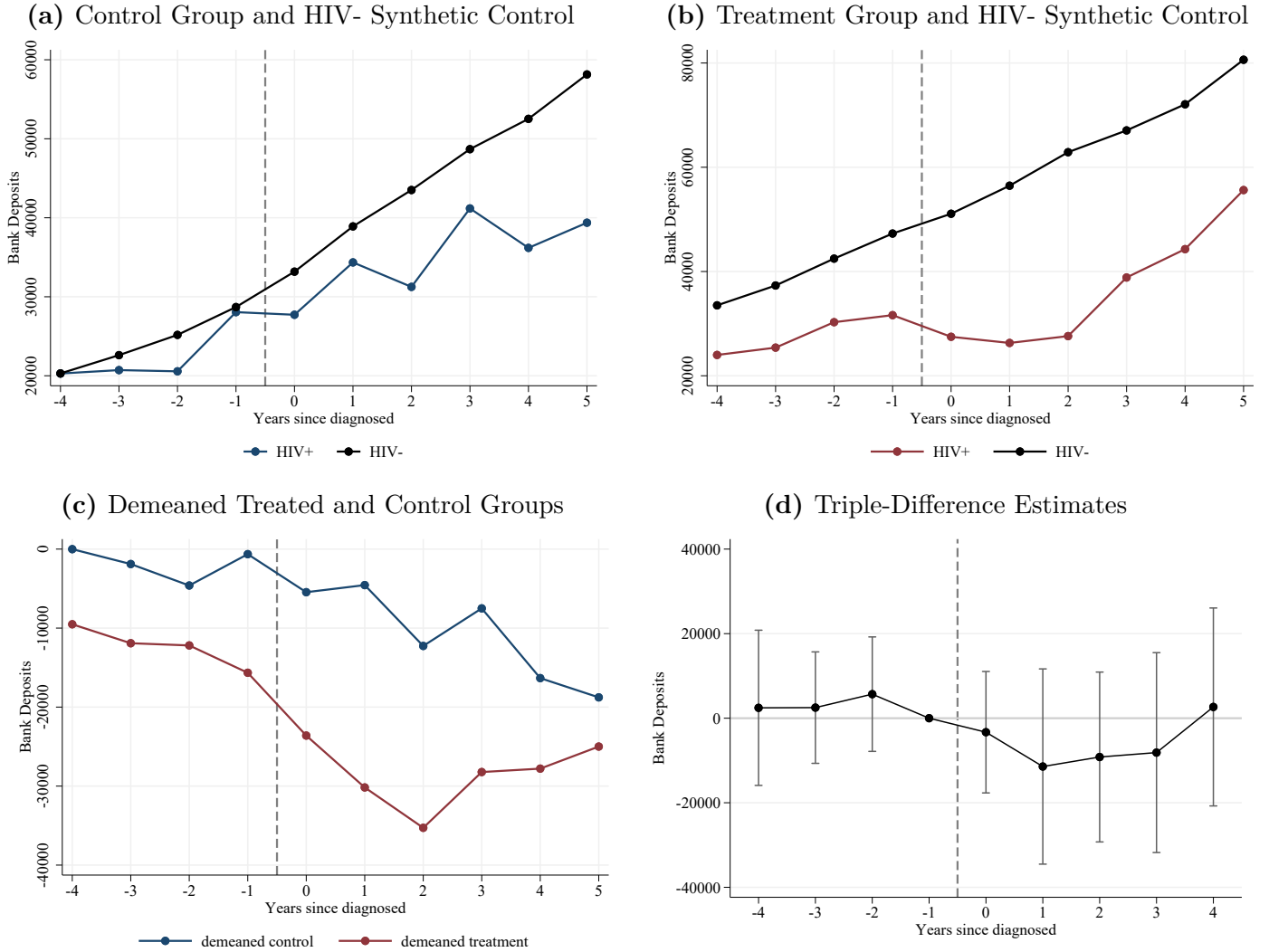
(d) Triple-Difference Estimates

Notes: These graphs plot the effects on disability benefits of the introduction of HAART medical innovation on individuals around the time when they are diagnosed with HIV. Graph (a) plots an event study for the control group (those diagnosed with HIV between 1990 and 1994, before the introduction of HAART) against a synthetic control of HIV– individuals matched in year -4 from the same cohort, age, gender, and education. Graph (b) plots an event study for the treatment group (those diagnosed with HIV between 1996 and 1999, after the introduction of HAART) against a synthetic control of HIV– individuals matched in year -4 from the same cohort, age, gender, and education. Graph (c) plots the control and treatment groups demeaned by their respective synthetic controls. Graph (d) plots the β_t estimates of the triple difference-in-differences model estimated in Equation (1), which identify dynamic causal effects of the introduction of HAART medical innovation. We report 95% confidence intervals calculated from clustered standard errors.

Figure A.5: Effects on Stocks Ownership from Medical Innovation around the Time of HIV Diagnosis

(a) Control Group and HIV- Synthetic Control

(b) Treatment Group and HIV- Synthetic Control

(c) Demeaned Treated and Control Groups
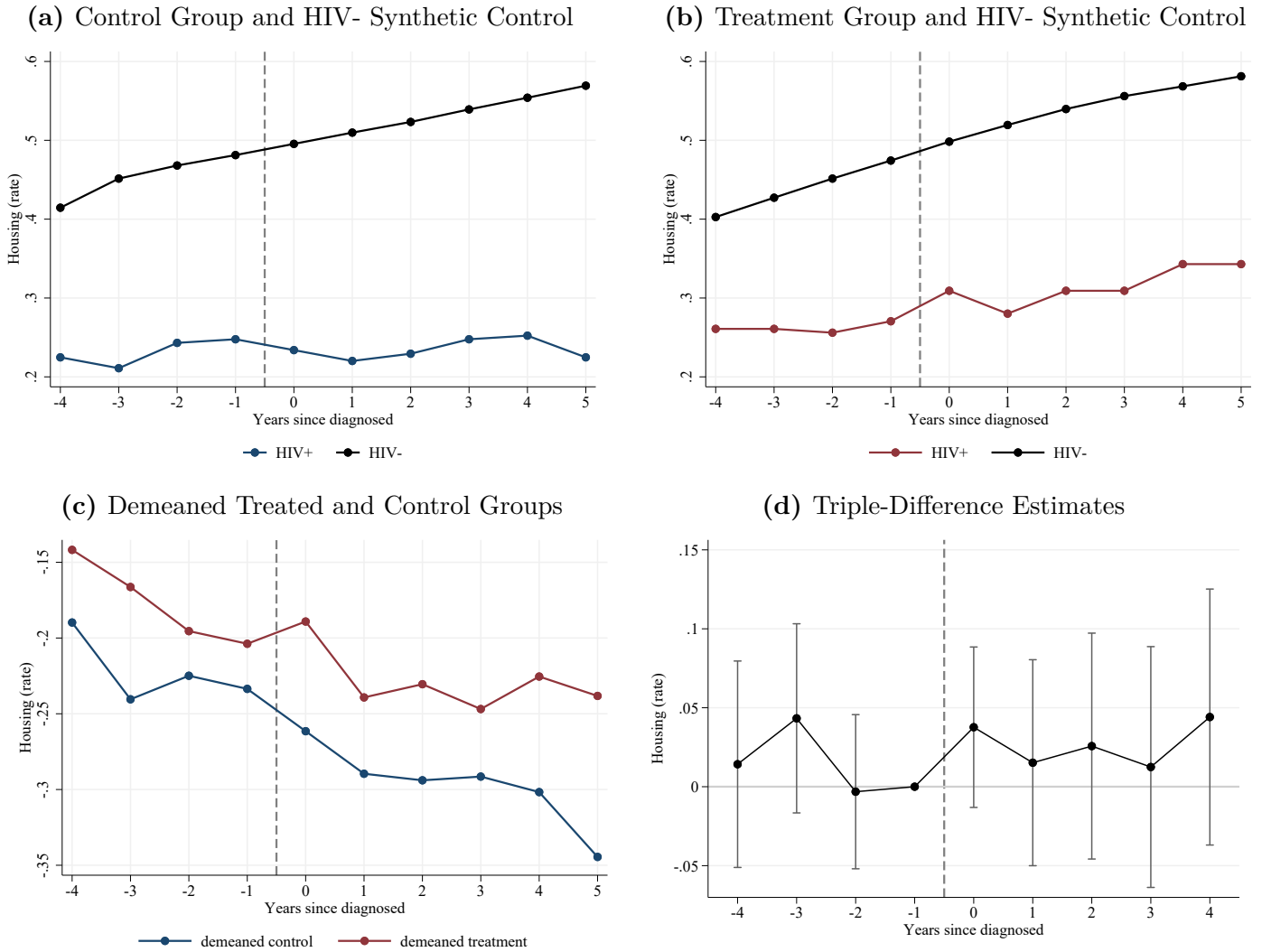

(d) Triple-Difference Estimates

Notes: These graphs plot the effects on stocks ownership of the introduction of HAART medical innovation on individuals around the time when they are diagnosed with HIV. Graph (a) plots an event study for the control group (those diagnosed with HIV between 1990 and 1994, before the introduction of HAART) against a synthetic control of HIV− individuals matched in year -4 from the same cohort, age, gender, and education. Graph (b) plots an event study for the treatment group (those diagnosed with HIV between 1996 and 1999, after the introduction of HAART) against a synthetic control of HIV− individuals matched in year -4 from the same cohort, age, gender, and education. Graph (c) plots the control and treatment groups demeaned by their respective synthetic controls. Graph (d) plots the β_t estimates of the triple difference-in-differences model estimated in Equation (1), which identify dynamic causal effects of the introduction of HAART medical innovation. We report 95% confidence intervals calculated from clustered standard errors.

Figure A.6: Effects on Bank Accounts from Medical Innovation around the Time of HIV Diagnosis

(a) Control Group and HIV- Synthetic Control

(b) Treatment Group and HIV- Synthetic Control

(c) Demeaned Treated and Control Groups
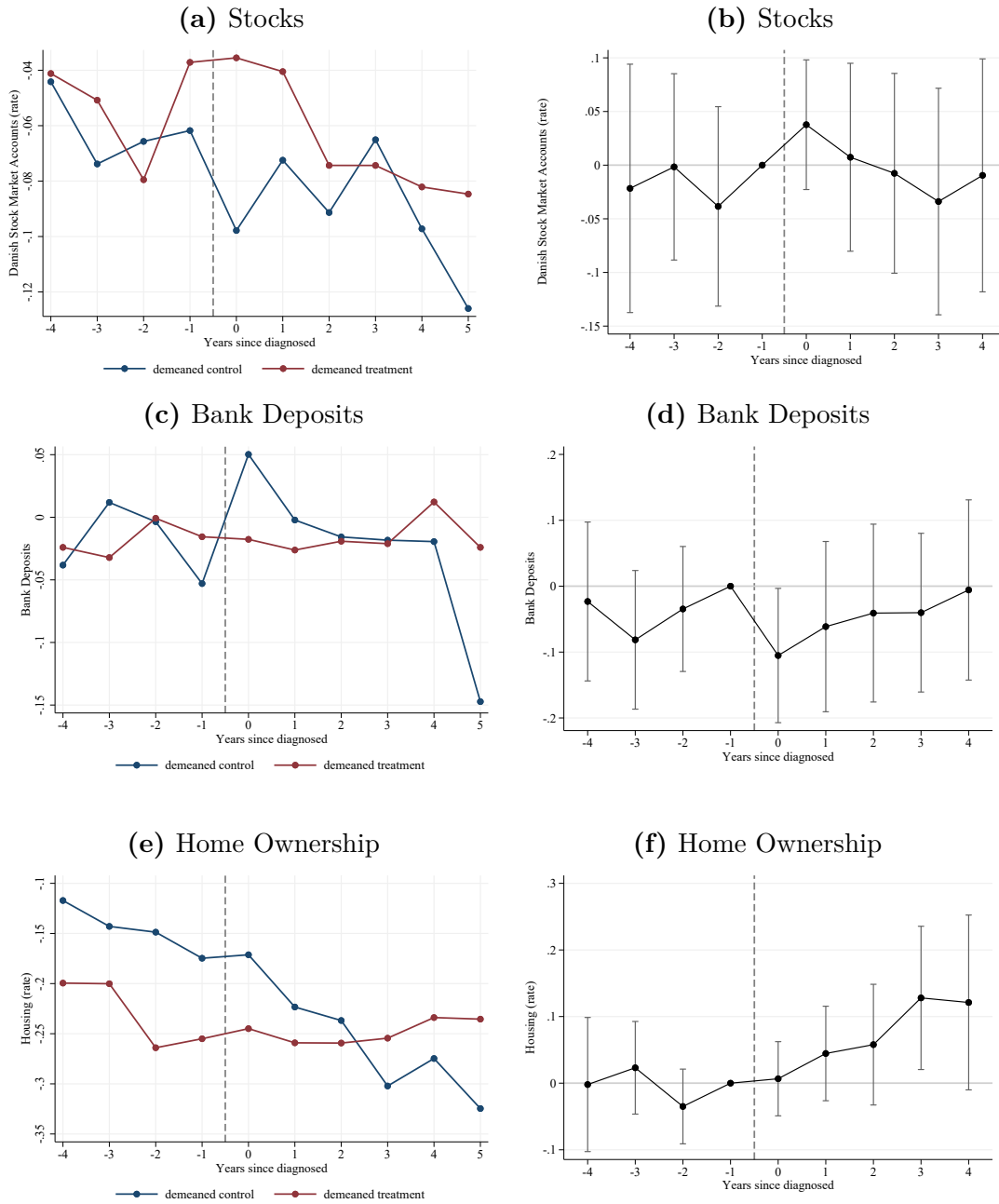

(d) Triple-Difference Estimates

Notes: These graphs plot the effects on bank accounts of the introduction of HAART medical innovation on individuals around the time when they are diagnosed with HIV. Graph (a) plots an event study for the control group (those diagnosed with HIV between 1990 and 1994, before the introduction of HAART) against a synthetic control of HIV− individuals matched in year -4 from the same cohort, age, gender, and education. Graph (b) plots an event study for the treatment group (those diagnosed with HIV between 1996 and 1999, after the introduction of HAART) against a synthetic control of HIV− individuals matched in year -4 from the same cohort, age, gender, and education. Graph (c) plots the control and treatment groups demeaned by their respective synthetic controls. Graph (d) plots the β_t estimates of the triple difference-in-differences model estimated in Equation (1), which identify dynamic causal effects of the introduction of HAART medical innovation. We report 95% confidence intervals calculated from clustered standard errors.

Figure A.7: Effects on Home Ownership from Medical Innovation around the Time of HIV Diagnosis

(a) Control Group and HIV- Synthetic Control

(b) Treatment Group and HIV- Synthetic Control

(c) Demeaned Treated and Control Groups
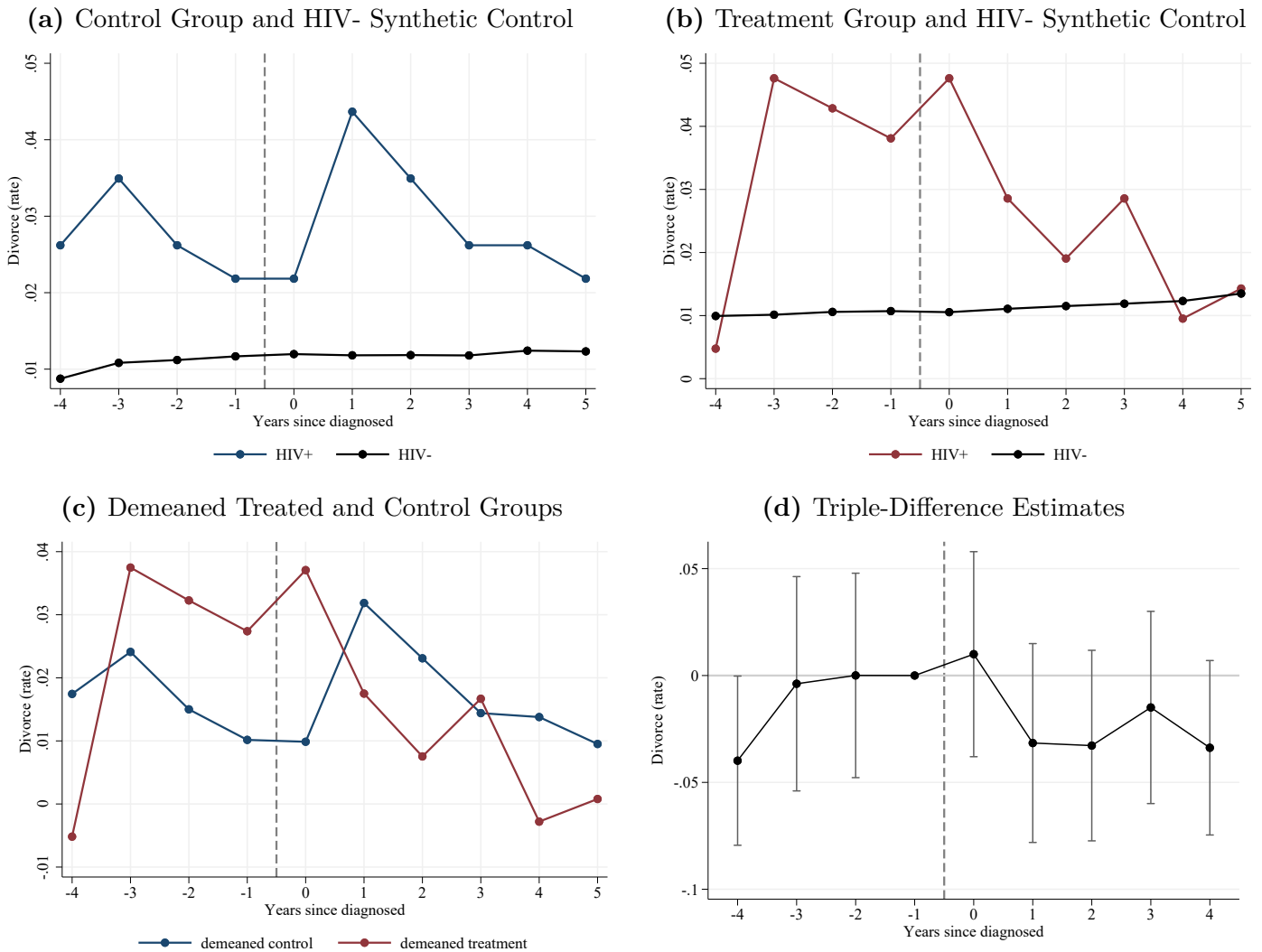

(d) Triple-Difference Estimates

Notes: These graphs plot the effects on home ownership of the introduction of HAART medical innovation on individuals around the time when they are diagnosed with HIV. Graph (a) plots an event study for the control group (those diagnosed with HIV between 1990 and 1994, before the introduction of HAART) against a synthetic control of HIV– individuals matched in year -4 from the same cohort, age, gender, and education. Graph (b) plots an event study for the treatment group (those diagnosed with HIV between 1996 and 1999, after the introduction of HAART) against a synthetic control of HIV– individuals matched in year -4 from the same cohort, age, gender, and education. Graph (c) plots the control and treatment groups demeaned by their respective synthetic controls. Graph (d) plots the β_t estimates of the triple difference-in-differences model estimated in Equation (1), which identify dynamic causal effects of the introduction of HAART medical innovation. We report 95% confidence intervals calculated from clustered standard errors.

Figure A.8: Wealth Outcomes for Sample of Individuals Diagnosed with Low CD4 Count (150-250)
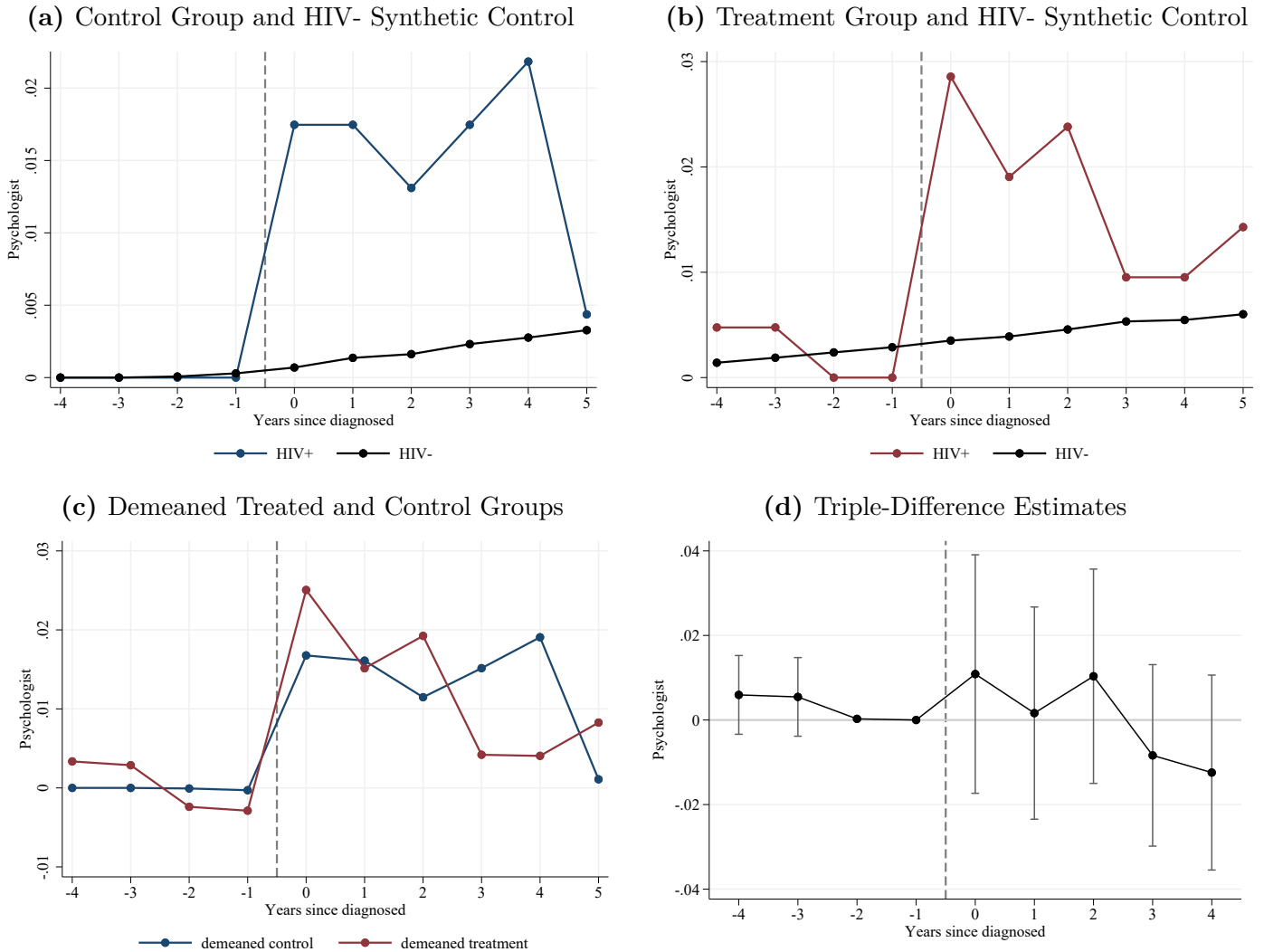

(a) Stocks

(b) Stocks

(c) Bank Deposits

(d) Bank Deposits

(e) Home Ownership

(f) Home Ownership

Notes: This Figure...

Figure A.9: Effects on Divorces from Medical Innovation around the Time of HIV Diagnosis

(a) Control Group and HIV- Synthetic Control

(b) Treatment Group and HIV- Synthetic Control

(c) Demeaned Treated and Control Groups
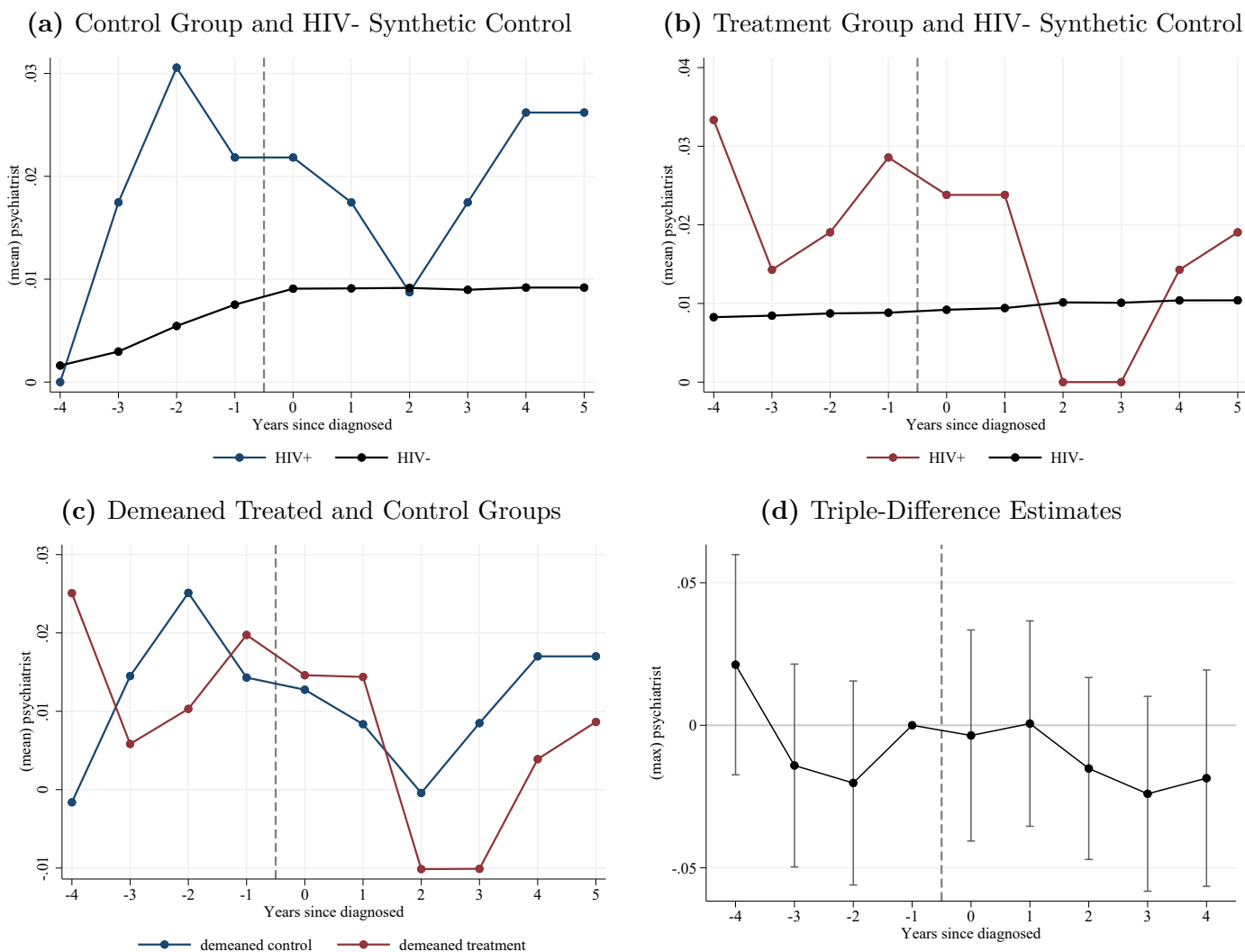

(d) Triple-Difference Estimates

Notes: These graphs plot the effects on divorce rate of the introduction of HAART medical innovation on individuals around the time when they are diagnosed with HIV. Graph (a) plots an event study for the control group (those diagnosed with HIV between 1990 and 1994, before the introduction of HAART) against a synthetic control of HIV– individuals matched in year -4 from the same cohort, age, gender, and education. Graph (b) plots an event study for the treatment group (those diagnosed with HIV between 1996 and 1999, after the introduction of HAART) against a synthetic control of HIV– individuals matched in year -4 from the same cohort, age, gender, and education. Graph (c) plots the control and treatment groups demeaned by their respective synthetic controls. Graph (d) plots the β_t estimates of the triple difference-in-differences model estimated in Equation (1), which identify dynamic causal effects of the introduction of HAART medical innovation. We report 95% confidence intervals calculated from clustered standard errors.

Figure A.10: Effects on Psychotherapy from Medical Innovation around the Time of HIV Diagnosis

(a) Control Group and HIV- Synthetic Control

(b) Treatment Group and HIV- Synthetic Control

(c) Demeaned Treated and Control Groups
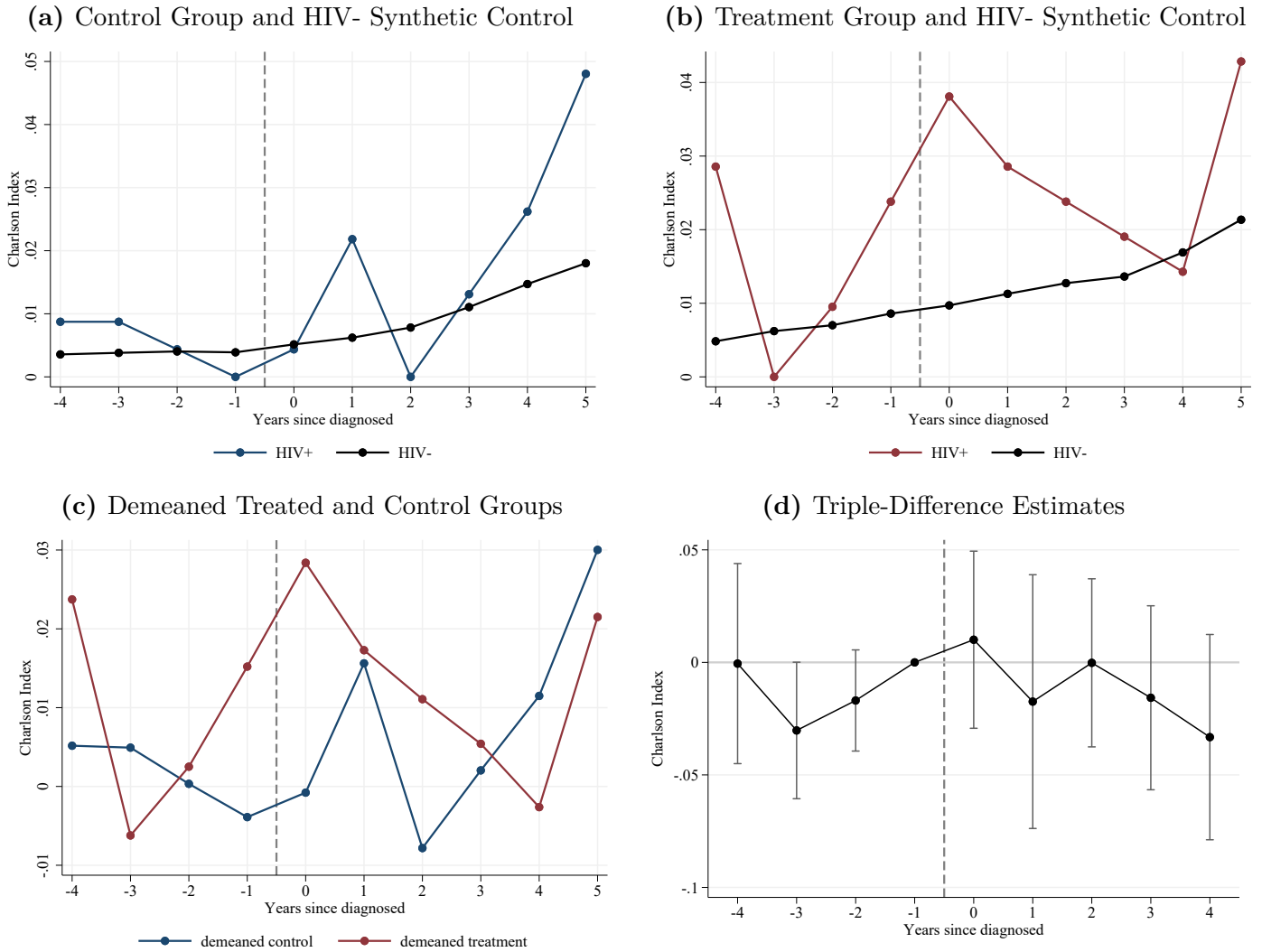

(d) Triple-Difference Estimates

Notes: These graphs plot the effects on psychotherapy of the introduction of HAART medical innovation on individuals around the time when they are diagnosed with HIV. Graph (a) plots an event study for the control group (those diagnosed with HIV between 1990 and 1994, before the introduction of HAART) against a synthetic control of HIV– individuals matched in year -4 from the same cohort, age, gender, and education. Graph (b) plots an event study for the treatment group (those diagnosed with HIV between 1996 and 1999, after the introduction of HAART) against a synthetic control of HIV– individuals matched in year -4 from the same cohort, age, gender, and education. Graph (c) plots the control and treatment groups demeaned by their respective synthetic controls. Graph (d) plots the β_t estimates of the triple difference-in-differences model estimated in Equation (1), which identify dynamic causal effects of the introduction of HAART medical innovation. We report 95% confidence intervals calculated from clustered standard errors.

Figure A.11: Effects on Psychiatrist from Medical Innovation around the Time of HIV Diagnosis

(a) Control Group and HIV- Synthetic Control

(b) Treatment Group and HIV- Synthetic Control

(c) Demeaned Treated and Control Groups
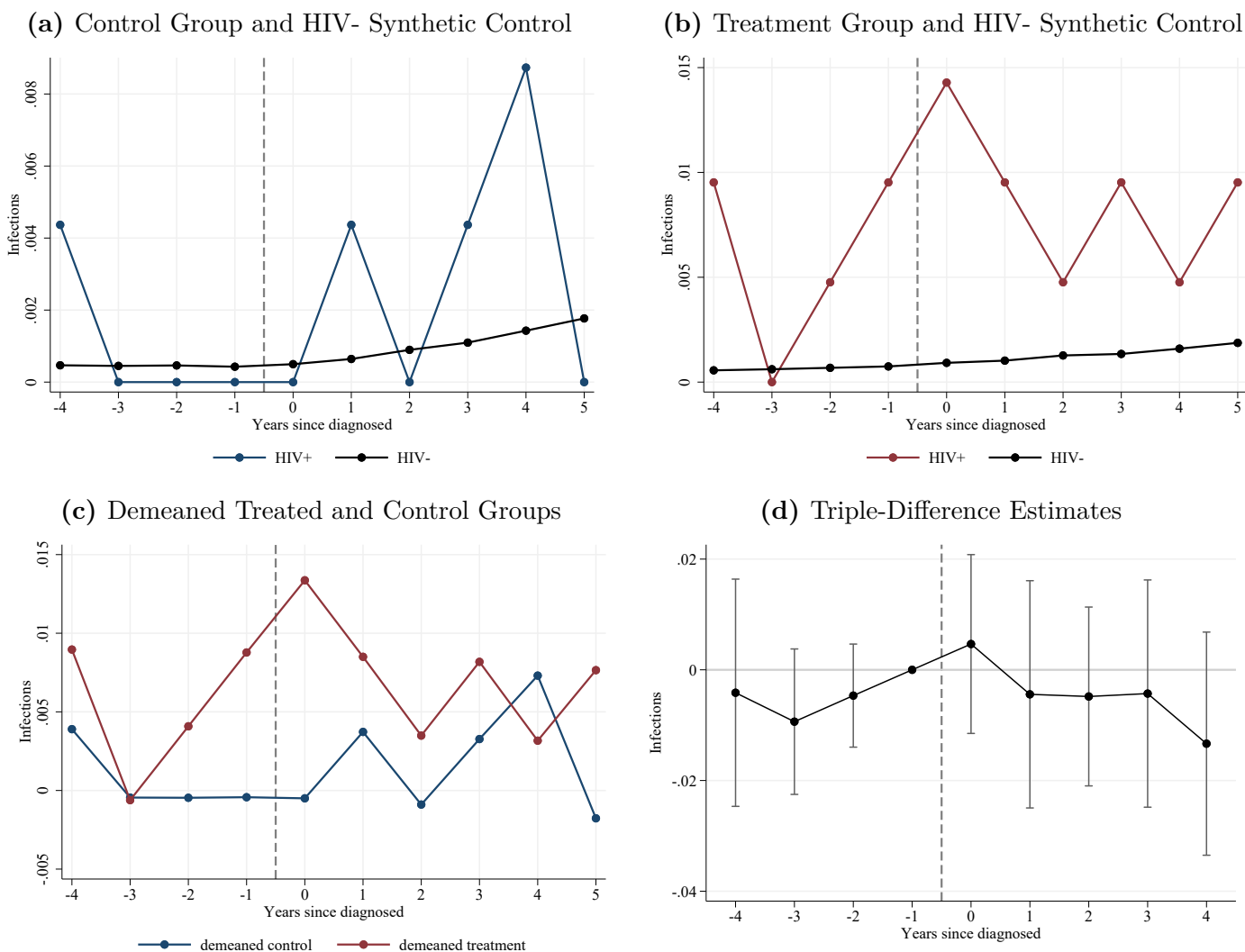

(d) Triple-Difference Estimates

Notes: These graphs plot the effects on psychiatrist of the introduction of HAART medical innovation on individuals around the time when they are diagnosed with HIV. Graph (a) plots an event study for the control group (those diagnosed with HIV between 1990 and 1994, before the introduction of HAART) against a synthetic control of HIV− individuals matched in year -4 from the same cohort, age, gender, and education. Graph (b) plots an event study for the treatment group (those diagnosed with HIV between 1996 and 1999, after the introduction of HAART) against a synthetic control of HIV− individuals matched in year -4 from the same cohort, age, gender, and education. Graph (c) plots the control and treatment groups demeaned by their respective synthetic controls. Graph (d) plots the β_t estimates of the triple difference-in-differences model estimated in Equation (1), which identify dynamic causal effects of the introduction of HAART medical innovation. We report 95% confidence intervals calculated from clustered standard errors.

Figure A.12: Effects on Charlson Index from Medical Innovation around the Time of HIV Diagnosis

(a) Control Group and HIV- Synthetic Control

(b) Treatment Group and HIV- Synthetic Control

(c) Demeaned Treated and Control Groups
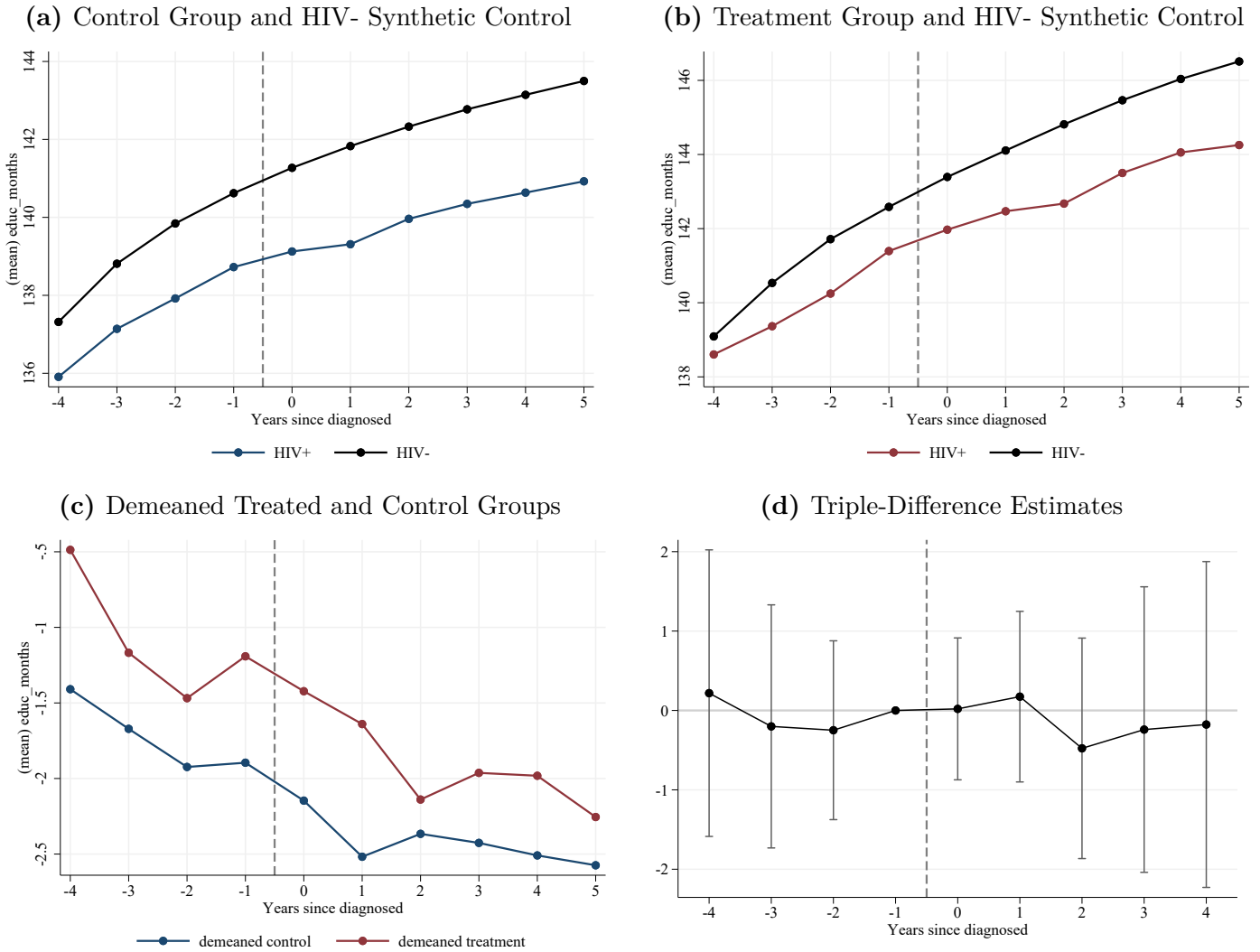

(d) Triple-Difference Estimates

Notes: These graphs plot the effects on the Charlson Index of the introduction of HAART medical innovation on individuals around the time when they are diagnosed with HIV. Graph (a) plots an event study for the control group (those diagnosed with HIV between 1990 and 1994, before the introduction of HAART) against a synthetic control of HIV– individuals matched in year -4 from the same cohort, age, gender, and education. Graph (b) plots an event study for the treatment group (those diagnosed with HIV between 1996 and 1999, after the introduction of HAART) against a synthetic control of HIV– individuals matched in year -4 from the same cohort, age, gender, and education. Graph (c) plots the control and treatment groups demeaned by their respective synthetic controls. Graph (d) plots the β_t estimates of the triple difference-in-differences model estimated in Equation (1), which identify dynamic causal effects of the introduction of HAART medical innovation. We report 95% confidence intervals calculated from clustered standard errors.

Figure A.13: Effects on Infections from Medical Innovation around the Time of HIV Diagnosis

(a) Control Group and HIV- Synthetic Control

(b) Treatment Group and HIV- Synthetic Control

(c) Demeaned Treated and Control Groups
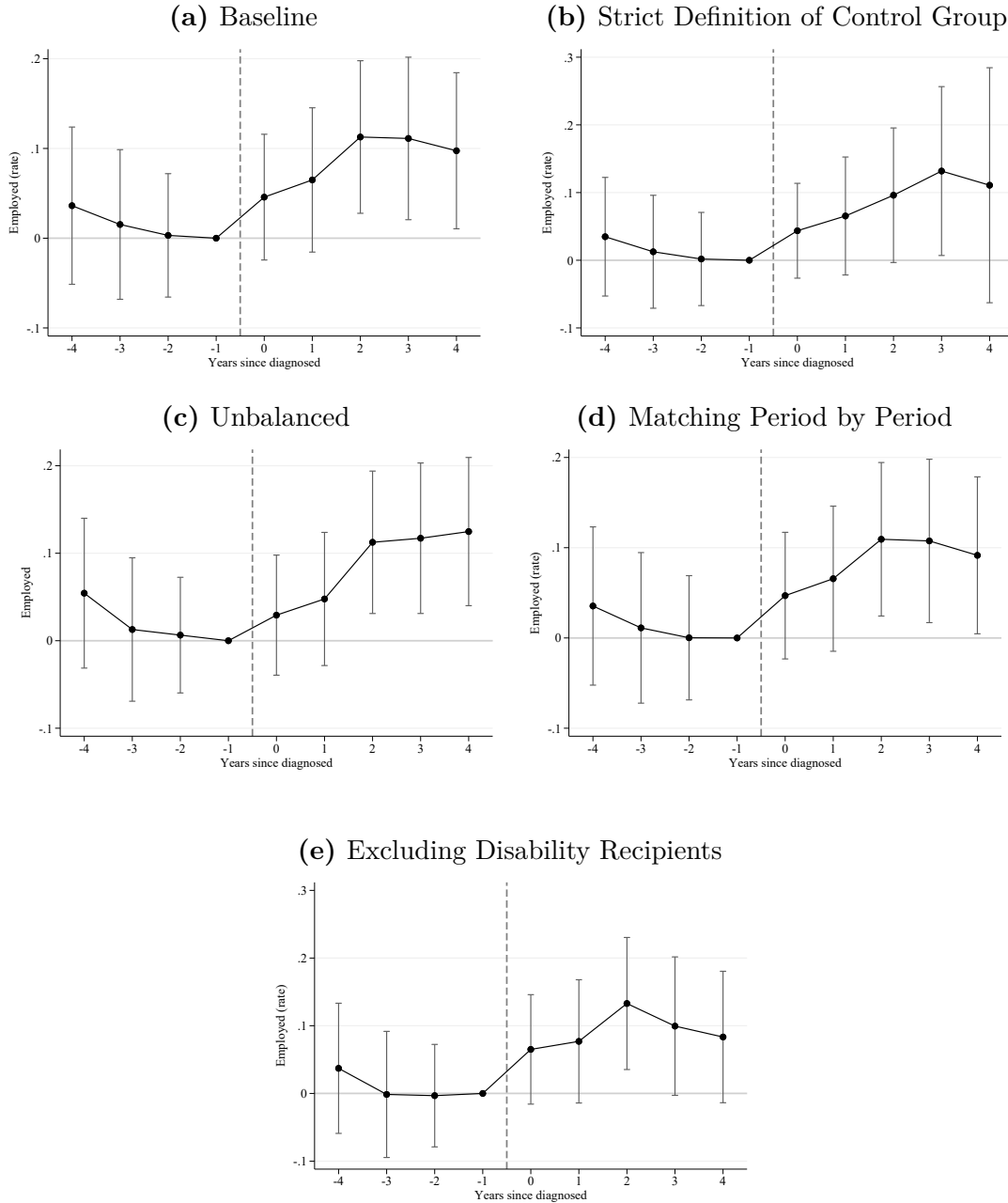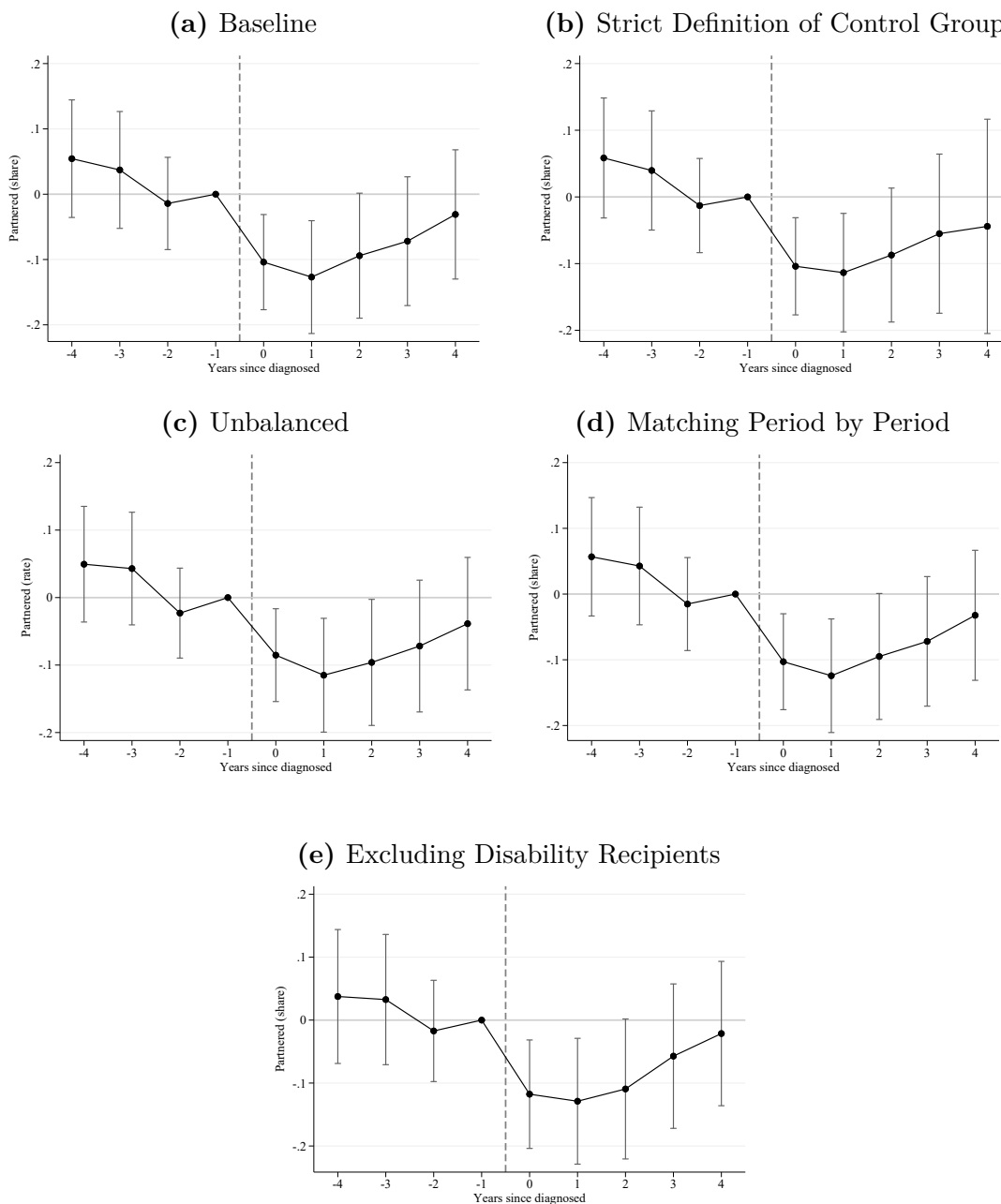

(d) Triple-Difference Estimates

Notes: These graphs plot the effects on Infections of the introduction of HAART medical innovation on individuals around the time when they are diagnosed with HIV. Graph (a) plots an event study for the control group (those diagnosed with HIV between 1990 and 1994, before the introduction of HAART) against a synthetic control of HIV– individuals matched in year -4 from the same cohort, age, gender, and education. Graph (b) plots an event study for the treatment group (those diagnosed with HIV between 1996 and 1999, after the introduction of HAART) against a synthetic control of HIV– individuals matched in year -4 from the same cohort, age, gender, and education. Graph (c) plots the control and treatment groups demeaned by their respective synthetic controls. Graph (d) plots the β_t estimates of the triple difference-in-differences model estimated in Equation (1), which identify dynamic causal effects of the introduction of HAART medical innovation. We report 95% confidence intervals calculated from clustered standard errors.

Figure A.14: Effects on Education from Medical Innovation around the Time of HIV Diagnosis

(a) Control Group and HIV- Synthetic Control

(b) Treatment Group and HIV- Synthetic Control

(c) Demeaned Treated and Control Groups

(d) Triple-Difference Estimates

Notes: These graphs plot the effects on education of the introduction of HAART medical innovation on individuals around the time when they are diagnosed with HIV. Graph (a) plots an event study for the control group (those diagnosed with HIV between 1990 and 1994, before the introduction of HAART) against a synthetic control of HIV– individuals matched in year -4 from the same cohort, age, gender, and education. Graph (b) plots an event study for the treatment group (those diagnosed with HIV between 1996 and 1999, after the introduction of HAART) against a synthetic control of HIV– individuals matched in year -4 from the same cohort, age, gender, and education. Graph (c) plots the control and treatment groups demeaned by their respective synthetic controls. Graph (d) plots the β_t estimates of the triple difference-in-differences model estimated in Equation (1), which identify dynamic causal effects of the introduction of HAART medical innovation. We report 95% confidence intervals calculated from clustered standard errors.

Figure A.15: Alternative Specifications for the Effect on Employment

(a) Baseline

(b) Strict Definition of Control Group

(c) Unbalanced

(d) Matching Period by Period


**(e)** Excluding Disability Recipients

Notes: This Figure shows the dynamic triple difference estimates under different specifications or sample definitions. Graph (a) shows our baseline definition. Graph (b) shows the result with a stricter definition of the control group, dropping observations beyond 1995 to avoid any potential contamination when these individuals gain access to the HAART treatment. Graph (c) shows the results for the unbalanced sample. Graph (d) shows the result when we match the synthetic group of HIV– individuals period by period, as opposed to matching based on period -4 only. Graph (e) restricts the analysis to individuals who do not receive disability benefits during the period of analysis.

Figure A.16: Alternative Specifications for the Effect on Partnership

(a) Baseline

(b) Strict Definition of Control Group

(c) Unbalanced

(d) Matching Period by Period

(e) Excluding Disability Recipients

Notes: This Figure shows the dynamic triple difference estimates under different specifications or sample definitions. Graph (a) shows our baseline definition. Graph (b) shows the result with a stricter definition of the control group, dropping observations beyond 1995 to avoid any potential contamination when these individuals gain access to the HAART treatment. Graph (c) shows the results for the unbalanced sample. Graph (d) shows the result when we match the synthetic group of HIV– individuals period by period, as opposed to matching based on period -4 only. Graph (e) restricts the analysis to individuals who do not receive disability benefits during the period of analysis.

Figure A.17: Marital Status for Heterosexuals

(a) Partnered

(b) Married

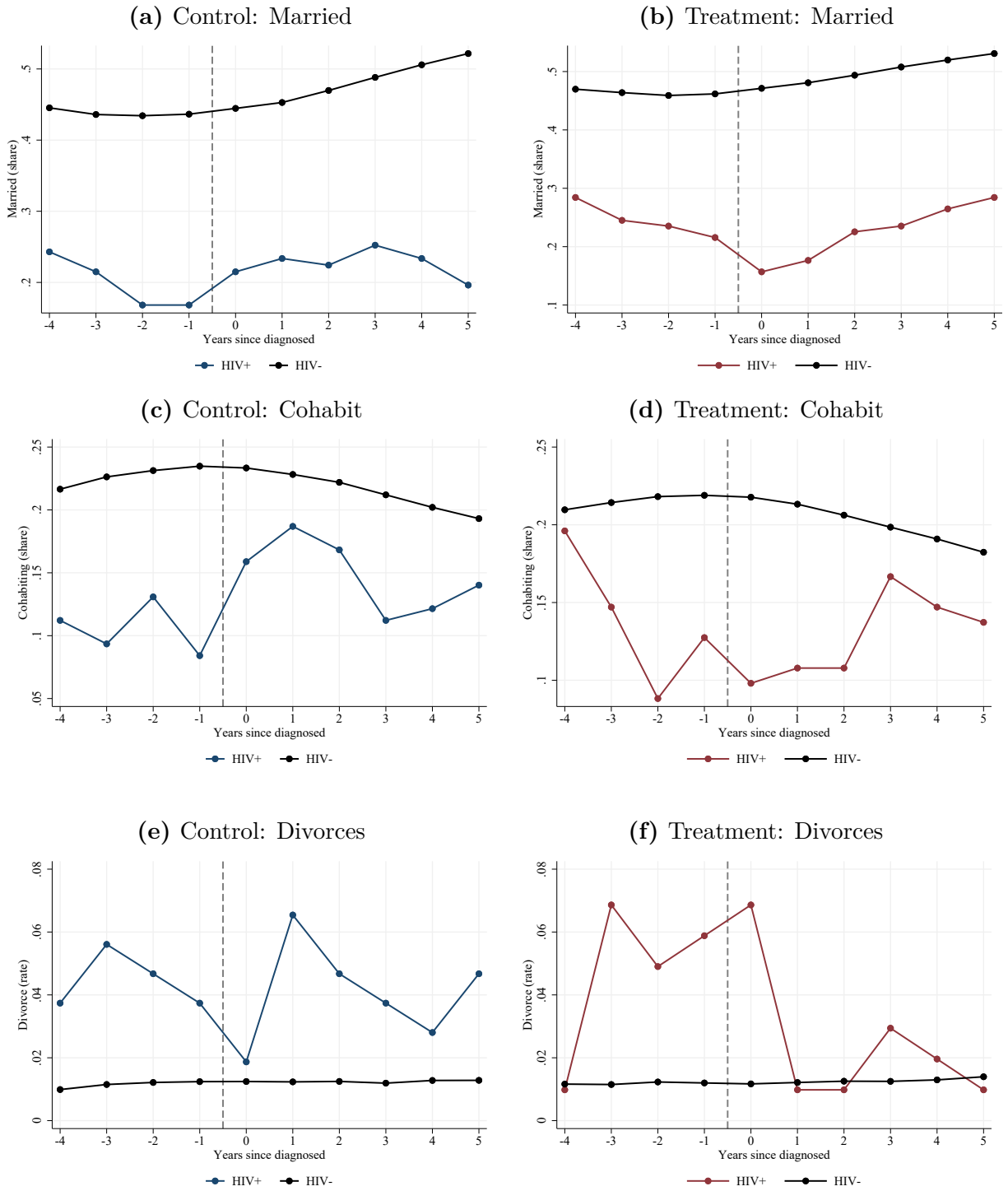

(c) Cohabiting

(d) Divorces (flow)
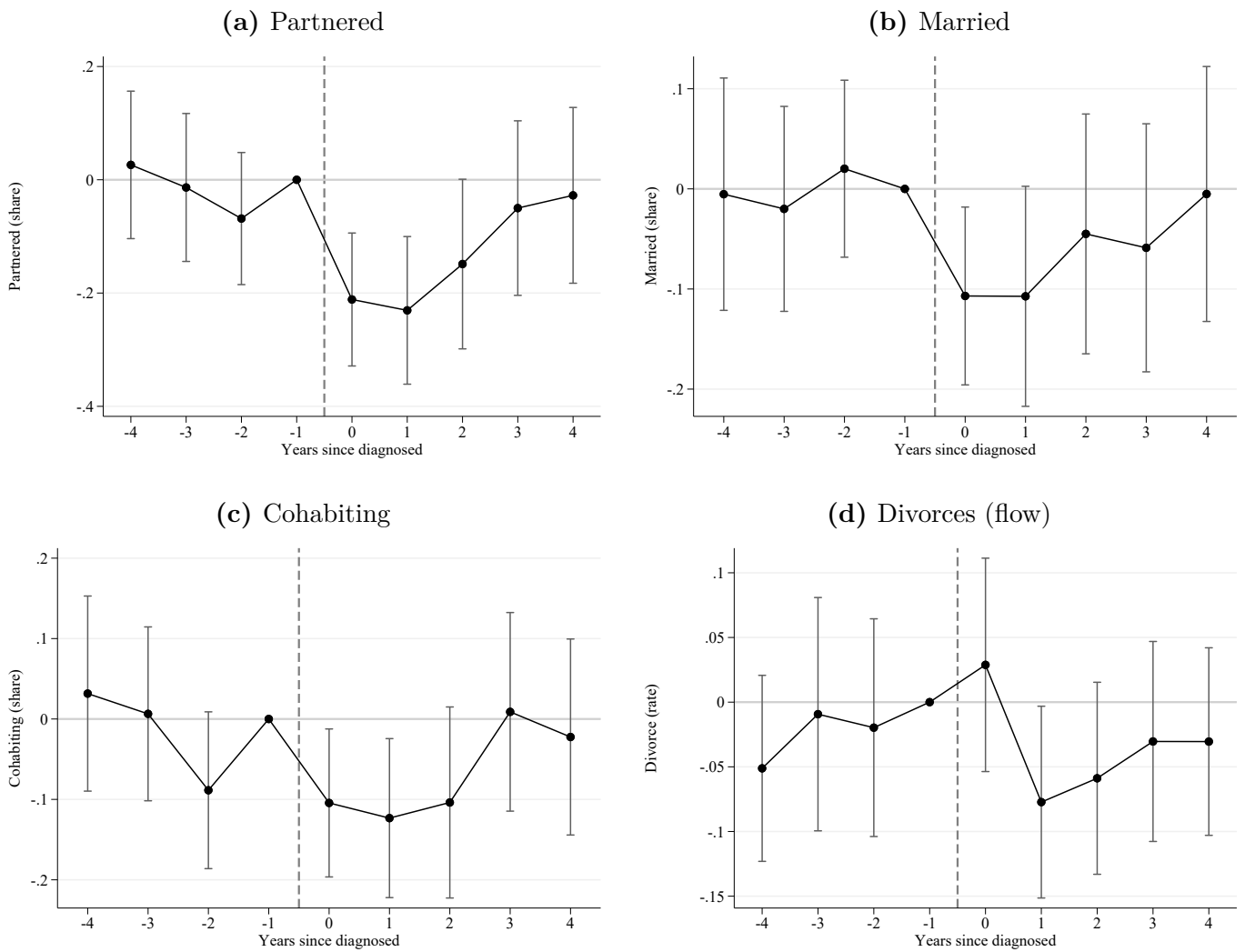
Notes: This Figures show the dynamic triple difference-in-differences. The underlying raw means for these estimates are reported in Figure A.19
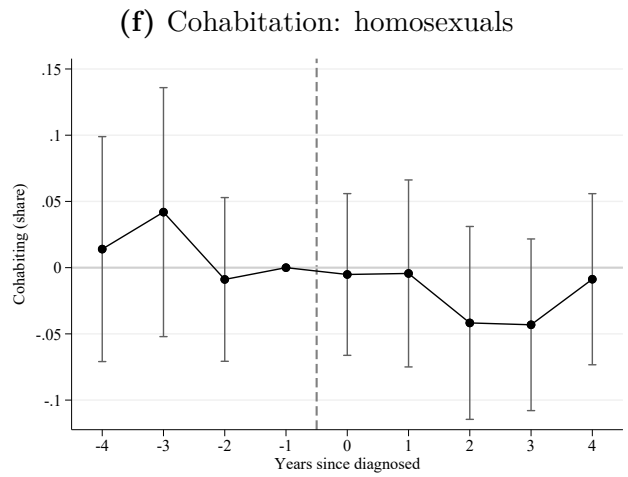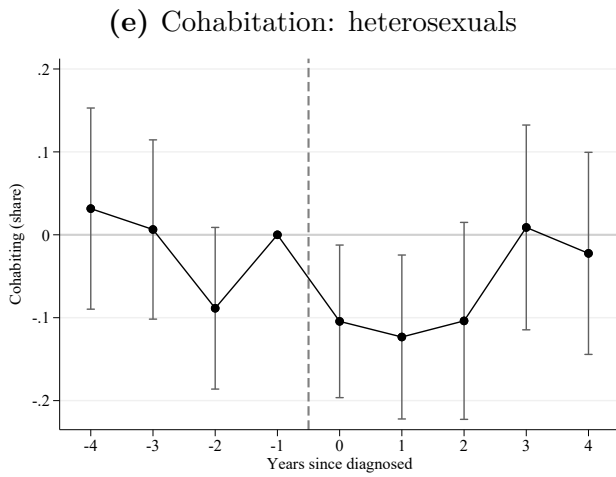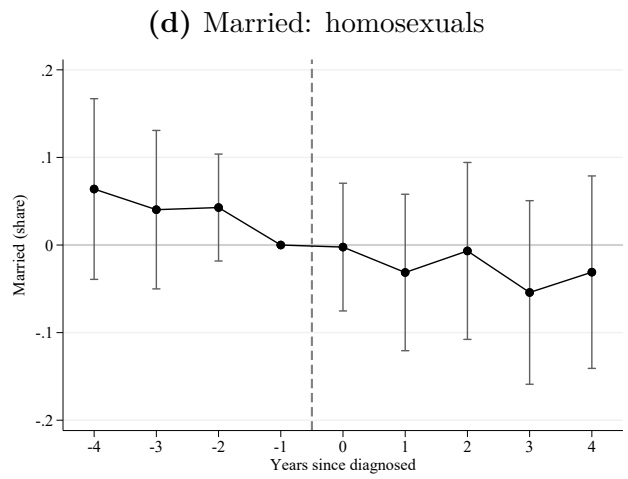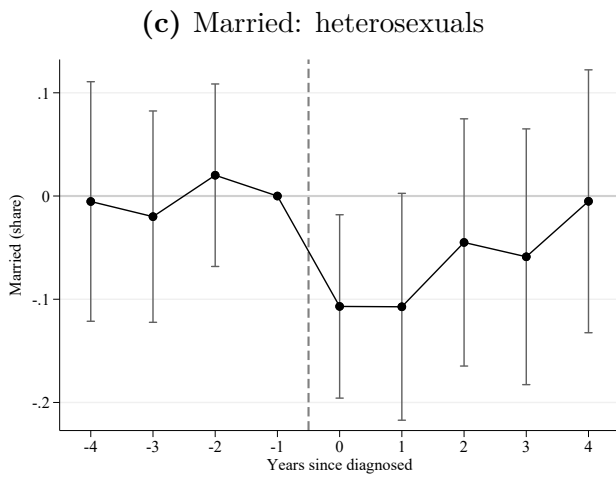
Figure A.18: Marital Status for Homosexuals

(a) Partnered

(b) Married

(c) Cohabiting

(d) Divorces (flow)
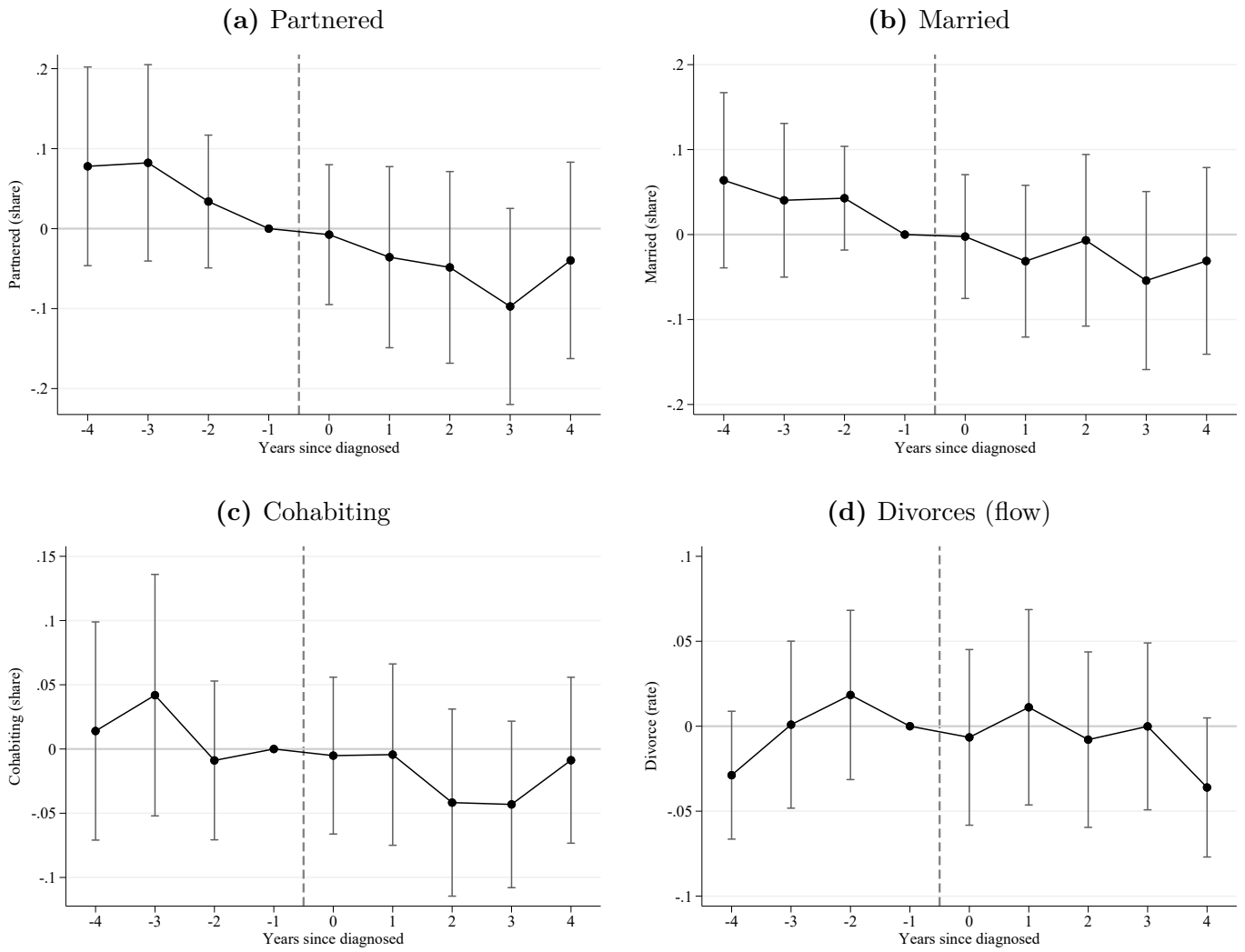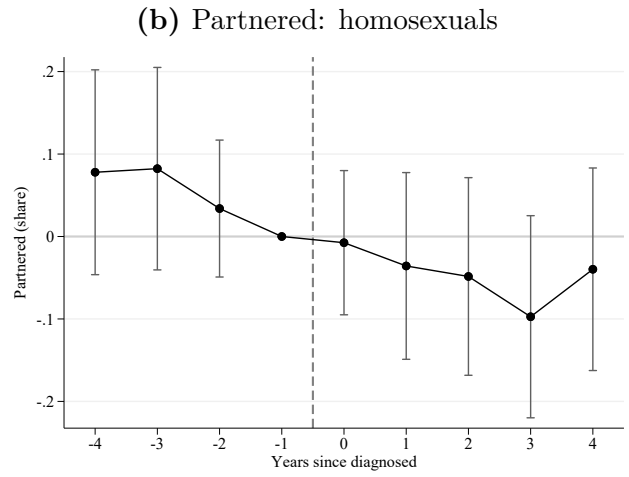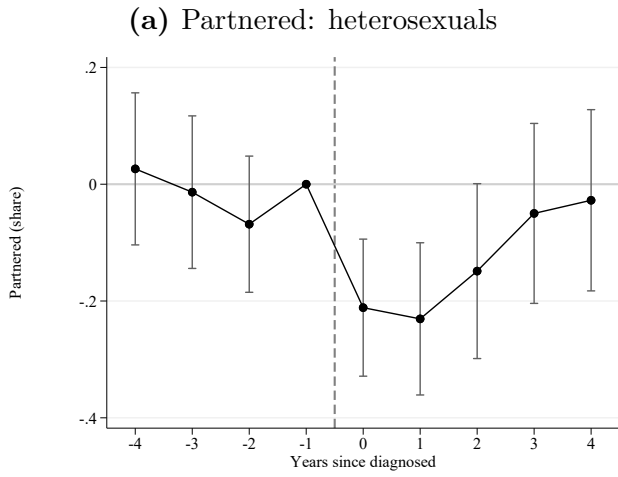

Notes: This Figures show the dynamic triple difference-in-differences....

Figure A.19: Row Means of Marital Outcomes for Heterosexuals

(a) Control: Married

(b) Treatment: Married

(c) Control: Cohabit

(d) Treatment: Cohabit

(e) Control: Divorces

(f) Treatment: Divorces

Notes: xxx

Figure A.20: Marital Status by Sexual Orientation

(a) Partnered: heterosexuals

(b) Partnered: homosexuals

(c) Married: heterosexuals

(d) Married: homosexuals

(e) Cohabitation: heterosexuals

(f) Cohabitation: homosexuals

Notes: