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

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Abstract

Longer life expectancy can affect individuals' incentives to work, save and marry, net of any changes in their underlying health. We test this hypothesis by using the sudden arrival of a new treatment in 1995 that dramatically increased life expectancy for HIV-infected individuals. We compare the behavioural responses of HIV-infected individuals who were still in good health but who differed in their access to the new treatment. Those with access to treatment work substantially more, marry later, but do not save more. Our results highlight the importance of accounting for such incentive effects when valuing increases in life expectancy.

Resumen

Una mayor esperanza de vida puede influir en los incentivos de las personas para trabajar, ahorrar o casarse, independientemente de cambios en su salud. En este trabajo evaluamos esta hipótesis utilizando la llegada repentina de un nuevo tratamiento en 1995, que aumentó drásticamente la esperanza de vida de las personas infectadas con VIH. Para ello, comparamos el comportamiento de personas infectadas con VIH que aún se encuentran en buen estado de salud, pero que difieren en su acceso al nuevo tratamiento. Aquellos con acceso al tratamiento trabajan considerablemente más, se casan más tarde, pero no aumentan su ahorro. Nuestros resultados resaltan la importancia de tener en cuenta estos efectos sobre los incentivos al evaluar aumentos en la esperanza de vida.

Palabras clave: esperanza de vida, empleo, matrimonio, VIH.

Códigos JEL: D84, I12, J12, J21.

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

In the last century, global life expectancy has shown a remarkable increase, owing to factors such as rising incomes, widespread health insurance, and advancements in medical treatments. Human capital theory posits that a longer life expectancy strengthens the incentives to invest in skill acquisition (Becker, 1964; Ben-Porath, 1967), forming the foundation for theories that link life expectancy to economic growth through a human capital channel (Kalemli-Ozcan, 2002; Soares, 2005; Murphy and Topel, 2006; Weil, 2007). A longer life expectancy has implications that extend beyond the realm of human capital theory, however, since major economic decisions about labor supply, savings, and marriage, are also taken under uncertainties about (remaining) life-span.[1] With a longer planning horizon, individuals' incentives regarding work-leisure trade-offs, retirement savings, and marriage market behavior are affected, with significant implications for both welfare and economic growth.

As longer lifespans are commonly associated with better health, often through medical innovations, it has proven difficult to empirically disentangle the pure incentive effect of longer life expectancy from that of improved health.[2] While some 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 (Oster et al., 2013b), there is little empirical evidence to date on the incentive effects on other major life choices, such as work, savings, and marital behavior. Understanding the role of these incentives is crucial not only for valuing increases in life expectancy in itself, but also for valuing the benefits of new medical technologies, as cost-benefit assessments are likely to underestimate their value if the pure incentive effects of a longer life expectancy are not properly accounted for.

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[1]See discussions in Blundell and MaCurdy (1999); Browning and Crossley (2001); Dynan et al. (2002, 2004); Cocco and Gomes (2012); Haan and Prowse (2014); Blundell and Macurdy (2017); Low et al. (2018).

[2]Healthier workers are more productive and improvements in health can also lead to better employment options and facilitate a longer work life (Dobkin et al., 2018; Fadlon and Nielsen, 2021; Stephens and Toohey, 2022). In addition, the marginal utility of consumption may increase with better health since many consumption goods, such as travel, are complements to good health (Finkelstein et al., 2013).

In this paper, we estimate the incentive effect of greater life expectancy on labor market outcomes, financial outcomes, and marriage market outcomes. We do so by focusing on a medical breakthrough that led to a sudden and dramatic increase in life expectancy for HIV-infected 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-infected individuals (Legarth et al., 2014; Mocroft and Lundgren, 2004). Before the introduction of HAART in 1995, a HIV diagnosis was associated with a substantially shortened life expectancy, and many patients died shortly after being diagnosed. Without treatment, newly infected individuals faced a remaining life expectancy of just 11 years (Papageorge, 2021). Individuals diagnosed before and after 1995 thus differed radically in their life expectancy, and the appearance of HAART has transformed HIV into a chronic illness that can be managed with medical treatment.

Specifically, we define our treatment group as individuals who received an HIV diagnosis after the introduction of HAART, whereas our control group consists of individuals diagnosed before the advent of HAART. Although both groups experience the shock of being diagnosed with HIV, the treatment group can anticipate a significantly enhanced life expectancy at the time of diagnosis owing to the availability of HAART.[3] It should be noted that individuals in the control group, who received their HIV diagnosis prior to the introduction of HAART, eventually gained access to this treatment in 1995, thereby attenuating differences in treatment status between our two comparison groups. We show in a robustness exercise that this attenuation bias does not appear to affect our results.

In our analyses, we utilize unique and high-quality longitudinal register data from Denmark on HIV-infected individuals, observed before and after they receive their HIV diagnosis. These data help us to address two main empirical challenges. First, they help in isolating the incentive effect of greater life expectancy from the effect on contemporaneous health. As the data includes clinical information on the stage of the disease, measured through blood samples and CD4 cell counts, we are able to select and study individuals who, at the time of the HIV diagnosis, were still in good health and would not face any symptoms in the following 4 to 5 years.[4] By comparing changes in outcomes before and after the HIV diagnosis across the two groups –those diagnosed before and those diagnosed after the introduction of

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[3]Prior to 1995 numerous clinical trials were conducted on anti-retroviral drugs, primarily involving HIV patients who were already in a relatively advanced stage of the disease, with CD4 levels below 300. However, the outcomes of these trials had not yet demonstrated significant success. (Hamilton et al., 2021). Also, Papageorge (2016) shows a significant increase in hopefulness about the future among HIV patients since 1995.

[4]We show that individuals in this sample, regardless of their access to HAART, are equally likely to remain above healthy levels of CD4 cell counts, exhibit equally low rates of infections and mental health diagnoses, and experience similar low mortality rates during the 4-5 year period following their HIV diagnoses. It should also be noted that we exclude drug addicts from our analyses.

HAART–, we can estimate the incentive effect of longer life expectancy, net of any changes in underlying physical health.

Second, as we are comparing changes in the outcomes of HIV-infected individuals over different periods of time, it is crucial to address potential confounding factors related to calendar time, such as business cycle, structural changes, or reforms. To account for such changes, we use data on the full population of Denmark and separately match a control group of non-HIV-infected (HIV-) individuals to the HIV-infected individuals (HIV+) in the treatment and control group, respectively, i.e. before and after the introduction of HAART. Our empirical analyses thus effectively corresponds to a triple-difference design.

Our first set of results shows that the introduction of HAART and the subsequent increase in life expectancy dramatically reduced the negative effect of receiving a HIV diagnosis on labor supply and earnings. In the four years following the HIV diagnosis, when health had not yet deteriorated, individuals with access to HAART had an 11 percent higher employment rate and 16 percent higher earnings compared to those without access. These effects are to a large extent driven by sharp reductions in employment and earnings among those diagnosed before 1995 who experienced a sharp drop in their life expectancy, suggesting a substitution towards leisure as life expectancy declined.

Our second set of results reveals that the increase in life expectancy resulting from HAART had only small effects on financial decisions, with bank account savings, stock market participation, and home ownership remaining largely unaffected. We interpret these results in the light of standard life-cycle theory; on the one hand, theory predicts that wealth is depleted as life expectancy is reduced following a health shock, but, on the other hand, a sharp reduction in life expectancy in the context of an HIV diagnosis could lead to increased precautionary savings in response to increased uncertainty (Davies, 1981; Kotlikoff, 1989). Furthermore, the impact of increased life expectancy on saving towards retirement may be limited in countries with generous pension systems and widespread passive savings behavior (Chetty et al., 2014; García-Miralles and Leganza, 2023) and in the presence of strong bequest motives (Hurd, 1987; Dynan et al., 2002; De Nardi et al., 2009).

Our third set of results suggests that HAART had 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 after being diagnosed. This finding can be interpreted in a family economics framework, where cohabitation and marriage provide important sources of private insurance against health shocks (Anderberg, 2007; Persson, 2020; Potoms and Rosenberg, 2021). In the absence of HAART, the insurance value of having a partner who can provide financial and practical caregiving support significantly increased in light of the large negative shock to life expectancy that followed an HIV diagnosis. Moreover, if relative preferences

for consumption and leisure change such that more weight is put on leisure relative to consumption, as the results above suggest, the utility of being in a couple could also increase if leisure complementarities are positive (Johnsen et al., 2022; Lalive and Parrotta, 2017; Browning et al., 2020; García-Miralles and Leganza, 2021; Georges-Kot et al., 2022).[5]

Our results highlight the importance of accounting for the incentive effect of greater life expectancy when valuing the gains from new medical technologies. While traditional cost-effectiveness studies typically account for only the direct health and socioeconomic effects following new medical treatments, our findings pinpoint that the pure anticipation of access to a new type of health technology in the future may have additional positive effects on economic outcomes, even before health starts to deteriorate. Such gains are of particular relevance for diseases with long time lags between receiving information about the disease and the outbreak of health symptoms. For such diseases, the incentive effect may constitute a non-negligible fraction of the total value of life-extending medical technology. We illustrate this phenomenon with a simple simulation exercise where we show that the incentive effect of HAART on employment may constitute as much as 18 percent of the total effect on employment during the first 15 years after a HIV diagnosis.

Our paper contributes to two strands of the literature. First, it contributes to the limited body of research that estimates the incentive effect of longer life expectancy on human capital investments (Fortson, 2011; Jayachandran and Lleras-Muney, 2009; Oster et al., 2013b).[6] We contribute to this literature by studying how longer life expectancy impacts an additional number of important (post-education) outcomes: employment, savings, and marital behavior. Our findings suggest that focusing solely on human capital investments may underestimate the total incentive effect of longer life expectancy. Moreover, the source of variation in life expectancy that we study originates from a well-defined medical breakthrough, rather than stemming from a combination of sources affecting mortality or from variation in naturally occurring mortality such as the Huntington's disease. Our results therefore speak more directly to cases where life expectancy is affected by new medical technology or by health policies that have a clear impact on individuals' perceptions of their life expectancy.[7]

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[5]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).

[6]Fortson (2011) found that regions in sub-Saharan Africa with higher HIV prevalence experienced relatively larger declines in schooling. Jayachandran and Lleras-Muney (2009) found that a sudden drop in maternal mortality in Sri Lanka in the 1950s, which sharply increased the life expectancy of girls, led to an increase in girls' education relative to that of boys. 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.

[7]In a contemporaneous working paper, Karparti (2022) studies the effect of life expectancy on wealth accumulation, using genetic testing data from a group of individuals at high risk of developing hereditary cancer syndrome.

Secondly, our paper is related to the literature that examines the effect of HAART treatment on different outcomes. One line of research focuses on the effect on health and labor market outcomes (Papageorge, 2016; Thirumurthy et al., 2008; Habyarimana et al., 2010; Thirumurthy and Zivin, 2012). Although these studies typically find large effects of HAART treatment on labor supply, they do not aim to disentangle the health effects of the treatment from the incentive effects of longer life expectancy. Another line of research, more closely related to our paper, aims to isolate the incentive effect of the longer life expectancy that results from HAART. Papageorge et al. (2021) use variation in disease progression across HIV positive women at the time of the introduction of HAART and find a larger reduction in domestic violence and drug use, as well as a larger increase in employment among women who were in a more advanced stage of the disease. Baranov et al. (2015) and Baranov and Kohler (2018) analyze the rollout of HAART treatment in Malawi and show that increased availability led to increased savings and labor supply, even among HIV negative individuals who anticipate a higher life expectancy in the event of future HIV infection. Our paper differs from Papageorge et al. (2021) in its focus on other important socio-economic outcomes, including earnings, bank holdings, stock market participation, housing and partnership formation, enabling us to paint a comprehensive picture of the consequences of increased life expectancy for major life decisions. Additionally, our paper employs a different empirical approach that relies on a more representative sample of HIV infected individuals, including both men and women of all income levels and excluding drug addicts from the analysis. We differ from Baranov et al. (2015) and Baranov and Kohler (2018) as we focus on a developed country where HIV had less dramatic overall effects on society, utilize data on actual disease progression, and ask a different question that focuses on the behavior of already-infected but healthy individuals.[8]

The paper unfolds as follows. In Section 2, we provide a brief overview of the institutional context of HIV and HIV treatment in Denmark, and we discuss how HAART dramatically changed the situation for HIV positive individuals. In Section 3, we describe the data sources used in our analyses. Section 4 outlines our empirical approach. We present our main results in Section 5, and Section 5.5 provides a set of robustness analyses. Finally, Section 6 concludes.

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[8]In a broader context, our paper relates to the literature on the effect of HIV treatment access on various behaviors. Hamilton et al. (2022) show that HIV-infected men often forego medication to avoid side effects, in part to remain employed. Lakdawalla et al. (2006) use variation in state-level eligibility rules for HAART and find that treating HIV individuals more than doubles their number of sex partners. Chan et al. (2015) develop a dynamic framework to show that HAART reduced the expected cost of an HIV infection by reducing the implicit price of risky sex. Keiser et al. (2010) and Shahid (2022) show that suicide rates of HIV patients decreased with the introduction of HAART in Switzerland and the US, respectively.

2 Background and institutional context

2.1 HIV and AIDS: Medical facts

HIV (Human Immunodeficiency Virus) is a chronic virus that impairs the immune system's ability to defend against ordinary infections, resulting in immunodeficiency. HIV is primarily transmitted through sexual contact and exposure to infected blood. The HIV virus attacks the immune system and especially the CD4 cells (T-cells). In the absence of treatment, HIV-infected individuals experience a progression of immunodeficiency, leading to the development of opportunistic infections and, ultimately, AIDS-related mortality.

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 by a blood test conducted by a health professional. Without HIV, a healthy immune system has a CD4 count between 500 and 1,600 cells per cubic millimeter of blood (cells/mm3). When the 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 discussed in the introduction, focusing on this particular subset of individuals constitutes an integral part of our empirical strategy, since physical symptoms cannot explain any differences in the behavioral responses to new HIV treatments within this group.

2.2 HIV in society

The first documented scientific report of HIV dates back to 1981, after which the number of cases increased dramatically throughout the 1980s worldwide. In many countries, the HIV/AIDS epidemic represented a significant demographic and economic shock (Karlsson and Pichler, 2015). To date, the disease has claimed the lives of over 32 million individuals across the globe. Prevalence is higher among homosexual men, people with haemophilia, drug addicts, and among individuals from sub-Saharan Africa.

In Denmark, the first reports about what is now recognized as HIV/AIDS appeared in Danish newspapers in late 1981. At that time, the disease was believed to be a type of cancer that primarily affected homosexual men. HIV testing became available in 1985, with individuals being able to obtain a blood test free of charge from their general practitioner (GP), at a hospital, or a clinic for sexual diseases. Some hospitals and clinics also offered anonymous testing options. The high mortality rate associated with AIDS quickly became apparent in Denmark. From the 1980s until the mid-1990s, AIDS claimed the lives of 175-240 individuals annually in Denmark, corresponding to a mortality rate of 3 out of 1000 individuals.

2.3   A medical breakthrough: HAART medication

The introduction of Highly Active Antiretroviral Therapy (HAART) medication to Danish patients in 1995 yielded rapid and substantial reductions in the number of individuals suffering from HIV/AIDS, with mortality rates plummeting to approximately one-third of their previous level. The breakthrough resulted from the combination of three anti-retroviral drugs, including a novel type of medication known as protease inhibitors. In June 1995, the Food and Drug Administration (FDA) approved the first protease inhibitors for the treatment of HIV patients, while the combination therapy was granted approval in December of that same year.[9] About six months later, in July 1996, the promising results of the new combined treatment were confirmed at the XI International AIDS Conference.

The media quickly disseminated information about these medical innovations to the general public. Anecdotal evidence and news articles from Denmark suggest that a sense of optimism began to emerge already in September 1995, with growing awareness that this marked the beginning of a new era with an effective treatment for HIV available (see Appendix A.1).

Anti-retroviral treatment works by inhibiting some of HIV's enzymes, reducing HIV in the body, and increasing 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 initiating HAART treatment when CD4 counts fall below 350.

HAART has transformed HIV into a chronic infection in the Western world, with survival rates similar to those of the general population. Figure 1 shows the survival rates by year following HIV diagnosis in Denmark. Patients diagnosed between 1990 and 1993 had low survival rates, with only 20 percent surviving five years after diagnosis. For patients diagnosed in 1994 or 1995, however, survival curves are much less steep, and at least 50 percent survived five years after the diagnosis. For patients diagnosed after 1996, 80 percent were still alive 5 years after diagnosis.

A study of the living conditions of those infected with HIV in Denmark points to remarkable changes in expectations and hopes for the future for patients diagnosed before and after the arrival of the HAART medication (Carstensen and Dahl, 2007). Patients diagnosed in the early period were more likely to discontinue their education or to report HIV infection as a reason for retirement than patients diagnosed in more recent years.

3   Data

Our study draws upon a novel data source that integrates longitudinal register data for the entire population from Statistics Denmark with a medical database containing

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

clinical health information on individuals diagnosed with HIV. This section outlines the key variables and provides a detailed description of the data.

3.1 Danish Registers

The data from Statistics Denmark allow us to follow the entire population of HIV positive individuals from the 1980s until 2000. The register data provides background information as well as socioeconomic outcomes and information on health care use. The socioeconomic outcomes studied in this paper include employment, income, savings, financial market participation, and housing. We deflate all monetary values to 1997 levels. The exchange rate in 1997 was approximately 10 Danish kroner (DKK) to 1.52 U.S. dollars (USD). In addition, the register data provides detailed information on cohabitation and marriage patterns. Statistics Denmark defines cohabitation as two adults who are not family-related, living at the same address, of opposite sexes, and with less than 15 years of age difference.[10] Finally, we utilize Statistics Denmark's healthcare registers to establish the timing of the first HIV test taken by each individual and to assess their general health status.

3.2 HIV Medical Database

To supplement our register data on socioeconomic characteristics and healthcare use, we obtained access to DANHIV, a comprehensive clinical database compiled by all public and private hospitals in Denmark since the 1990s.[11] The data in DANHIV covers all patients diagnosed with HIV (ICD-10 codes B20-24) who were alive in 1995 when the database was initiated. It is important to note, however, that the database also contains retrospective information on the date of HIV diagnosis and the source of infection for those diagnosed before 1995. Furthermore, each individual in DANHIV has been linked to Statistics Denmark's anonymized register data. Three variables from DANHIV are of particular importance for our analysis:

CD4 Counts. This variable is the leading indicator of immune system health and provides information about the progression of the HIV disease. It is a key variable for defining our sample, which we restrict to healthy HIV-positive individuals whose immune system has not yet deteriorated.

On average, individuals have their CD4 counts measured 2.5 times per year in our sample. To ensure compatibility with our annual outcome measures, we construct an annual CD4 count measure for each individual by taking the mean of all their

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[10]Since a significant portion of our sample comprises homosexual men, we are also interested in studying partnering among men. To this end, we use information on registered partnerships, which were introduced in Denmark in 1989 as a legal equivalent to marriage for same-sex couples. Unlike cohabitation among people of opposite sexes, there is no information in Statistics Denmark's registers on cohabitation between people of the same sex.

[11]DANHIV is described in Obel et al. (2008). More information on DANHIV (in Danish): https://www.rkkp.dk/kvalitetsdatabaser/databaser/Dansk-HIV-database-/.

CD4 count measurements in a given year. It should be noted that HIV patients are regularly informed of their CD4 counts during their visits to the doctor, as this is a critical indicator of their current health status.

The HIV medical database only includes CD4 counts from 1995 onwards. For individuals diagnosed before 1995, we therefore impute their pre-1995 CD4 counts based on the counts observed since 1995. It is important to note that these imputed values are not included as a variable in any regression analyses, but are used solely to identify individuals with sufficient health status at the time of diagnosis.

Our preferred imputation method estimates a quadratic model with individual fixed effects, with CD4 counts as a function of time from diagnosis. We estimate this model using observations between the time of diagnosis and the start of HAART treatment, as CD4 counts can be affected by the treatment. 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 that captures differences in levels across the different individuals. The slope parameters β_1 and β_2 capture the annual change in individual's CD4 counts that occurs, from the time they are diagnosed with HIV until the start of HAART treatment. We then use the estimated parameters to impute CD4 count values that are missing between the time of diagnosis and the start of HAART treatment.[12]

Source of Infection. The medical database contains information on the source of infection, which is self-reported by the patient. The source of infection can be classified as sexual transmission, including heterosexual transmission or transmission by men having sex with men (MSM), or non-sexual transmission such as drug abuse, transmission from mother to child during pregnancy, blood transfusions, or unknown. This information is crucial for defining our sample as we focus on individuals who were infected through the two most prevalent sources of infection: heterosexual contact and men having sex with men. We also use this information to exclude individuals who were infected through drug use from our sample and to examine whether the effects of our interventions vary by sexual orientation.

Time of HAART treatment. The DANHIV database provides information on both the date of HIV diagnosis and the date when HAART treatment commenced. During our period of analysis, the latter date was determined based on the CD4 counts of each individual.

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[12]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, which is composed by healthy HIV+ individuals, in Appendix Figure A.2 we report 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 analytical sample, we impose four restrictions. First, we select the 2,153 individuals who were diagnosed with HIV either in the five years preceding the introduction of HAART (1990-1994) or in the five years following its introduction (1995-1999). Second, we exclude individuals who are likely to be 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 those 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. This reduces the sample to 596 individuals, with 289 diagnosed before 1995 and 307 diagnosed after 1995.

Fourth, we balance the sample by only including individuals who are observed annually from 4 years before diagnosis until 4 years after, resulting in a final sample of 443 individuals. The control group comprises 230 individuals diagnosed before 1995, while the treatment group comprises 213 individuals diagnosed from 1995. We demonstrate in the robustness section that our results are robust to this restriction and other sample definitions.

3.4   Descriptive Statistics and Balancing Tests

Columns 1 and 2 of Table 1 show summary statistics for key background and outcome variables for our 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 comprises individuals diagnosed with HIV between 1995 and 1999, after HAART became available, while the control group includes those diagnosed between 1990 and 1994, before the advent of HAART. Some notable features of the sample are that males are heavily over represented (about 80 percent) and that the average age is about 34. Heterosexual individuals represent 43 percent of the sample.

Column 3 provides a comparison of means between the treatment and control groups, while column 4 reports the *p-values* for tests of equality of means. These tests indicate that the treatment and control groups exhibit substantial similarity across most observable characteristics. Among the 17 variables considered, only two exhibit a statistically significant difference: higher earnings in nominal terms in the treatment group (because of calendar time effects) and a greater proportion of stock holdings in the control group. We note that the existence of HAART treatment might have altered risk behaviors, as suggested by Chan et al. (2015), and this could lead to a change in the composition of HIV positive individuals after 1996. Our results from the balancing show, however, that our two groups are comparable along observable

characteristics. Finally, column 5 displays the means for the HIV-negative matched synthetic control group, which we further describe in Section 4.

3.5   Balancing on Health

A key feature of our empirical design is our focus on HIV positive individuals who exhibit high CD4 counts, indicating good health and an absence of physical symptoms. By focusing on 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. To substantiate this claim, we present four lines of evidence:

First, Table 1 shows that the CD4 counts are high in our sample and hardly differ between the treatment and control groups (619 vs. 620). This indicates that both groups have CD4 counts that well exceed the threshold for physical symptom onset. Second, Table 1 reveals that rates of infection and Charlson Index scores are comparably low in both groups.[13] Third, the treatment and control groups face high and similar survival rates during the first years after diagnosis, as demonstrated in Appendix Figure A.3. Note that, by construction, individuals in the control group must survive at least one year after diagnosis to be included in the medical database and in our sample. If we impose the same requirement on the treatment group, the survival curves align even more closely, as evidenced by the dotted and red lines.[14]

Fourth, we can examine the proportion of individuals in the treatment and control groups who fall below certain CD4 thresholds over time. Importantly, in Figure 2 we show that the share of individuals below CD4 thresholds at which individuals typically start experiencing negative physical symptoms remains small and similar in both treatment and control groups. A negligible share of individuals in our sample exhibit CD4 counts below 200 in the four years after diagnosis. Even if we adopt a more conservative threshold of 300, only a small proportion of individuals (8 percent) display CD4 counts below this threshold, and both treatment and control groups exhibit similar patterns of evolution. In sum, these four findings demonstrate that our sample is healthy and well balanced across socioeconomic, demographic, and health-related factors.

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[13]The Charlson Index is a weighted index that predicts mortality on the basis of pre-existing conditions. We follow Quan et al. (2011) in defining the index as a weighted sum of comorbidity conditions based on the ICD-10 classification. These include congestive heart failure, dementia, chronic pulmonary disease, connective tissue disease, liver disease, diabetes, hemiplegia, renal disease, tumors, leukemia, and lymphoma. We exclude HIV from the index.

[14]As survival probabilities are similar in the control and treatment groups, the sample selection in the control group does not seem to pose a problem. In fact, if anything, it is likely that the control group is somewhat healthier than the treatment group, implying that any effect of HAART treatment may be underestimated.

4   Methodology

Our empirical approach involves comparing the evolution of outcomes of individuals diagnosed with HIV before and after the introduction of HAART in 1995. Specifically, we consider individuals diagnosed between 1995 and 1999 as our treatment group, and those diagnosed between 1990 and 1994 as our control group. To ensure that the groups are healthy and have not experienced immune system deterioration, as documented in section 3.5, we restrict our sample to HIV-infected individuals with high CD4 counts.

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 demonstrate that matching year by year yields comparable results.

With our matched synthetic controls, we estimate the following standard dynamic triple difference specification:

$$
\begin{aligned}
Y_{it} =& \alpha_0 + \sum_{j \neq -1} \beta_j \cdot Treat_i \cdot Inf_i \cdot Time_{j=t} + \sum_{j \neq -1} \gamma_j \cdot Inf_i \cdot Time_{j=t} \\
& + \sum_{j \neq -1} \eta_j \cdot Treat_i \cdot Time_{j=t} + \sum_{j \neq -1} \theta_j \cdot Time_{j=t} \\
& + \phi_1 \cdot Treat_i \cdot Inf_i + \phi_2 \cdot Inf_i + \phi_3 \cdot Treat_i + X_{it} \cdot \Phi_4 + \epsilon_{it},
\end{aligned}
\tag{1}
$$

where Y_{it} is the outcome variable of interest for individual i in time t, $Treat_i$ 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_i is an indicator that takes one if an individual is ever infected with HIV and zero otherwise, that is if the individual belongs to the synthetic sample of individuals who are not diagnosed with HIV.[15] X contains the control variables: age dummies, gender, and a dummy for being a Danish citizen.

The β_j coefficients identify the causal effect of the introduction of HAART medication on various outcomes. By plotting β_j over time t we are able to evaluate the

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

identifying assumption that both treatment and control groups move in parallel before the HIV diagnosis that occurs at $t = 0$. We present and discuss these graphical results in Section 5.

To quantify the average effect of the introduction of HAART, we also 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 $t = 0$.

$$\begin{aligned} Y_{it} = &\beta_0 + \beta_1 \cdot Treat_i \cdot Inf_i \cdot Post_{it} + \beta_2 \cdot Inf_i \cdot Post_{it} + \beta_3 \cdot Treat_i \cdot Post_{it} \\ &+ \beta_4 \cdot Post_{it} + \beta_5 \cdot Treat_i \cdot Inf_i + \beta_6 \cdot Inf_i + \beta_7 \cdot Treat_i + X_{it} \cdot \Gamma + \epsilon_{it}. \end{aligned} \tag{2}$$

individuals who were diagnosed before 1995 gained access to HAART if they survived until 1995 and, thus, became treated. In section 5.5, we show that our results are robust to excluding observations on these individual from 1995 onwards, when HAART became available to all. Another concern is that changes in life expectancy, especially drastic ones, can have significant effects on mental health, which may in turn affect our results. We address these concerns in section 5.5, where we estimate the effect of HAART on a range of mental health measures, using the same triple difference specification as for life expectancy. We find no effects on these outcomes. In addition to these concerns, we also investigate the robustness of our results to alternative specifications and sample definitions. Overall, our robustness checks provide strong support for our main findings, suggesting that our results are not driven by any particular specification or assumption.

5   Results

In this section, we report our main findings on the impact of HAART medication on the outcomes of HIV-positive individuals. To illustrate the dynamic effects of the treatment, we first present the results in four-field figures, which show the contrasts used in the Triple Difference specification. Additionally, we provide static regression results based on Equation (2)

In each of the four-field graphs in Figures 3, 4 and 6, Graph (a) plots the evolution of the outcome before and after the HIV diagnosis for the control group (those diagnosed with HIV between 1990 and 1994, before the introduction of HAART) against the corresponding evolution for the matched synthetic control group of HIV negative individuals. In Graph (b), we show the corresponding evolution for the treatment group (those diagnosed with HIV between 1995 and 1999, after the introduction of HAART) and its matched synthetic control of HIV-negative individuals.

duction of HAART) and its matched synthetic control of HIV-negative individuals. Graph (c) plots the evolution of the control and treatment groups, demeaned by their respective synthetic control groups. Finally, Graph (d) presents the event study estimates, β_t, of the triple difference-in-differences model estimated in Equation (1), which identifies dynamic causal effects of the introduction of HAART treatment.

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 displays the results for employment, where we observe that the treatment and control groups faced similar declining employment trends in the years before the diagnosis (Graphs (a) and (b)). In both cases, the trends depart from the employment trends among the synthetic controls, highlighting 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. This difference in employment trends suggests that access to HAART may have a positive impact on employment.

The divergence in employment trends between the treatment and control groups 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, after which they start to diverge. Recall that the divergence cannot be explained by any divergence in health between the groups, as the analysis focuses on HIV positive individuals who are still in good health, where the share of individuals with CD4 counts below critical thresholds for symptoms is similar and very 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 supply incentives.

Graph (d) presents the event study estimates from the triple difference specification and provides a similar picture. The estimates confirm that the groups face parallel employment trends before the diagnosis but depart afterward. The employment rate is about 10 percentage points higher in the treatment group than the control group in the third to fourth year after diagnosis. On average, in the years following the diagnosis, the treatment group experiences a 7 percentage point increase in employment, corresponding to an 11 percent difference (Panel A of Table 2).

We next investigate the impact on labor earnings, considering that a sharp change in life expectancy may affect labor supply decisions at both the extensive and in-

tensive margins, as well as affecting productivity. Figure 4 reveals large effects on labor earnings. Before the diagnosis, the treatment and control groups follow 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 diverge, with a sharp decline in earnings in the control group. The triple difference estimates in Graph (d) show that the effects are large, with earnings being 20,000 to 30,000 DKK greater in the treatment group in the year following the diagnosis, while no such difference can be seen in the years before the diagnosis. Table 2 shows that the average effect across all post-treatment years amounts to 20,517 DKK, which corresponds to a 16 percent difference in earnings (Panel A). When we restrict the sample to individuals who participated in the labor market throughout the study period, the corresponding amount is 23,676 DKK (see also Figure A.4)

5.2 Savings, Housing, and Stock-holding

As the introduction of HAART dramatically changed the life expectancy of HIV positive individuals, it also affected their financial investment horizon. We continue by investigating how the increased life expectancy affected a) bank account savings, b) stock-holding, and c) home ownership. Figure 5 shows the triple difference event study estimates for these outcomes, while Figures A.6, A.5 and A.7 in the appendix provide further comparison between the treatment and control groups and their matched synthetic controls.

Our results reveals that the group gaining access to HAART experienced a larger decrease in bank account savings (Graph a in Figure 5). Table 2 shows that the effect amounts to a reduction of 10,628 DKK, corresponding to a 39 percent decrease. While this a large effect, it is also imprecisely estimated and not significant. Figure A.5 in the appendix further illustrates this finding, as it shows that the effect is driven by a decline in the treatment group, while the savings in the control group appear to be surprisingly unaffected by receiving an 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 that stock ownership does not evolve very differently in the treatment and control groups. This is further illustrated in Graphs (a) and (b) of Figure A.6 in the appendix, where stock ownership in both the treatment and control groups largely follow the trends of their synthetic control groups. Table 2 reveal that there was a 4 percentage point decline in stock ownership during the post-treatment years, which was statistically insignificant.

Did the change in life expectancy resulting from HAART treatment impact other significant long-term decisions, such as home ownership? We obtain no such evidence, 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 a HIV diagnosis.

The absence of an effect is confirmed in Table 2, where the average effect is small and insignificant.

Overall, the results suggest that the dramatically increased life expectancy due to HAART had surprisingly small effects on important economic decisions such as 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 and who face uncertain life expectancies. While life expectancy after a HIV diagnosis is on average shorter in the control group, some individuals will live longer than expected. The fact that assets are not depleted in the control group could be attributed to precautionary saving motives, reflecting such uncertainty in remaining life expectancy. It is also plausible that the depletion of assets mainly occurs closer to death, when health has deteriorated. Figure A.8 in the appendix does not suggest so, however, as the patterns closer to death are similar in the treatment and control groups. Another explanation for the lack of asset depletion could be that individuals in our sample possess strong bequest motives.

Furthermore, the absence of any effects could be attributed to the generosity of the Danish public pension system, where there is limited necessity to save for one's own retirement.

Taken together, the results from the analyses of earnings and the various wealth components suggest that consumption patterns may have been affected by the arrival of HAART. Notably, the sharp decline in earnings in the control group at the point of HIV diagnosis, while other significant wealth components remained unaffected, suggests that consumption levels were reduced. This could reflect a substitution from labor to leisure when otherwise healthy individuals in the control group learn about their limited life expectancy.[16]

5.3   Marriage market outcomes

Drastic changes in life expectancy may affect the incentives for partnership formation. In Figure 6, we illustrate the effects on partnership formation, defined as marriage or cohabitation. In Graph (a), we observe a notable increase in the likelihood of having a partner in the control group immediately following a HIV diagnosis, whereas no such increase is observed in the synthetic control group. In contrast, in the treatment group, we observe a decrease in the likelihood of having a partner at the time of diagnosis (Graph b). The different patterns in the treatment and control groups are further illustrated in Graph (c), and Graph (d), which presents the triple difference estimates. These estimates demonstrate a negative effect of the increased life expectancy resulting from HAART on partnership formation, reflecting the dif-

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[16]Since we do not observe all components of wealth, we cannot rule out that some other components of wealth were depleted, in order to maintain the same level of consumption.

ferential patterns observed in the treatment and control groups. Table 3 shows that this negative effect amounts to about 10 percentage points across the post-treatment years.

How should we interpret the different patterns observed in the treatment and control groups? One possibility is to interpret the increase in partnership formation in the control group, who faced a negative shock to life expectancy following their HIV diagnosis, in a family economics framework. According to this perspective, cohabitation and marriage can serve as vital sources of intra-marriage insurance against health shocks (Anderberg, 2007; Persson, 2020; Potoms and Rosenberg, 2021). As one's health declines and the need for care increases, having a partner to provide support and assistance becomes increasingly valuable. Another possible interpretation for the observed increase in partnership formation in the control group is that individuals who are HIV positive and have few years left may prioritize leisure over consumption, thus increasing the utility of being in a couple. This interpretation is consistent with the results of the previous section, which showed that a HIV diagnosis was associated with decreased consumption in the control group.[17] In contrast, individuals diagnosed after 1995 did not have an insurance motive for partnership formation, and their "option value of waiting" for the right match was higher due to the introduction of HAART and the resulting higher life expectancy. The decline in marriage and cohabitation in this group may also reflect social stigma, reduced attractiveness on the marriage market, and marriage turmoil following from HIV diagnosis.[18]

To improve the understanding of the mechanisms behind the effects, it can be informative to study whether the effect on partnership formation reflects changes in marriage, cohabitation, or divorce rates. Table 3 and Figures A.9 and A.10 in the Appendix reveal that both marriage and cohabitation rates are reduced in the treatment group. While it is possible that part of this decrease reflects an increase in divorces, the results shown in Appendix Figure A.11 and Table 3 rule out this possibility, indicating that divorces from marriage turmoil are not the driving factor.[19] Instead, the divorce rates decrease slightly in the treatment group in the years following the diagnosis, although this effect is not statistically significant. Thus, the

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[17]We have further investigated whether the increase in partnerships of individuals in the control group comes at the cost of match quality, as there is less time to find a good match, and since acquiring an HIV diagnosis reduces their attractiveness on the marriage market. Specifically, we looked at income and education differences between partners based on their observed levels before the date of diagnosis, and computed means of these pre-defined variables for partners before and after diagnosis, for the treatment and control group separately. While we did observe that individuals in the control group, after diagnosis, formed partnerships with spouses of lower income and education levels, the results should be interpreted with caution due to imprecise estimates.

[18]HAART also reduced the risk of spreading HIV infection through sexual activity. Some evidence suggests that the introduction of HAART may have led to an increase in the number of sexual partners, potentially contributing to marriage turmoil (Lakdawalla et al., 2006)

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

observed decrease in partnership formation in the treatment group occurs despite the slight decrease in divorces.

The observed increase in both marriage and cohabitation rates in the control group suggests that bequest motives are unlikely to be the main driving force behind the observed increase in partnership formation. Bequest motives would typically have a stronger effect on marriages, as the surviving spouse in Denmark automatically inherits the estate of the deceased spouse. To further explore this, we also examined whether the effects differ by sexual orientation, since the strength of any bequest motives may vary due to differences in the presence of children in the household. However, as demonstrated in the second and third columns of Table 3, the effects are significant and similar in size for both heterosexual and homosexual couples.

5.4 Alternative mechanisms: Impact of Mental Health on Behavior

In Section 3.4, we have shown that our treatment and control groups consist of HIV positive individuals who were in good physical health, based on their CD4 counts at the time of diagnosis. However, one remaining concern is that drastic changes in life expectancy can have effects on mental health and mood that can potentially affect behavior. Specifically, it is reasonable to think that a positive shock to life expectancy can improve mental health. While such changes in mental health can be part of the causal chain through which life expectancy affects behavior, it is a distinct mechanism from the pure incentive effect that arises from changes in life expectancy (Oster et al., 2013b).

The descriptive statistics in Table 1 provide little reason for concern, however. In both the treatment and control groups, only 3 percent have seen a psychiatrist and less than 1 percent have visited a psychologist for psychotherapy, which are smaller shares than those observed for the HIV–group. Furthermore, the results in Table 4 show that we obtain small and insignificant estimates when we run Equation (2) on mental and physical health outcomes. Overall, these findings suggest that changes in mental health are unlikely to be the driving force behind the observed differences in behavior between the treatment and control groups.

5.5 Robustness

We next demonstrate the robustness of our main results to alternative specifications and sample definitions. Specifically, for each of our primary outcomes, we present our baseline triple difference event study estimates in panel (a) in Appendix Figures A.12 to A.15, while panels (b) to (d) display the corresponding estimates from alternative specifications and sample definitions.

Stricter definition of the control group. An implication of our research design is that individuals in the control group (diagnosed with HIV between 1990 and 1994, before the introduction of HAART) eventually become treated. This is because we only include individuals who were alive in 1995 when HAART was introduced and when CD4 counts start being measured in our data. As a result, the control group also gains access to HAART at some point, which may bias our estimates as the behavior of the control group becomes more similar to that of the treatment group.

To address this concern, we replicate our analysis on a more restrictive sample, where we exclude observations on individuals in the control group beyond 1995. Individuals diagnosed in 1993, for instance, are kept up to two years from the diagnosis, while individuals diagnosed in 1991 are kept up to 4 years after their diagnosis. As shown in Figures A.12 to A.15, our results for earnings and partner formation are robust to imposing this stricter sample restriction.

Unbalanced sample. Our main analyses utilize a balanced sample, observed from 4 years before diagnosis until 4 years after. Figure A.3 showed that both the treatment and control groups exhibited similar survival rates in the years following their diagnosis, indicating that the balancing of the sample does not induce any differential sample selection between the two groups. Nevertheless, Graph (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 four years prior to the HIV diagnosis, and then follow both groups over time. An alternative approach is to match individuals on a yearly basis, using the same characteristics as before. Graph (d) shows that this alternative matching approach yields identical results.

5.6 The incentive effect and cost-effectiveness of new medical innovations

Our findings suggest that economic behaviors could be influenced by the anticipation of future access to life-extending medical technologies. Such incentive effects, which can manifest even before the onset of any health symptoms, are typically not accounted for in the assessment of the value of new medical technology. In this section, we demonstrate how the benefits of new medical innovations may, therefore, be underestimated in general, using the expected employment gains from HAART as an illustrative example.

To illustrate the incentive effect, we consider two hypothetical scenarios for HIV patients: one in which HAART is not yet available, and another in which HAART exists and treatment is initiated when the patient's CD4 counts fall below 350.[20] We

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[20]This was the guideline in the 1990's.

assume that the patient in both scenarios is a Danish man, 34 years of age, with 11.9 years of education, and has a CD4 count of 500 at the time of HIV diagnosis. First, we estimate the development of CD4 over time with and without the HAART treatment. We then estimate the impact of CD4 counts on employment in both scenarios, using data from the period 1995-2005. The details of the calculations are presented in Appendix A.2.

The top panel of Figure 7 displays the predicted development of CD4 in the two scenarios. In the absence of HAART, CD4 counts decline steadily over time. With access to HAART, CD4 counts decline for the first five years following the HIV diagnosis until they reach 350, after which HAART treatment is initiated, and CD4 counts start to increase again. Using these predicted CD4 counts, we can calculate the implied employment for both scenarios. The lower panel of Figure 7 shows that even in the period before HAART treatment is initiated (when the CD4 counts are similar in both scenarios), the employment level in the scenario with HAART is higher. This difference is due to the incentive effect of higher life expectancy, resulting from individuals anticipating future access to HAART at the time when they will experience HIV symptoms (dark gray area). The incentive effect is estimated to about 0.42 years of employment (dark grey area) during the first five years.

During the period where HAART has been initiated, the difference between the two scenarios is instead due to both a pure health effect and the incentive effect (light gray area). The accumulated differences in employment between those who receive HAART and those who do not from 5 to 15 years after the HIV diagnosis is 1.88 years of employment. The total employment benefit of HAART treatment is therefore the sum of the health and incentive effects, but only the former is typically accounted for. In this example, the total difference in employment between those with access to HAART and those without is 2.30 years of employment during the the first 15 years after diagnoses (the light grey and dark grey area). This calculation indicates that the total employment effect of HAART treatment during the first 15 years is underestimated by about 18 percent if the incentive effect is not accounted for. This back-of-the-envelope calculation demonstrates that the full benefits of introducing medical technologies, such as HAART, may be underestimated unless both types of effects are accounted for.

6 Conclusion

Over the last century, populations in the Western world have experienced remarkable gains in life expectancy (Case and Deaton, 2020). This increase in life expectancy alters individual incentives to work, save, and marry, but it has proven challenging to distinguish the pure incentive effect of a longer planning horizon from that of improved health, as the two usually go hand in hand. We overcome this challenge by

examining the impact of a sudden and dramatic rise in life expectancy caused by a crucial medical innovation: 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, allowing us to isolate the pure incentive effect of increased life expectancy.

Our empirical results reveal that the rise in life expectancy that followed the introduction of HAART had substantial effects on the labor market behaviors of HIV positive but otherwise healthy individuals. Labor supply and earnings were considerably higher for those diagnosed after HAART became available, suggesting that the increase in life expectancy strengthened incentives to work. HIV-positive individuals without access to HAART, however, may have substituted labor for leisure, given their short remaining life expectancy.

On the other hand, we find no evidence that the positive shock to life expectancy had any large effect on financial decisions, such as savings and stock market participation among HIV positive individuals. While perhaps surprising, in light of standard life-cycle theory, we attribute this finding to strong bequest and precautionary savings motives.

Our results also indicate that the introduction of HAART had an effect on marriage market incentives. Partnership formation after a HIV diagnosis declined for those with access to HAART, while it increased for those without access. We interpret this finding through a family economics lens, where cohabitation and marriage serve as vital sources of private insurance against health shocks.

Our research adds to the limited literature that demonstrates how longer life expectancy strengthens the incentives for human capital investments, which, in turn, affect economic growth. We show that longer life spans affect people's labor market and marriage market behavior, implying that life expectancy affects welfare and economic growth both through a human capital and labor market channel.

Finally, our findings have important implications for the valuation of life-extending medical technologies. Cost-effectiveness analyses typically do not account for the incentive effects that arise from increases in life expectancy due to new medical technologies, such as HAART. Based on our estimates, we conduct a simple simulation exercise, showing that the incentive effect may account for up to 20 of the total effect on employment during the first 15 years after an HIV diagnosis. This highlights the importance of considering labor market incentives when evaluating the value of life-extending medical technologies, as failure to account for these effects may lead to underestimation of their true value.

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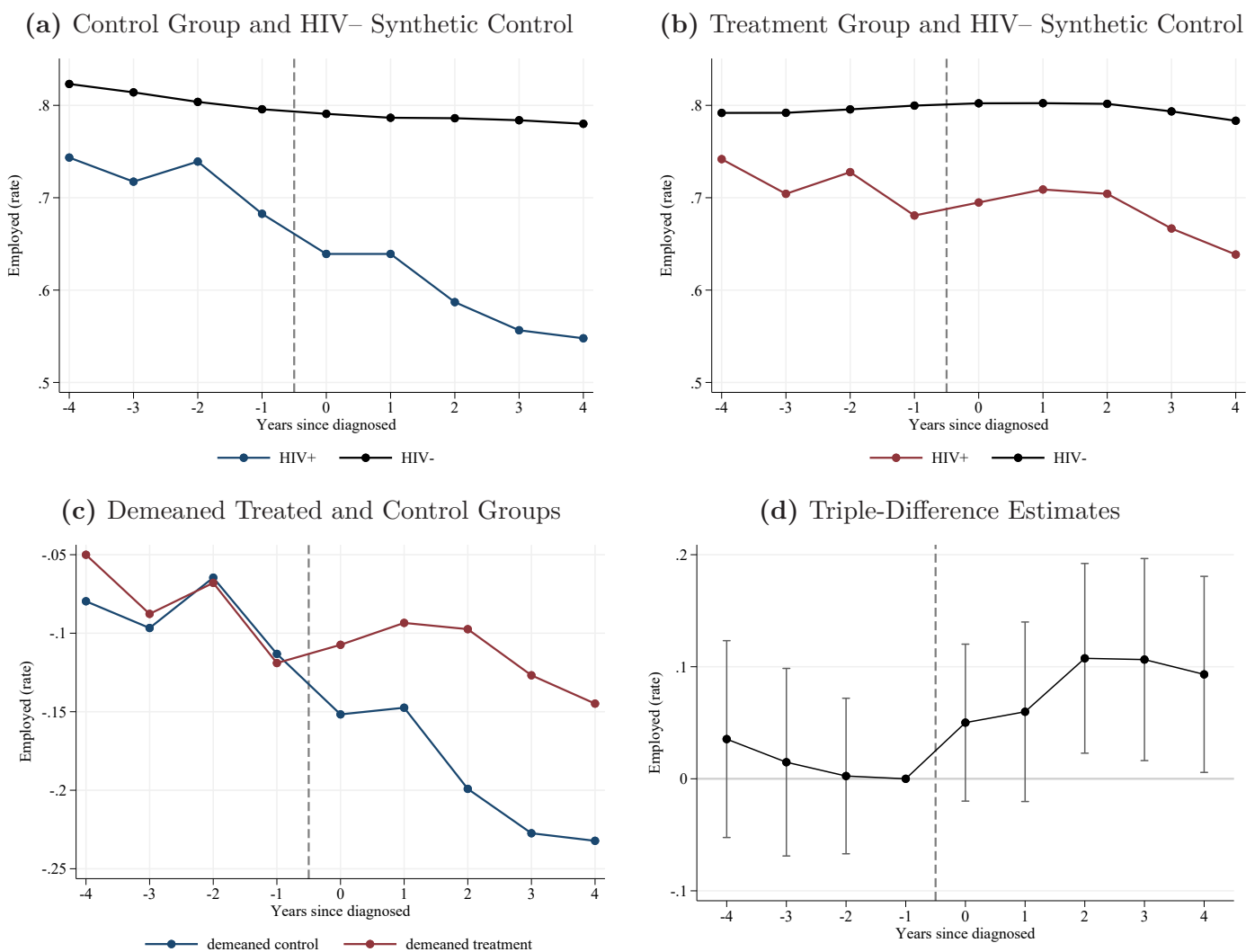**Figure 1:** Survival by Year of HIV Diagnosis
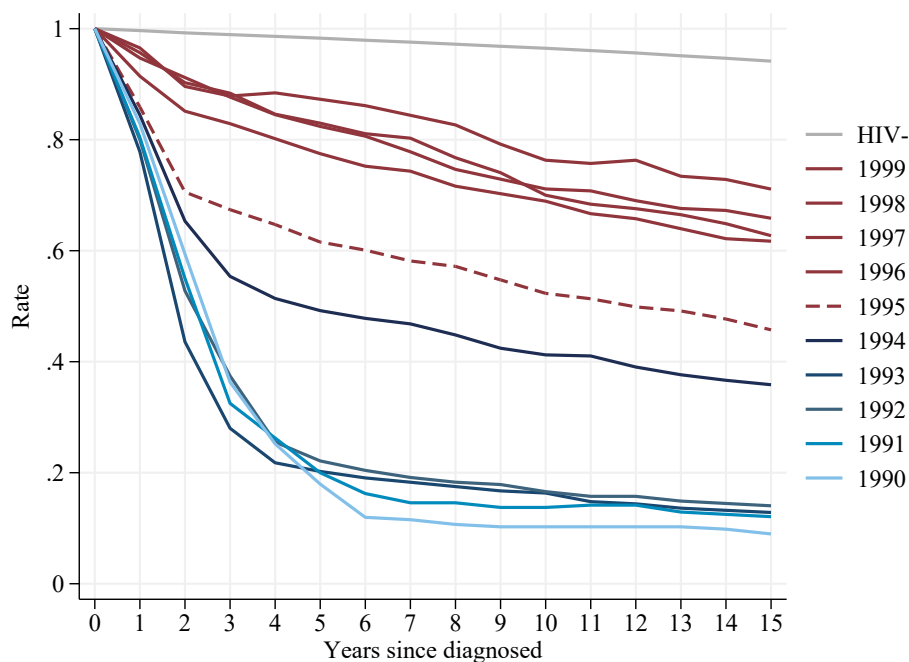
Notes: This figure plots the survival rate of individuals diagnosed with HIV distinguishing by year of diagnosis. The Figure illustrates that individuals diagnosed during earlier calendar years face sharp drops in their survival rates in the years following the diagnosis, while individuals diagnosed 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) Under 200

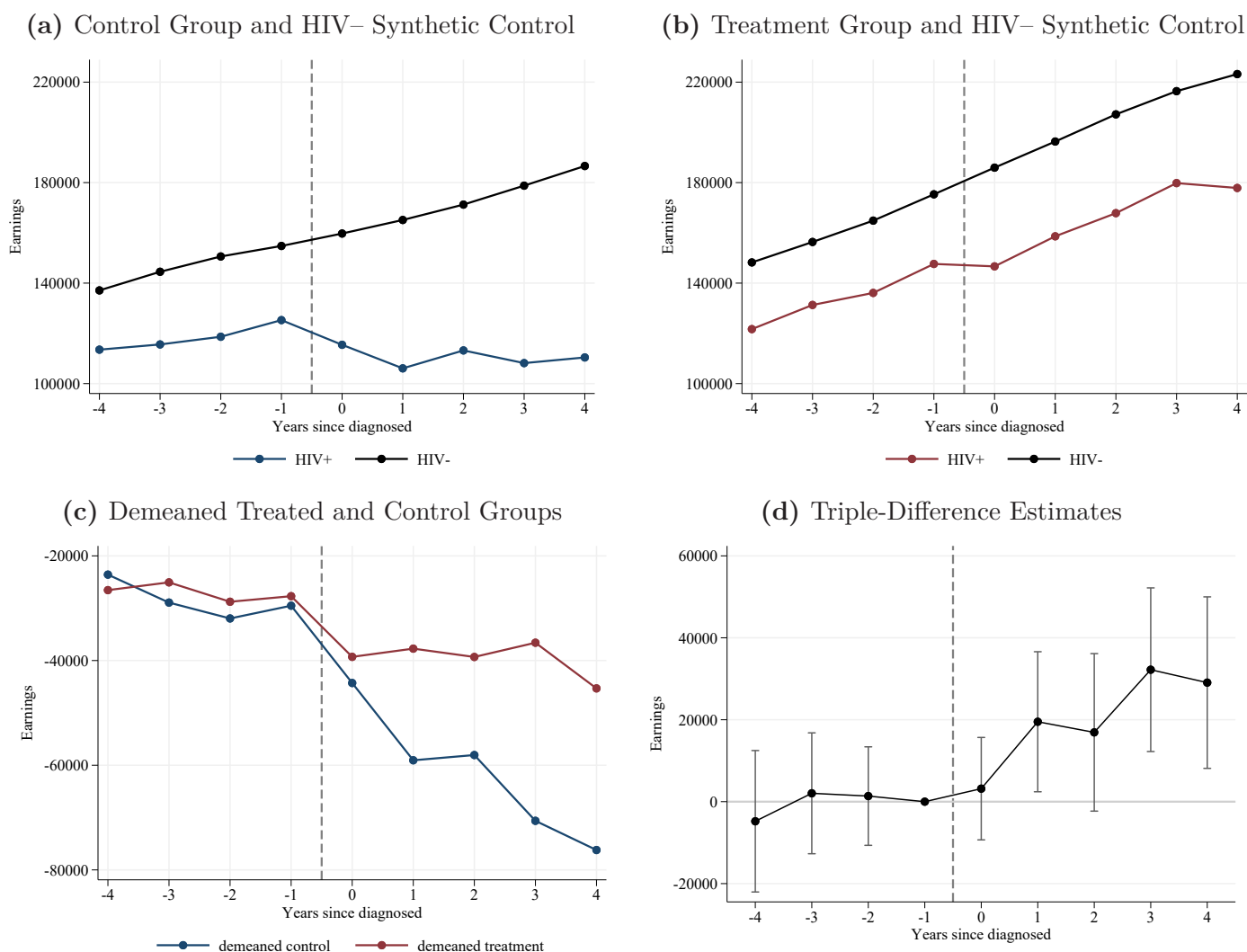**(b)** Under 250

(c) Under 300
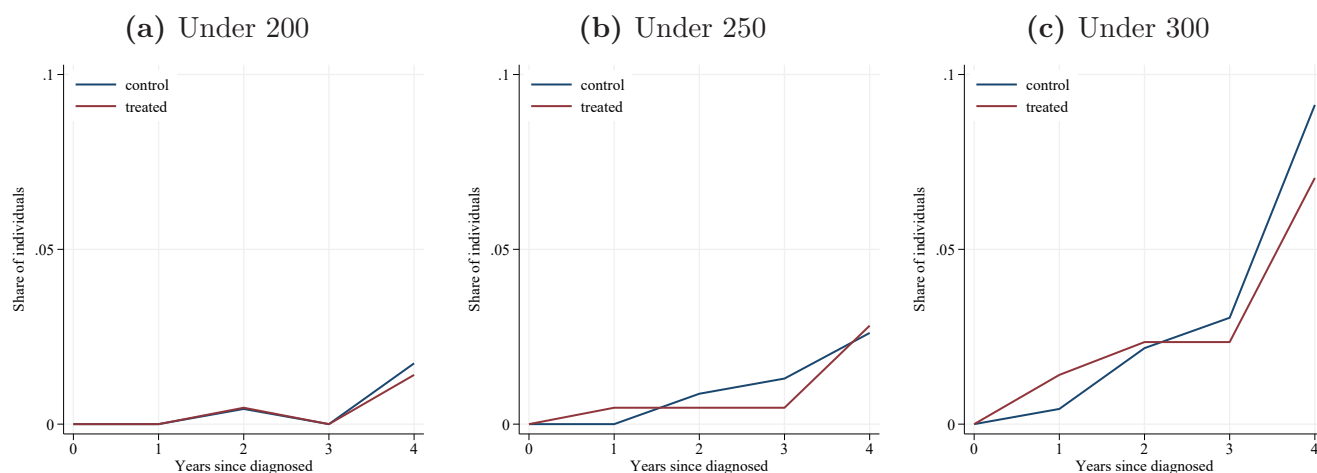
Notes: These figures display the proportion of individuals in the treatment and control groups whose CD4 counts fall below a particular threshold. In panel (a), the threshold is 200, which is regarded as the level where AIDS can initiate. In panel (b), the threshold is 250. In panel (c), the threshold is 300.

Figure 3: Effects on Employment of the introduction of HAART treatment 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
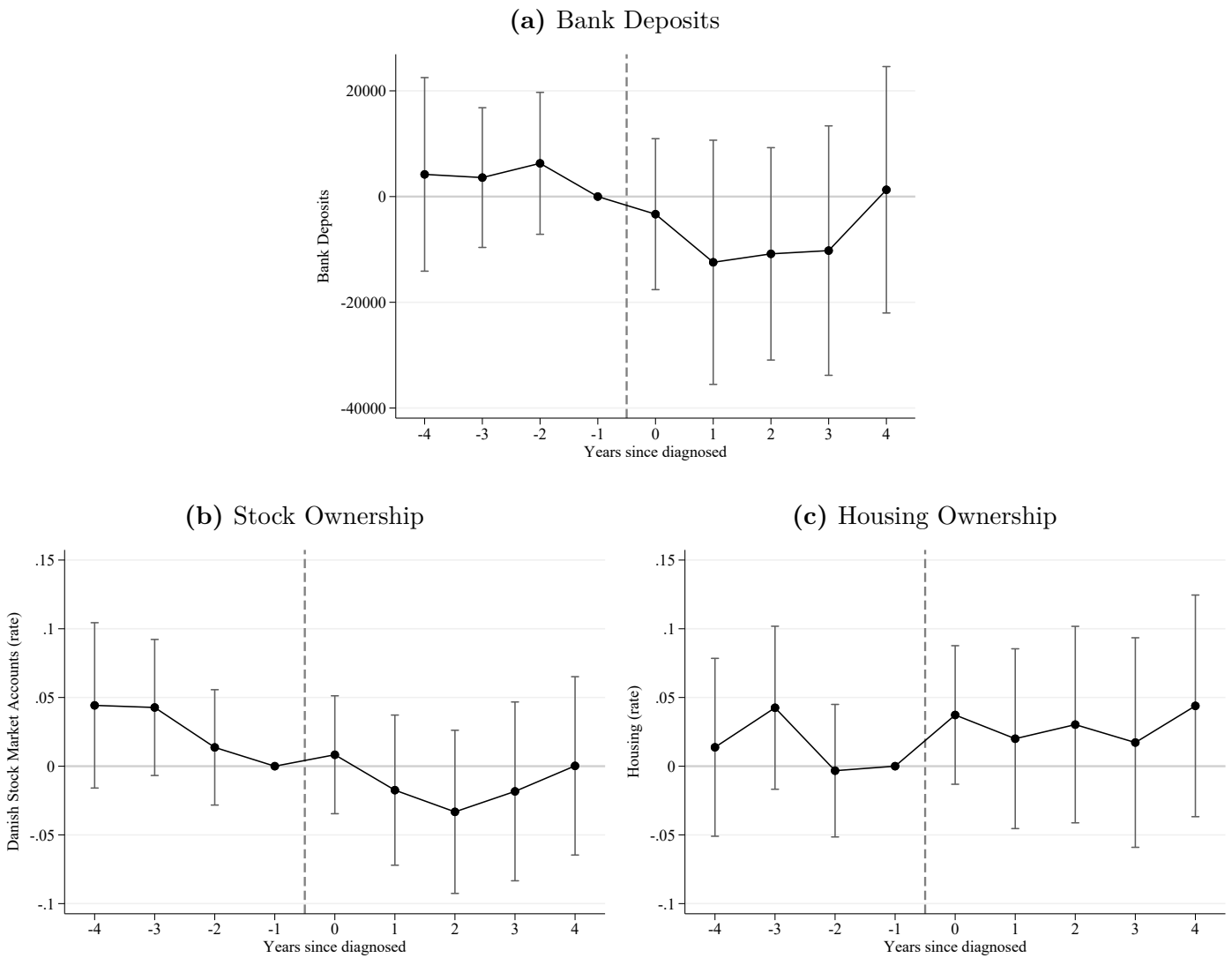

(d) Triple-Difference Estimates

Notes: These graphs plot the effects on employment of the introduction of HAART treatment 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 1995 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 treatment. We report 95% confidence intervals calculated from clustered standard errors.

Figure 4: Effects on Earnings of the introduction of HAART treatment 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
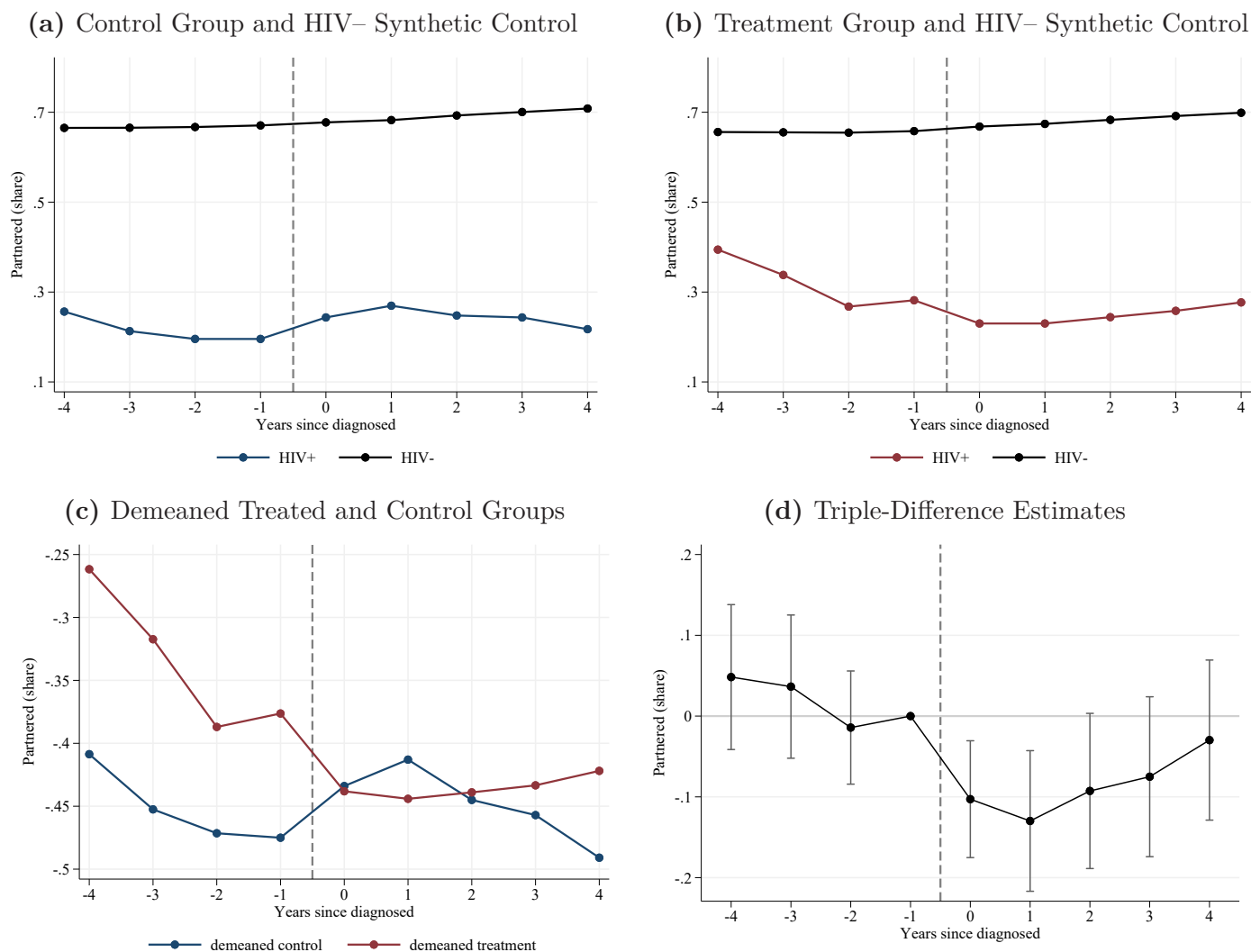

(d) Triple-Difference Estimates

Notes: These graphs plot the effects on earnings of the introduction of HAART treatment 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 1995 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 treatment. We report 95% confidence intervals calculated from clustered standard errors.

Figure 5: Triple-Difference Estimates of the Effects on Bank Account Savings, Stocks, and Housing of the introduction of HAART treatment around the Time of HIV Diagnosis

(a) Bank Deposits

(b) Stock Ownership


(c) Housing Ownership

Notes: These graphs plot the effects on bank account savings, stocks, and housing of the introduction of HAART treatment 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 treatment. We report 95% confidence intervals calculated from clustered standard errors.

Figure 6: Effects on Partnership of the introduction of HAART treatment 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 treatment 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 1995 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 treatment. We report 95% confidence intervals calculated from clustered standard errors.

Figure 7: CD4 and Employment with and without HAART
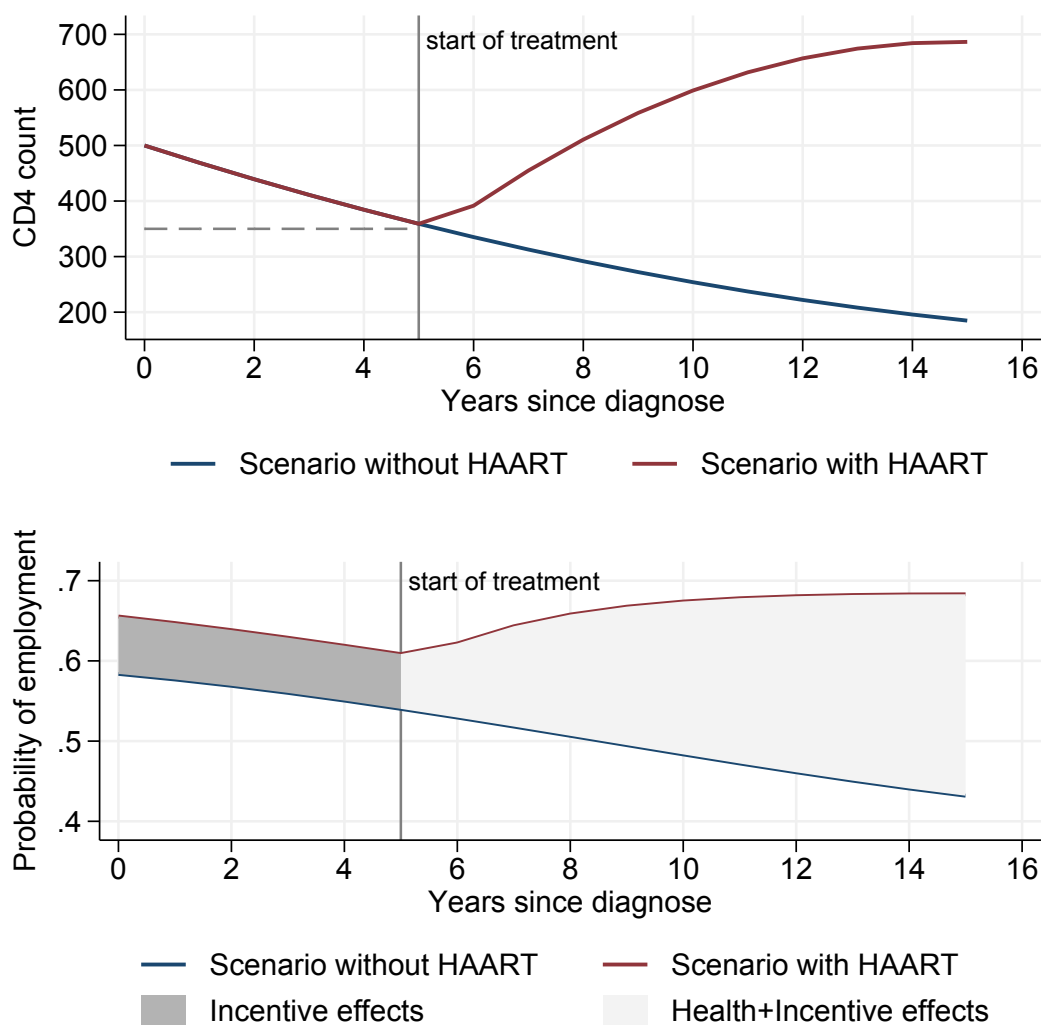
Notes: Top panel: The figure is based on two fixed effect regressions of CD4 counts on time since diagnosis and time since treatment initiated, see appendix A.2, step 1. The lower panel is calculated based on estimates from a regression of employment on CD4 counts. We allow the effect to depend on whether HAART was available at diagnosis, see Appendix A.2

Table 1: Descriptive Statistics

	Control	Treated	Difference	*P-value*	HIV–
	(1)	(2)	(3)	(4)	(5)
Demographics					
Age	33.60	34.54	-0.94	0.27	34.04
Male	0.82	0.80	0.02	0.53	0.81
Years of education	11.6	11.8	-0.21	0.37	12.1
Dane	0.93	0.91	0.03	0.26	0.96
Economic outcomes					
Employed	0.68	0.68	0.00	0.97	0.81
Earnings	125,273	147,638	-22,366	0.06	169,700
Earnings (percentile)	41.52	43.95	-2.43	0.38	50.5
Home Owner	0.24	0.27	-0.02	0.56	0.49
Stocks Ownership	0.14	0.07	0.07	0.01	0.16
Marital					
Married	0.13	0.18	-0.06	0.10	0.44
Cohabiting	0.07	0.10	-0.03	0.27	0.23
Health					
Psychotherapy	<0.01	0.02	0.00	1.00	0.002
Psychiatry	0.02	0.03	-0.01	0.66	0.008
Charlson Index	<0.01	0.02	-0.02	0.08	0.006
Infections	<0.01	0.01	-0.01	0.14	0.001
DANHIV					
CD4 Count	618.79	619.87	-1.08	0.95	–
Heterosexual	0.43	0.44	-0.01	0.82	–
Observations	230	213			443,000

Notes: This table presents summary statistics of key variables for different samples, measured one year before diagnosis, except CD4 counts that are measured in 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 Access to HAART on Labor and Wealth Outcomes.
Triple-Difference Estimates

	Estimate (1)	Mean (2)
A: Labor Outcomes		
Employment	0.0702** (0.029)	0.666
Earnings	20,517*** (7,521)	130,457
Earnings (cond. part.)	23,676*** (8,501)	179,479
B: Assets		
Bank Accounts	-10,628 (7,679)	27,425
Any Stocks	-0.0373 (0.024)	0.117
Home Ownership	0.0165 (0.030)	0.264
Observations	4,990,987	4,990,987
N. Individuals	443,443	–

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 4 years following diagnosis. Column (2) reports the average value of a given outcome measured the year before HIV diagnosis for the sample of analysis. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$

Table 3: Marital Effects of the introduction of HAART Treatment after HIV Diagnosis. Triple-Difference Estimates.

	Full Sample (1)	Hetero. (2)	Homo. (3)	Mean (4)
Partnered	-0.104***	-0.119**	-0.0922**	0.24
	(0.036)	(0.057)	(0.044)	
Married	-0.0596*	-0.0634	-0.0574	0.15
	(0.032)	(0.050)	(0.041)	
Cohabiting	-0.0441**	-0.0559	-0.0349	0.09
	(0.022)	(0.039)	(0.022)	
Divorce	-0.0086	-0.0134	-0.0036	0.03
	(0.010)	(0.017)	(0.012)	
Observations	3,990,987	1,891,890	2,099,097	3,990,987
N. Individuals	443,443	210,210	233,233	443,443

Notes: Columns (1) to (3) in this table report the coefficient of interest, β_1, estimated in Equation (2), that captures the causal effect of the introduction of HAART treatment that extended life expectancy on different outcomes, for the full sample as well as distinguishing by sexual orientation. Column (4) reports the average value of a given outcome measured the year before HIV diagnosis for the full sample. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$

Table 4: Health Effects of the introduction HAART Treatment after HIV Diagnosis.
Triple-Difference Estimates

	Estimate (1)	Mean (2)
A: Physical Health		
Charlson Index	0.0002	0.020
	(0.009)	
Infections	0.0001	0.007
	(0.005)	
B: Mental Health		
Psychologist	-0.0026	0.023
	(0.008)	
Psychiatrist	0.0087	0.023
	(0.010)	
Observations	3,990,987	3,990,987
N. Individuals	443,443	–

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 4 years following diagnosis. Column (2) reports the average value of a given outcome measured the year before HIV diagnosis for the sample of analysis. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$

Appendix A

A.1 The dissemination of news on the HAART treatment

The HAART medication was introduced in 1996 in Denmark, but in 1995 the first positive indications of the new treatment were already presented. In the mid-1990s there was a lot of attention for the medical innovations on HIV treatment and the media were frequently reporting from scientific conferences and events. In 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 was disseminated to the Danish population. An 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 translated 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 ddl and ddC.*[21] illustrates the growing optimism and the beginning of a new era with an effective treatment of HIV.

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 for 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*

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

A.2   Calculations of incentive effects

In this section, we present "a back of the envelope calculation" of the benefits from introducing a new medical treatment. The calculations illustrate that the benefits are underestimated if the incentive effects prior to the actual treatment are not accounted for. The calculations are made in three steps. In all the steps, we use data on all HIV patients diagnosed from 1985-2005 who were still alive in 1995 except drug addicts. We exclude extreme observations with CD4 counts above 1600.[22] In the analysis, we use data from the period 1995-2005. The data contains 18,262 observations from 2,519 individuals. Note that in this exercise we use the actual CD4 counts and not the imputed ones.

Step 1: To make the calculations, we need to estimate the development in CD4 counts before the HIV patient receives the HAART treatment and the development after receiving the HAART treatment. We do this by estimating two fixed effect regressions of CD4 counts: one for individuals who are diagnosed but have not yet received HAART treatment, and the one for individuals after the HAART treatment has started. The estimation results are shown in the following equations:

$$
\begin{aligned}
CD4_{it} &= \underset{(6.26)}{539.50} - \underset{(1.76)}{31.53} \cdot ysd_{it} + \underset{(0.11)}{0.70} \cdot ysd_{it}^2 + \widehat{\alpha_i} + \widehat{\epsilon_{it}} \\
CD4_{it} &= \underset{(2.29)}{262.64} + \underset{(1.34)}{71.67} \cdot ysh_{it} - \underset{(0.16)}{3.81} \cdot ysh_{it}^2 + \widehat{\alpha_i} + \widehat{\epsilon_{it}},
\end{aligned}
\tag{3}
$$

where ysd is year since diagnosis and ysh is years since HAART treatment initiated.

Step 2: We estimate a flexible function for how the CD4 counts affect employment. In the specification, we allow the impact of CD4 to depend on whether individuals were diagnosed before or after the introduction of HAART.[23] We use the following model:

$$
\begin{aligned}
Empl_{it} =& \gamma_0 + \delta_0 \cdot Treat_i + X_{it}\beta \\
&+ \sum_{s=1}^{3} \gamma_s 1(CD4_{it})^s + \sum_{s=1}^{3} \delta_s (CD4_{it})^s \cdot Treat_i + u_{it},
\end{aligned}
\tag{4}
$$

where X contains age, months of education, a dummy for female, a dummy for being Dane and calendar year dummies.[24] The estimation results are shown in Figure A.1. The predicted employment rate is shown for a Danish man who is 34 years old

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[22]We drop 22 observations out of more than 19.000 observations

[23]We define individuals diagnosed between 1985-1994 as the group diagnosed without HAART and individuals diagnosed from 1995-2005 as diagnosed with HAART.

[24]For comparison, we also estimate a specification with indicator functions:

$$
\begin{aligned}
Empl_{it} =& \gamma_0 + \delta_0 \cdot Treat_i + X_{it}\beta \\
&+ \sum_{s} \gamma_s 1(CD4_{it} \in I_s) + \sum_{s} \delta_s 1(CD4_{it} \in I_s) \cdot Treat_i + u_{it},
\end{aligned}
\tag{5}
$$

where 1 is an indicator function, and I_s is an interval of size 50.

Figure A.1: Employment and CD4 counts
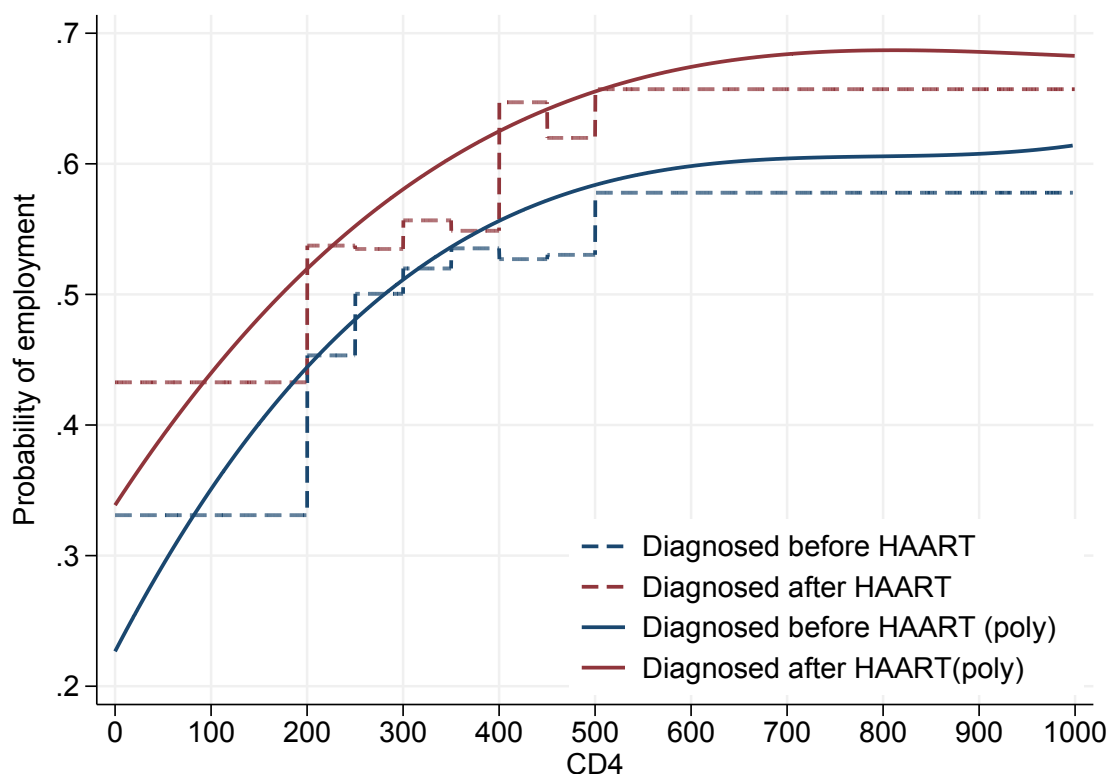
Notes: This figure shows estimates of the impact of CD4 counts on employment. The estimated effects are calculated for a man aged 34 with 143 months of education. The year is set to 1995. The blue curve is estimates for a patient without access to the HAART treatment while the red curve is for a patient with access to HAART treatment. The solid lines refer to the estimates are obtained from an OLS regression of equation (4) while the dashed lines refer to equation (5).

and has 143 months of education and is shown for year 1995. The results confirm that even at the same level of CD4 counts the cohorts diagnosed after the HAART treatment was available are more likely to work.

Step 3: We now calculate the predicted probability of employment and compare the predictions for an individual in the two scenarios, one where HAART treatment exists and one where HAART does not exist. We assume that HAART treatment is initiated when the CD4 counts fall below 350 as the guidelines stipulated in the 1990s. To illustrate the benefits from the new medical innovation, we calculate the probability of employment for a Danish man aged 34 who has 143 months of education. In both scenarios, we assume that the individual has CD4 counts at 500 at the time of the diagnose. We use the estimated equations in (3), to predict the development of CD4 in the two scenarios, see top panel of Figure 7. The HAART treatment in the scenario with HAART, will be initiated 5 years after diagnosis since the CD4 will be below 350. Based on the (predicted) CD4 counts, we use figure A.1 to calculate the implied predicted employment. The lower panel in Figure 7 shows

that even before the individuals receive the treatment (where the CD4 counts are similar in the two scenarios), employment in the scenario with HAART is higher. The difference is due to the incentive effect of individuals who already anticipate that they will have access to the treatment when they experience symptoms of HIV (dark gray area). The difference after the treatment has started is due to both a health and an incentive effect (light gray area). The total employment benefit of the HAART innovation should, therefore, be the sum of two effects. The example illustrates that unless the incentive effects prior to start of treatment are included, the total benefit of introducing HAART is underestimated.

A.3 Supplementary Tables and Figures

Figure A.2: Distribution of individual's Observed CD4 Count Changes
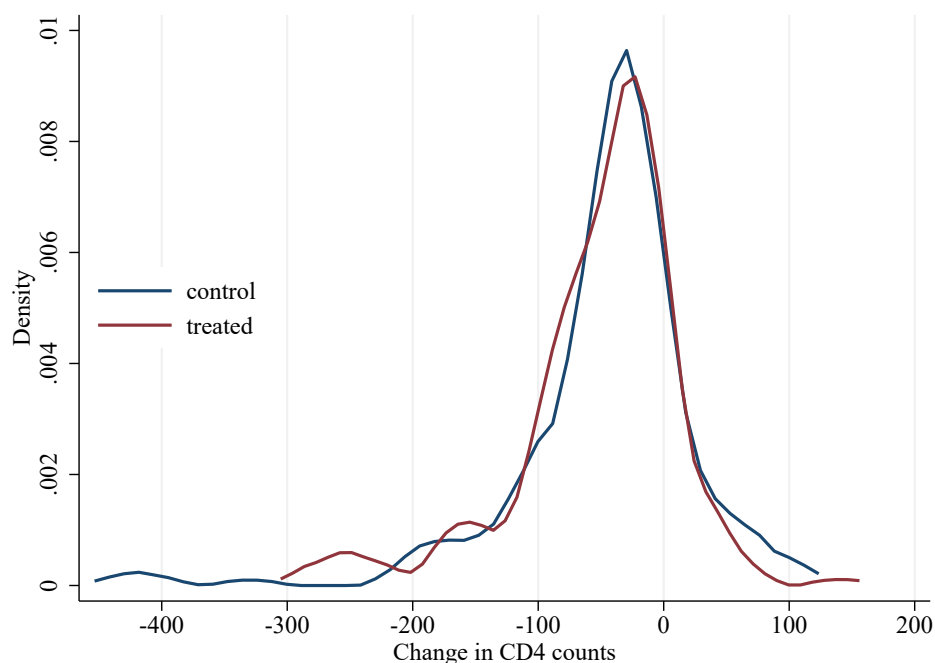
Notes: This figure shows the distribution of the slope parameters resulting from estimating the following linear equation: $CD4_{it} = \alpha + \beta * time_i + \epsilon_{it}$ for each individual observed at least two times between being diagnosed with HIV and receiving HAART treatment. $CD4_{it}$ refers to observed CD4 counts, and $time$ is 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 treatment.

Figure A.3: Survival by Treatment Group
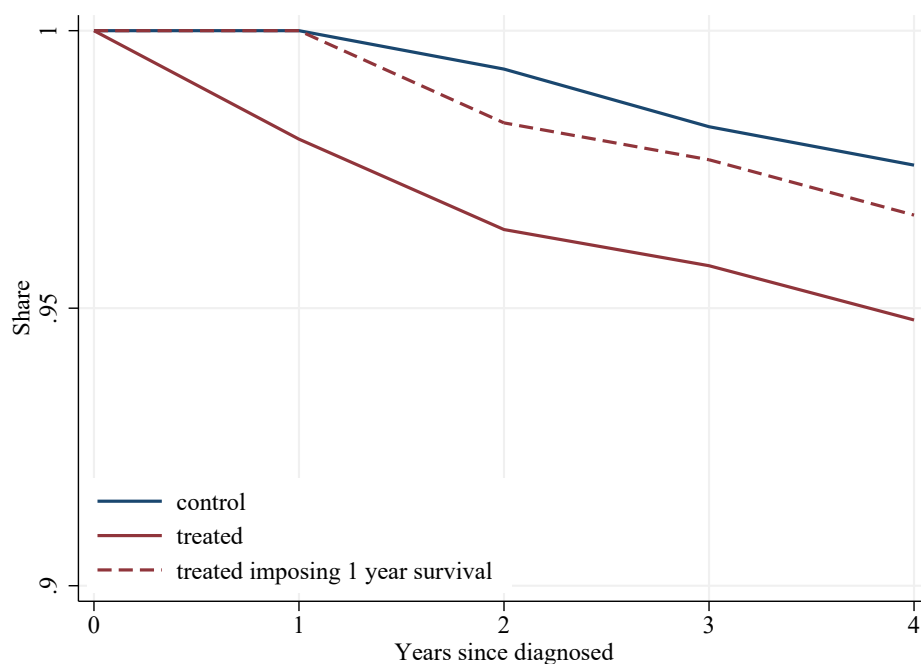
Notes: This figure shows the share of individuals who were alive up to 4 years after diagnosis distinguished by treatment and control group before we impose a balancing of the sample (that is, before we keep only those individuals who are alive and observed every year from four years before diagnosis 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.4: Effects on Earnings (Conditional on Labor Market Participation) of the introduction of HAART Treatment 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
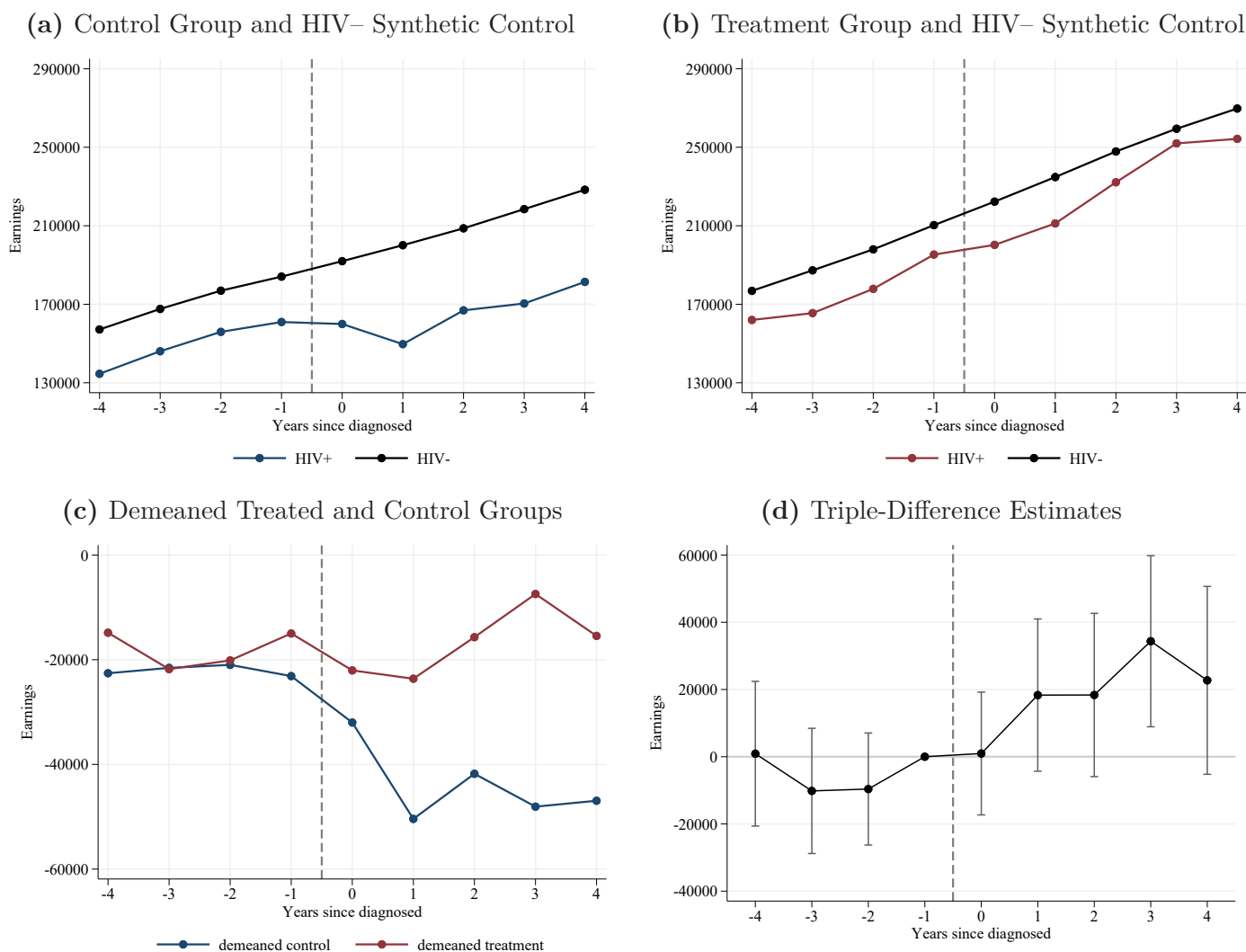
Notes: These graphs plot the effects on earnings conditional on participation of the introduction of HAART treatment on individuals around the time when they are diagnosed with HIV. Graph (a) plots an event study for the control group (those diagnosed with HIV between 1990 and 1994, before the introduction 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 1995 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 treatment. We report 95% confidence intervals calculated from clustered standard errors.

Figure A.5: Effects on Bank Account Savings of the introduction of HAART Treatment 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
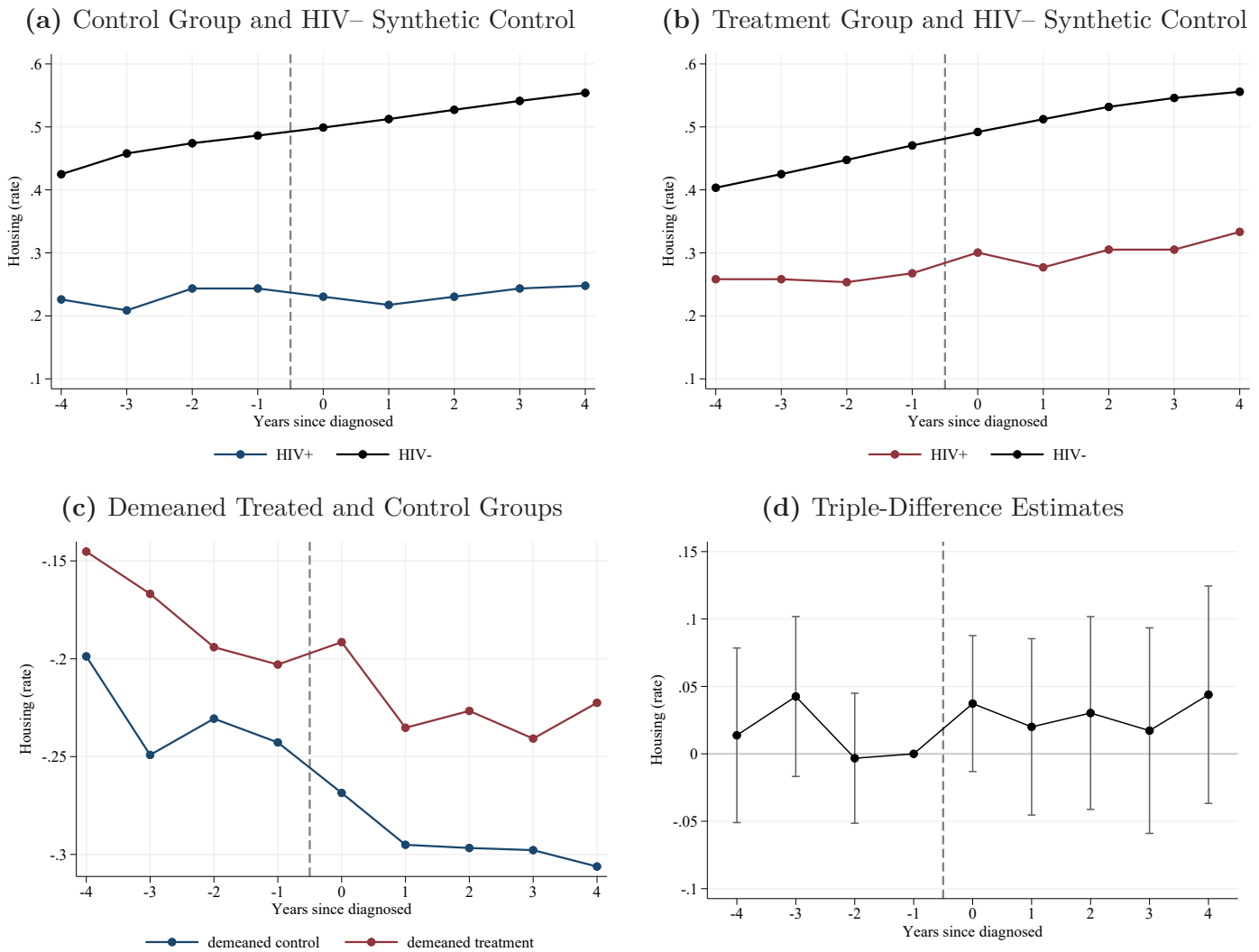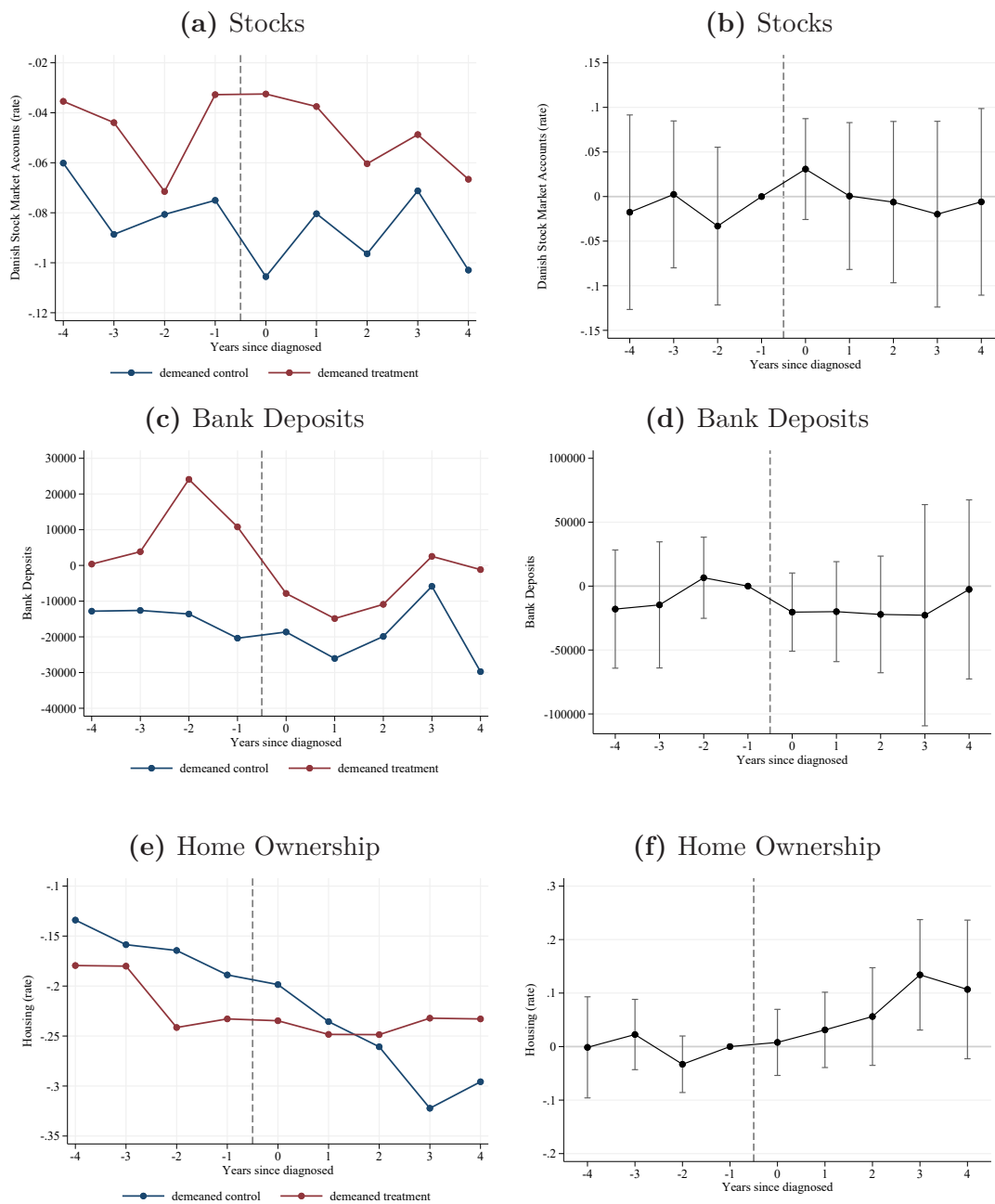

(d) Triple-Difference Estimates
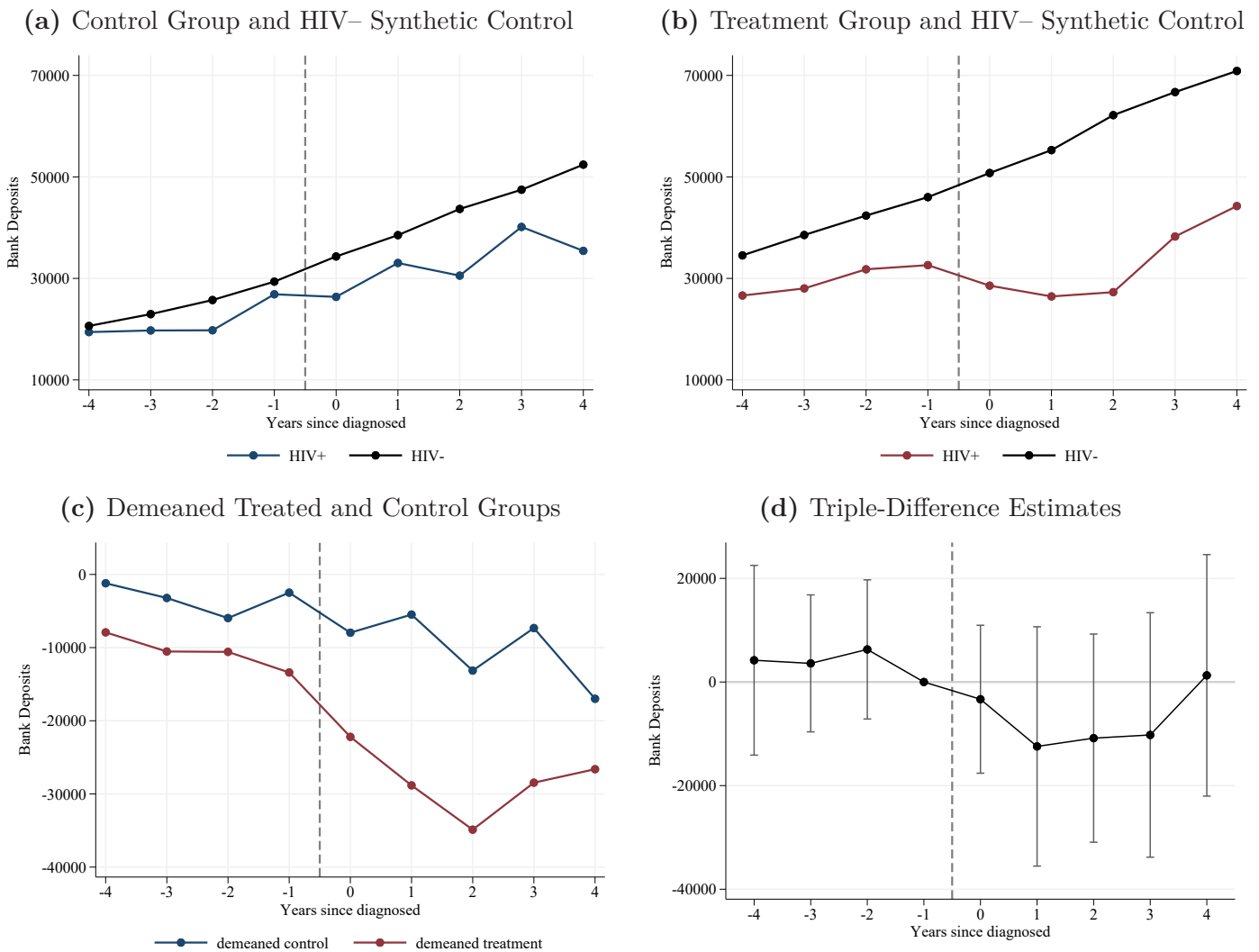
Notes: These graphs plot the effects on bank accounts of the introduction of HAART treatment on individuals around the time when they are diagnosed with HIV. Graph (a) plots an event study for the control group (those diagnosed with HIV between 1990 and 1994, before the introduction of HAART) against 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 1995 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 treatment. We report 95% confidence intervals calculated from clustered standard errors.

Figure A.6: Effects on Stocks Ownership of the introduction of HAART Treatment 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
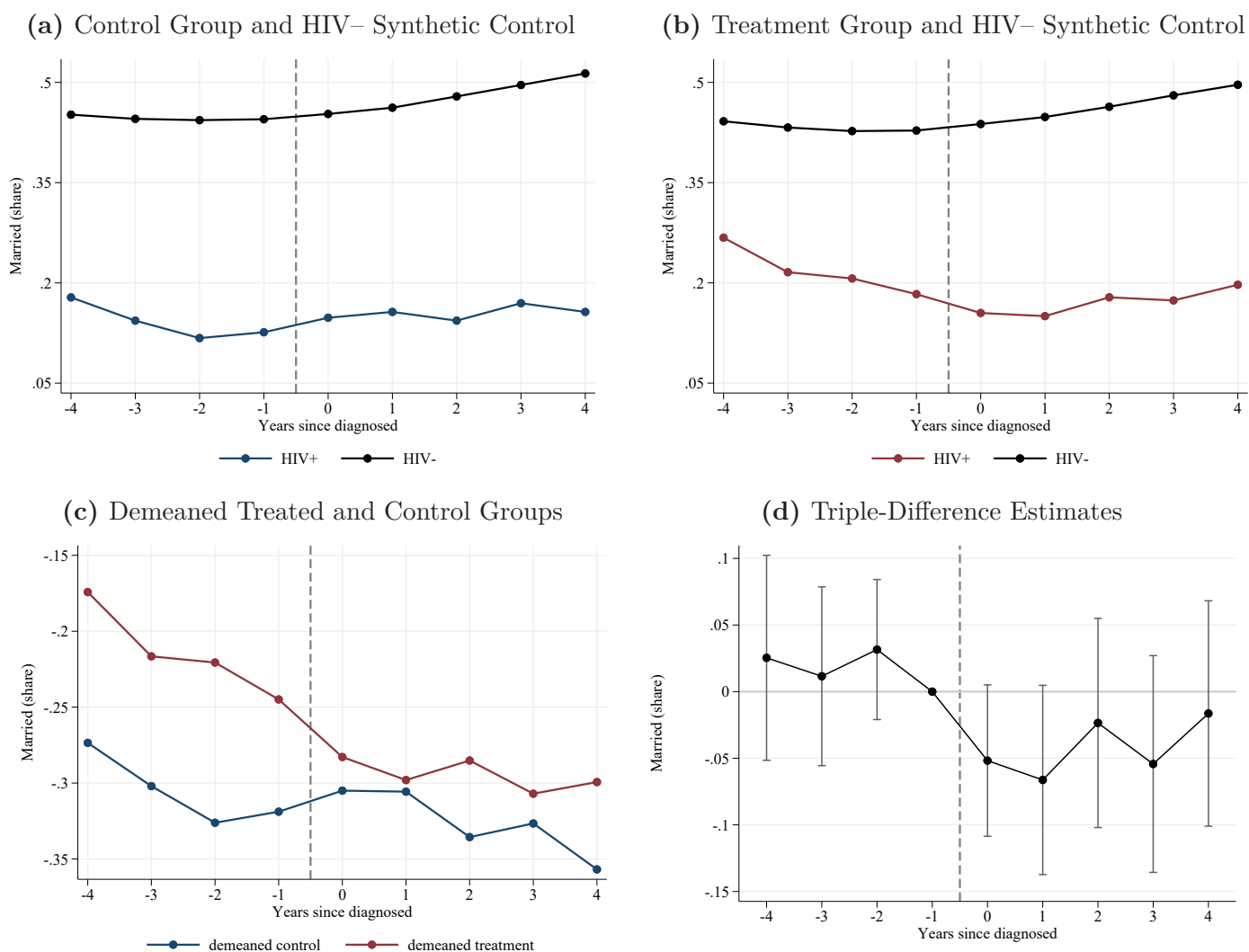

(d) Triple-Difference Estimates
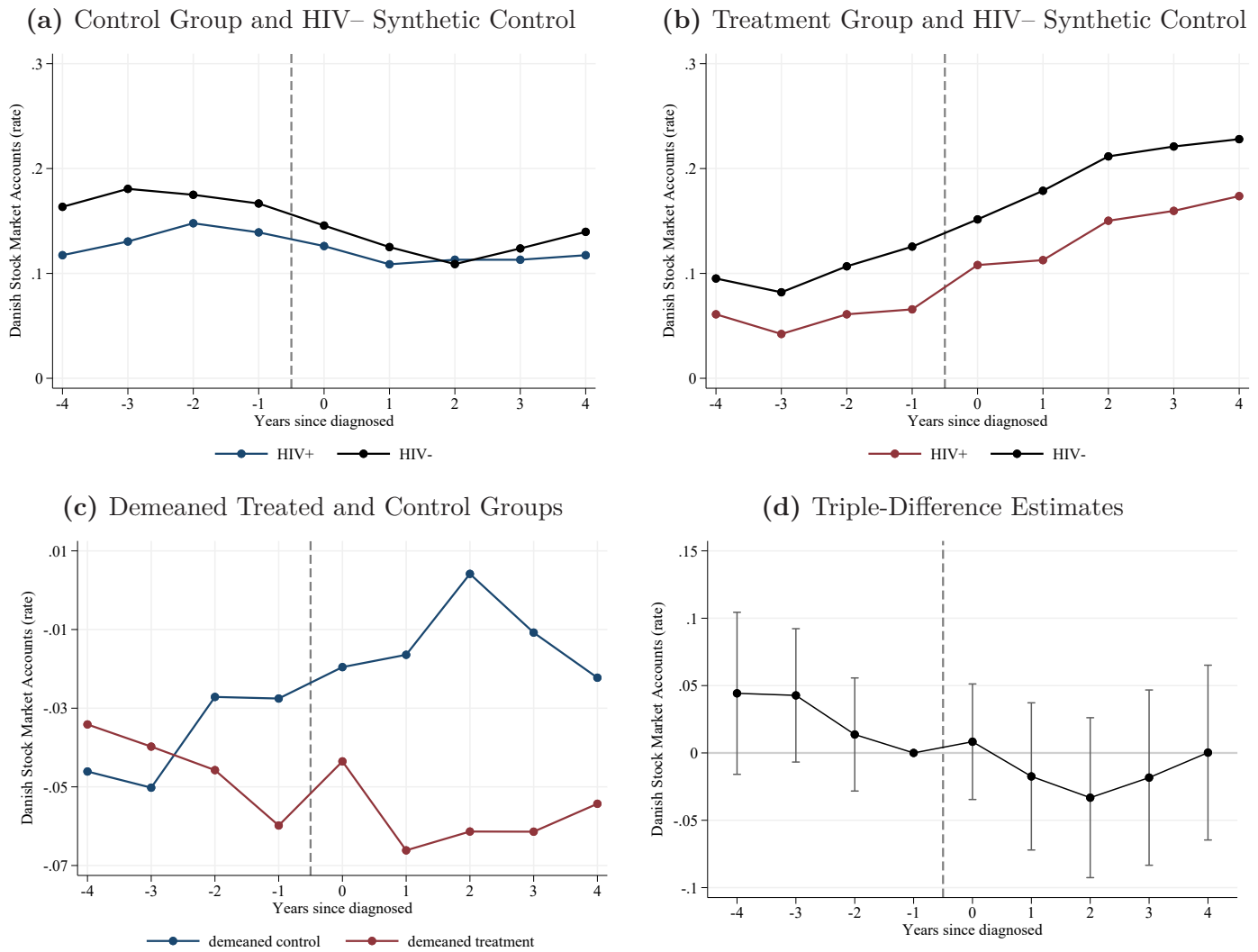
Notes: These graphs plot the effects on stocks ownership of the introduction of HAART treatment on individuals around the time when they are diagnosed with HIV. Graph (a) plots an event study for the control group (those diagnosed with HIV between 1990 and 1994, before the introduction of HAART) against 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 1995 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 treatment. We report 95% confidence intervals calculated from clustered standard errors.

Figure A.7: Effects on Home Ownership of the introduction of HAART Treatment 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
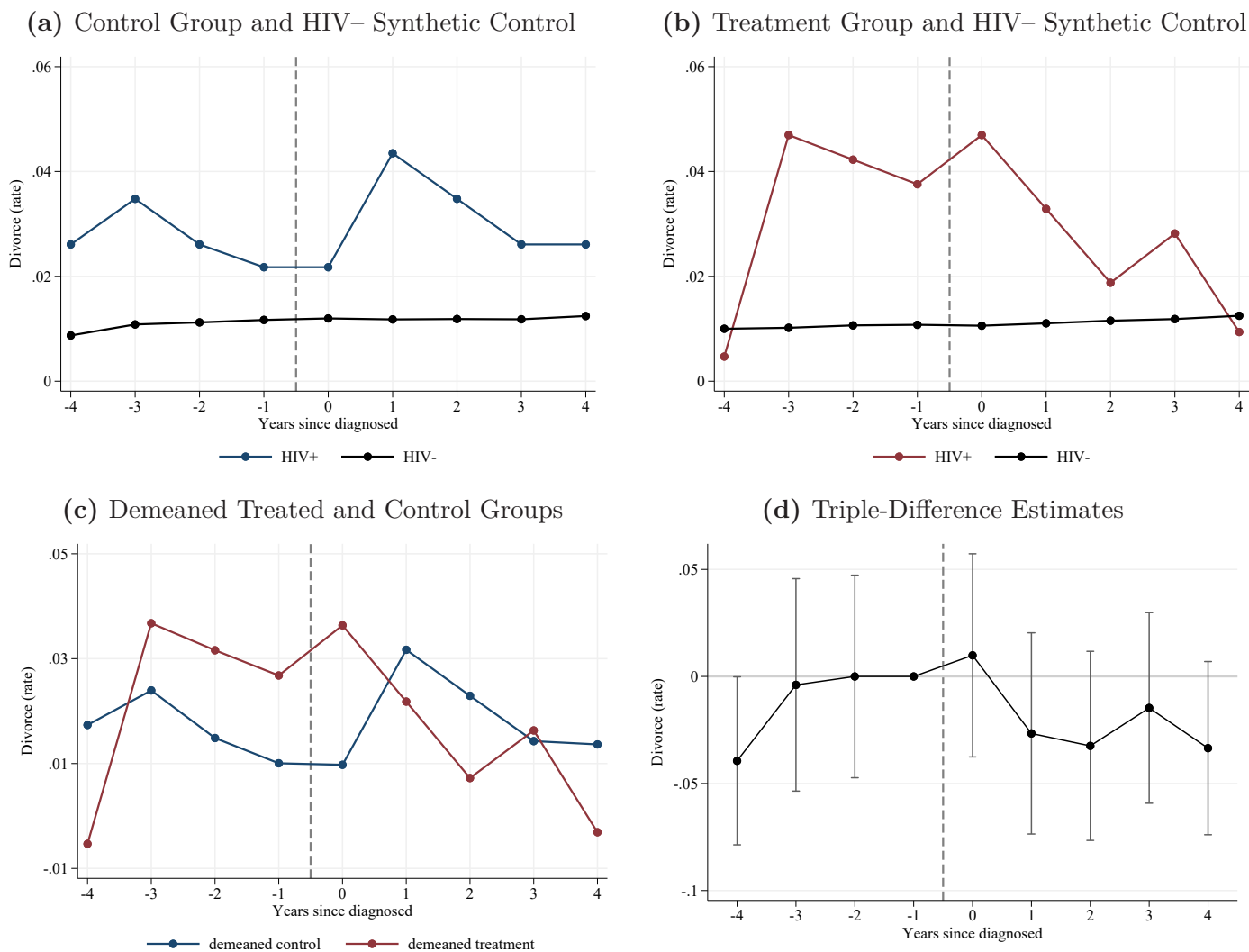
(d) Triple-Difference Estimates

Notes: These graphs plot the effects on home ownership of the introduction of HAART treatment 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 1995 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 treatment. We report 95% confidence intervals calculated from clustered standard errors.

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

(a) Stocks

(b) Stocks

(c) Bank Deposits

(d) Bank Deposits

(e) Home Ownership

(f) Home Ownership

Notes: This figure shows estimates of wealth effects for a group of individuals with a lower CD4 count (150-250) than the main sample. Graphs (a, c and e) on the left plot the control and treatment groups demeaned by their respective synthetic controls. Graphs (b, d and f) on the right plot the estimates of the dynamic triple difference-in-differences model estimated in Equation 1

Figure A.9: Effects on Marriage of the introduction of HAART Treatment 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
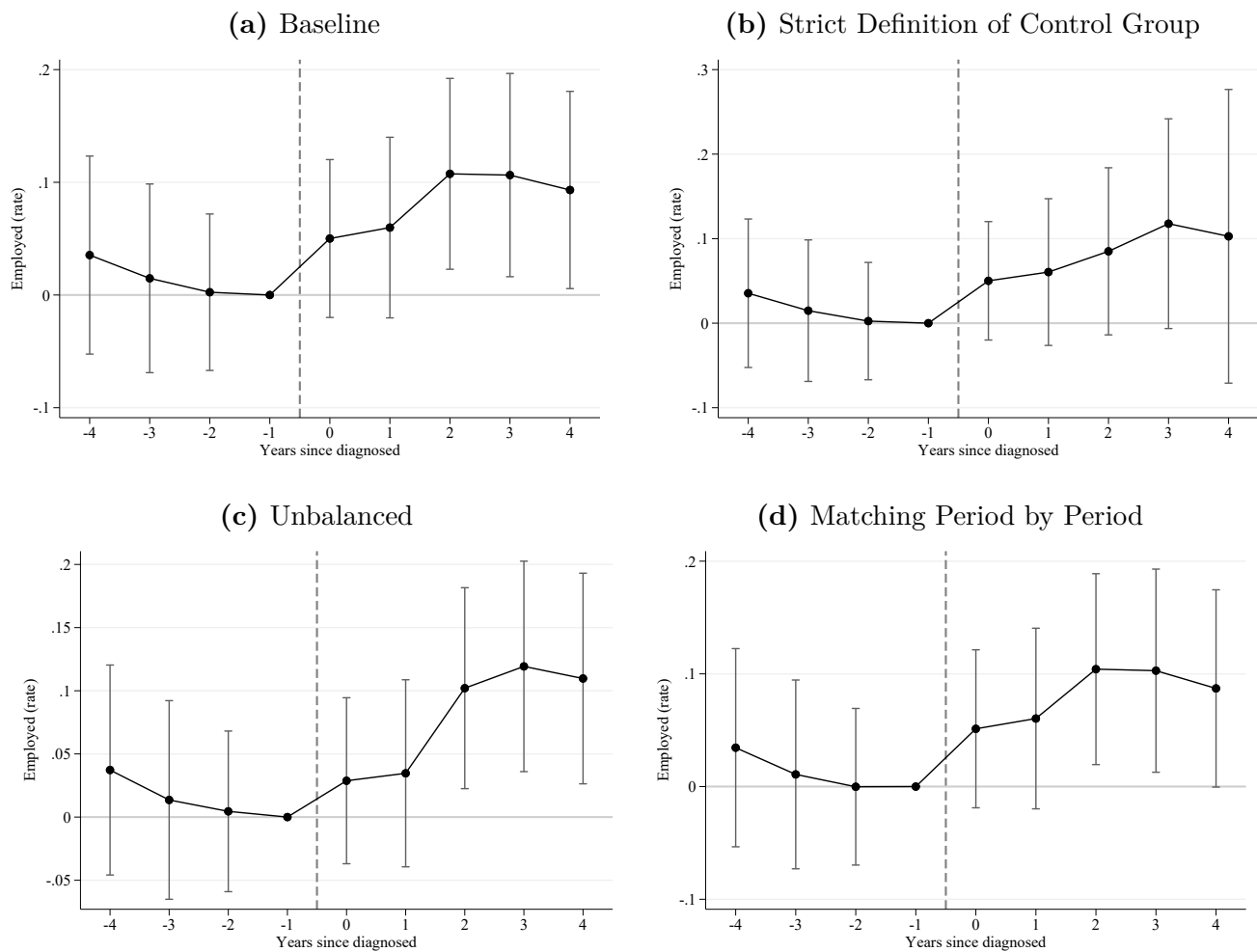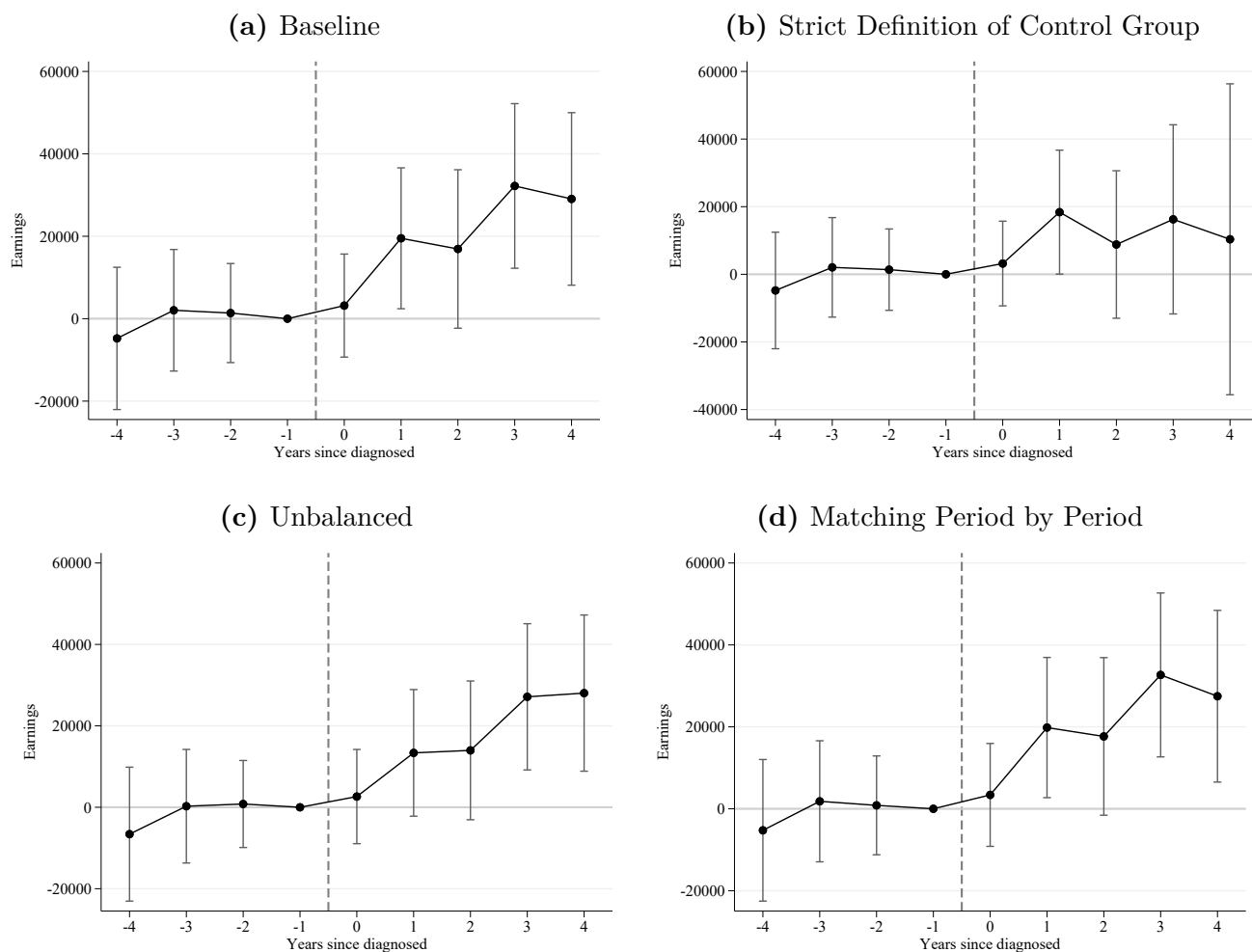

(d) Triple-Difference Estimates

Notes: These graphs plot the effects on marriage of the introduction of HAART treatment 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 1995 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 treatment. We report 95% confidence intervals calculated from clustered standard errors.

Figure A.10: Effects on Cohabitation of the introduction of HAART Treatment 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 treatment 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 1995 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 treatment. We report 95% confidence intervals calculated from clustered standard errors.

Figure A.11: 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 treatment 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 1995 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 treatment. We report 95% confidence intervals calculated from clustered standard errors.

Figure A.12: Alternative Specifications for the Effect on Employment

(a) Baseline

(b) Strict Definition of Control Group

(c) Unbalanced
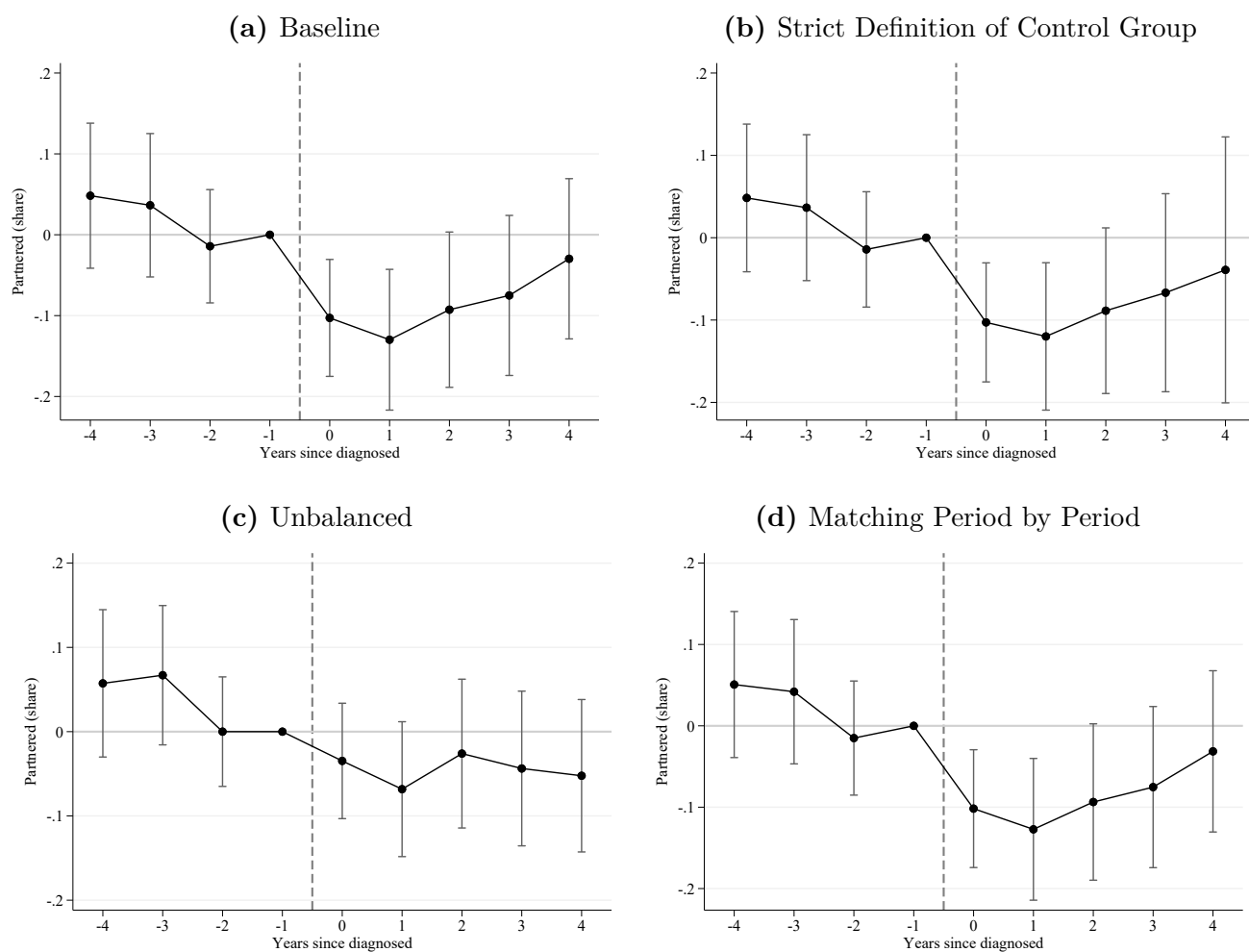

(d) Matching Period by Period

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.

Figure A.13: Alternative Specifications for the Effect on Earnings

(a) Baseline

(b) Strict Definition of Control Group

(c) Unbalanced

(d) Matching Period by Period

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.

Figure A.14: Alternative Specifications for the Effect on Bank Accounts
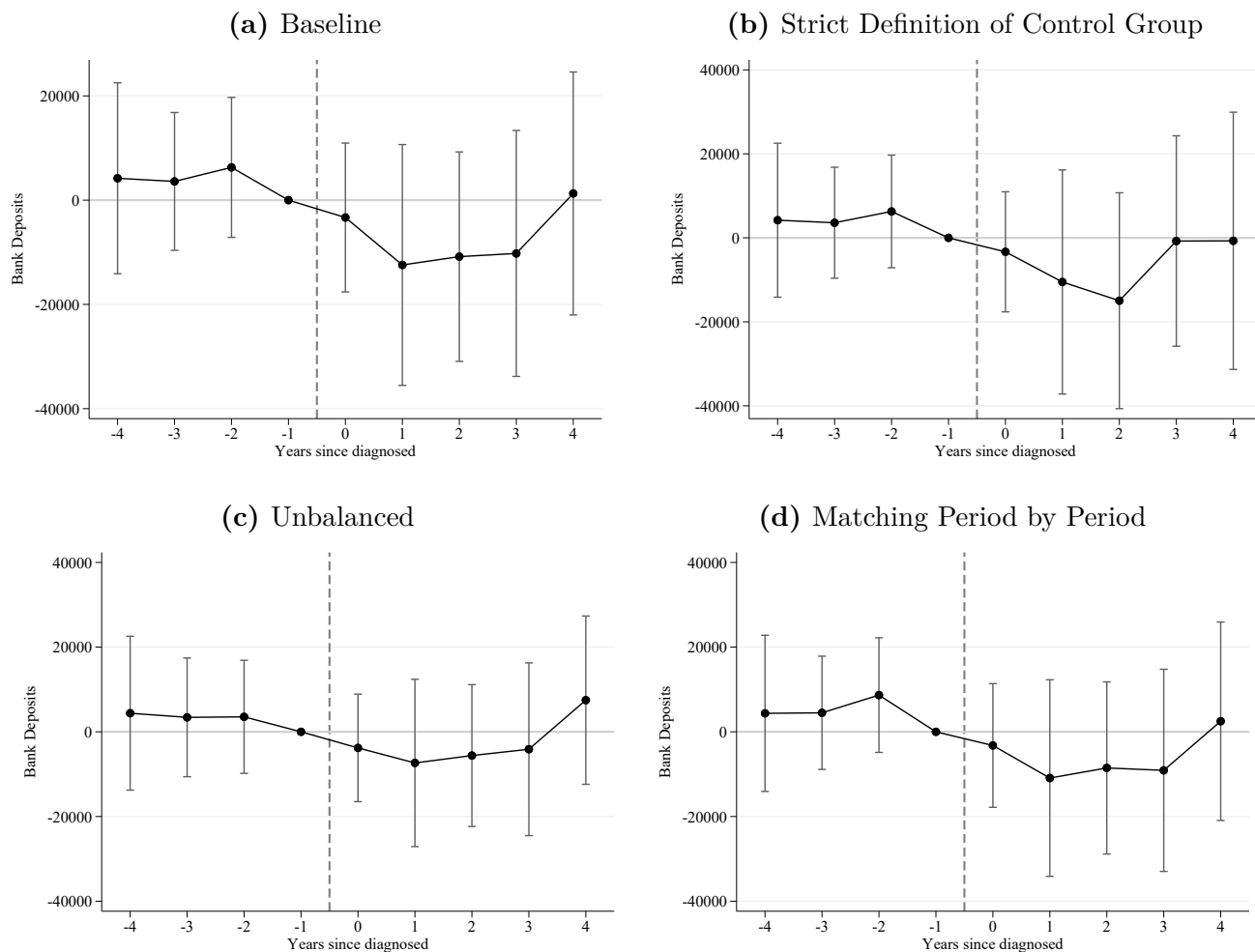
(a) Baseline

(b) Strict Definition of Control Group

(c) Unbalanced

(d) Matching Period by Period

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.

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

(a) Baseline

(b) Strict Definition of Control Group

(c) Unbalanced

(d) Matching Period by Period

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.

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