Universitat Autònoma de Barcelona

# Three Essays in Macroeconomics: Family, Health and Policy Evaluation

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Universitat Autònoma de Barcelona

Doctoral Thesis

# Three Essays in Macroeconomics: Family, Health and Policy Evaluation

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A dissertation submitted to the *Departament d'Economia i d'Història Econòmica* and the *International Doctorate in Economic Analysis* (IDEA) in partial fulfillment of the requirements for the degree of Doctor of Philosophy.

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*A la meva yaya, Blanca Yañez, que ens estimava molt.*

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# Contents

# Abstract

With this dissertation I aim to contribute to three different areas in Economics: (1) family economics, (2) health and development economics and (3) quantitative methods for policy evaluation. In this doctoral thesis I make use of quantitative structural modeling as a tool to highlight the mechanisms behind the empirical facts observed in the data.

Chapter 1 is in the field of family economics. I use information on divorce filings, (that is, whether the husband or the wife initiated the legal divorce proceedings) to identify (latent) gender specific match quality within the dyad. Using data for the United States (U.S.), I quantitatively assess the role of divorce filings in explaining divorce rates over a period of 46 years starting in 1970. To this purpose, I construct a life cycle model of endogenous marriage and unilateral divorce with endogenous labor supply and savings that jointly explains divorce filings and divorce rates over time. I use my model to measure the contribution of changes in the gender-wage gap, property division laws and child custody arrangements in explaining the observed changes in divorce rates. I find that the decrease in the gender-wage gap and the increase in the probability of getting child custody for men are major drivers behind the changes in divorce rates and in divorce filings, respectively. First, the reduction in the gender-wage gap generates two opposing effects. On the one hand, the reduction of the gender-wage gap increases the value of divorce for married women (a direct effect) and, on the other hand, unmarried women become more selective in the marriage market thus raising the quality of newly formed matches (a selection effect). Second, children increase the value of divorce for the custodial parent; so a higher probability of getting child custody raises his/her chances of filing for divorce. Third, a higher share of assets assigned to wives upon divorce can either increase or decrease divorce rates by altering the savings decision of the household. These results go hand in hand with the proposed identification of the gender specific match quality. In particular, I show that failure to match the composition of divorce filings can deliver opposite results, thus giving wrong divorce rate predictions. My results pave the road for further work studying the relationship between family dissolution and labor markets and its policy implications.

Chapter 2 is joint work with Prof.Daniela Iorio and Prof.Raül Santaeulàlia-Llopis. We provide an innovative algorithm that normalizes country-specific paths of HIV epidemic to a stylized path that tracks the course of the epidemic in terms of *stages*. This normalization uncovers heterogeneity in the *stage* of the epidemic across countries at any given point in time. We combine this heterogeneity with micro survey data to show that the relationship between education and the probability of HIV infection is

U-shaped (positive-zero-positive) over the course of the epidemic. In contrast, the relationship between education and knowledge about the process of HIV infection follows an inverted U-shaped pattern. We develop a non-stationary quantitative macroeconomic theory with heterogeneous agents that is consistent with these facts. Our theory endogeneizes the entire course of the HIV epidemic across different (aggregate) stages: a pre-HIV epidemic stage; a myopic HIV stage in which agents are not aware of the process of HIV infection; a learning stage in which agents heterogeneously—across education groups—learn about the process of infection; and an anti-retroviral (ARV) stage that modifies the effects of HIV infection on individuals. Results show that asymmetric learning is key to reproduce both the micro patterns documented and the aggregate evolution of the HIV epidemic. In further counterfactual experiments, we assess the effects of an early understanding of the virus and its mode of infection, improvements in the composition of education, the earlier (and universal) adoption of ARVs and the use of PrEP to prevent further spread.

Chapter 3 is joint with Prof.Christopher Busch, Prof.Alexander Ludwig and Prof.Raül Santaeulàlia-Llopis. We develop a novel method that identifies the effects of policy implemented nationwide—i.e. across all regions at the same time. Starting point is the insight that the dynamics of many outcome variables can be tracked over *stages*. A *stage* is defined as the location of a regional outcome on a reference outcome path. Our method proceeds in two steps. First, we conduct a normalization that maps the time-path of regional outcomes onto a reference outcome path using only pre-policy data. After normalization, the pre-policy outcome paths mapped onto the reference region are identical across regions which implies that the normalization controls for pre-policy regional heterogeneity (the so-called "parallel trends") without taking a stand on its source, (un)observability or (non)constancy. Since regions can differ by *stage* at any point in time, the normalization uncovers variation in the *stage* at the time of policy implementation—even in instances where the implementation occurs at the same time across regions. Second, we use this *stage* variation at the time of policy implementation for a clean identification of the nationwide policy effect: a *stage*-leading region delivers the counterfactual path of the outcome variable after policy. Since the non-leading regions react to policy, our identification of policy effects is not subject to the Lucas critique. Our identification assumption is that the normalization conducted using pre-policy data holds post policy. We validate our method with a set of Monte-Carlo experiments that include unobserved heterogeneity. We show several applications including public health stay-home policies (i.e. the national lockdown against Covid-19 in Spain), the effects of the pill (i.e. the FDA nationwide approval of oral contraceptives in 1960 in the U.S.) on women's career choice and fertility; and growth policy (e.g. German Reunification).

# Chapter 1

# Kramer vs. Kramer, On the Importance of Children and Divorce Filings for Understanding Divorce Rates in the U.S.

## 1.1 Introduction

Divorce rates and the composition of divorce filings[1] in the United States (U.S.) have changed substantially since the 1970s. While the family economics literature has studied divorce rates and their relation with the major labor market changes happening in the U.S. since the 70s, it has done so without using information on divorce filings. Using actual divorce filing data for the U.S., I quantitatively assess the role of divorce filings in explaining divorce rates over a period of 46 years. In particular, I use the information of divorce filings to identify gender specific match quality within a couple. Moreover, I find that matching divorce filing data moments is relevant for accurately quantifying the effects of labor market changes on the structure of the family.

I document that around 70%[2] of the divorces were initiated by wives in the early 70s, since then this number experienced a large decline, reaching 56% in 2015. What's more, divorce filing data exhibits important heterogeneity across education groups and between couples who had or not children. In 1970, 75% of all divorces were initiated by wives when the couple had children, 83% when a college educated woman was married to a non-college man and 56% when non-college women were married to college men.[3] At the same time, divorce rates sharply increased

[1] Alleging to whether it was the wife or the husband the one who initiated the legal divorce proceedings in court. Evidence shows that there is a high correlation between the record of who files for divorce and the person who actually "wanted more" the divorce, Allen and Brinig (2000), Sayer et al. (2011), Rosenfeld (2018).

[2] This number is based on divorce filing records for a sample of 31 states, refer to Section 1.2 for a more detailed discussion.

[3] I classify an individual as "college" if he/she reported to have 16 years of education or more. Refer to the data Section 1.2 for a more detailed description of the data used in this paper.

since 1960 until they reached a peak in 1981 and thereafter continuously declined, thus exhibiting a hump shape pattern, see Figure 1.1.[4]

The purpose of this paper is to understand the above described patterns of divorce rates and divorce filings, in a world with higher assortative mating, lower fertility, rising female wages, more custodial fathers and changing property division laws. The analysis aims to study the complete evolution of divorce rates and divorce filings starting in 1970 until 2015, thus encompassing 46 years of divorce data.[5] I emphasize the importance of explaining divorce rates together with divorce filings, since abstracting from the latter may lead to different conclusions about the response of divorce rates to changes in labor market outcomes. I construct and estimate a life cycle model of endogenous marriage and unilateral divorce, where agents differ in age, gender, marital status, and education attainment. Married agents make joint decisions on consumption, savings and labor supply. If agents are single they randomly meet unmarried people of the opposite gender and decide whether to marry or not. In order for the marriage to happen, both people need to agree to marry, in this sense this will be an equilibrium match. Once married, spouses can choose to divorce unilaterally. Utility is non transferable, this implies that if one spouse would rather divorce but the other prefers marriage, then he or she cannot convince the divorcing spouse to stay married. Fertility is exogenous and children bring additional utility to their parents, however upon divorce only one of the parents gets full custody of the children with a given exogenous probability, under this setting, the odds of child custody will alter the expected value of divorce for the custodial parent (more often the mother)[6] In my model men and women enjoy married life differently (i.e marital quality/love is gender specific). This a specific feature that I use to match divorce filing moments. This is not a new feature to the literature, a similar way to model love/match quality can be seen in Rios-Rull et al. (2010), however they do not explicitly target divorce filing moments in their estimation, which in contrast is the purpose of my study.

There are three main mechanism in my model occurring through: (1) the gender-wage gap in the labor market, (2) child custody arrangements upon divorce and (3) property (assets) division upon divorce: First, in terms of the labor market, rising female wages affect martial outcomes in two opposite directions in the model, a direct and selection effect respectively. On the one hand, a reduction of the gender-wage gap increases the value of divorce for married women, this makes it easier for women to divorce and leave inconvenient marriage arrangements, thus increasing divorce rates and the number of divorces filed by women (direct effect). On the other hand, a

[4]The hump shape patters is also present by state and by education of the couple members.

[5]To the best of my knowledge there is no study that quantitatively explains the hump shape pattern of divorce rates using a unifying framework. This study also fills this gap.

[6]In recent years, joint custody became more common, in my model I do not allow for joint custody arrangements, however joint custody would entail that both parents share the children, as opposed to only having one custodial parent. See González-Val and Marcén (2012) for a discussion on the implications of joint custody for divorce.

reduction of the gender-wage gap increases the value of singlehood for unmarried single women making them more selective in the marriage market (selection effect), this means that potential candidates that she would have married with the previous income arrangement might get rejected under the current arrangement. In other words, women can now afford to wait longer in the marriage market with the intention of securing a better match for themselves. This stronger selection mechanism translates into a delay in marriage and a lower number of marriages being formed. These new marriages exhibit higher average match quality for women, thus reducing their willingness to divorce and lowering the share of divorces initiated by wives. It is the case, the direct effect dominates between 1970 and 1980, however from 1980 on wards the selection effect was stronger thus explaining the hump shape in divorce rates.

Second, in terms of children and their custodial arrangements upon divorce, I model children as a public good that brings additional utility to the couple; thus making marriage more attractive for both parties. Upon divorce, one of the couple members is randomly chosen to become the custodial parent, moreover, only the custodial parent enjoys the additional utility coming from the child. Under this set up, children increase the value of divorce for the custodial parent, thus raising his/her chances of filing for divorce. In my model, I inform the odds of custody by gender as I find in the data. In particular, in the latest decades the probability of fathers in getting full custody of their children has increased, therefore increasing the number of divorces initiated by husbands, which is in line with the observed reduction of the share of divorce filings done by wives.

Third, in terms of property (asset) division upon divorce, in my model assets are divided according to an exogenous splitting rule. The implications of changes in the splitting rule on divorce rates are ambiguous. On the one hand, higher share of assets for wives increases the value of divorce for women thus increasing the number of divorces by wives, on the other hand, lower share of assets for men reduce the value of divorce for men, which decreases the number of divorces by men. The way assets are split upon divorce also alters the savings decision of married households, which in turn also affects the value of divorce for both partners. From 1970 to 1985 reforms were aimed to give a larger share of assets to wives, this created a more convenient situation for divorcing wives, which is in line with the increase in divorce rates between that period.

In order to assess the role of each of these mechanisms in explaining divorce filings and divorce rates, the model is set to match a set of relevant data moments of 1970.[7] I take the 1970's economy as the status quo, I then use the model to get a counterfactual prediction for 1985. To do this I set (exogenously) the education composition of the population, fertility, the gender-wage gap, child custody arrangements and property division rules to their 1985 values. The model predicted values are then compared with their data counterparts, I repeat the same

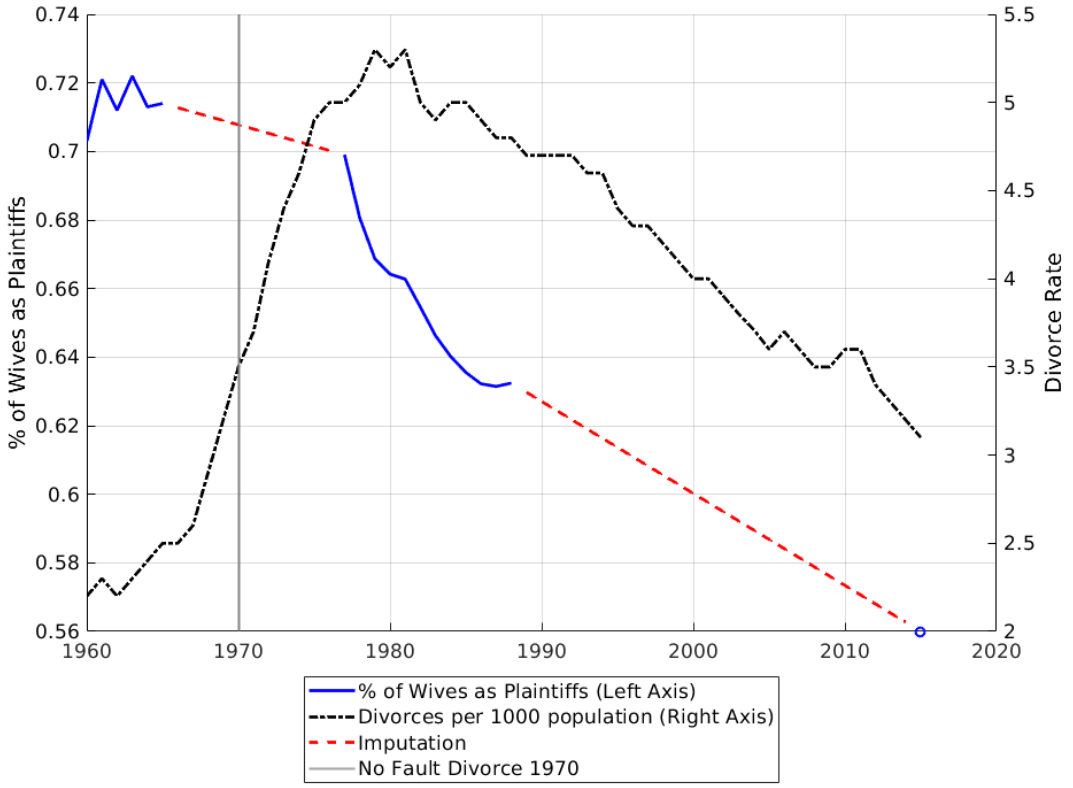
[7] Importantly, I target divorce rates and divorce filings by education of the couple.

exercise for the period 1985 to 2015. The estimation of the model for 1970 reveals that women enjoy married life less than men and that children bring additional utility to the household they live in. This explains why, conditional on the presence of children, women file more for divorce than men. Furthermore, we can see that the model predictions for 1985 are in line with the changes observed in the data. The model accounts for 41% of the rise in divorce rates and 53% of the decline in divorce filings from 1970 to 1985, and 50% of the decline in divorce rates and 52% of the decline in divorce filings between 1985 and 2015.

Next, I decompose the above predictions by measuring the individual contribution of each of the three drivers: the gender-wage gap, property division laws and child custody arrangements. To this end, I keep the 1985 parametrization fixed but set the gender-wage gap to its 1970 value. I do the same for the odds of child custody and the property division splitting rule. I repeat the same set of exercises for the period 1985 to 2015. These counterfactual experiments show that the reduction of the gender-wage gap can explain large percentages of the changes seen in both time intervals. Rising relative wages account for 91% of the increase in divorce rates (direct effect) and 29% of the decline in divorce filings between 1970 to 1985. And 62% of the decrease in divorce rates (selection effect) and 17% of the overall decline in divorce filings by women between 1985 to 2015. Results show that the change in child custody arrangements is the most important driver behind the reduction in the share of divorce initiated by wives, it accounts for 77% of the decline between 1970 and 1985 and 83% variation between 1985 and 2015. Changes in property division explain 50% of the rise in divorce rates from 1970 to 1985.

I then turn to analyse the relevance of using divorce filing information in getting the above results and show that failing to match divorce filing moments leads to opposite counterfactual results. To do this, I propose an alternative model that abstracts from the features that would allow it to match divorce filing moments; namely gender specific match quality and higher probability for the wife to get custody of the children. In the alternative model both men and women enjoy marriage equally and both have the same chance of becoming the custodial parent in case of divorce. When estimating the alternative model for 1970 I target divorce rates, but no longer target the share of divorces initiated by wives. First, the estimation of the alternative model for 1970 has no problem in replicating the divorce rate of 1970 however, the model fails to get the share of divorces initiated by wives, predicting this to be 48% as opposed to 71%, as observed in the data for 1970. Second, I then use the alternative model to obtain a counterfactual prediction for 1985. In this case the model predicts a decline in divorce rates and an increase of the divorce filings by wives; results that are contrary to what is observed in the data. This occurs because in the alternative model most of the divorces come from husbands (as opposed to wives as it is the data); therefore an increase in female wages will make women more economically attractive to men, since men can now work less and their wives more, thus reducing the number of divorces

Figure 1.1: Divorce filings and divorce rates




Notes: Divorce filing data comes from the NBER collection of Marriage and Divorce, refer to NBER (1995). Divorce rates are taken from the CDC/NCHS National Vital Statistics System reports, refer to Section 1.2 for more details.

initiated by husbands.

**Relation to the literature** This paper speaks to the family economics literature that studies the links between marital structures and people's economic choices i.e labor market participation, wealth accumulation, investment in education and fertility among others (Greenwood et al. (2017), Yamaguchi et al. (2014)). See a comprehensive summary of the state of the art concerning the economics of the family in Doepke and Tertilt (2016). However, to the best of my knowledge this paper is the first to quantitatively asses the role of divorce filings in explaining divorce rates using actual divorce filing data. I build on the work done by Rios-Rull et al. (2010), where the authors measure the contribution of changing wages on the share of single female households and other demographic facts. My framework is similar to theirs in that I also allow

the utility of married individuals to differ by gender; which has the potential to generate gender asymmetries in divorce filings, however the authors do not explicitly target divorce filing moments, as I do. In addition, their model presents a similar selection mechanism where rising female wages induces single women to wait for a better match and makes men more willing to marry since the earning power of their potential partners increased. My work can be seen as an extension to their framework by adding wealth accumulation and child custody arrangements, and a simplification by removing endogenous education attainment/sorting and fertility. The framework I present here is also closely related to Santos and Weiss (2014), where they focus on the effect of the rise in income volatility on the delay and decline of first-marriages. In their model, marriage entails consumption commitments that affect the gains from marriage. Increased volatility leads to agents waiting longer to get a high income draw before getting married, this in order to be able to meet the consumption commitments that come with marriage. In their analysis they abstract from modeling divorce decisions, therefore abstracting from modeling divorce filings. For further work focusing on changes in the wage structure and marriage and intrahousehold decisions refer too Goussé et al. (2017) and Ciscato (2018). Santos and Weiss (2015) links the decline of divorce to the rise of income volatility. Higher volatility leads to less divorce because married couples value spousal insurance more. This channel reduces divorce risk associated to negative income shocks. Their model extends the framework in Santos and Weiss (2014) by including unilateral divorce and bargaining over consumption, labor supply and savings decisions within the household. Their model explains both the decline in divorce rates and the rise in elderly divorce, however information on divorce filings is not reported. Greenwood et al. (2016) present an economy with declining marriage, increasing divorce and rising assortative mating. Within that context they explain how the changing wage structure and the reduction in the price of durables affect marital composition, education attainment and female labor force participation and how all of these factors jointly determine income inequality. The authors explain how, in the presence of better technology at home, the economies of scale from marriage are lower, thus reducing the incentives to marry and promoting divorce. In contrast to Greenwood et al. (2016), I focus on explaining both the rise and fall of divorce rates overtime, together with the decline in the share of divorces initiated by wives. I do not model home production, but at first glance a reduction in the price of durables would reduce the value of marriage for married men more than for women, therefore contributing to the decline in filings done by wives this would be a useful extension to my model.

My paper is also related to Guvenen and Rendall (2015), who explore the role of education as insurance against bad marriages. They build on the fact that future returns on human capital are not divided upon divorce, this makes education a good insurance against divorce risk. In their model women endogenously respond to the change from mutual consent divorce to unilateral

divorce by increasing their college enrollment. In my model I take the education composition of the population as given, but this does not hinder college educated wives from being more prone to end bad marriages than their non-college counterparts. Knowles (2005) uses a model of marital bargaining to explain the trends in U.S. labour supply since 1970. The author shows that the standard model without bargaining predicts a large decline in married-male labor supply in response to the reduction of the female to male gender-wage gap since 1970. The author emphasizes that although bargaining has a small impact on aggregate labor supply it is critical in explaining the trends in female labor supply observed in the data. In contrast to my framework, the author explicitly models intra-household bargaining, divides time use within the household in three categories, leisure, work in the office and work at home, finally he includes income taxes into the analysis. However, the author abstracts from modeling wealth accumulation, fertility and child custody arrangements plus does not explicitly target divorce filing moments. Regarding bargaining, I too propose a version of my model where the intra-household allocation weights are set through Nash Bargaining. After estimating my model for 1970, the obtained endogenous the pareto weights were similar to the ones found by Knowles (2005), importantly I found that there was little change in the endogenous pareto weights when generating model predictions for 1985. Following this insight, I keep the pareto weights constant throughout my analysis.

My work is also related to the literature addressing changes in divorce laws and child custody settlements. Fernández and Wong (2017) study the welfare effects of switching from mutual consent divorce to unilateral divorce. They use a model with endogenous family formation, where children stay with the mother upon divorce. They calibrate their model to match moments of the 1940 cohort, and conclude that women were better off under the mutual consent regime vs. unilateral divorce. They model love as a public good but explore the possibility of allowing love draws to differ across partners, however they assume a common distribution of love across partners. I relax this assumption by allowing the mean of the love distribution to differ by gender, furthermore I discipline these means to match divorce filing data by education and conditional on the presence of children. In Fernández and Wong (2017) the welfare losses from divorce are larger for women than for men, this happens because women earn less than men and bear a larger share of child rearing costs, thus more likely to benefit from a mutual consent regime. Voena (2015), studies the effects of switching from mutual consent divorce to unilateral divorce in states with a title based regime vs. states with community property or an equitable distribution regime.[8] She finds that the introduction of unilateral divorce in states with equal division of property, resulted in higher savings and lower female labor force participation. She estimates a model featuring limited commitment to marriage, imperfectly transferable utility between spouses, and remarriage. In my model, utility is non-transferable and I abstract from the possibility of remarriage.

[8]For the definition of title based regime, community property and equitable distribution refer to Footnote **??**.

Shephard (2019) presents a limited commitment overlapping generations framework with both within and across cohort marital matching. The model is able to explain why men marry women younger than themselves and why labor supply of married women is lower the older is her husband. Furthermore the author quantifies the effect of decline in the gender-wage gap on the marriage age gap; he finds that one third of the reduction of marital age gap is associated to the reduction of relative wages. Marcassa (2013) measures the effects of changes in financial settlements, namely changes in property division, alimony transfers and child custody arrangements and time assignments on the rise of divorce rates in the U.S. since late 1960's. To do so she calibrates a model of wealth accumulation, labor supply and limited commitment to marriage. She finds that financial settlement can account for 30% of the rise in divorce rates. Moreover, she finds that after accounting for all financial settlements, the role of switching from mutual consent to unilateral divorce is significantly smaller. In later counterfactual experiments she finds that changes in the wage structure explain 15% of the increase in divorce rates. In her model agents solely divorce due to marital quality shocks, save for retirement and to mitigate divorce risk. Arpad and Sarolta (2015) study how divorce and asset division rules affect intra-household risk sharing. In a model with lack of commitment and efficient separations calibrated for the UK, they compute optimal property division upon divorce. They find that optimal property division rules balance the trade-off between risk sharing within the couple, and consumption smoothing across marital states (married vs. divorced). In a static setup Weiss and Willis (1985) study endogenous child custody settlements. In their model expenditure on children is a public good enjoyed by both the father and the mother; upon divorce the non-custodial parent looses control over the child expenditure allocations. Under this set up optimal marriage contracts are constructed where couples decide the allocations within marriage and child custody settlements upon divorce. If one party is committed to provide the other with a high level of utility, then he may be better off assigning custody as well, thus benefiting from the high level of child expenditures the other party will choose. I would like to stress that none of the above mentioned research papers focus on matching who files for divorce and its role in explaining divorce behaviour, aspect that is the backbone of my analysis.

Regarding the literature studying divorce filings and its determinants, most work is empirical and comes from sociology and law. Empirically, I contribute to this literature by documenting the steady decline in the share of divorce filings initiated by wives since 1970. I show that this decline is not purely a composition effect by showing that divorce filings by wives decline both by education group of the couple and by the presence of children in the household, see Figure 1.2. Quantitatively, I estimate a structural model to measure and decompose the effects of the main driving forces behind changes in divorce rates and divorce filings by women. Friedman and Percival (1976) make a complete historical analysis on the evolution of divorce and divorce filings

in the U.S. since 1870 until the late 1970's. Allen and Brinig (2000) show empirically that filing behaviour depends on the spouse's relative power within the marriage, financial independence and anticipation of child custody, the latter being the most important determinant. Allen and Brinig (2000) were concerned by the fact that most (if not all) couples experience a separation time until the final divorce decision takes place, therefore the divorce filing record might not necessarily reflect the intention of the filing member to divorce when the separation period is long (larger than two years). They find evidence that within a period of two years between separation and divorce, the divorce filing does not appear to be simply a matter of convenience. Following this insight I control for the time form separation to divorce and only use divorce records where the time from separation to divorce did not exceed six years. Then the median time from separation to divorce used to produce the statistics in this paper is two years. Dixon and Weitzman (1982) show that husbands who filed for divorce also revealed a strong preference for becoming the custodial parent. Kalmijn and Poortman (2006) emphasize that child custody affects the husband's decision to divorce more than for it does for wives. Fox and Kelly (1995) study the determinants of child custody arrangements upon divorce. They find that the odds of father custody were enhanced when the children where older but reduced by parental unemployment and prior child support arrangements. Sayer et al. (2011) show that female employment made women more likely to leave the marriage. Additionally they found that men's unemployment affects equally the probability of either partner to file for divorce. Gunter and Johnson (1978) finds evidence that the passage of no-fault divorce was partially responsible for an increase in male divorce filings, suggesting that cultural and societal changes had a secondary role. Finally, Rosenfeld (2018) document that women tend to report lower marital quality than husbands, which is in line with the estimation results coming from my model.

The remainder of the paper is organized as follows. Section 1.2 presents the data used in this paper. Section 1.3 describes a simple model of divorce filings, and explains its mechanism. Section 1.4 describes the fully fledged model to be estimated. Section 1.5 explains the calibration and estimation procedure. Section 1.6 and 1.7 describe the main results and counterfactual experiments. Finally, Section 1.8 concludes.

## 1.2 Data

This paper collects information from various data sets, these data sets are: The Panel Study of Income Dynamics (PSID), Current Population Survey (CPS), divorce filing data from the National Vital Statistics System complemented with survey data coming from the HCMST project, see Rosenfeld (2018). Otherwise stated the numbers reported/used in this paper come from data extracts from the Current Population Survey that were accessed through IPUMS. Moreover, the

relevant statistics were computed for couples where the husband was between 21 and 60 years old. In addition, I classify an individual as "college" if he/she reported to have 16 years of education or more. I now describe in detail the relevant variables and their sources.

**Divorce filing data** This paper uses data on divorce filing records that were directly collected from divorce certificates available for 31 states across the U.S. from 1968 to 1995, see the complete list of sample states in Appendix A.2. This data set was compiled by the National Vital Statistics System of the National Center for Health Statistics, the data set can be downloaded from the NBER collection of Marriage and Divorce Data, refer to NBER (1995). The collection of this data stopped in 1995 due to lack of funds. Unfortunately data for all states was not available, however the information available for the 31 sample states can be very well used to conduct inference at the national level. In Table 1.1, I show that the divorce rates at the national level and those computed using the sample of 31 states are not so different from one another.

The data set consists of more than 3 million observations and includes the relevant variables necessary for my analysis: level of education of the husband and wife, number or children under 18, marriage duration, time from separation to divorce, age, race, and state of residency. Detailed information on the plaintiff (the person who filed for divorce) is only available from 1977 to 1988, therefore the average share of divorces initiated by wives for 1970 was imputed by interpolating between the value for 1960 (reported by Friedman and Percival (1976)), which was 72% and the value for 1977. The respective numbers by education of the couple and filings in the presence of children were imputed accordingly. In addition, following Allen and Brinig (2000) I control for the time form separation to divorce and only use records where the time from separation to divorce did not exceed six years, this makes the median time form separation to divorce to be two years in the sample I use.

The value for the share of divorce initiated by wives in 2015 comes from survey data collected for the project How Couples Meet and Stay Together, see Rosenfeld (2018). This data set consists of 3,009 records of married individuals and their partners. Follow-up surveys were conducted one and two years after the original wave. After classifying divorces by the education of the couple, the surviving number of observations was to low to produce reliable statistics. Because of this reason I do not analyze divorce filings by education of the couple for the period 1981 to 2015.

**Divorce rates** Divorce rates were computed using PSID data. First, married heads in the PSID were paired with their respective spouses, thus creating a data set at the couple level. I then follow these couples across years, recording if the couple changed its marital status from married to divorced from one year to the next. I then compute the divorce rate as the relative number of

couples who switched marital status from married to divorced between survey dates.[9] Separated couples are not included in the analysis. Between 1968 and 1997, PSID interviews were conducted annually. Since then, interviews have been biennial, thus the calculation for the divorce rate for 2015 has been adapted accordingly.

For the quantitative exercise I use divorce rates at the national level, instead of divorce rates computed for the sample of 31 states for which divorce filing data is available. Table 1.1 shows that the divorce rates at the national level and those computed using the sample of 31 states are not so different from one another, thus justifying the use of divorce rates at the national level.

Table 1.1: Divorce rates (in %) for all states vs. sample states

| | Year | | | | | |
|---|---|---|---|---|---|---|
| | 1970 | 1975 | 1980 | 1985 | 1990 | 2015 |
| All States | 1.30 | 1.42 | 1.85 | 1.87 | 1.60 | 1.30 |
| Sample States* | 1.29 | 1.38 | 1.75 | 1.93 | 1.68 | 1.22 |

*For the complete list of sample states refer to List A.2. Source: PSID

Furthermore, Figure A.1 in Appendix A.2 shows the divorce rate trends computed from the CDC National Vital Statistics Reports. The figure conveys the same message as Table 1.1; it shows that the divorce rates trends from the sample states are close to the ones at the national level. Additionally it shows the divorce rate trends for selected states. Oklahoma and Arizona which are the two states with the two highest divorce rates 1970. Mississippi and Ohio with divorce rates closest to the median for the U.S. Finally, New Jersey and New York with the lowest divorce rates in 1970.

**Fertility** Data on fertility was collected from the OECD reports on fertility by mother's age at childbirth. The OECD collected data for the U.S. from 1960 until the present. Table 1.5 shows the evolution of fertility rates by age group of the mother for 1970, 1985 and 2015 .

[9] Alternatively, the crude divorce rate can be computed as the number of divorces per 1000 population.

Table 1.2: Fertility: Births per 1000 women, U.S.

| | Age Group | | | | | | |
|---|---|---|---|---|---|---|---|
| Year | 15-19 | 20-24 | 25-29 | 30-34 | 35-39 | 40-44 | 45-49 |
| 1970 | 68.3 | 167.8 | 145.1 | 73.3 | 31.7 | 8.1 | 0.5 |
| 1985 | 51.0 | 108.3 | 111.0 | 69.1 | 24.0 | 4.0 | 0.2 |
| 2015 | 22.3 | 76.8 | 104.3 | 101.5 | 51.8 | 11.0 | 0.8 |

Notes: Source, OECD Stats, Fertility rates by mother's age at childbirth, five-year age groups, 1960-2019

**Labor market variables** The gender-wage gap, female labor force participation and the average weekly number of hours worked by women were calculated from the Current Population Survey using CPS-IPUMS extracts. I use the sample for all married couples where the husband is between 21 ant 60 years of age. As with the PSID, I first pair up husbands with their respective wives thus creating a sample at the couple level, the CPS provides an easy way of linking spouse records. The CPS provides information on labor earnings per year, thus hourly salaries were computed by dividing the yearly labor earnings by the number of hours worked reported that year. I then proceed to calculate life cycle trends; different trends were computed by education (college, non-college) and gender, these trends were later smoothed using a quadratic fit on age: $\mho_{g,e}(age) = \alpha_0 + \alpha_1\, age + \alpha_2\, age^2$.

The CPS reports the employment status of the household members. It also reports the average number of hours worked per week if the respondent was active in the labor force. Using this information it is straight forward to calculate the female labor force participation and the female to male hours worked ratio $h_f/h_m$.

**Assortative mating** The education composition of the population was calculated from the Current Population Survey. This involves computing the relative weight of the respective education groups: (1) College educated women married to college educated men, (2) college educated women married to non-college men, (3) non-college women married to college men, finally (4) non-college women married to non-college men. Table 1.3 shows the respective shares for 1970 and 1980. The degree of assortiative mating between those years increased, the Pearson's correlation coefficient between the education of the wife and the husband in 1970 was 0.47 and 0.52 in 1985. For a more detailed analysis on the rise of assortative mating see Greenwood et al. (2016), Greenwood et al. (2017) and Liu (2020).

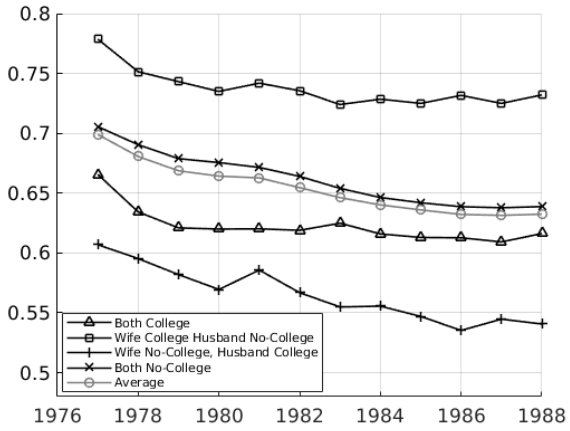
**Trends in divorce and divorce filings** Between 1970 and 1985 the percentage of divorces initiated by wives in the U.S. declined from 70% to 63% (a 9.7% reduction) and from 63% to 56% (a 11.1% decline) from 1985 to 2015. The crude divorce rate increased, from 3.5% to 5.3% (increase of 51%) between 1970 and 1980, reached a peak in 1981 and declined ever since. By 2015 they had reached levels close to the ones seen in the early 70's. Figure 1.1 summarizes these trends.

The declining trend in divorce filings by wives is also present when looking at cuts by education of the couple members and in the presence of children, see Figure 1.2. We observe that the decline is larger for non-college women married to college men (a 10.9% decline), whereas smaller for college women married to non-college men (a 5.9% decline). Note that at every point in time college educated women married to non-college educated men filed more, having a cross-year average of 73% and non-college women married to college men filed the least, 56%. The other two groups were in between, with 62% of women filing when both had a college education and 66% when none of the partners where college educated.

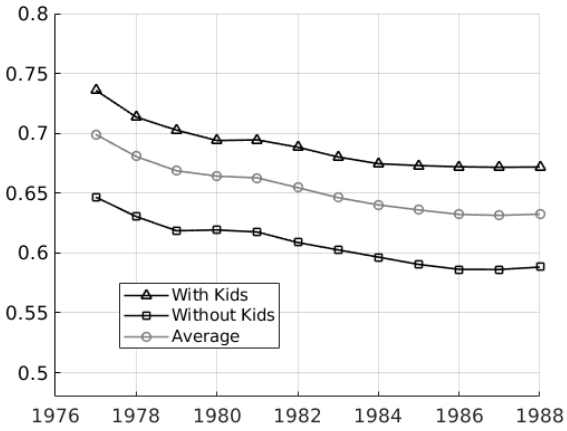
When looking at divorce filings in the presence of children we can see that kids are an important factor behind women's divorce filing behavior. In 1977, 74% of all divorces where initiated by women when the couple had children and 64% in the absence of children. Moreover, the declining trend is present for both groups, the reduction has been larger in magnitude for those couples who didn't have children a 9.7% reduction (from 64% to 58%) and a slightly smaller decline for couples with children, a 8.6% reduction (from 74% to 67%).

Figure 1.2: Percentage of divorces initiated by wives by education of the couple and presence of children

(a) By education




(b) By presence of Children




Source: NBER collection of Marriage and Divorce, refer to NBER (1995).

Table 1.3 summarizes information about divorce filings, divorce rates, gender-wage gap, assortative mating and female labor supply by education groups for 1970 and 1985. We can see that average relative wages $\bar{w}_f/\bar{w}_m$ are positively correlated with the percentage of divorces initiated by wives; the Pearson's correlation coefficient is $0.7$ between the two variables. This suggest a strong connection between wives filing behaviour and their income. We see that for 1970, the wage gap of college women married to non-college men is the lowest, with such women earning 69% their husbands wage; for non-college women married to college men the gap is the largest, with 37%. The gender-wage gap of college women married to college men was 55% and for non-college women married to non-college men was 47%. On the contrary the divorce rate for college women married to college men is the lowest, and the divorce rate of college women married to non-college men is the largest.

Table 1.3: Divorce rates, divorce filings and labor market variables by education, U.S. 1970, 1985

| | Education of the couple | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | Both College | | Wife College Husband Not | | Husband College Wife Not | | None College | |
| | 1970 | 1985 | 1970 | 1985 | 1970 | 1985 | 1970 | 1985 |
| Divorce rate (%) | 0.85 | 0.92 | 1.87 | 2.06 | 1.37 | 1.60 | 1.27 | 1.99 |
| % of divorces initiated by wives | 66.56 | 61.29 | 77.87 | 72.51 | 60.70 | 54.74 | 70.56 | 64.18 |
| gender-wage gap $\bar{w}_f/\bar{w}_m$ | 0.55 | 0.60 | 0.69 | 0.74 | 0.37 | 0.44 | 0.47 | .54 |
| Working hours ratio $h_f/h_m$ | 0.72 | 0.8 | 0.85 | 0.83 | 0.69 | 0.74 | 0.78 | .82 |
| Married female labor force participation (%) | 47.44 | 67.42 | 61.26 | 71.73 | 31.85 | 54.12 | 40.72 | 54.04 |
| Sample shares (%) | 8.10 | 14.87 | 4.73 | 7.34 | 8.69 | 9.68 | 78.48 | 67.97 |

Sources: Divorce filing data is from the NBER collection of Marriage and Divorce, refer to NBER (1995). Divorce rates from PSID. gender-wage gap was computed form CPS-IPUMS extract.

## 1.3 A Simple Model of Divorce Filings

The purpose of this section is to present a simple two period model of divorce filings to better understand the main mechanisms of interest: (1) relative wages (gender wage gap) (2) probability of child custody (3) property division rules. This simple model paves the road for the fully fledged quantitative model developed in Section 1.4.

Consider a simple economy where agents live only for two periods and are indexed by their gender $g \in \{m, f\}$ and marital status $\omega \in \{\mathcal{M}, \mathcal{D}\}$, married and divorced respectively. Individuals are born married and with children. Agents can only be married to an individual of the opposite sex, therefore a family is conformed by a husband, a wife and children. All families are born with zero assets $a_{t=1} = 0$. As a simplification, children are modeled as a public good that bring additional utility $\eta > 0$ to each parent in the household; children do not make any decisions. Each couple member is born with a given gender specific match quality $q_g$ that is randomly drawn from the gender specific distribution $\mathcal{N}(\alpha_g, \sigma_{\epsilon_\alpha})$. At every point in time agents make decisions over consumption $c$ and the number of hours to work $h$ at a given stochastic gender specific wage rate $w_g$. log wages follow a Markov process with persistence $\rho$. Compute the average female to male gender-wage gap as $\bar{w}_f/\bar{w}_m$. Additionally, every period married agents decide unilaterally whether or not to get divorced. This means that although all agents are born married they can choose to divorce in the first period, furthermore a couple that decided to stay together in the

first period can choose to split in the second period. In period two, individual match quality is redrawn from the same distribution, but only for married couples; there is no remarriage. Match quality takes the value of zero $q_g = 0$ if divorced. Upon divorce one of the parents keeps the children with probability $\nu_g$, such that $\nu_m + \nu_f = 1$, this means that the non custodial parent looses any utility gains $\eta$ he/she used to get from the children, whilst the custodial parent keeps them. Importantly, in period one, all agents (married or not) must also choose how much to save for the second period $a'$. Upon divorce, assets are split according to the asset splitting rule $0 \leq \kappa_g \leq 1$, such that $\kappa_m + \kappa_f = 1$, there are no additional costs to divorce. Following the above description the period utility for an individual is $u(c_g, h_g) + q_g(\omega) + \mathbf{1}_{k=1}\eta$, where $\mathbf{1}_{k=1}$ is an indicator variable that takes the value of $1$ in case the individual lives with the child, zero otherwise. I assume that agents are risk averse and dislike working.

**The problem of married couples** Married couples maximize the weighted sum of each member's individual utility according to the pareto weights $\mu_g$, [10], subject to the budget constraint, $c_m + c_f + a' = w_m h_m + w_g h_f$ in period one and $c_m + c_f = w_m h_m + w_g h_f + a'(1 + r)$ in period two, where $r$ is the risk free interest rate. We can see from the borrowing constraint that agents pool income and make joint consumption and savings decisions. Denote the value of marriage for an agent of gender $g$ at period $t$ as $V_g^{\mathcal{M}}(t)$, where $V_g^{\mathcal{M}}(t)$ is an equilibrium object coming from the maximization problem of the married household.

**The problem of the divorced** A divorced individual maximizes his/her own utility, subject to $c_g + a' = w_g h_g$ in period one, $c_g = w_g h_g + a'(1 + r)$ in period two if divorced in period one and $c_g = w_g h_g + a'(1 + r)\kappa_g$ in period two if divorced in period two. Denote the value of divorce for an agent with gender $g$ at period $t$ as $V_g^{\mathcal{D}}(t, k)$, where $k$ takes the value 1 if parent $g$ is the custodial parent. Denote $\mathbb{E}$ as the expectation operator over the probability of getting custody of the child upon divorce.

**The divorce decision** Divorce occurs at any period $t$ if either $\mathbb{E}(V_m^{\mathcal{D}}(t)) > V_m^{\mathcal{M}}(t)$ or $\mathbb{E}(V_f^{\mathcal{D}}(t)) > V_f^{\mathcal{M}}(t)$, or both, thus divorce is unilateral when either the husband or wife is better off in the divorce state and divorce is mutual when both partners are at the same time better off divorced. Under this set up it is straight forward to compute the number of divorces that were initiated by wives, husbands or both.

In the next subsection I parametrize, solve and simulate the above described two period model. Subsequently, I provide an explanation of the relevant mechanisms of interest.

[10] Such that the value for a couple is: $V_t^{\mathcal{C}}(w, w^*, a, q_m, q_f, k) = \max_{c_m, c_f, h_m, h_f, a'} \mu^f u(c_f, h_f, q_f) + \mu^m u(c_m, h_m, q_m) + \eta$ subject to the period specific budget constraint.

### 1.3.1 Mechanisms

In this section I solve the two period model and conduct a comparative statics exercise to better understand the response of divorce rates and the share of divorces initiated by women when varying respectively, (1) the gender-wage gap $\bar{w}_f/\bar{w}_m$, (2) the asset sharing rule upon divorce $\kappa_g$ and (3) child custody arrangements $\nu_g$. Figures 1.3 and 1.4 present the results from the comparative statics exercise. 1.4 illustrates the response of divorce rates and divorce filings when varying the unobservable gender specific match quality $Ł_m, Ł_f$.
I propose four alternative parametrizations of the model aimed to shed light on the importance of the odds of child custody and the role of the gender specific match quality component:

1. **Reference model:** Represents a gender equality scenario in which there are no gender differences in the model. This scenario sets $\bar{w}_f/\bar{w}_m = 1, \kappa_m = 0.5, \nu_m = 0.5, \psi_m = \psi_f, \mu_g = 0.5, Ł_m = Ł_f = 4$. For the rest of the parameters see Appendix A.1.

2. **Alternative model 1:** Sets somewhat more realistic gender differences, as found in the literature, $\bar{w}_f/\bar{w}_m = 0.55, \kappa_m = 0.6, \psi_m < \psi_f, \mu_m = 0.6$ but keeps the mean of the individual match quality $Ł_g$ and the probability of child custody $\nu_g$, symmetric between men and women $Ł_m = Ł_f$ and $\nu_m = \nu_f$.

3. **Alternative model 2:** Keeps the parameter values of Alternative model 1, but sets $Ł_m > Ł_f$, explicitly $Ł_m = 4$ and $Ł_f = 3$, that is men an women enjoy married life differently. With women having lower match quality than men.

4. **Alternative model 3:** Keeps the parameter values of Alternative model 2, but sets $\nu_m = 0.1$, that is women have a higher probability of getting custody of the children upon divorce.

The divorce rates in panels (a), (c) and (e) of Figure 1.3 and panels (a) and (c) of Figure 1.4 are normalized to the values of the reference/equality model that is $\kappa_m = 0.5, \nu_m = 0.5, Ł_m = 4$ respectively, except for panel (a) in Figure 1.3, that is normalized to a gender-wage gap of $\bar{w}_f/\bar{w}_m = 0.5$ for illustration purposes.

**Discussion on the effect of relative wages** The most interesting result comes from Figure 1.3 panel (a) and (b). In these figures we see that on the reference/equality model, the relationship between the gender-wage gap and divorce rates is negative, and its relationship with the share of divorces initiated by wives is positive. However we can see, that the maximum share of divorces by wives that can be reached under this set up is just $50\%$, by setting $\bar{w}_f/\bar{w}_m = 1$. This changes when we move to Model 1. On one hand, by adding gender differences such as: the way

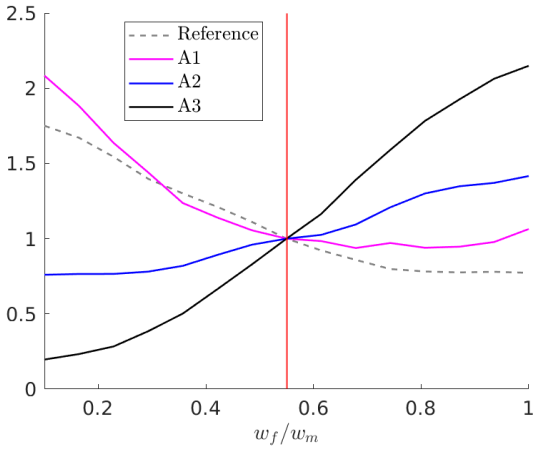
consumption is split within the household, asset splitting rules and making women dislike work more than men, we see that the slope of the pink) line in panel (b) becomes steeper. On the other hand we observe that the relationship between the gender-wage gap and divorce rates takes a U shape, reaching a minimum right around a gender-wage gap of $\bar{w}_f/\bar{w}_m = 0.8$, which happens to be the value where the share of divorce initiated by wives surpasses $50\%$. For Model 2, the slope of the blue line in panel (b) becomes even steeper and the relationship between divorce rates and the gender-wage gap becomes positive. This becomes more evident when looking at Model 3, that makes women more prone to get custody of the children. Moreover, panel (a) and (b) illustrate that only the Models 2 and 3 are capable or reaching a share of divorce filings by wives that we observe in the data (around $70\%$). Note that Models 2 and 3 imply different responses of aggregate divorce rates to changes in the gender-wage gap, than Models 1 and 2, which do not reach a higher level of divorce filings by women. This aspect is very important at the moment of quantifying the individual effects of changes in the gender-wage gap, more so in the presence of heterogeneity in divorce filings and wages across education groups. For example for college women married to non-college men the share of divorces initiated by wives is higher and the gender-wage gap lower, which would put this group above $\bar{w}_f/\bar{w}_m = 0.5$ in panels (a) and (b), but for non-college women married to college men, the gender-wage gap will be around $0.5$ and the share of divorces initiated by wives lower, position this group of people at the middle. We can see that higher relative wages increases divorce rates, this happens because higher wages increases the value of divorce for women relative to marriage, then already married women who were stuck in inconvenient marriage arrangements can now afford to divorce (a direct effect).

In this model is also possible to look at the relationship between unobservable individual match qualities, divorce rates and divorce filings by women. These shown in Figure 1.4 and are straight forward: higher match quality either for men or women, lower divorce rates and lower share of divorces initiated by husbands and wives respectively. However we are interested in the interaction between the realized match quality and higher wages for women. This relationship is more involved and results in a selection effect. Consider that the higher the value of single-hood for women associated to higher wages, the longer they will wait for a higher match quality. In the long run, this will result in the new matches having a higher average match quality than before (a selection effect), thus exhibiting a lower aggregate level of divorce coming from women. In this way, women become more selective in the marriage market which translates into an increasing average age at first marriage and contributing in building a larger pool of never married individuals. On the contrary, higher wages for women would make men eager to get married even if this means settling for a lower match quality. It is in this two ways (direct effect and selection effect) that higher relative wages contribute to the rise and later decline in divorce rates and the overall decline in the share of divorces initiated by wives.

Figure 1.3: Comparative Statics, Observables

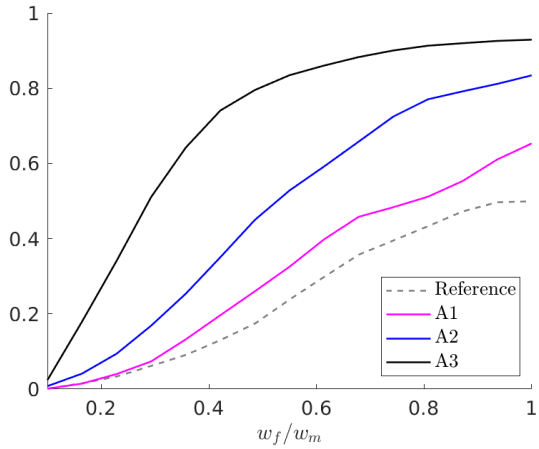
Divorce Rate

(a) Relative Wages $\bar{w}_f/\bar{w}_m$

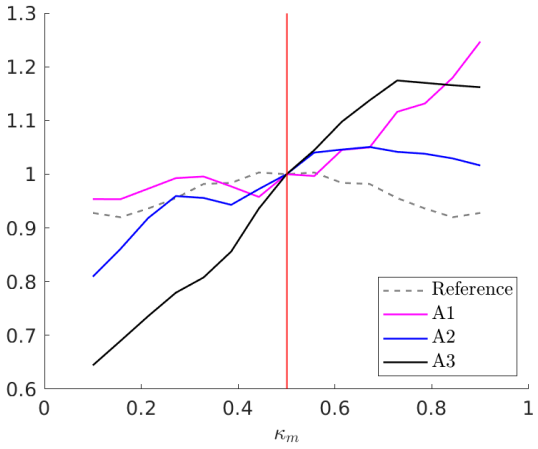



Share of divorces initiated by wives (%)

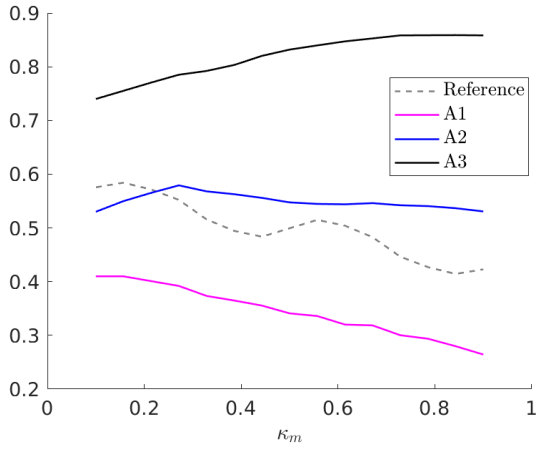
(b) Relative Wages $\bar{w}_f/\bar{w}_m$



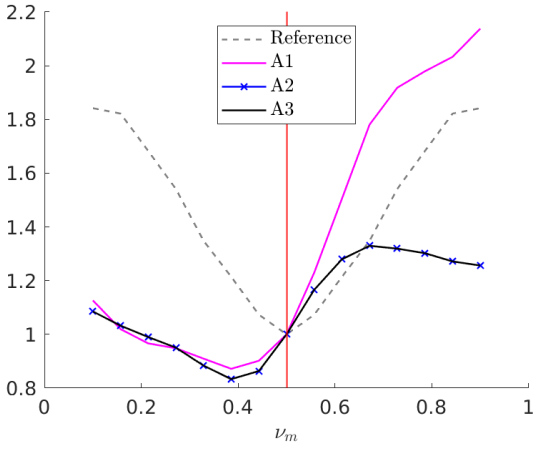

(c) Asset Sharing Rule $\kappa_m$



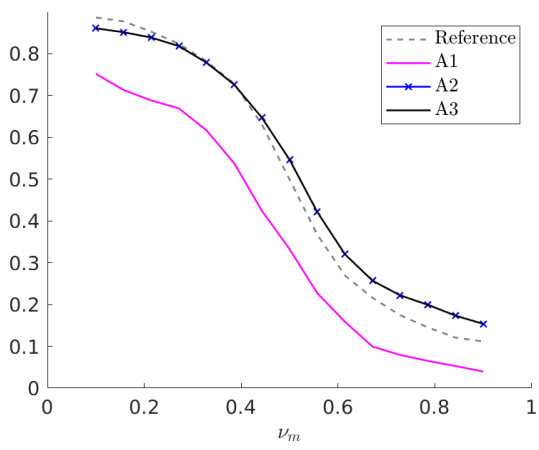

(d) Asset Sharing Rule $\kappa_m$




(e) Child Custody $\nu_m$




(f) Child Custody $\nu_m$

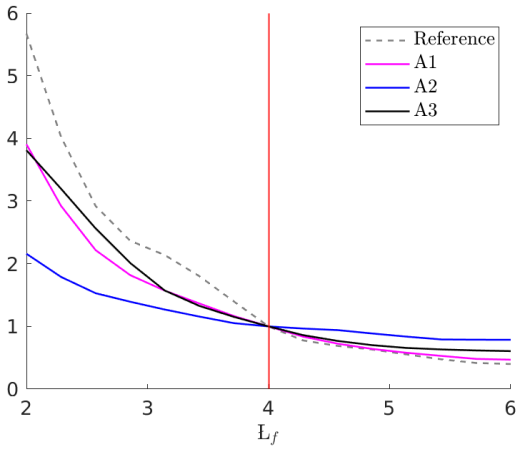



Notes: The reference line solves the model with $\bar{w}_f/\bar{w}_m = 1, \kappa_m = 0.5, \nu_m = 0.5, \phi_m = \phi_f, \mu_m = 0.5$, and $Ł_m = Ł_f = 4$ The Model 1 uses $\bar{w}_f/\bar{w}_m = 0.55, \kappa_m = 0.6, \nu_m = 0.5, \phi_m < \phi_f, \mu_m = 0.67$ and $Ł_m = Ł_f = 4$. Model 2 sets $Ł_f = 3$ and $Ł_m = 4$ in addition to Model 1. Model 3 sets $\nu_m = 0.1$ on top of Model 2. See Appendix A.1 for the values of the rest of the parameters used in this example.

Figure 1.4: Comparative Statics, Unobservables

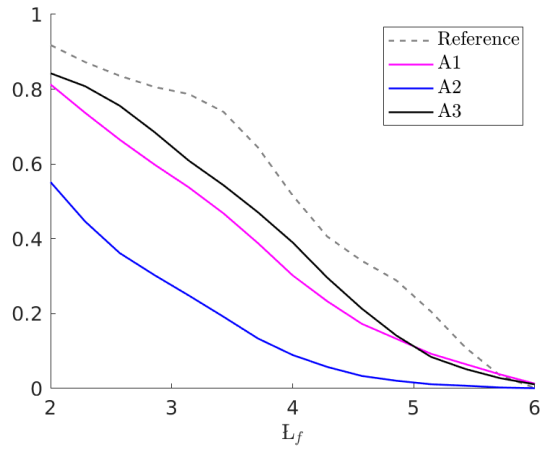
Divorce Rate

(a) Wife's Match Quality Ł$_f$

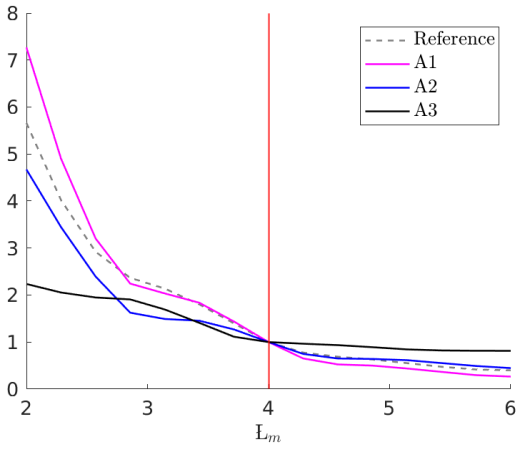



Share of divorces initiated by wives (%)

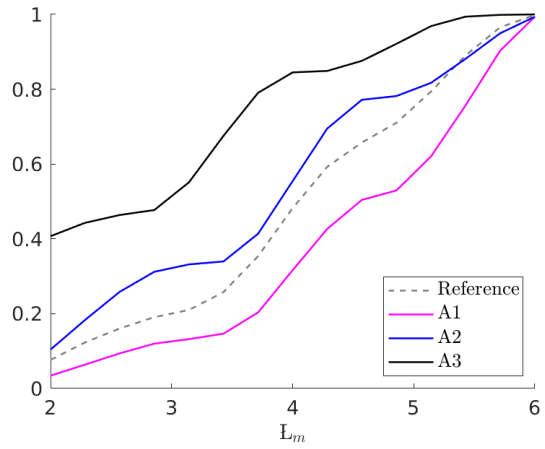
(b) Wife's Match Quality Ł$_f$




(c) Husbands's Match Quality Ł$_m$




(d) Husbands's Match Quality Ł$_m$




Notes: The reference line solves the model with $\bar{w}_f/\bar{w}_m = 1, \kappa_m = 0.5, \nu_m = 0.5, \phi_m = \phi_f, \mu_m = 0.5$, and Ł$_m$ = Ł$_f$ = 4. Model 1 uses $\bar{w}_f/\bar{w}_m = 0.55, \kappa_m = 0.6, \nu_m = 0.5, \phi_m < \phi_f, \mu_m = 0.67$ and Ł$_m$ = Ł$_f$ = 4. Model 2 sets Ł$_f$ = 3 and Ł$_m$ = 4 in addition to Model 1. Alternative 3 sets $nu_m = 0.1$ on top of Model 2. See Appendix A.1 for the values of the rest of the parameters used in this example.

## 1.4 The Model

In this section I present a life cycle model featuring endogenous marriage, unilateral divorce, wealth accumulation and female labor supply, both at the intensive and extensive margins. I start by describing the demographics, namely living arrangements, fertility and the education composition composition of the population. Next I describe child custody arrangements and property division upon divorce. I then describe the income process together with its life cycle component. I next turn to preferences, and the timing that governs decision making and shapes the law of motion of the population. Finally, I provide a definition for the stationary equilibrium and outline the steps of a solution algorithm.

### 1.4.1 Demographics

The economy is populated by generations of equal number of men and women, who at every point in their life are indexed by their age/generation $t$, gender $g \in \{m, f\}$, education attainment $e \in \{c, nc\}$ college, non-college respectively, marital status $\omega \in \{\mathcal{NM}, \mathcal{M}, \mathcal{D}\}$, never married, married and divorced respectively.[11] Additionally, married couples are indexed by the presence of children or not in the household $k \in \{1, 0\}$.
Men and women are born single and enter the model at age $t_m = 21$ and $t_f = 19$ respectively. Agents age deterministically and live a total of $39$ periods, that is, men live until age $T_m = 60$ and women up to $T_f = 58$. A difference of 2 years between men and women has been chosen since men tend to marry women who are on average two years younger than them. In the model a generation $t$ consists of all men aged $t_m$ and women aged $t_m - 2$. For the rest of the document, otherwise stated, $t$ will refer to the age of the man and his associated generation. .

**Living arrangements** Every period, single and married agents decide over their marital status. Single agents decide whether or not to get married to another single individual of the opposite sex but from the same generation and education group. For simplicity I restrict marriage to happen only between individuals from the same generation and education group, that is, there is no intergenerational marriage. Married individuals decide whether to divorce their current partner or remain married for the rest of the period, after divorce there is no possibility of remarriage, therefore divorce is an absorbing state. Notice that since agents are born single, divorce cannot happen at $t = 21$.

**Education** For simplicity, the education composition and the degree of assortative mating across education groups will be exogenously fed into the model. The education composition of

[11] For the rest of the document the term "never married" and "single(s)" will be used interchangeably

the married population is summarised in contingency Table 1.4, where the shares $\phi_{e,e^*}$ will be directly taken from the data.

Table 1.4: Education composition of the married population

| | | Husbands's Education | |
|---|---|---|---|
| | | College | Non College |
| Wife's Education | College | $\phi_{c,c}$ | $\phi_{c,nc}$ |
| | Non College | $\phi_{nc,c}$ | $\phi_{nc,nc}$ |

Such that: $\phi_{c,c} + \phi_{nc,c} + \phi_{c,nc} + \phi_{nc,nc} = 1$

The population in the model is then divided into four groups: $G1(c,c)$: composed of college men and college women, $G2(nc,c)$: non-college men and college women, $G3(c,nc)$: college men and non-college women, $G4(nc,nc)$: non-college men and women. Under this set up agents are restricted to interact solely with agents of their respective group, for example, a college woman of group $G1(c,c)$ can only meet and marry a college man of the same group and generation, but cannot marry a non-college man of group $G2(nc,c)$, only college women of group $G2(nc,c)$ can marry with non-college men.

**Fertility** Fertility is exogenous; children can be born within a dyad with probability $\varphi_t$, conditional on the couple not having children. There are no children born out of the wedlock and upon divorce the child stays with one of the parents. Children stick around with the couple or the custodial parent forever, see below.

### 1.4.2 Child Custody and Divorce Laws

Upon divorce one of the parents gets full custody of the child with exogenous probability $\nu_g$, such that $\nu_m + \nu_f = 1$, where $\nu_m$ is the probability that the father gets full custody. The custodial parent inherits all the costs and benefits of raising the child.

Divorce causes the couple's savings $(a_t)$ to be split among couple members according to parameter $\kappa_g$. Where $\kappa_m$ is the share of the assets that the man keeps. Moreover, divorce has no additional cost therefore $\kappa_m + \kappa_f = 1$.

### 1.4.3 Income

Agents provide labor supply $h$ and receive wages $w_t^{g,e}$ that depend on gender and education. Wages are stochastic around a deterministic trend $\mho_t$ and assumed to follow an AR(1) process

with persistence $\rho$ and transition matrix $\Pi$:

$$log(w_t^{g,e}) = \mho_{t,g,e} + F_t \qquad \text{where: } F_t = \rho\, F_{t-1} + \epsilon_w \qquad \text{with: } \epsilon_w \sim \mathcal{N}(0, \sigma^2_{\epsilon_w})$$

Define $\delta = \bar{w}_f/\bar{w}_m$ as the average gender-wage gap and $z = \bar{w}_c/\bar{w}_{nc}$ as the average college wage premium. Note that the income process does not depend on the agents marital status and so the income processes between spouses are independent of each other.
Markets are incomplete and households can save/borrow assets $a$, at a risk-free interest rate $r$. There are no borrowing constraints, no intergenerational transmission of assets and agents must pay all their debt before they die, that is $a_{T+1} \geq 0$.

### 1.4.4 Preferences

Agents are risk averse, enjoy consumption and dislike working. When married they get additional individual utility $q_g$ associated to the current match. Individual match quality $q_g$ has a permanent component $\alpha_g$ that is drawn from distribution $\mathcal{N}(Ł_g, \sigma^2_{\epsilon_\alpha})$ at the time the couple met, and a stochastic component $\epsilon_q$, marital bliss, that follows an i.i.d process distributed $\mathcal{N}(0, \sigma^2_{\epsilon_q})$. Moreover, if the couple has children $k = 1$, each couple member enjoys extra utility $\eta$. In addition, there is a fixed utility cost $f$ to the period utility of married individuals when female hours worked are strictly positive $h_f > 0$. This cost is not present for neither single nor divorced women. Considering the above described features, individual period utility is given by:

$$u_g(c,h,q) + \mathbf{1}_{k=1}\eta + \mathbf{1}_{h_f>0}\, f(\omega) = \frac{c^{1-\sigma}}{1-\sigma} - \psi_g \frac{h^{1+\frac{1}{\xi}}}{1+\frac{1}{\xi}} + q_g(\omega) + \mathbf{1}_{k=1}\eta + \mathbf{1}_{h_f>0}\, f(\omega)$$

$$q_g(\omega) = \begin{cases} \alpha_g + \epsilon_q \quad \epsilon_q \sim \mathcal{N}(0, \sigma^2_{\epsilon_q}) & \text{if } \omega = \{\mathcal{M}\} \\ 0 & \text{if } \omega = \{\mathcal{NM}, \mathcal{D}\} \end{cases}$$

$$f(\omega) = \begin{cases} 0 & \text{if } \omega = \{\mathcal{NM}, \mathcal{D}\} \\ < 0 & \text{if } \omega = \{\mathcal{M}\} \end{cases}$$

- Where $\alpha_g$ is drawn at the time of marriage from distribution $\mathcal{N}(Ł_g, \sigma^2_{\epsilon_\alpha})$.
- $\psi_g$ governs the disutility from labor supply.
- $\xi$ is the Frish elasticity of labor supply.
- Denote $A_q$ the transition matrix associated to $q$, and $B_k$ the transition matrix governing

fertility.[12]

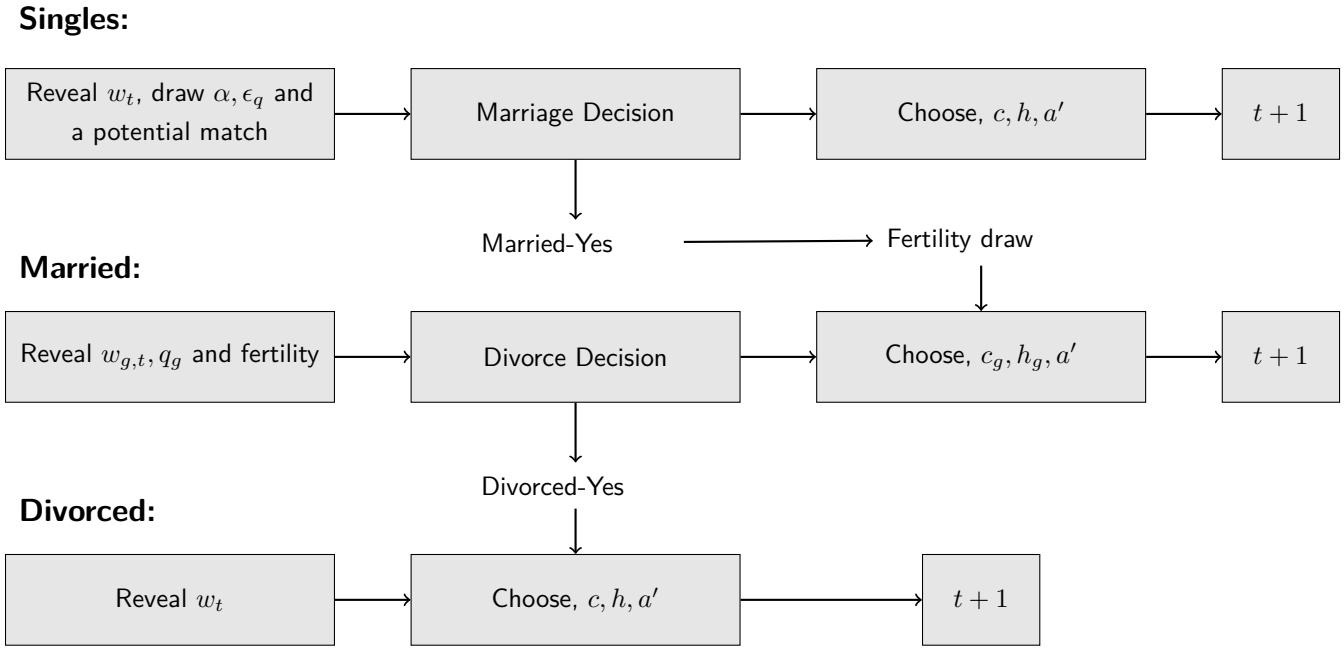
- People discount their future at a rate $\beta$.

We are now ready to describe the timing and decision making process.

### 1.4.5 Timing

The sequence of events occurring on a given period is summarized in Figure 1.5. At the beginning of the period income and marital shocks are revealed; single agents draw a random partner from the pool of singles ($\mathcal{NM}$), get a permanent match quality draw from $\mathcal{N}(Ł_g, \sigma^2_{\epsilon_\alpha})$ and a temporary marital bliss shock $\epsilon_q$. Married couples get a temporary bliss shock and a fertility shock if they didn't have children already.
Next, married couples decide whether to get divorced from their current partner, and the never married/single agents decide to marry or not their match. After the marriage decision has taken place, fertility is revealed for the newly wedded couple. The marriage decision is taken before fertility is revealed in order to avoid selection into marriage associated to the presence of children. Divorce is an absorbing state, there is no possibility of remarriage. Finally, consumption, work and savings decisions are executed.

Figure 1.5: Timing




[12] $A_q$ is an $N_A \times N_A$ matrix where $N_A$ is the number of discretized states of $q \in Q$, the elements of each row of $A_q$ equal $1/N_A$, such that they sum to one. $B_k$ is a $2 \times 2$ matrix with $b_{1,1} = 1 - \varphi(t), b_{1,2} = \varphi(t), b_{2,1} = 0$ and $b_{2,2} = 1$, where $\varphi_t$ is the fertility of women of generation $t$.

### 1.4.6 Decision Making

**Singles** Single agents must decide how much to consume $c$, save $a$ and whether to marry of not the person they randomly meet at the beginning of the period. Single agents solve the following dynamic problem:

$$Vg_t^{\mathcal{NM}}(w,a) = \max_{c,h,a'} \quad u(c,h,0) + \beta \int_{\Omega^{g*}_{\mathcal{NM}}} \sum_{w'} \sum_{q'} \sum_{k'} \pi_{w'|w} A_{q'|q} B_{k'|k} \tag{1.1}$$

$$\times \left\{ \mathscr{M}_{t+1} Vg_{t+1}^{\mathcal{M}}(w', w^{*\prime}, a', q', q^{*\prime}, k') + (1 - \mathscr{M}_{t+1}) Vg_{t+1}^{\mathcal{NM}}(w', a') \right\} d\Omega^{g*}_{\mathcal{NM}}(t+1)$$

s.t.

$$c + a' = w_t^{g,e} h + (1+r)a \tag{1.2}$$

- Where $\mathscr{M}(t)$ is the marriage decision policy at period $t$, takes value of one if the 'potential' couple decided to marry, zero otherwise, see expression 1.3.
- $\Omega^{g*}_{\mathcal{NM}}(t)$ is the normalized distribution of singles of the opposite sex at period $t$.
- Define the policy functions of the singles problem as follows: consumption policy $\mathcal{P}g_c^{\mathcal{NM}}(w,a,t)$, savings policy $\mathcal{P}g_a^{\mathcal{NM}}(w,a,t)$ and labor supply policy $\mathcal{P}g_h^{\mathcal{NM}}(w,a,t)$.

Marriage, occurs only if both parties agree, that is:

$$\mathscr{M}_t = \begin{cases} 1 & \text{only if } \mathbb{E}_\varphi(Vm_t^{\mathcal{M}}) > Vm_t^{\mathcal{NM}} \text{ and } \mathbb{E}_\varphi(Vf_t^{\mathcal{M}}) > Vf_t^{\mathcal{NM}} \\ 0 & otherwise \end{cases} \tag{1.3}$$

- Where $\mathbb{E}_\varphi(Vm_t^{\mathcal{M}})$ is the expected value of marriage before the arrival of children, see 1.6.

Note that in order to solve the above problem single agents must be completely aware of the distribution of singles in $t+1$.

**Married** Married couples jointly decide how much to consume $c_m, c_f$, how much the wife works $h_f > 0$ or if she doesn't work at all $h_f = 0$, labor supply of the husband $h_m$ and how much to save $a'$. The couple maximizes the weighted sum of each members utility according to pareto weights $\mu_m + \mu_f = 1$.[13] Married individuals decide unilateraly whether they separate from their current partners. Because the pareto weights are fixed there is no mechanism by which a partner

[13] Consistent with the unitary framework of the household as described in Chiappori and Donni (2009)

convinces the other to stay in case of divorce. A married household solves the following dynamic problem:

$$V_t^{\mathcal{C}}(w_m, w_f, a, q_m, q_f, k) = \max_{c_m, c_f, h_m, h_f, a'} \eta \mathbf{1}_{k=1} + \mu^f u(c_f, h_f, q_f) + \mu^m u(c_m, h_m, q_m) + f\mathbf{1}_{h_f>0} \tag{1.4}$$

$$\begin{aligned} &+ \beta \sum_{w'_g} \sum_{q'_g} \sum_{k'} \pi_{w'_m|w_m} \pi_{w'_f|w_f} \; A_{q'_m|q_m} \; A_{q'_f|q_f} \; B_{k'|k} \\ &\times \Big\{ \mathscr{D}_{t+1} \left[ \mu^f \mathbb{E}_\nu \left( V f_{t+1}^{\mathcal{D}}(w'_f, a'\kappa_f, k') \right) + \mu^m \mathbb{E}_\nu \left( V m_{t+1}^{\mathcal{D}}(w'_m, a'\kappa_m, k') \right) \right] \\ &+ (1 - \mathscr{D}_{t+1}) V_{t+1}^{\mathcal{C}}(w'_g, a', q'_g, k') \Big\} \end{aligned}$$

s.t.

$$c_m + c_f + a' = w_t^{m,e} h_m + w_t^{f,e^*} h_f + (1+r)a \tag{1.5}$$

- Where $\mathscr{D}(t)$ is the divorce policy at $t$, taking the value one if the couple divorced and zero otherwise.
- The operator $\mathbb{E}_\nu$ is the expectation over custody of the child. The custodial parent gets custody of the child with probability $\nu_g$, such that $\nu_m + \nu_f = 1$.
- $\kappa_m$ is the share of assets kept by the husband after divorce. The wife keeps $\kappa_f = 1 - \kappa_m$.
- Define the policy functions of the married individuals as follows: consumption policy $\mathcal{P}g_c^{\mathcal{M}}(w, w^*, a, t)$, savings policy $\mathcal{P}g_a^{\mathcal{M}}(w, w^*, a, t)$, labor supply policy $\mathcal{P}g_h^{\mathcal{M}}(w, w^*, a, t)$, and married female labor force participation $\mathcal{P}f_f^{\mathcal{M}}(w, w^*, a, t)$.
- After solving the optimization problem described in 1.4, we can define the value of a married individual of gender $g \in \{f, m\}$ as:

$$\begin{aligned} Vg_t^{\mathcal{M}}(w_m, w_f, a', q_m, q_f, k) &= \eta \mathbf{1}_{k=1} + u(c, h, q) + f\mathbf{1}_{h_f>0} \\ &+ \beta \sum_{w'_g} \sum_{q'_g} \sum_{k'} \pi_{w'_m|w_m} \pi_{w'_f|w_f} \; A_{q'_m|q_m} \; A_{q'_f|q_f} \; B_{k'|k} \\ &\times \Big\{ \mathscr{D}_{t+1} \mathbb{E}_\nu \left( Vg_{t+1}^{\mathcal{D}}(w'_g, a'\kappa_g, k') \right) \\ &+ (1 - \mathscr{D}_{t+1}) Vg_{t+1}^{\mathcal{M}}(w'_m, w'_f, a', q'_m, q'_f, k') \Big\} \end{aligned} \tag{1.6}$$

- Define $\mathbb{E}_\varphi(Vg_t^{\mathcal{M}}) = \varphi_t * Vg_t^{\mathcal{M}}(k=1) + (1-\varphi_t) * Vg_t^{\mathcal{M}}(k=0)$, as the expected value of marriage before the arrival of children.

**Divorced** Divorced agents choose how much to consume $c$ and save $a'$. They solve the following dynamic problem:

$$Vg^{\mathcal{D}}_{t+1}(w,a,k) = \max_{c,h,a'} \quad \eta \mathbf{1}_{k=1} + u(c,h,0) + \beta \sum_{w'} \pi_{w'|w} Vg^{\mathcal{D}}_{t+1}(w',a',k') \tag{1.7}$$

s.t.

$$c + a' = w^{g,e}_t h + (1+r)a \tag{1.8}$$

- Define the policy functions of the divorced agents as follows: consumption policy $\mathcal{P}g^{\mathcal{D}}_c(w,a,t)$, savings policy $\mathcal{P}g^{\mathcal{D}}_a(w,a,t)$ and labor supply policy $\mathcal{P}g^{\mathcal{D}}_h(w,a,t)$,

Divorce occurs unilaterally, that is:

$$\mathscr{D}_t = \begin{cases} 1 & \text{if } \mathbb{E}_\nu(Vm^{\mathcal{D}}_t) > Vm^{\mathcal{M}}_t \text{ and/or } \mathbb{E}_\nu(Vf^{\mathcal{D}}_t) > Vf^{\mathcal{M}}_t \\ 0 & \text{otherwise} \end{cases} \tag{1.9}$$

- Where $\mathbb{E}_\nu(Vg^{\mathcal{D}}_t)$ is the expected value of divorce over the probability of obtaining custody of the child, that is $\mathbb{E}_\nu(Vg^{\mathcal{D}}_t) = \nu_g * Vg^{\mathcal{D}}_t(w,a,k=1) + (1-\nu_g) * Vg^{\mathcal{D}}_t(w,a,k=0)$.

### 1.4.7 Endogenous Population Dynamics

**Singles** The law of motion for single agents of gender $g$ and education group $G(e,e^*)$ is:

$$\Omega^g_{\mathcal{NM}}(w',a',t+1) = \int_{\Omega^{g^*}_{\mathcal{NM}}(w^*,a^*)} \sum_w \sum_q \sum_k \Big\{ \mathbf{1}_{a'(w,a)\in A} \pi_{w'|w} A_{q'|q} B_{k'|k} \times (1 - \mathscr{M}_t(w,w^*,a,q,q^*,k)) \Omega^g_{\mathcal{NM}}(w,a,t) \Big\} d\Omega^{g^*}_{\mathcal{NM}}(w^*,a^*,t) \tag{1.10}$$

Normalize as needed for Problem 1.1:

$$\Omega^g_{\mathcal{NM}}(y,a,t) = \Omega^g_{\mathcal{NM}}(y,a,t) \Big/ \int d\Omega^g_{\mathcal{NM}}(y,a,t)$$

**Married** The law of motion for married agents is:

$$\begin{aligned}\Omega^g_{\mathcal{M}}(w', w^{*\prime}, a', q', q^{*\prime}, k', t+1) = &\sum_{w,w^*}\sum_{q,q^*}\sum_{k}\Big\{\mathbf{1}_{a'(w,w^*,a)\in A}\pi_{w'|w}\pi_{w^{*\prime}|w^*}\ A_{q'|q}\ A_{q^{*\prime}|q^*}\ B_{k'|k} \\ &\times (1-\mathscr{D}_t(w,w^*,a,q,q^*,k))\ \Omega^g_{\mathcal{M}}(w,w^*,a,q,q^*,k,t)\Big\} \\ &+\int_{\Omega^{g^*}_{\mathcal{NM}}(w^*,a^*)}\sum_{w}\sum_{q}\sum_{k}\Big\{\mathbf{1}_{a'(w,w^*,a+a^*)\in A}\pi_{w'|w}A_{q'|q}B_{k'|k} \\ &\times \mathscr{M}_t(w,w^*,a,q,q^*,k)\ \Omega^g_{\mathcal{NM}}(w,a,t)\Big\}d\Omega^{g^*}_{\mathcal{NM}}(w^*,a^*,t)\end{aligned} \tag{1.11}$$

Normalized:

$$\Omega^g_{\mathcal{M}}(w,w^*,a,q,q^*,t) = \Omega^g_{\mathcal{M}}(w,w^*,a,q,q^*,t)\Big/\int d\Omega^g_{\mathcal{M}}(w,w^*,a,q,q^*,t)$$

**Divorced** The law of motion for divorced agents is:

$$\begin{aligned}\Omega^g_{\mathcal{D}}(w',a',k',t+1) = &\sum_{w}\Big\{\mathbf{1}_{a'(w,a)\in A}\pi_{w'|w}\ \Omega^g_{\mathcal{D}}(w,a,k,t)\Big\} \\ &+\sum_{w,w^*}\sum_{q,q^*}\sum_{k}\Big\{\mathbf{1}_{a'(w,a,\kappa_g)\in A}\pi_{w'|w}\pi_{w^{*\prime}|w^*}\ A_{q'|q}\ A_{q^{*\prime}|q^*}\ B_{k'|k} \\ &\times \mathscr{D}_t(w,w^*,a,q,q^*,k)\ \Omega^g_{\mathcal{M}}(w,w^*,a,q,q^*,k,t)\Big\}\end{aligned} \tag{1.12}$$

Normalized:

$$\Omega^g_{\mathcal{D}}(w,a,t) = \Omega^g_{\mathcal{D}}(w,a,t)\Big/\int d\Omega^g_{\mathcal{D}}(y,a,t)$$

Define the period $t$ divorce rate for education group $G(e,e^*)$ as:

$$DIV(e,e^*) = \int\Big(\mathscr{D}(t)\times\Omega^g_{\mathcal{M}}(t)\Big)\,d\Omega^g_{\mathcal{M}}(w_{g,e},w_{g,e^*},a,q,q^*,t) \tag{1.13}$$

And the aggregate divorce rate

$$\widehat{DIV} = \sum_{e}\sum_{e^*}\phi(e,e^*)DIV(e,e^*) \tag{1.14}$$

### 1.4.8 Definition for the Stationary Equilibrium

A Stationary Equilibrium is a set of value functions by gender $g$ and age $t$: for never married individuals $Vg_t^{\mathcal{NM}}(w,a)$, divorced $Vg_t^{\mathcal{D}}(w,a)$ , married households $V^{\mathcal{C}}(w,w^*,a,q_m,q_f,k)$, consumption policy functions, $\mathcal{P}g_c^{\mathcal{NM},\mathcal{D}}(w,a)$ and $\mathcal{P}_c^{\mathcal{M}}(w,w^*,a)$, policy functions for savings $\mathcal{P}g_a^{g,\mathcal{NM},\mathcal{D}}(w,a)$ and $\mathcal{P}_a^{\mathcal{M}}(w,w^*,a)$, policy functions for labor supply $\mathcal{P}g_h^{\mathcal{NM},\mathcal{D}}(w,a)$ and $\mathcal{P}_h^{\mathcal{M}}(w,w^*,a)$, labor force participation for married women $\mathcal{P}_f^{\mathcal{M}}(w,w^*,a)$, divorce and marriage policy functions $\mathscr{D},\mathscr{M}$ respectively, and stationary distributions of singles $\Omega_{\mathcal{NM}}^g$, married individuals $\Omega_{\mathcal{M}}^g$ and divorced $\Omega_{\mathcal{D}}^g$, such that:

- The policy functions $\mathcal{P}g_c^{\mathcal{NM}}(w,a),\mathcal{P}g_a^{\mathcal{NM}}(w,a),\mathcal{P}g_h^{\mathcal{NM}}(w,a)$, together with the marriage decision rule $\mathscr{M}$ and the distribution of potential partners $\Omega_{\mathcal{NM}}^g$, solve the singles problem described in 1.1.
- The policy functions $\mathcal{P}g_c^{\mathcal{M}}(w,w^*,a),\mathcal{P}g_a^{\mathcal{M}}(w,w^*,a),\mathcal{P}g_h^{\mathcal{M}}(w,w^*,a),\mathcal{P}g_f^{\mathcal{M}}(w,w^*,a)$, together with the divorce decision rule $\mathscr{D}$ solve the married household problem 1.4.
- The policy functions $\mathcal{P}g_c^{\mathcal{D}}(w,a),\mathcal{P}g_a^{\mathcal{D}}(w,a),\mathcal{P}g_h^{\mathcal{D}}(w,a)$ solve the problem for the divorced, problem 1.7.
- The Marriage policy $\mathscr{M}$ is computed according to Equation 1.3, given $Vg_t^{\mathcal{NM}}(w,a)$ and $Vg_t^{\mathcal{M}}(w,w^*,a,q_m,q_f,k)$.
- The Divorce policy $\mathscr{D}$ is computed according to Equation 1.9, given $Vg_t^{\mathcal{D}}(w,a)$ and $Vg_t^{\mathcal{M}}(w,w^*,a,q_m,q_f,k)$.
- The stationary distributions for singles $\Omega_{\mathcal{NM}}^g$, married individuals $\Omega_{\mathcal{M}}^g$ and divorced $\Omega_{\mathcal{D}}^g$ are induced by the equilibrium policy functions.

### 1.4.9 Solution Algorithm

We are interested in finding the Stationary Equilibrium described above for a given set of parameters $\Theta=\{\sigma,\xi,\mu_m,\rho,\sigma^2_{\epsilon_w},\mho_t,\varphi_t,\beta,r,\kappa_m,\nu_m,Ł_m,Ł_f,\sigma_{\epsilon_\alpha},\sigma_{\epsilon_q},\phi_m,\phi_f,\eta\}$. Since agents are fully rational, this requires single agents to know the exact distribution of potential partners $\{\Omega_{\mathcal{NM}}^g(w,a,t)\}_{t=21}^{T=60}$ at every point in their lives, this will involve making a guess of the underlying marital distribution of the population over the life cycle. The reasoning goes as follows the decision to marry today depends on the value of waiting to marry tomorrow, which in turn depends on the distribution of potential partners tomorrow, this extends until the last period of life. The problem requires solving the optimal allocations backwards starting from the last

period $T = 60$ until $t = 21$. Below I describe in detail the computational steps to solve for the equilibrium:

***Algorithm No.1*: Computation of the stationary equilibrium:**

Step 0: Provide a guess for the distribution of potential partners $\{\Omega^g_{\mathcal{NM}}(w, a, t)\}_{t=21}^{T=60}$ for every $t$.

Step 1: Compute the equilibrium policy functions and the value functions iterating backwards from the last period $T = 60$ until $t = 21$.

Step 2: With the help of the optimal policy functions, simulate forward the evolution of the population distribution from $t = 21$ to $T = 60$. Store the simulated distribution of never married individuals at every $t$, $\{\widehat{\Omega}^g_{\mathcal{NM}}(w, a, t)\}_{t=21}^{T=60}$ .

Step 3: Compare the simulated distribution of potential partners $\{\widehat{\Omega}^g_{\mathcal{NM}}(w, a, t)\}_{t=21}^{T=60}$ with the initial guess of the same object. If they are not the same update the guess and go back to Step 1.

## 1.5 Calibration Strategy

In this Section I describe the calibration strategy. The model is calibrated to the U.S. in 1970. Parameters which have direct observable data analogs were assigned its respective values, some other parameters take values that are commonly used in the literature $(\sigma, \xi, \mu_m, \rho, \sigma^2_{\epsilon_w}, \mho_t, \varphi_t, \beta, r, \kappa_m, \nu_m)$. The rest of the parameters $(Ł_m, Ł_f, \sigma_{\epsilon_\alpha}, \sigma_{\epsilon_q}, \phi_m, \phi_f, \eta)$ were picked to match several moments in the data. I select the same number of data moments (7) than the number of free parameters in the model (7), thus achieving exact identification.

This procedure involves finding a set of parameters $\Theta$ that minimizes the distance between the model generated moments and the moments observed in the data. Specifically, let the targeted moments be $\mathcal{M}(\Theta) = [\mathbf{m} - \widehat{\mathbf{m}}(\Theta)]$ where $\mathbf{m}$ is a vector of observed moments and $\widehat{\mathbf{m}}(\Theta)$ is the vector of model generated moments given parametrization $\Theta$. Then, we can construct the objective function $\min_\Theta \ \mathcal{M}(\Theta)^T W \mathcal{M}(\Theta)$, where the weighting matrix $W$ is the diagonal matrix.

A summary of the parameter values resulting from the calibration exercise for 1970 is presented in Table 2.7. Below I describe in detail the calibration choices for 1970.

### 1.5.1 Calibration to 1970

#### Externally calibrated parameters

We have to choose values for $\sigma, \xi, \mu_m, \rho, \sigma^2_{\epsilon_w}, \mho_t, \varphi_t, \beta, r, \kappa_m, \nu_m$.

**Coefficient of relative risk aversion** $\sigma$ Agents are risk averse with coefficient of relative risk aversion $\sigma$. Estimates for the coefficient of relative risk aversion range from $1$ to $4$. I select a coefficient of risk aversion of $\sigma = 2$, which is standard in the literature, Ortigueira and Siassi (2013), Ríos-Rull (1996), Santos and Weiss (2014).

**Frish elasticity of labor supply** $\xi$ Estimates for the Frish elasticity for labor supply vary, see Bredemeier et al. (2021) and Domeij and Floden (2006). Blundell et al. (2016) estimate the Frish elasticity for men to be $0.52$ and $0.85$ for women. Ortigueira and Siassi (2013) target a value of $0.5$ for men and $0.85$ for females. Following these references I choose $\xi = 0.5$.

**Husbands pareto weight** $\mu_m$ There is very little guidance on how to select this parameter and choices in the literature are diverse. Cubeddu and Rios-Rull (1997) choose a value of $0.5$, and in later robustness select $0.4$. They select a lower pareto weight because it is usually the case that the wife keeps custody of the children after divorce, as they don't model child custody explicitly. Voena (2015) estimates a value of $0.75$, following this insight Fernández and Wong (2017) use a value of $0.7$. Knowles (2005) estimates a value of $0.67$ for 1970 and a value of $0.57$ for 1990. I set, $\mu_m = 0.67$ and keep it constant thoughout, I later conduct robustness on this value. Other papers determine the weights through Nash bargaining Knowles (2005), Greenwood et al. (2002), I too propose a version of the model where the weights are set though Nash Bargaining, it turns out the estimates for 1970 were around $0.6$ and there was little change for 1985.

**Income process** I borrow the estimates for $\rho$ and $\sigma^2_\epsilon$ from Santos and Weiss (2014). They estimate the income process using PSID data from 1964 and 2009, for individuals aged 18 to 64 years old. Their estimates yield a highly persistent process with $\rho = 0.98$ and variance $\sigma^2_\epsilon = 0.011$. The life cycle trend component $\mho_{age,g,e}$ was computed using CPS data extracts for 1970, 1985 and 2015 for couples where the husband was between 21 and 60 years of age. Different trends were computed by education (college, non-college) and gender. The trends were later smoothed using a quadratic fit on age.[14] The average gender-wage gap and college premium were calculated from $\mho_{age,g,e}$. For the purpose of the model I take non-college males aged 21 as the reference group, that is $w(t = 21, g = m, e = nc) = 1$. Note that the life cycle trend component does not

[14] That is $\mho_{age,g,e} = \alpha_0 + \alpha_1\, age + \alpha_2\, age^2$

depend on marital status, in the model more productive individuals are endogenously sorted into marriage.

**Fertility** $\varphi_t$ The OECD reports fertility rates by mother's age at childbirth for the U.S. from 1960 until the present. Table 1.5 shows the values for 1970, 1985 and 2015 used in the calibration.

Table 1.5: Fertility: Births per 1000 women, U.S.

| | Age Group | | | | | | |
|---|---|---|---|---|---|---|---|
| Year | 15-19 | 20-24 | 25-29 | 30-34 | 35-39 | 40-44 | 45-49 |
| 1970 | 68.3 | 167.8 | 145.1 | 73.3 | 31.7 | 8.1 | 0.5 |
| 1985 | 51.0 | 108.3 | 111.0 | 69.1 | 24.0 | 4.0 | 0.2 |
| 2015 | 22.3 | 76.8 | 104.3 | 101.5 | 51.8 | 11.0 | 0.8 |

Notes: Source, OECD Stats, Fertility rates by mother's age at childbirth, five-year age groups, 1960-2019

**Property division rules** $\kappa_m$ Around the 1970's, title based regimes were predominant across the majority of stated in the U.S., Voena (2015) estimates that in title based states [15], around 60% of the assets went to the husband. I follow that estimate and set $\kappa_m = 60\%$ for the 1970 calibration. By 1985, most states transited to either equitable distribution or community property regimes, therefore I set $\kappa_m = 50\%$ for the 1985 and 2015 model simulations.

**Child custody arrangements** $\nu_m$ The Census Bureau[16], reports that the percentage of children living only with their father was 10% in 1970, 15% in 1985 and 20% in 2015, I take this as a proxy for the evolution of the probability of the husband in getting full custody of the child $\nu_m$. For a more exhaustive analysis on the characteristics of single-father headed families with children see Meyer and Garasky (1993). In recent years, joint custody became more common, in my model I do not allow for joint custody arrangements.

**Other parameters** I choose a discount factor of $\beta = 0.98$ and a yearly real risk-free interest rate of $r = 2\%$ following Fernández and Wong (2017) and Blundell et al. (2016) respectively.

[15] The title based regime assigns assets to the respective title owner, community property makes a 50/50 split, and equitable distribution involves the courts discretion so that assets are "fairly" divided, but this is not necessarily 50/50. For a detailed timeline showing the years in which states switched from title based regimes to equitable distribution or community property see Voena (2015).

[16] In a report authored by Hemez and Washington (2021)

**Estimated Parameters and Targeted Moments**

The following parameters, $Ł_m, Ł_f, \sigma_{\epsilon_\alpha}, \sigma_{\epsilon_q}, \phi_m, \phi_f, \eta$ were picked to match several data moments. The average marital quality by gender $Ł_m, Ł_f$ were chosen to match divorce rates and the average share of divorces initiated by wives. The value of the public good $\eta$, is aimed to match the share of divorces initiated by wives with children. The standard deviation of the initial match quality $\sigma_{\epsilon_\alpha}$ and the standard deviation of the marital quality shock $\sigma_{\epsilon_q}$ govern the percentage of never married agents in the economy and the median age at first marriage. Finally, the female and male dis-utilities from work $\phi_m$ and $\phi_f$ are directly set to match the amount of work hours supplied by each couple member, see values in Table 1.3. I normalize the amount of hours worked supplied by men to unity. Divorce filing data is taken from the NBER collection of Marriage and Divorce Data NBER (1995), divorce rates were computed from the PSID, the rest of the data moments were computed from CPS-IPUMS data extracts. I compute these moments for couples where the husband had between 21 and 60 years old, see Section 1.2 for a more detaield description of the data treatment.

### 1.5.2 Estimation Results

Table 2.7 shows the estimation results for 1970. We can see that $Ł_m < Ł_f$, this means that on average women enjoy marriage less than men, this result is not new in the literature, Rios-Rull et al. (2010) find results in the same direction, moreover, this result is consistent with marital satisfaction surveys where wives report on average lower marital quality than their husbands Rosenfeld (2018). We can also see that $\eta > 0$ meaning that children bring additional utility to the marriage, thus making marriage more attractive for both men and women, however, since wives have a higher probability of keeping the children upon divorce, children raise the value of divorce for wives more than for husbands which justifies why the percentage of wives filing for divorce is larger in the presence of children. Finally, the estimation makes $\phi_m < \phi_f$, meaning that men have a comparative advantage in the labor market, relative to women.

**Table 1.6: Calibration for 1970**

| Description | Symbol | Value |
|---|---|---|
| Preferences | | |
| Risk aversion | $\sigma$ | 2 |
| Frish elasticity | $\xi$ | 0.5 |
| Disutility from work Men | $\phi_m$ | 0.98 |
| Disutility from work Women | $\phi_f$ | 2.7 |
| Value of the public good/children | $\eta$ | 1.35 |
| Husbands utility share | $\mu_m$ | 0.67 |
| Income Process | | |
| Persistence | $\rho$ | 0.98 |
| Variance wage shock | $\sigma_\epsilon^2$ | 0.011 |
| Gender wage gap | $w_f/w_m$ | 0.55 |
| Life cycle trend | $\mho_t$ | CPS |
| Marital Shocks | | |
| Average marital quality Man | $Ł_m$ | 2.9 |
| Average marital quality Woman | $Ł_f$ | 2.2 |
| St. deviation of initial marital quality draw | $\sigma_{\epsilon_\alpha}$ | 1.01 |
| St. deviation of marital quality shock | $\sigma_\epsilon$ | 11.0 |
| Fertility | | |
| Fertility | $\varphi_t$ | OECD Charts |
| Other | | |
| Discount factor | $\beta$ | 0.98 |
| Risk free rate | $r$ | 0.02 |
| Husbands share of assets | $\kappa_m$ | 0.6 |
| Wife share of assets | $\kappa_f$ | 0.4 |
| Probability the husbands gets child custody | $\nu_m$ | 0.1 |

Notes: Parameters in red are those which were estimated rather than set apriori.

**Model fit 1970** Table 1.7 shows the performance of the model in matching the targeted moments. We see that the model performs fairly well in matching most of the targeted moments.

Table 1.7: Model Fit 1970

| Moment | 1970 | |
|---|---|---|
| | Model | Data |
| Divorce Rate | 1.34 | 1.30 |
| % of wives as plaintiffs | 71.59 | 71.60 |
| % of wives w/kids as plaintiffs | 76.94 | 75.80 |
| % of Never married | 11.48 | 12.40 |
| Male hours | 1.05 | 1.00 |
| Female hours | 0.75 | 0.77 |
| Male median age at first marriage | 25.45 | 22.00 |

## 1.6 Counterfactual experiments

In this section I conduct a set of counterfactual experiments. First, I generate counterfactual predictions for 1985 and 2015 where I measure the combined contribution of changes in the population composition, fertility, gender-wage gap, child custody arrangements and property division laws. Next I propose a decomposition exercise where I individually quantify the effects of the main drivers of interest.

### 1.6.1 Counterfactual Prediction for 1985

The purpose of this exercise is to generate model predictions for 1985. To do so, I exogenously change the value of fertility, the gender-wage gap, child custody arrangements, and asset splitting rules and set them to their 1985 values. With these values, the model is simulated and the moment predictions compared with their data counterparts for 1985.

From 1970 to 1985 the average relative wages increased from $\bar{w}_f/\bar{w}_m(1970) = 0.55$ to $\bar{w}_f/\bar{w}_m(1985) = 0.65$, the probability of the husband in getting custody of the children increased from $10\%$ to $15\%$, the share of assets that went to the husband after divorce decreased from $60\%$ to $50\%$. Finally, fertility decreased, see Table 1.5. For 1985 I re-estimate the values of $\phi_m$ and $\phi_f$ such that they match the new ratio between male hours and female hours $h_f/h_f(1985) = 0.8$, otherwise the rise in relative wages causes male hours to reduce and female hours to increase.

**Model predictions for 1985** Table 1.8 shows that the model can account for $40\%$ of the total change in divorce rates between 1970 and 1985, $53\%$ of the change in the share of divorces initiated by wives, $33\%$ in the rise of the share of never married individuals and $30\%$ in the delay in marriage, reflected by an increase in the median age at first marriage.

Table 1.8: Model Fit 1985

| Moment | 1970 | | 1985 | | %Δ | |
|---|---|---|---|---|---|---|
| | Model | Data | Model | Data | Model | Data |
| Divorce Rate | 1.34 | 1.30 | 1.58 | 1.87 | 17.79 | 43.85 |
| % of wives as plaintiffs | 71.59 | 71.60 | 66.13 | 61.30 | -7.63 | -14.39 |
| % of wives w/kids as plaintiffs | 76.94 | 75.80 | 62.28 | 64.70 | -19.05 | -14.64 |
| % of Never married | 11.48 | 12.40 | 13.98 | 20.50 | 21.82 | 65.32 |
| Male hours | 1.05 | 1.00 | 1.08 | 1.00 | - | - |
| Female hours | 0.75 | 0.77 | 0.83 | 0.80 | - | - |
| Male median age at first Marriage | 25.45 | 22.00 | 26.54 | 25.00 | 4.25 | 13.64 |

### 1.6.2 Counterfactual Prediction for 2015

The objective is to generate model predictions for 2015 and compare them with their respective data counterparts. I follow the same logic that is described above. Between 1985 and 2015 relative wages increased from $\bar{w}_f/\bar{w}_m(1985) = 0.65$ to $\bar{w}_f/\bar{w}_m(2015) = 0.80$, the probability of the husband in getting custody of the children increased from $15\%$ to $20\%$, the share of assets that went to the husband after divorce remained constant, fertility decreased, see Table 1.5. This time the values of $\phi_m$ and $\phi_f$ were kept as in 1985 since the new ratio between male hours and female hours remained roughly constant $h_f/h_f(2015) = 0.83$.

**Model predictions for 2015** We can see from Table 1.9 that the change in divorce rates generated by the model between 1985 and 2015, represents $95\%$ of the total change in the data, $52\%$ of the change in the share of wives as plaintiffs, $4\%$ in the share of never married and $26\%$ of the change in the median age at first marriage of males. Moreover simple calculations, show that the model accounts for $54\%$ of the overall decline in divorces initiated by wives between 1970 and 2015, $17\%$ in the rise of the share of never married and $26\%$ of the rise in the median age at first marriage for men.

Table 1.9: Model Fit 2015

| Moment | 1985 | | 2015 | | %Δ | |
|---|---|---|---|---|---|---|
| | Model | Data | Model | Data | Model | Data |
| Divorce Rate | 1.58 | 1.87 | 1.12 | 1.30 | -28.96 | -30.48 |
| % of wives as plaintiffs | 66.13 | 61.30 | 63.47 | 56.60 | -4.02 | -7.67 |
| % of wives w/kids as plaintiffs | 62.28 | 64.70 | 54.40 | 59.00 | -12.65 | -8.81 |
| % of Never married | 13.98 | 20.50 | 14.21 | 29.20 | 1.66 | 42.44 |
| Male hours | 1.08 | 1.00 | 1.08 | 1.00 | - | - |
| Female hours | 0.83 | 0.80 | 0.90 | 0.84 | - | - |
| Male median age at first Marriage | 26.54 | 25.00 | 27.62 | 29.00 | 4.10 | 16.00 |

### 1.6.3 Decomposition of Effects

I'm interested in measuring the contribution of each driver: the rise of mean relative wages $\bar{w}_f/\bar{w}_m$, the change in the probabilities of becoming custodial parent $\nu_m$ and the change in property division laws $\kappa_m$, in explaining the observed rise and later decline of divorce rates and the reduction of the share of dorced initiated by wives. To this end I propose the following counterfactual experiments:

1. Take the parametrization used for the 1985 simulation but set $\bar{w}_f/\bar{w}_m$ to its value in 1970 $(\bar{w}_f/\bar{w}_m(1970) = 0.55)$.
2. Take the parametrization used for the 1985 simulation but set $\kappa_m$ to its value in 1970 $(\kappa_m(1970) = 0.6)$.
3. Take the parametrization used for the 1985 simulation but set $\nu_m$ to its value in 1970 $(\eta_m(1970) = 0.1)$.

I repeat the above experiments for the period 1985 to 2015, this involves fixing the parameters used for the 2015 simulation and setting one by one the values of $\bar{w}_f/\bar{w}_m, \kappa_m$ and $\nu_m$ to their respective 1985 levels, that is: $\bar{w}_f/\bar{w}_m(1985) = 0.65, \kappa_m(1985) = 0.5$ and $\eta_m(1985) = 0.15$. The Results for these experiments are shown in Tables 1.11 to 1.13.

Tables 1.11 to 1.13 show the percentage of the data variation than can be explained by each of the drivers. I then proceed to measure the contribution of each driver on the total effect shown in Tables 1.8 and 1.9. This is done by computing a residual by subtracting the individual effects (taken from tables 1.11 to 1.13) from the total effect. I then divide the residual by the number

of drivers (three in this case) and add this value to each of the individual effects. The sum of the residual and the individual effect by driver gives a new set of individual effects such that the sum of all individual effects adds up to the total effect. Results of this exercise are shown in 1.10.

From table 1.10 we see that the change in relative wages is the main driver of the rise in divorce rates from the period 1970 to 1985 (direct effect). Changes in the splitting rule contribute positively to the rise in divorce rates in the same time frame. On the contrary changes in child custody arrangements act in the opposite direction reducing divorce rates. We see that Changes in child custody arrangements are the main driver of the reduction in the share of wives as plaintiffs, accounting for 77% of the decline, the rest is mostly explained by changes in the gender-wage gap.

For the period 1985 to 2015 the selection effect arising from the rise in female wages accounts for 62% of the decline in divorce rates, the rest is driven by changes in child custody arrangements. During this period it is still the case that most of the reduction in the share of divorces initiated by wives, around 82%, is explained by the increase in the probability of the husband becoming the custodial parent.

Table 1.10: Decomposition of Effects

| | Shutting Down: | | | Total | Contribution of: (%) | | |
|---|---|---|---|---|---|---|---|
| | $\Delta w_f/w_m$ | $\Delta \kappa_m$ | $\Delta \nu_m$ | $\frac{\Delta Model}{\Delta Data}$ | $w_f/w_m$ | $\kappa_m$ | $\nu_m$ |
| | | | | 1970 to 1985 | | | |
| Divorce Rate | 37.02 | 20.09 | -16.53 | 40.57 | 91.24 | 49.51 | -40.75 |
| % of wives as plaintiffs | 15.31 | -3.28 | 40.99 | 53.02 | 28.88 | -6.18 | 77.31 |
| % of wives w/kids as plaintiffs | 31.66 | 41.97 | 56.49 | 130.12 | 24.33 | 32.25 | 43.41 |
| | | | | 1985 to 2015 | | | |
| Divorce Rate | 59.20 | 0.00 | 35.81 | 95.01 | 62.31 | 0.00 | 37.69 |
| % of wives as plaintiffs | 9.04 | 0.00 | 43.38 | 52.41 | 17.24 | 0.00 | 82.76 |
| % of wives w/kids as plaintiffs | 0.82 | 0.00 | 142.77 | 143.59 | 0.57 | 0.00 | 99.43 |

### Shutting down relative wages ($\bar{\mathbf{w}}_\mathbf{f}/\bar{\mathbf{w}}_\mathbf{m}$)

When relative wages don't increase we see that the share of divorces initiated by wives slightly increases and the aggregate divorce rate slightly decreases. This suggests that relative wages are a major driver of both divorce rates and divorce filings by wives. Raising relative wages to their 1985 levels would increase divorce rates by $20\%$ and reduce divorce filings by women by $8.39\%$, which account for $47\%$ and $53\%$ of the variation that we see in the data.

Table 1.11: Shutting Down Relative Wages $\bar{w}_f/\bar{w}_m$

| Moment | 1970 | 1985 | | Experiment: 1985 with $\bar{w}_f/\bar{w}_m$ of 1970 | |
|---|---|---|---|---|---|
| | Model | Model | Data | Result | % of data variation explained by $\Delta\bar{w}_f/\bar{w}_m$ |
| Divorce Rate | 1.34 | 1.58 | 1.87 | 1.31 | 47.54 |
| % of wives as plaintiffs | 71.59 | 66.13 | 61.30 | 71.68 | 53.87 |
| % of wives w/kids as plaintiffs | 76.94 | 62.28 | 64.70 | 62.80 | 4.68 |
| % of Never married | 11.48 | 13.98 | 20.50 | 12.30 | 20.74 |
| Male hours | 1.05 | 1.08 | 1.00 | 1.02 | - |
| Female hours | 0.75 | 0.83 | 0.80 | 0.77 | - |
| Male median age at first Marriage | 25.45 | 26.54 | 25.00 | 25.85 | 22.83 |
| Moment | 1985 | 2015 | | Experiment: 2015 with $\bar{w}_f/\bar{w}_m$ of 1985 | |
| | Model | Model | Data | Result | % of data variation explained by $\Delta\bar{w}_f/\bar{w}_m$ |
| Divorce Rate | 1.58 | 1.12 | 1.30 | 1.21 | 14.65 |
| % of wives as plaintiffs | 66.13 | 63.47 | 56.60 | 67.02 | 75.49 |
| % of wives w/kids as plaintiffs | 62.28 | 54.40 | 59.00 | 51.75 | -46.52 |
| % of Never married | 13.98 | 14.21 | 29.20 | 12.37 | 21.12 |
| Male hours | 1.08 | 1.08 | 1.00 | 1.02 | - |
| Female hours | 0.83 | 0.90 | 0.84 | 0.80 | - |
| Male median age at first Marriage | 26.54 | 27.62 | 29.00 | 25.92 | 42.60 |

### Shutting down changes in property division ($\kappa_{\mathbf{m}}$)

If in 1985 women would be getting the same share of marital wealth as in the 1970's, divorce rates wouldn't have risen as much as in the data, likewise the share of divorces initiated by wives wouldn't have reduced as much. In addition the share of never married increases as well as the median age at first marriage.

Table 1.12: Shutting down changes in property division $\kappa_m$

| Moment | 1970 | 1985 | | Experiment: 1985 with $\kappa_m$ of 1970 | |
|---|---|---|---|---|---|
| | Model | Model | Data | Result | % of variation explained by $\Delta\kappa_m$ |
| Divorce Rate | 1.34 | 1.58 | 1.87 | 1.41 | 30.61 |
| % of wives as plaintiffs | 71.59 | 66.13 | 61.30 | 69.76 | 35.28 |
| % of wives w/kids as plaintiffs | 76.94 | 62.28 | 64.70 | 63.95 | 14.99 |
| % of Never married | 11.48 | 13.98 | 20.50 | 14.84 | -10.62 |
| Male hours | 1.05 | 1.08 | 1.00 | 1.12 | - |
| Female hours | 0.75 | 0.83 | 0.80 | 0.85 | - |
| Male median age at first Marriage | 25.45 | 26.54 | 25.00 | 26.77 | -7.88 |
| Moment | 1985 | 2015 | | Experiment: 2015 with $\kappa_m$ of 1985 | |
| | Model | Model | Data | Result | % of variation explained by $\Delta\kappa_m$ |
| Divorce Rate | 1.58 | 1.12 | 1.30 | 1.12 | 0.00 |
| % of wives as plaintiffs | 66.13 | 63.47 | 56.60 | 63.47 | 0.00 |
| % of wives w/kids as plaintiffs | 62.28 | 54.40 | 59.00 | 54.40 | 0.00 |
| % of Never married | 13.98 | 14.21 | 29.20 | 14.21 | 0.00 |
| Male hours | 1.08 | 1.08 | 1.00 | 1.08 | - |
| Female hours | 0.83 | 0.90 | 0.84 | 0.90 | - |
| Male median age at first Marriage | 26.54 | 27.62 | 29.00 | 27.62 | 0.00 |

**Shutting down changes in the probability of becoming the custodial parent ($\nu_{\mathbf{m}}$)**

In the absence of changes in the probability of the husband in becoming the custodial parent, the share of divorces initiated by women decrease very little, from 71.50% to 68%. This suggests that the change in child custody arrangements is the main driver of the reduction in the share of divorce filings by wives, as it accounts for almost all the variation observed in the data. Furthermore, if wives would have the same chance of getting custody of the children the divorce rates would reach higher (lower) levels for the period 1970 to 1985 (1985 to 2015) thus suggesting that changes in child custody arrangements have an impact on the composition of divorces but seem to keep the divorce rates at the same level.

Table 1.13: Shutting down changes in the probability of the father in getting full custody of the child $\nu_m$

| Moment | 1970 | 1985 | | Experiment: 1985 with $\nu_m$ of 1970 | |
|---|---|---|---|---|---|
| | Model | Model | Data | Result | % of variation explained by $\Delta\nu_m$ |
| Divorce Rate | 1.34 | 1.58 | 1.87 | 1.61 | -6.01 |
| % of wives as plaintiffs | 71.59 | 66.13 | 61.30 | 74.32 | 79.55 |
| % of wives w/kids as plaintiffs | 76.94 | 62.28 | 64.70 | 65.56 | 29.51 |
| % of Never married | 11.48 | 13.98 | 20.50 | 14.11 | -1.56 |
| Male hours | 1.05 | 1.08 | 1.00 | 1.08 | - |
| Female hours | 0.75 | 0.83 | 0.80 | 0.82 | - |
| Male median age at first Marriage | 25.45 | 26.54 | 25.00 | 26.58 | -1.54 |
| Moment | 1985 | 2015 | | Experiment: 2015 with $\nu_m$ of 1985 | |
| | Model | Model | Data | Result | % of variation explained by $\Delta\nu_m$ |
| Divorce Rate | 1.58 | 1.12 | 1.30 | 1.07 | -8.74 |
| % of wives as plaintiffs | 66.13 | 63.47 | 56.60 | 68.64 | 109.83 |
| % of wives w/kids as plaintiffs | 62.28 | 54.40 | 59.00 | 59.84 | 95.43 |
| % of Never married | 13.98 | 14.21 | 29.20 | 14.21 | 0.03 |
| Male hours | 1.08 | 1.08 | 1.00 | 1.08 | - |
| Female hours | 0.83 | 0.90 | 0.84 | 0.90 | - |
| Male median age at first marriage | 26.54 | 27.62 | 29.00 | 26.61 | 25.42 |

## 1.7 Importance of Matching Divorce Filings

In this section I emphasize the importance of using the information on divorce filings to explain the observed trends in divorce rates since 1970. I show that failure to match "who" files for divorce delivers different (potentially misleading) counterfactual results.

Divorce rates in the U.S. have been predominantly driven by wives, but would aggregate divorce rates respond differently to the observed labor market changes if divorces were not mainly triggered by wives? The answer to this question is not obvious, more so since the proposed model exploits fundamental differences in the utility that men and women get from marriage and child custody arrangements to match the share of divorces initiated by wives. To address this question, one would need to abstract from these mechanisms and reassess the model's performance in reproducing the divorce rate trends observed in the data. To this end, I propose a restricted version of the model where I abstract from the model features that allow it to match divorce filings. I set the restriction that $Ł_m = Ł_f$, that is, now men and women on average enjoy married life equally, and I set $\nu_m = 0.5$, that is, both the husband and wife have the same probability of getting the custody of the child after divorce. With this restrictions I re-estimate the model to match the data moments of 1970 described in Section 1.5.1, except for divorce filing moments. I repeat the exercise described in Section 1.6.1 to get the prediction for 1985, then I conduct the corresponding decomposition exercise to disentangle the effects of relative wages, child custody arrangements and property division laws. Results are shown in Tables 1.15 to 1.16.

**Alternative model fit to 1970** The newly estimated parameter values are shown in Table 1.14. We can see that the average match quality for men and women in the alternative model is lower than in the benchmark estimation. In addition, the variance of the initial marital quality draw and the standard deviation of the marital quality shock are smaller, making marriages in this world less susceptible to divorce risk coming from marital quality. Table 1.15 shows that the alternative model matches the targeted moments very closely, however delivers a large share of divorce filings coming from men $52\%$ compared to $29\%$ in the data.

Table 1.14: Estimated Parameters, alternative model

| Description | Symbol | Value |
|---|---|---|
| Average marital quality | $Ł_m = Ł_f$ | 1.49 |
| St. deviation of the initial marital quality draw | $\sigma_{\epsilon_\alpha}$ | 0.7 |
| St. deviation of the marital quality shock | $\sigma_{\epsilon_q}$ | 8.5 |
| Disutility from work Men | $\phi_m$ | 0.93 |
| Disutility from work Women | $\phi_m$ | 2.1 |

**Alternative model predictions for 1985** The alternative model predicts a decline in divorce rates and a slight increase of the divorce filings by wives, these results are opposite to what is observed in the data. The intuition behind the results is the following: because in the alternative model most of the divorces come from husbands (as opposed from wives like in the data),an increase in female wages will make women more attractive to men, since if married, men can work less and wives more, thus reducing the number of divorces initiated by husbands. Moreover, the median age at first marriage and the percentage of never married individuals barely increased compared to the benchmark scenario, this suggests that women's incentives to wait to obtain better matches are not as strong as in the benchmark scenario.

Table 1.15: Model Fit 1985

| Moment | 1970 | | 1985 | | $\%\Delta$ | |
|---|---|---|---|---|---|---|
| | Model | Data | Model | Data | Model | Data |
| Divorce Rate | 1.34 | 1.30 | 1.02 | 1.87 | -23.94 | 43.85 |
| % of wives as plaintiffs | 48.09 | 71.60 | 48.95 | 61.30 | 1.79 | -14.39 |
| % of wives w/kids as plaintiffs | 50.54 | 75.80 | 41.79 | 64.70 | -17.30 | -14.64 |
| % of Never married | 13.38 | 12.40 | 13.47 | 20.50 | 0.64 | 65.32 |
| Male hours | 1.03 | 1.00 | 1.00 | 1.00 | -3.05 | - |
| Female hours | 0.72 | 0.77 0 | 0.79 | 0.80 | 8.99 | - |
| Male median age at first Marriage | 25.65 | 22.00 | 26.20 | 25.00 | 2.15 | 13.64 |

Table 1.16: Counterfactual experiments

| Moment | 1970 | 1985 | | Experiment: 1985 with $\bar{w}_f/\bar{w}_m$ of 1970 | |
|---|---|---|---|---|---|
| | Model | Model | Data | Result | % of data variation explained by $\Delta\bar{w}_f/\bar{w}_m$ |
| Divorce Rate | 1.34 | 1.02 | 1.87 | 1.21 | -34.14 |
| % of wives as plaintiffs | 48.09 | 48.95 | 61.30 | 59.03 | 97.80 |
| % of wives w/kids as plaintiffs | 50.54 | 41.79 | 64.70 | 66.36 | 221.30 |
| % of Never married | 13.38 | 13.47 | 20.50 | 12.62 | 10.47 |
| Male hours | 1.03 | 1.00 | 1.00 | 1.02 | - |
| Female hours | 0.72 | 0.79 | 0.80 | 0.82 | - |
| Male median age at first Marriage | 25.65 | 26.20 | 25.00 | 25.87 | 11.11 |
| Moment | 1970 | 1985 | | Experiment: 1985 with $\kappa_m$ of 1970 | |
| | Model | Model | Data | Result | % of variation explained by $\Delta\kappa_m$ |
| Divorce Rate | 1.34 | 1.02 | 1.87 | 1.16 | -24.64 |
| % of wives as plaintiffs | 48.09 | 48.95 | 61.30 | 41.38 | -73.57 |
| % of wives w/kids as plaintiffs | 50.54 | 41.79 | 64.70 | 34.70 | -63.89 |
| % of Never married | 13.38 | 13.47 | 20.50 | 12.97 | 6.15 |
| Male hours | 1.03 | 1.00 | 1.00 | 1.00 | - |
| Female hours | 0.72 | 0.79 | 0.80 | 0.76 | - |
| Male median age at first Marriage | 25.65 | 26.20 | 25.00 | 25.61 | 19.66 |

## 1.8 Conclusions

In this paper I quantitatively assess the role of divorce filings in explaining divorce rate trends in the U.S. from 1970 until 2015. To do so I construct and estimate a model with endogenous marriage and unilateral divorce that matches divorce filing data for the U.S. in 1970. The model includes two features that allow it to match divorce filing data: first, gender specific marital quality/love, meaning that men and women enjoy married life differently. Second, child custody arrangements that favoring wives in getting the custody of the children after divorce. Both being crucial for obtaining accurate counterfactual results. Equipped with this model I generate counterfactual predictions for 1985 and 2015 and quantify the effects of changes in the gender-wage gap, property division laws and child custody arrangements. I show that the proposed model does a good job in describing the joint dynamics of divorce rates and divorce filings. The model accounts for roughly 50% of the variation in divorce rates and divorce filings by wives between

1970 and 2015.

In further counterfactual experiments I decompose the effects of the three drives (the gender-wage gap, property division laws and child custody arrangements), in accounting for the observed trends in divorce rates and divorce filings by wives. Results show that changes in relative wages and changes in child custody arrangements are largely responsible for the rise and later decline in divorce rates, plus the overall decline in divorce filings by women. Rising relative wages account for approximately 91% of the increase of divorce rates between 1970 to 1985 (direct effect) and 62% of their decrease between 1985 and 2015 (selection effect). They also account for 23% of the overall decline in divorce filings by women between 1970 to 2015. Child custody arrangements are the most important driver behind the reduction in the share of divorce initiated by wives. The change in the probability of the husband in getting custody of the children accounts for almost 77% of the decline in divorce filings by wives between 1970 and 1985, and 83% of the variation between 1985 and 2015.

Importantly, I show that failing to match divorce filing data delivers opposite counterfactual results, thus stressing the importance of matching who files for divorce when explaining divorce rates. An alternative version of the model that does not match divorce filing moments predicts a decline in divorce rates and an increase of the share of divorce filings done by wives for the period 1970 to 1985, result that is opposite to what is observed in the data. Behind this result lies the fact that it is relevant to distinguish whether divorce rates are driven by husbands or by wives. If divorces are driven by husbands (opposite to what we see in the data) higher relative wages for women will make women more financially attractive to men, thus reducing the number of divorces triggered by men and as a consequence reducing aggregate divorce rates.

The model presented in this paper can be extended to study other related questions, like the rise in married female labor force participation or welfare implications of changes in child custody laws or property division rules upon divorce. Furthermore, I currently make steady state comparisons between 1970 and 2015; ideally one would want to endogenize the full path for divorce rates and divorce filings by computing transitional dynamics between these years.

# Chapter 2

# A Quantitative Theory of the HIV Epidemic: Education, Risky Sex and Asymmetric Learning

*Written jointly with Daniela Iorio and Raül Santaeulàlia-Llopis.*

## 2.1 Introduction

In Sub-Saharan Africa (SSA) most HIV infections are due to heterosexual intercourse, and risky sexual behavior is one of the most relevant margins that policy intervention can affect (Behrman and Kohler, 2012; DePaula et al., 2014; Greenwood et al., 2013; Nyqvist et al., 2015). If risky sexual behavior, and in turn HIV exposure, differs across education groups as the HIV epidemic evolves, then the timing of policy interventions targeted to specific educational groups is crucial for the effectiveness of these policies. Nowadays, however, the major international donors in the fight against HIV do not provide any guidance on specific targeting strategies across education groups.[1] This lack of policy advice could be explained by the fact that the current understanding of the sign and size of the relationship between education and HIV status lacks consensus (Beegle and de Walque, 2009). That is, the knowledge of which education groups are at major risk of being infected with HIV remains unclear to scholarship, with a large body of mixed evidence that we review below. We provide a potential explanation that could reconcile the mixed evidence,

[1] According to Kates et al. (2011), $6.9 billion was given by donor governments to international AIDS assistance in 2010. The United States is the largest resource provider for the global fight against AIDS, and it channels its aid through the President's Emergency Plan for AIDS Relief (PEPFAR). Initiated by President George W. Bush for 2003-2008, PEPFAR has continued its activity under the mandate of President Barack Obama, who renewed the efforts for 5 years with few changes in policy implementation. However, they increased the amount of money—to about 50% in countries with a generalized epidemic— spent on preventing sexual transmission via abstinence, delay of age of first sexual intercourse, monogamy, fidelity, and reduction in the number of sex partners. More recently, new PEPFAR funds have been channeled to include "men who have sex with men, people who inject drugs, and sex workers", see the remarks of the Secretary of State John Kerry for the PEPFAR 10th annivarsary celebration at http://www.state.gov/secretary/remarks/2013/06/210770.htm. See also UNAIDS (2015).

departing from the observation that the existing works have, almost invariably, used data from different aggregate stages of the HIV epidemic, while the education gradient in HIV may vary over the evolution of the epidemic.

That the HIV epidemic in SSA evolves differently across countries and that these countries are at different stages of the HIV epidemic at any point in time is practically self-evident. In particular, we observe that the peak of HIV prevalence, the year of the HIV peak, the time it takes each country to reach its own peak, and the pace at which each country moves away from its peak differs greatly across SSA countries and over time. Based on these considerations, we propose an innovative unified macro framework that consists of a two-dimensional normalization of the HIV epidemic. Our definition of the stages of the HIV epidemic are analogous to the definition of stages of economic development (Herrendorf et al., 2014b; Lucas, 2004a) or the stages of the demographic transition (Galor and Weil, 2000a; Greenwood et al., 2005a; Lee, 2003). In the context of HIV, our normalization adjusts for both the country-specific size of the epidemic (HIV prevalence rate) and the associated time paths of the epidemic (speed at which HIV epidemic reaches its peak and it moves away from it) in a comparable manner across countries. This way, our macro framework systematically defines aggregate stages of the HIV epidemic taking into account the large degree of cross-country heterogeneity in both the HIV prevalence rates and the speed at which the HIV epidemic evolves. We then use the heterogeneity in the stages of the HIV epidemic in 39 Demographic and Health Surveys (DHS) to document the stylized dynamic relationship between education and individual HIV status across the stages of the HIV epidemic.

Our main finding is that the education gradient in HIV follows a significant U-shaped (positive-zero-positive) pattern as the epidemic evolves. In particular, when individuals live in an economy that is at the early stages of the epidemic, the HIV-Education gradient is significantly positive and remarkably high: one additional year of education is associated with 1.12 percentage point increase in the probability of being HIV-positive. In other terms, completing five additional years of schooling doubles on average the likelihood of being infected (the HIV prevalence is 5.18% in our sample). Interestingly, the educational disparities in HIV gradually vanish as the epidemic progresses past these early stages, to then revert to a positive education gradient in HIV in the more advanced stages of the epidemic, where an additional five years of education result in a 2.40 percentage point increase in the probability of being HIV positive. This U-shaped pattern is also significant when controlling for country and year effects, although the size of the effects of education on HIV is smaller. We then explore whether there are heterogeneous effects by gender. While women and men share the U-shaped pattern of the education gradient in HIV, its magnitude is larger for women than for men. This gap is largest at the early stages of HIV development, and tends to disappear later on. Regarding women, completing five additional years

of schooling is associated with a 7.40% rise in the probability of being HIV positive at the early stages of the epidemic, that is, an increase twice as large as that of men, 3.80% per five years of education. Thereafter, the education gradient in HIV substantially declines until it vanishes for women who live in the middle of the HIV epidemic. At this stage, the decline in the gradient is even sharper for men, and changes its sign, reaching a -0.65% per five years of schooling. Interestingly, the gradient reverts to positive in the more advanced stages of the epidemic for both women and men, respectively, 2.70% and 1.75% per five years of education.

To gain a better understanding of the dynamic relationship between education and the probability of infection, we explore educational disparities in the actual risky sexual behavior. Remarkably, the pattern of the education gradient in HIV closely resembles the pattern of the educational disparities in risky sexual behavior. While more education is associated with more extramarital partners at early stages of the epidemic (0.19 per five more years of schooling for women and 0.15 for men), this relationship rapidly and significantly declines in mid stages of the epidemic (0.06 and 0.05 extramarital partners per five more years of schooling for, respectively, women and men). Interestingly, in the most advanced stages of the epidemic the relationship between education and the number of extramarital partners significantly increases (0.11 per five more years of schooling for women and 0.15 for men). The fact that the HIV-Education gradient closely follows education disparities in risky sexual behavior across stages of the epidemic points out the important role of educational disparities in determining the HIV incidence.[2] We develop a non-stationary quantitative macroeconomic theory with heterogeneous agents that is consistent with these facts. Our theory endogeneizes the entire course of the HIV epidemic across different (aggregate) stages: a pre-HIV epidemic stage; a myopic HIV stage in which agents are not aware of the process of HIV infection; a learning stage in which agents heterogeneously—across education groups—learn about the process of infection; and an anti-retroviral (ARV) stage that modifies the effects of HIV infection on individuals. Results show that asymmetric learning is key to reproduce both the micro patterns documented and the aggregate evolution of the HIV epidemic. In further counterfactual experiments, we assess the effects of an early understanding of the virus and its mode of infection, improvements in the composition of education, the earlier (and universal) adoption of ARVs and the use of PrEP to prevent further spread.

The rest of our paper is organized as follows. In the next paragraph we discuss the related literature. In Section **??**, describes the empirical evidence. We document the heterogeneity of the

[2]The prevalence of HIV is determined by the newly infected individuals as well as by the survival probabilities of the individual being infected in the past. Therefore, the educational disparities in the HIV prevalence could be the result of educational disparities in the incidence of HIV and/or educational disparities in the survival probabilities. The DHS data allow us only to focus on individual attitudes towards risky sexual behavior that might increase the probability of being HIV infected. Later on, we discuss the potential role that educational disparities in survival rates might play in shaping the HIV-Education patterns as the HIV epidemic evolves.

epidemic across SSA countries and propose a unified macro framework to define the aggregate stages of the HIV epidemic in a comparable manner across time and space in Section 2.2.2. The estimates of the stylized evolution of the HIV-Education gradient across the aggregate stages of the epidemic are in Section 2.2.3. Section 2.3 presents the quantitative model that endogeneizes the entire course of the HIV epidemic across aggregate stages. Section 2.4 presents the model estimation. Section 2.5 and Section 2.6 describe the main results and counterfactual experiments. Section 2.7 concludes.

**Related literature** We are not the first to investigate the HIV-Education gradient. A large number of epidemiological studies and small-scale studies examined socioeconomic disparities in HIV without reaching neither conclusive nor generalizable answers.While the current mixed evidence is likely to reflect differences in methodology, sampling strategy, and measures of socioeconomic indicators and HIV status, this may not entirely explain the differing conclusions reached by previous studies, which for instance overlooked the large differences in the evolution of the HIV epidemic across SSA countries. In a review of epidemiological studies Gregson et al. (2001) conclude that there could be temporal dynamics of the influence of socio-economic development on rates of HIV transmission, and in particular that the greater vulnerability of individual with a high socioeconomic status may be a transient feature of the early stages of epidemics. The findings of de Walque (2007) point in the same direction in his analysis of the HIV-education gradient in rural Uganda between 1989 and 2001. Importantly, these studies did not expect a rebound in the education gradient in HIV that we document. Recently, using nationally representative data from the Demographic and Health Surveys (DHS) for five SSA countries, Fortson (2008) finds education has a positive association with HIV status: adults with primary school are one half more likely to be infected than adults with no schooling conditioning on age, sex and area of residence (urban/rural). Using the DHS wealth index, de Walque (2009) also finds that wealth displays a positive association with HIV status. Mishra et al. (2007) find similar results for eight DHS countries in SSA.

We contribute to this literature in two major respects. First, we uncover the evolution of the HIV epidemic using a unified macro framework that systematically defines the aggregate stages of the HIV epidemic in a comparable manner across countries. Our approach addresses similar data challenges as those faced when defining the stages of economic development (Herrendorf et al., 2014b; Lucas, 2004a) or the stages of the demographic transition (Galor and Weil, 2000a; Greenwood et al., 2005a; Lee, 2003). Second, in the context of this unified macro framework, and using repeated cross-sections of DHS surveys, our analysis exploits a rich variation in the aggregate stages of the HIV epidemic to document the stylized dynamic behavior of the HIV-Education gradient along the course of the epidemic, as well as the evolution of educational

disparities in sexual responses. Our findings emphasize that while at early stages of the epidemic the HIV-Education gradient is large and positive (roughly three times larger than its stationary counterpart, à la Fortson (2008)), the gradient decreases to levels that are not significantly different from zero as the epidemic evolves; a macro cross-country decline that resembles the results by de Walque (2007) for several sites in rural Uganda. The time span of our data allows us to pick a rebound in the education gradient in HIV in mature epidemics, which we link to a positive change in risky sexual behavior among highly educated individuals. Interestingly, this U-shaped pattern is much more prominent for women than for men.

Due to the fact that heterosexual intercourse is the major mode of HIV infection (Behrman and Kohler, 2012), the relationship between HIV, risky sexual behavior, and HIV knowledge has been extensively studied. For example, information campaigns that improve the knowledge about HIV risk infection may induce people to adopt safer lifestyles. In this direction, de Walque (2007) documents substantial behavioral change in rural Uganda associated with the ABC campaign (Abstinence, Be faithful, and use Condoms).

Finally, our study is broadly related to the literature examining educational disparities in health outcomes. See Cutler and Lleras-Muney (2011) for a review of the studies examining this relationship in both developed and developing countries. Within this group of studies our work relates more closely to those that allow the relationship between education and health to be nonstationary. This is the case of the "fundamental cause" literature described in Cutler et al. (2006)in which the diffusion of information on technological improvements is an argument used to explain the changes in the education gradient in health.

## 2.2 Empirical Evidence

### 2.2.1 Data

The core of our exercise consists of examining the relationship between education and HIV education over the stages of the HIV epidemic. To address this question it would be ideal to use nationally representative long panel data for several SSA countries starting in the pre-HIV era. Unfortunately, available nationally representative data are neither long nor panel. However, from a macroeconomic perspective, we show it is possible to construct a normalized path of the patterns of HIV infection by education groups over the stages of the epidemic for several SSA countries to recover stylized patterns between education and HIV. To do so, we combine two sources of data: (i) cross-sectional data from the DHS, and (ii) aggregate data from the most recent World Population Prospects (WPP) provided by the United Nations.

Table 2.1: The DHS Sample Characteristics (across Countries)

| (A) Women | Mean | Median | Min. | Max. | Gini |
|---|---|---|---|---|---|
| HIV Prevalence (%) | 6.1 | 4.1 | 0.5 | 31.2 | 0.54 |
| Years of Schooling | 3.2 | 2.8 | 0.6 | 4.8 | 0.26 |
| Age | 28.2 | 28.2 | 27.7 | 29.4 | 0.01 |
| Urban (%) | 32.7 | 33.6 | 10.6 | 88.3 | 0.25 |
| Extramarital Partners, $n$ | 0.15 | 0.17 | 0.01 | 0.56 | 0.37 |
| $n = 0$ (%) | 87.4 | 84.9 | 57.9 | 99.2 | 0.07 |
| $n = 1$ (%) | 11.5 | 14.3 | 0.8 | 35.9 | 0.36 |
| $n = 2$ (%) | 1.0 | 1.2 | 0.0 | 6.3 | 0.47 |
| $n \geq 3$ (%) | 0.1 | 0.1 | 0.0 | 0.0 | 0.66 |
| Frequency of Condom Use (%) | 9.5 | 8.7 | 0.5 | 37.7 | 0.42 |
| Not Sexually Active (%) | 15.6 | 12.8 | 4.3 | 30.7 | 0.28 |

| (B) Men | Mean | Median | Min. | Max. | Gini |
|---|---|---|---|---|---|
| HIV Prevalence (%) | 4.1 | 2.2 | 0.4 | 19.7 | 0.56 |
| Years of Schooling | 3.8 | 3.2 | 1.3 | 5.3 | 0.18 |
| Age | 28.2 | 28.5 | 25.9 | 30.3 | 0.02 |
| Urban (%) | 33.7 | 35.6 | 15.4 | 87.6 | 0.24 |
| Extramarital Partners, $n$ | 0.41 | 0.48 | 0.08 | 1.17 | 0.30 |
| $n = 0$ (%) | 73.7 | 65.7 | 36.8 | 93.5 | 0.14 |
| $n = 1$ (%) | 19.7 | 28.0 | 5.1 | 43.8 | 0.22 |
| $n = 2$ (%) | 4.5 | 5.2 | 0.8 | 14.4 | 0.36 |
| $n \geq 3$ (%) | 0.7 | 0.7 | 0.1 | 2.8 | 0.42 |
| Frequency of Condom Use (%) | 20.5 | 21.4 | 4.2 | 49.3 | 0.31 |
| Not Sexually Active (%) | 21.7 | 17.9 | 7.0 | 35.9 | 0.24 |

*Notes*: The computation of these statistics is performed by using individual HIV weights provided by the DHS to compute the mean. Then, country-specific population weights (i.e., the population size of each country provided by World Population Prospects) are used to compute the statistics across countries. The number of extramarital partners refers to the last 12 months. The frequency of condom use refers to the last sexual intercourse. Our sample is based on 39 DHS with 25 countries and a total of 227,935 women and 174,852 men.

**The Demographic and Health Surveys.** The DHS are based on nationally representative samples and are available for a large set of SSA countries. We consider the full sample of SSA DHS surveys for which individual HIV testing has been conducted (and available as of July 2014): Burkina Faso (2003, 2010), Burundi (2010), Cameroon (2004, 2011), Congo (2007), Côte d'Ivoire (2005, 2011), Democratic Republic of Congo (2007), Ethiopia (2005, 2011), Gabon (2012), Ghana (2003), Guinea (2005, 2012), Kenya (2003, 2008), Lesotho (2004, 2009), Liberia (2007), Malawi (2004, 2010), Mali (2006), Mozambique (2009), Niger (2006), Rwanda (2005, 2010), Senegal (2005, 2010), Sierra Leone (2008), Swaziland (2006), Tanzania (2003, 2007, 2011), Uganda (2011), Zambia (2007) and Zimbabwe (2005, 2010), for a total of 25 DHS countries. For a number of countries the survey was conducted in two consecutive years, so we can exploit variation across 51 country-year pairs, which provide sufficient observational heterogeneity on the stages of the HIV epidemic to obtain reliable estimates of the evolution of the education gradient over these stages.

While the DHS are primarily health interviews, they also contain cross-sectional information on individual socioeconomic characteristics, knowledge on HIV, several measures of risky sexual behavior (e.g., number of extramarital relationships and condom use) and most importantly, a large proportion of adult respondents have been tested for HIV.[3] We use this cross-referenced individual information harmonically collected across SSA countries. Our whole sample consists of a total of 402,670 individuals, of which 56.5% are women. We choose to explore HIV infection risk by education groups separately for women and men. The average HIV prevalence is 6.1% for women and 4.1% for men (respectively, panel A and B, Table 2.1). There is a substantial degree of heterogeneity in HIV prevalence across countries. The Gini index for the HIV prevalence across these 39 DHS (25 countries) is 0.54 for women and 0.56 for men, with a range from 0.5 to 31.2 for women and from 0.4 to 19.7 for men. Note that there is a substantial HIV gender gap of 2% that is roughly half of the total HIV prevalence for men. Interestingly, as we show below, this gender gap in HIV prevalence evolves across the stages of the HIV epidemic. We restrict our attention to HIV-tested adults men and women 15-49 years old who reported their schooling achievement, which is on average 3.2 for women and 3.8 for men. The urban population is roughly one third for both women and men.

Several aspects make DHS datasets appealing for our exercise. An important advantage of these data is that they provide unambiguous individual measures of individual HIV status,

[3]The proportion of respondents who did not take the HIV test is .318 in the original whole sample (.098 and .432 among men and women, respectively). However, we find that the association between the likelihood of taking the HIV test and the educational attainment is virtually zero in the DHS sample. Further, our evidence suggests the DHS non-response bias for HIV testing is minimal. We find that statistics for age, schooling, and residence computed for the sample of HIV-tested adults resemble the analogous ones in the overall male sample. These results are available upon request. See also the discussions in Fortson (2008).

education, knowledge on HIV, and risky sexual behavior in a comparable manner across SSA countries. First, regarding individual HIV status, the DHS provides a direct measure as individuals have blood testing for HIV, so we do not rely on indirect proxy for HIV obtained from other health outcomes or biomarkers. Second, regarding education variables, DHS collect data on education (i.e., number of years of schooling and maximum degree attained) and also provide an asset-based wealth index.[4] Our preferred choice for measuring education is years of schooling—perhaps the most commonly used measure for education in the previous literature. The reasoning for our choice of years of schooling—rather than the DHS wealth index— is that while wealth is influenced by subsequent negative health conditions (such as HIV), or other shocks that will potentially determine one's health status in adulthood, educational attainment is not because, typically, education is completed before individuals in our sample—adults between 15 and 49 years of age—enter adulthood. However, we cannot entirely rule out the fact that investments in education might respond to changes in life expectancy. Indeed, Fortson (2011) suggests a significant negative effect of HIV on investment in children in a model where agents explicitly consider mortality risk when making human capital decisions.

Finally, regarding risky sexual behavior, we focus on (i) the number of sex partners (i.e., the extensive margin) in the past 12 months other than spouses and (ii) condom use in last intercourse (i.e., the intensive margin, quality). The number of extramarital partners is on average 0.15 for women and 0.41 for men (panel A and B, Table 2.1). This is consistent with the population of women not having extramarital partners being larger than men's, respectively 87.4% and 73.7%. The frequency on condom use is 9.5% for women and 20.5% for men. That is, women not only report less extramarital partners than men, but also less condom use in the last sexual intercourse than men. These statements are consistent, as men's last sexual intercourse for casual sex is more common than women's. These figures are conditional on individuals being sexually active. Interestingly, the proportion of women not sexually active is smaller, 15.6%, than the one of men, 21.7%.[5]

**The World Population Prospects.** To uncover the evolution of the HIV epidemic we use the data from the 2015 revision of the World Population Prospects (WPP) constructed by the United Nations (Department of Economic and Social Affairs, Population Division). The WPP 2015 provides estimates of the HIV prevalence rates from 1980 until 2014 (at the country level),

[4]Unfortunately, the DHS do not collect data on income, with very few exceptions that document wage earnings. This task is not easy as many SSA populations are mostly rural and a large proportion of these households' resources come from unsold agricultural production; see a discussion in De Magalhães and Santaeulàlia-Llopis (2015).

[5]The Gini index for the number of extramarital partners across these 39 DHS (25 countries) is 0.37 for women and 0.30 for men, with a range from 0.01 to 0.56 for women and from 0.08 to 1.17 for men. The Gini index for the frequency of condom use in last sexual intercourse is 0.42 for women and 0.31 for men, with a range from 0.5% to 37.7% for women and from 4.2% to 49.3% for men (Table 2.1).

and their projections from 2014 onward for a large set of SSA countries. [6] The additional data on country-specific ART coverage used in our robustness exercise are also from the WPP.[7]

Finally, for all our SSA countries we use data on real output per capita from the Penn World Tables and data on agricultural share of output from the World Bank Development Indicators. We use these data in our empirical analysis to control for country-specific stages of aggregate economic development.

### 2.2.2 The Stages of the HIV Epidemic

This section describes the stages of the HIV epidemic. First, we discuss a set of challenges that we argue a useful definition of the stages of the HIV epidemic must address (Section 2.2.2). From our macroeconomic perspective these challenges arise from country differences in HIV prevalence across time (i.e., the evolution of the HIV epidemic within a country) and across space (i.e., heterogeneity of HIV prevalence across countries within a given period). Second, we provide an algorithm that circumvents those challenges by normalizing the HIV epidemic in both dimensions, time and space (Section 2.2.2). Our definition is provided in Section 2.2.2.

[6] The WPP data represent the official 2014 estimates of UNAIDS. Until 2006 the UNAIDS estimates relied mostly on data aggregations collected from antenatal clinics that overestimated prevalence levels. Since 2008, the UNAIDS data belong to a downward revision largely originated by the appearance of nationally representative surveys such as the DHS, and do not suffer from overestimation problems. Indeed, the HIV prevalence levels computed from our DHS samples and the HIV prevalence levels from WPP are very similar. See also a detailed discussion of these data in Bongaarts et al. (2008).

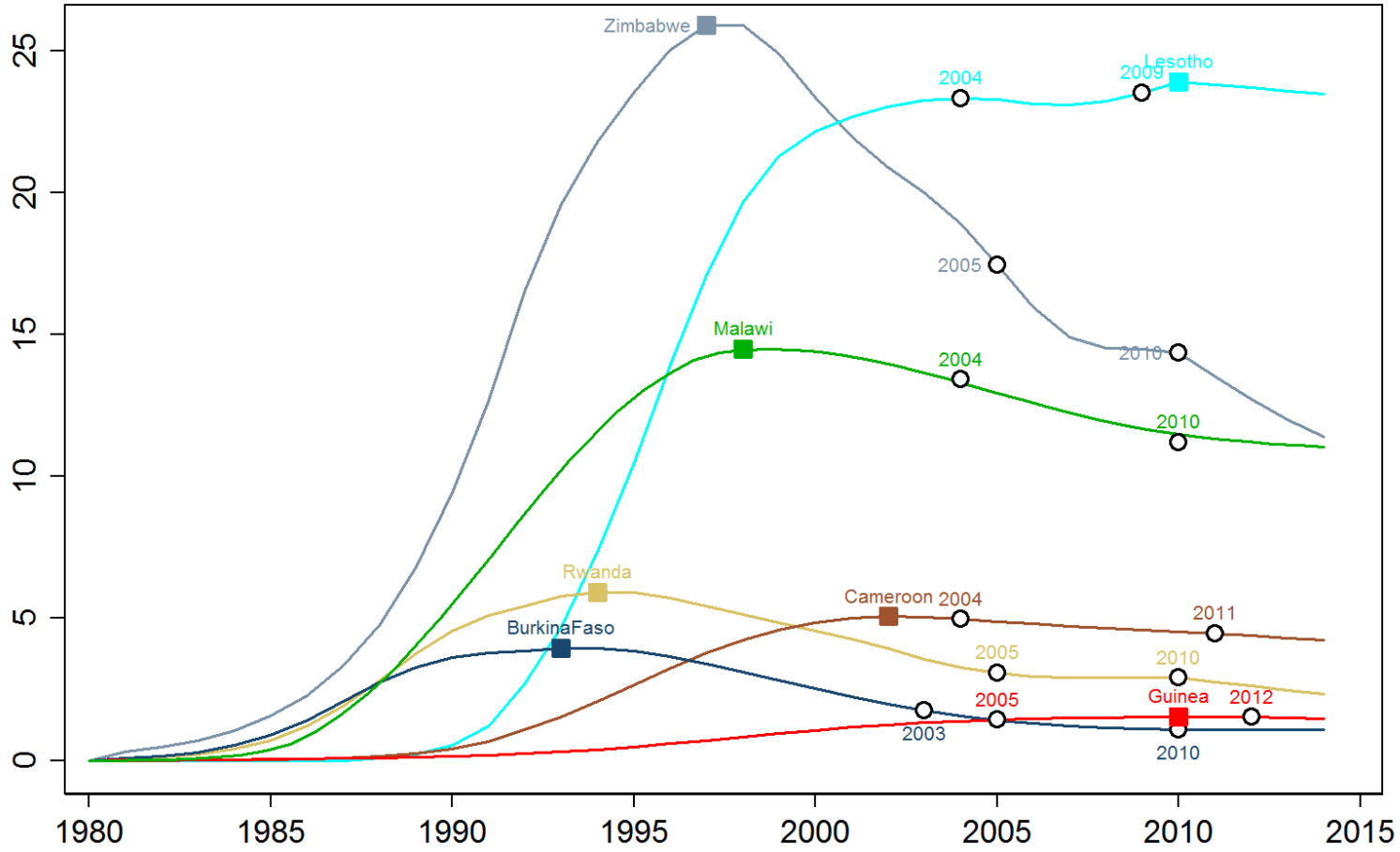
[7] Source: United Nations, Department of Economic and Social Affairs, Population Division: World Population Prospects. Unpublished Data - Special Tabulations. We thank Patrick Gerland for sharing these data.

Table 2.2: The Evolution of the HIV Epidemic across Sub-Saharan Countries: The DHS Sample

| | DHS Obs. | | Peak | | DHS/Peak | |
|---|---|---|---|---|---|---|
| Country | $t_i$ | $HIV_i$ | $t_{i,*}$ | $HIV_{i,*}$ | $t_i - t_{i,*}$ | $\frac{HIV_i}{HIV_{i,*}}$ |
| Burkina Faso | 2003 | 1.76 | 1993 | 3.96 | -10 | 0.44 |
| Burkina Faso | 2010 | 1.10 | 1993 | 3.96 | -17 | 0.27 |
| Burundi | 2010 | 1.39 | 1996 | 4.47 | -14 | 0.31 |
| Cameroon | 2004 | 4.98 | 2002 | 5.06 | -2 | 0.98 |
| Cameroon | 2011 | 4.47 | 2002 | 5.06 | -9 | 0.88 |
| Congo Brazaville | 2009 | 2.74 | 1995 | 4.80 | -14 | 0.57 |
| Cote d'Ivoire | 2005 | 4.79 | 1999 | 6.18 | -6 | 0.77 |
| Cote d'Ivoire | 2011-12 | 3.41 | 1999 | 6.18 | -12 | 0.55 |
| D.R. Congo | 2007 | 1.30 | 2001 | 1.43 | -6 | 0.91 |
| Ethiopia | 2005 | 2.28 | 1999 | 3.23 | -6 | 0.70 |
| Ethiopia | 2011 | 1.32 | 1999 | 3.23 | -12 | 0.41 |
| Gabon | 2012 | 4.16 | 2003 | 5.53 | -9 | 0.75 |
| Ghana | 2003 | 2.00 | 2000 | 2.15 | -3 | 0.93 |
| Guinea | 2005 | 1.43 | 2010 | 1.54 | 5 | 0.93 |
| Guinea | 2012 | 1.52 | 2010 | 1.54 | -2 | 0.98 |
| Kenya | 2003 | 7.08 | 1996 | 9.16 | -7 | 0.77 |
| Kenya | 2008-09 | 5.84 | 1996 | 9.16 | -12 | 0.64 |
| Lesotho | 2004-05 | 23.33 | 2010 | 23.91 | 6 | 0.98 |
| Lesotho | 2009-10 | 23.52 | 2010 | 23.91 | 1 | 0.98 |
| Liberia | 2006-07 | 1.34 | 2002 | 1.84 | -5 | 0.73 |
| Malawi | 2004 | 13.41 | 1998 | 14.99 | -6 | 0.89 |
| Malawi | 2010 | 11.21 | 1998 | 14.99 | -12 | 0.75 |
| Mali | 2006 | 1.34 | 1999 | 1.71 | -7 | 0.78 |
| Mozambique | 2009 | 9.98 | 2008 | 10.01 | -1 | 1.00 |
| Niger | 2006 | 1.00 | 2002 | 1.20 | -4 | 0.83 |
| Niger | 2012 | 0.60 | 2002 | 1.20 | -10 | 0.50 |
| Rwanda | 2005 | 3.09 | 1994 | 5.93 | -11 | 0.52 |
| Rwanda | 2010 | 2.90 | 1994 | 5.93 | -16 | 0.49 |
| Senegal | 2005 | 0.90 | 2004 | 0.90 | -1 | 1.00 |
| Senegal | 2010-11 | 0.70 | 2004 | 0.90 | -6 | 0.78 |
| Sierra Leone | 2008 | 1.66 | 2008 | 1.66 | 0 | 1.00 |
| Swaziland | 2006-07 | 23.36 | 2010 | 24.46 | 4 | 0.95 |
| Tanzania | 2003-04 | 6.04 | 1996 | 7.36 | -7 | 0.82 |
| Tanzania | 2007-08 | 5.30 | 1996 | 7.36 | -11 | 0.72 |
| Tanzania | 2011-12 | 4.81 | 1996 | 7.36 | -15 | 0.65 |
| Uganda | 2011 | 6.55 | 1991 | 12.62 | -20 | 0.52 |
| Zambia | 2007 | 12.87 | 1998 | 14.77 | -9 | 0.87 |
| Zimbabwe | 2005-06 | 17.46 | 1997 | 25.90 | -8 | 0.67 |
| Zimbabwe | 2010-11 | 14.35 | 1997 | 25.90 | -13 | 0.55 |

Notes: $t_i$ is the calendar year of DHS data collection for country $i$; $HIV_i$ is the prevalence rate for country $i$ the year of DHS data collection; $t_{i,*}$ is the year country $i$ reaches its HIV prevalence peak; $HIV_{i,*}$ is the peak prevalence rate for country $i$. Sources: United Nations, Department of Economic and Social Affairs, Population Division: World Population Prospects: The 2015 Revision, Medium-Variant Estimation and Projection.

Figure 2.1: Challenges for the Definition of Stages of the HIV: Epidemic. The Evolution of the HIV Epidemic for a DHS Subsample.




*Source*: United Nations, Department of Economic and Social Affairs, Population Division: World Population Prospects: The 2015 Revision, Medium-Variant Estimation. *Notes*: The solid square on each HIV time path displays the HIV prevalence at the peak year, and the open black circle on each HIV time path displays the HIV prevalence at the year that the DHS data were collected.

### Challenges for a Definition of the Stages of the HIV Epidemic

The evolution of the HIV epidemic is largely heterogeneous. To illustrate this, we show in Figure 2.1 the country-specific time path of the epidemic for a selected subsample of countries.[8] In addition, we provide country-specific statistics of the HIV epidemic for our entire sample of 39 DHS country-year surveys (Table 2.2). The following patterns arise across time and space.

**HIV Prevalence Differences Across Space** While the HIV prevalence levels largely differs across countries (columns 1 and 2, Table 2.2),[9] two countries with the same HIV prevalence are not necessarily at the same epidemiological stage.

**Remark 1.** *The HIV prevalence alone is not sufficient to define the aggregate stage of the HIV epidemic.*

We show this argument with two straightforward counterexamples. Although Malawi in 1998 and Zimbabwe in 2010 both share the same HIV prevalence of 14.4, Malawi reaches this infection rate at its HIV peak while Zimbabwe reaches it only 13 years after its HIV peak (Figure 2.1). Indeed, in 2010 Zimbabwe's HIV prevalence is 55% of its HIV peak prevalence (column 6, Table 2.2). Another interesting counterexample arises from the comparison of the DHS observations of Zimbabwe and Lesotho. The DHS observation of Zimbabwe in 2005 delivers an HIV prevalence of 19.2%, lower than that of Lesotho in 2004, 23.4%. Looking at this statistic only, we would infer that Lesotho is at later stages of the epidemic than Zimbabwe. However, we actually know that Zimbabwe's HIV peak occurred at a higher level and earlier, 29.1% at 2009, than that of Lesotho, 23.8% at 2007, which suggests an opposite ordering over stages. That is, the ordering of DHS countries by HIV prevalence is a mere artifact of the years in which DHS were collected.

One step to address the problematic use of the absolute size of the HIV prevalence as a measure of the stages of the epidemic is to compute relative size of HIV prevalence dividing country-specific observations of HIV prevalence by their corresponding HIV peaks. However, this poses a new set of drawbacks because countries not only differ in the HIV peak level but also in the year of their HIV peak (columns 3 and 4, Table 2.2).

[8] This subsample of DHS countries consists of Burkina Faso, Cameroon, Guinea, Lesotho, Malawi, Rwanda and Zimbabwe. This subsample serves expositional purposes only as it is useful to highlight the heterogeneity of the evolution of the HIV epidemic across countries as we describe next. Many other subsample choices would be equally useful.

[9] For example, in year 2010, the HIV prevalence (in percentages) is 1.1 in Burkina Faso, 5.1 in Cameroon, 1.9 in Guinea, 11.7 in Malawi, 3.1 in Rwanda and 18.0 in Zimbabwe. Across all SSA countries, the inequality in HIV prevalence remains high across time with a Gini coefficient of 0.63 in 1990, 0.56 in 2000 and 0.57 in 2010. The SSA set consists of 44 countries. Similar figures are attained with our sample of 25 DHS countries with Gini's coefficients of 0.55 in 1990, 0.51 in 2000 and 0.55 in 2010.

**Remark 2.** *The relative HIV prevalence alone is not sufficient to define the aggregate stage of the HIV epidemic.*

To see this, note, for example, that while the DHS observations of Guinea in 2005 and Ghana in 2003 share the same relative HIV prevalence of .93 (column 6, Table 2.2), Guinea attains that relative size 5 years before reaching its peak, and Ghana does so 3 years after reaching its peak (column 6, Table 2.2). This observation suggests that two countries can be at different stages of the epidemic despite having the same relative HIV prevalence. Another interesting example is the one posed by the DHS observations of Rwanda 2005 and Uganda 2011. Both surpassed their respective peaks, and have the same relative prevalence level of .52 (column 6, Table 2.2). However, it took Rwanda 11 years to move from its peak to this relative prevalence, while it took Uganda almost twice as much time, 20 years, to reach the same relative prevalence (column 5, Table 2.2). The fact that the transition away from the peak is slower in Uganda than in Rwanda is, in itself, a phenomenon to which we would like our definition of the stages of the epidemic to be invariant. Constructively, the arguments posed here against the sole use of the absolute (or the relative) HIV prevalence to define stages of the epidemic also suggest what we need to add to our definition of the stages to resolve the exposed problems: some properties of the time path of the HIV epidemic.

**HIV Prevalence Differences Across Time** The time-path of HIV epidemic largely differs across countries. In particular, a large degree of heterogeneity exists for the HIV peak year across SSA countries (column 3, Table 2.2). In our DHS sample, the peak of the HIV year ranges from 1991 in Uganda to 2010 in Guinea, Lesotho and Swaziland. This leads to the following remark.

**Remark 3.** *Time (calendar year) alone is not sufficient to define the aggregate stage of the HIV epidemic.*

This remark states that two countries that suffer the HIV epidemic are not necessarily at the same epidemiological stage at the same calendar year. This is straightforward. Lesotho and Guinea reach the peak of their respective HIV epidemic in 2010, while Zimbabwe is at more advanced stage of its epidemic in 2010, precisely 13 years ahead of its HIV peak in 1997 (Figure 2.1).

One step to correct for the country-specific year of the HIV peak is to compute the relative time, i.e., calendar year minus year of HIV peak (column 5, Table 2.2). However, a large degree of heterogeneity exists for the speed by which SSA countries move to the respective HIV peaks and the speed by which SSA countries move away from their respective HIV peaks. This leads to the following remark.

**Remark 4.** *Relative time (calendar year minus year of HIV peak) alone is not sufficient to define*

*the aggregate stage of the HIV epidemic.*

To see this, note that the DHS observations of Ethiopia in 2011 and Malawi in 2010 both share the same time distance with respect to their own HIV; in both cases 12 years have passed between the peak and the DHS data collection. However, in those 12 years Ethiopia has managed to decrease its relative HIV prevalence to 0.41 (column 6, Table 2.2), while Malawi has only managed to decrease its relative HIV prevalence to 0.75. Again, as we noted for remark 2, the fact that the transition away from the peak of Malawi is slower than that of Ethiopia is, in itself, a phenomenon to which we would like our definition of the stages of the epidemic to be invariant. The relative time does not suffice to define stages of the HIV epidemic.

To address these four remarks at once, we propose a two-dimensional (2D) algorithm that normalizes both the HIV prevalence level and time.

### A Two-Dimensional Normalization of the Evolution of the Epidemic Across Time and Across Space

This section builds a 2D algorithm that, for all countries, normalizes the country-specific level and time path of the epidemic, thereby making the evolution of the HIV epidemic comparable across countries. Once the evolution of the epidemic is normalized for all countries, the position of each DHS dataset on its associated epidemiological stage readily follows.

**Algorithm 1. [A Two-Dimensional Normalization of the Evolution of the HIV Epidemic]** *Given the time series of the level of HIV prevalence of each $i$, we follow three steps to conduct a 2D normalization of the level and time path of the HIV epidemic:*

1. *Interpolate the country-specific time path of prevalence for each country $i$, $\{\lambda_{i,t}\}_{t_0}^{t_p}$, for $p+1$ interpolation points (years), where $p$ is a positive integer. Then, interpolate the aggregate (across countries) prevalence path as $\lambda_t = \frac{\sum_i^n \lambda_{i,t}\mu_{i,t}}{\sum_i^n \mu_{i,t}}$, where $n$ is the total of number of countries and $\mu_{i,t}$ is the population level of country $i$ at period $t$. Denote the country-specific interpoland function as $s_i : t \to [0, \max_t \lambda_{i,t}]$, where $\max_t \lambda_{i,t} \in [0,1]$ and $s_i \in \mathcal{S}$, where $\mathcal{S}$ is the collection of functions that can be written as a linear combination of a set of $n$-known linearly independent basis functions $\psi_j$, $j = 1, ..., n$,*

$$s_i(t) = \sum_{j=1}^{n} \theta_j \, \psi_j(t)$$

*with $n$ unknown $\theta_j$ coefficients. Denote the aggregate interpoland as $s(t)$ where $s(t)$*

*shares the same properties as the country-specific interpolands $s_i(t)$. Importantly, note that $\max_t s_i(t)$ is not necessarily identical across countries or to the aggregate $\max_t s(t)$.*

2. *Level normalization*

   (a) *Compute the country-specific peak prevalence,*

   $$s_i(t_*^i) = \max_t s_i(t), \tag{2.1}$$

   *where $t_*^i = \arg\max_t s_i(t)$ is the period country $i$ reaches its peak, $s_i(t_*^i)$. Redo equation (2.1) to obtain the aggregate peak $s(t_*)$ and aggregate peak period, $t_* = \arg\max_t s(t)$.*

   (b) *Normalize the country-specific and aggregate interpolands by their respective peak prevalence,*

   $$\widetilde{s}_i(t) = \frac{1}{s_i(t_*^i)}\, s_i(t) \quad \textit{and} \quad \widetilde{s}(t) = \frac{1}{s(t_*)}\, s(t),$$

   *where $\widetilde{s}_i, \widetilde{s} : t \to \Lambda = [0,1]$ and $\arg\max_t s_i(t) = t_*^i = \arg\max_t \widetilde{s}_i(t)$. Note now that $\widetilde{s}_i(t_*^i) = \widetilde{s}(t_*) = 1\ \forall i$.*

3. *Time normalization*

   (a) *For $t_0^i < t^i \leq t_*^i$, normalize the time interval between the initial period for which data are available, $t_0^i = 1980$, and the country-specific peak period, $t_*^i$, by the time interval between the aggregate initial period, $t_0 = 1980$, and the aggregate peak period, $t_*$. To do so, we compute the constant of time proportionality for the pre-peak era,*

   $$\alpha_i^L = \frac{t_* - t_0}{t_*^i - t_0^i}.$$

   *For $t^i > t_*^i$, normalize the time between the peak period, $t_*^i$, and the period $t_\gamma^i$ in which country $i$ reaches a given threshold $\gamma \in [0,1]$, that is, $t_\gamma^i = \widetilde{s}_i^{-1}(\gamma)$, over the analogous aggregate interval with $t_*$ and $t_\gamma = \widetilde{s}^{-1}(\gamma)$,*

   $$\alpha_i^R(\gamma) = \frac{t_\gamma - t_*}{t_\gamma^i - t_*^i}.$$

   *Here, note that $t_\gamma^i$ and $t_\gamma$ may not occur at an interpolation node but elsewhere along their respective interpoland.*

(b) *Normalize the time input of the country-specific interpolands by* $\alpha_i^L$ *and* $\alpha_i^R$,

$$\tau = \alpha_i^L(t - t_*^i) \quad \textit{for} \quad t \leq t_*^i \tag{2.2}$$

$$\tau = \alpha_i^R(t - t_*^i) \quad \textit{for} \quad t > t_*^i \tag{2.3}$$

*where* $\tau \in T$ *are the normalized units of time. Operations (2.2) and (2.3) compress/stretch the interpoland*[10] *to ensure that for* $\tau \leq \tau_*$ *(before the peak) the number of normalized periods* $\tau$ *that it takes each country to move from* $\tau_0$ *to the peak are the same across countries,*

$$\widetilde{s}_i^{-1}(1) - \widetilde{s}_i^{-1}(0) = \widetilde{s}_j^{-1}(1) - \widetilde{s}_j^{-1}(0) = \widetilde{s}^{-1}(1) - \widetilde{s}^{-1}(0) \qquad \forall\, i, j\ ,$$

*and for* $\tau > \tau_*$ *(after the peak) the normalized periods* $\tau$ *that it takes each country to move from the peak to a threshold of prevalence* $\gamma$ *is the the same across countries,*

$$\widetilde{s}_i^{-1}(\gamma) - \widetilde{s}_i^{-1}(1) = \widetilde{s}_j^{-1}(\gamma) - \widetilde{s}_j^{-1}(1) = \widetilde{s}^{-1}(\gamma) - \widetilde{s}^{-1}(1) \qquad \forall\, i, j.$$

*This allows us to define the evolution of the epidemic for each country and the aggregate,*

$$\widetilde{s}_i : \tau \to \Lambda \qquad \textit{and} \qquad \widetilde{s} : \tau \to \Lambda, \tag{2.4}$$

*in the same—hence comparable—2D normalized space* $(T, \Lambda)$.

To implement the algorithm we need to make two choices: the shape of the basis functions, $\psi(\tau)$, and the prevalence threshold $\gamma$ for the time normalization after the peak. First, we specify $\widetilde{s}(\tau)$ as a B-spline with cubic pieces and solve for the $\theta_j$ coefficients accordingly. Our choice of splines as interpolands obeys our desired manageability of the interpoland given the size of the Lagrangian interpolation problem poised by 71 (1980-2050) interpolation data points. Second, our choice of $\gamma$ responds to balance between minimizing the use of projection of U.N. data for our set of DHS countries and maximizing the number of countries that have already surpassed the threshold $\gamma$ at the time of DHS data collection. Our search for this balance suggests a value of $\gamma = .8$. This choice of $\gamma$ implies that more than half of the countries in our dataset have already passed the threshold. Our results are robust to alternative choices of $\gamma$. To see this, note that the value of $\gamma$ does not alter the ranking of countries across stages of the epidemic.

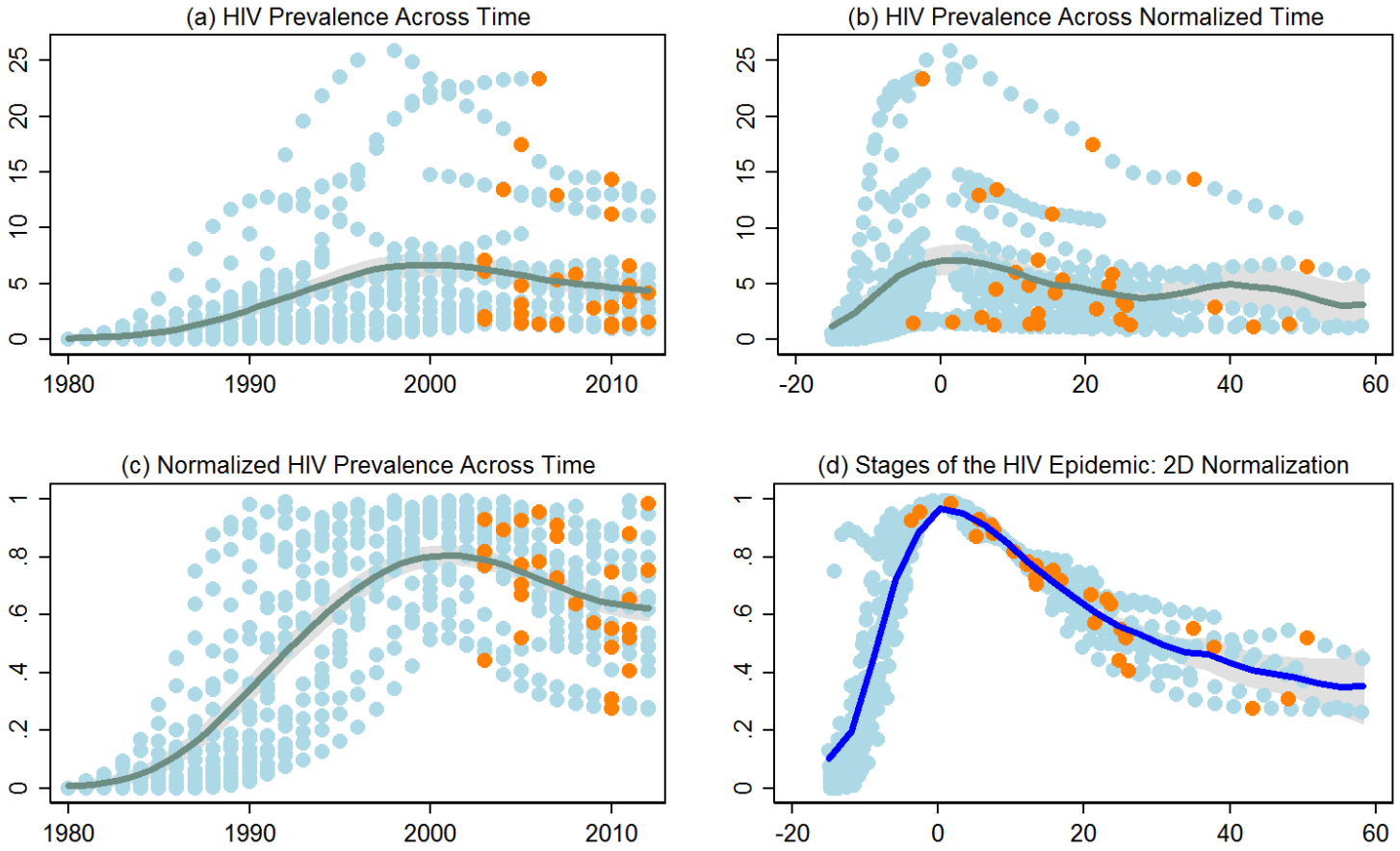
We next apply our algorithm to the SSA countries for which U.N. estimates and projections

[10] The interpoland $s_i$ horizontally compresses when $\alpha_i^k > 1$ with $k = \{L, R\}$ and expands otherwise.

of the HIV prevalence time path are available.[11] The results are depicted in Figure 2.2. Panel (a) shows the HIV prevalence level across time for each and all countries. We highlight (in orange) the country-year observations for which DHS data with HIV testing results are available. This panel portrays all challenges discussed in Section 2.2.2. Panel (b) shows the result of the first normalization of our algorithm, the HIV prevalence level normalization, which tackles the issues associated with the absolute HIV prevalence, but does not correct for having HIV peaks at different calendar years for different countries. Panel (c) shows the result of the second normalization of our algorithm, the time normalization, which forces all countries to peak at the same calendar year but does not resolve the issues associated with the absolute HIV prevalence level. Finally, panel (d) closes our algorithm by jointly applying the level and the time normalizations. These 2D normalization resolves the full set of challenges posed in Section 2.2.2 as the relative HIV prevalence peaks in all countries at the same period. As it is obvious from panel (d), after the 2D normalization the level and time path of the epidemic are entirely comparable across countries.

[11] The interpolation Lagrangian points, that is, the country-specific prevalence time series, $\lambda_{i,t}$, are retrieved from the U.N. population division estimates and projections until 2050 (medium-variant).

Figure 2.2: Defintion of Stages of the HIV Epidemic




*Source*: Outcome of our 2D-normalization algorithm (Subsection 2.2.2) implemented using WPP, 2015, data for SSA. The vertical axis in the top panels is HIV prevalence. The vertical axis in the bottom panels is normalized HIV prevalence. The horizontal axis in the left panels is time. The horizontal axis in the right panels is normalized time. This way, the 2D-normalization is operative in panel (d). In each panel, the orange markers in the scatterplots represent a DHS dataset. The plotted trends are locally weighted polynomials with 95% confidence intervals.

### A Definition of the Stages of the HIV Epidemic

Note that the position of each country $i$ on its normalized HIV time path at the period $t_{DHS}$ at which its respective DHS data were collected can be easily computed by solving for $\tau_i$ in

$$\widetilde{s}_i^{-1}\left(\frac{\lambda_{i,t_{DHS}}}{s_i(t_*^i)}\right) = \tau_i.$$

Then, the stage of the HIV epidemic is the continuous real variable,

$$\omega(\tau,\zeta) = \frac{\zeta}{\tau - \tau_*} \to \mathcal{R}^1, \tag{2.5}$$

with the pair $(\tau,\zeta)$ belonging to the 2D normalized space, $T\times\Lambda$. Geometrically, $\omega(\tau,\zeta)$ represents the slope of the arrays from the origin in the $(T,\Lambda)$ space with the following limiting properties:

$$\lim_{\tau\to\tau_*^-}\omega(\tau,\zeta) = -\infty,\ \lim_{\tau\to\tau_*^+}\omega(\tau,\zeta) = \infty,\ \text{ and }\ \lim_{\tau\to-\infty}\omega(\tau,\zeta) = \lim_{\tau\to+\infty}\omega(\tau,\zeta) = 0.$$

To conduct our empirical exercise, we discretize the continuous variable that defines the epidemiological stages in (2.5).[12]

**Definition 1. [Stages of the HIV Epidemic]** *Given a set of stage thresholds* $\{\zeta_0,...,\zeta_j,...,\zeta_n\}$ *with* $\zeta_j > \zeta_{j+1}$ *for all* $j$*, the stage* $j$ *of the HIV epidemic consists of all pairs* $(\tau,\zeta)\in T\times\Lambda$ *such that* $\omega(\widetilde{s}^{-1}(\zeta_{j+1}),\zeta_{j+1}) \leq \omega(\tau,\zeta) \leq \omega(\widetilde{s}^{-1}(\zeta_j),\zeta_j)$*, where* $\widetilde{s}(\tau)$ *is the normalized (population-weighted) aggregate of the HIV epidemic defined in (2.4).*

Our choice for the stage thresholds $\{\zeta_0,...,\zeta_j,...,\zeta_n\}$ pursues the maximization of both countries per stage of the epidemic and number of stages. To do so, we set $\zeta_0 = 1$ and $\zeta_j = \zeta_1 - .05j\ \forall j$. The results of this exercise are shown in Figure 2.3 where each data point $(\tau_i,\widetilde{s}_i)$ represents a DHS dataset. This implies the following allocation of DHS datasets, $\omega(\tau_i,\widetilde{s}_i(\tau_i))$, over stages of the epidemic as follows:

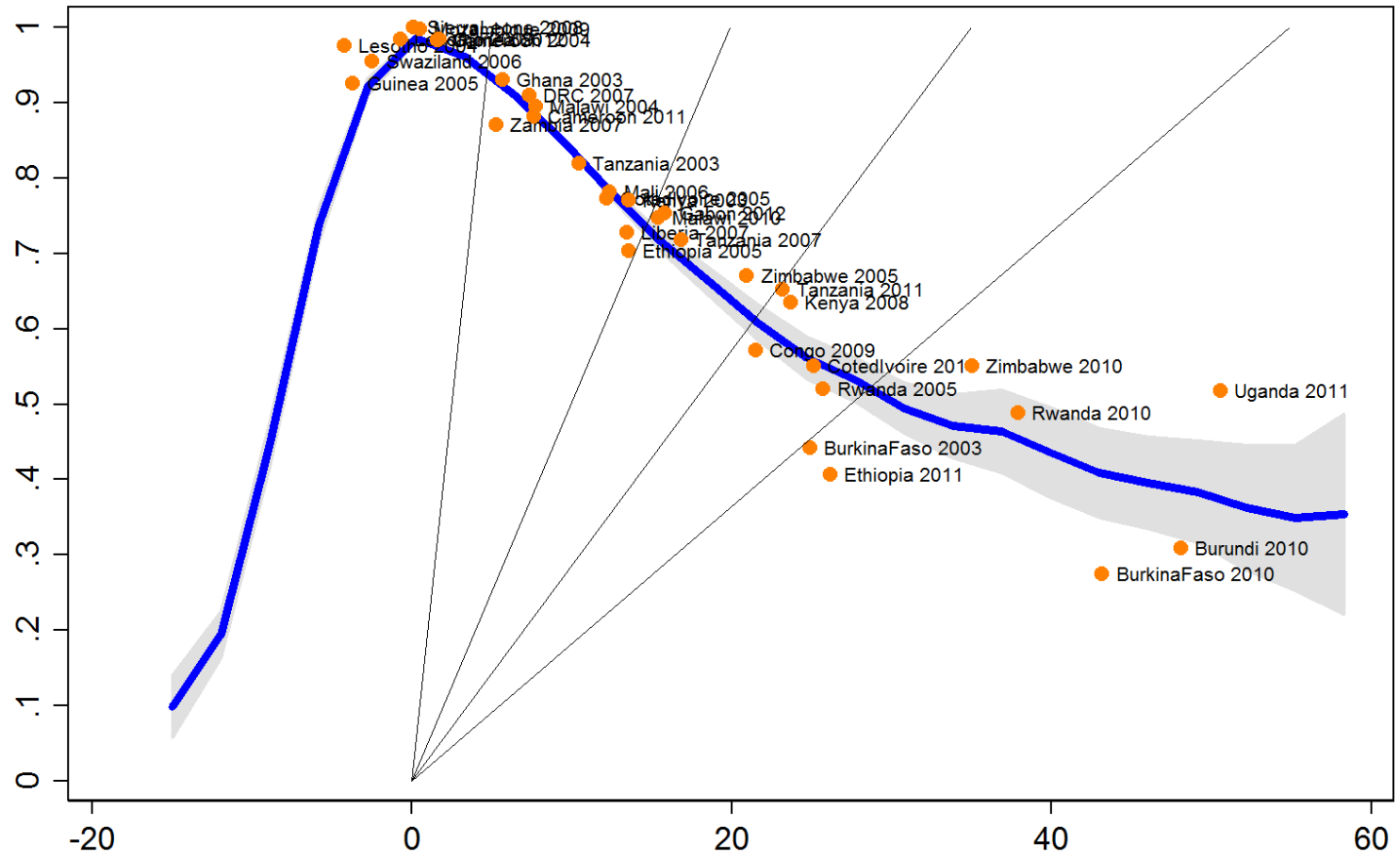
- Stage $\leq$0: Cameroon 2004, Guinea 2005, Guinea 2012, Lesotho 2004/05, Lesotho 2009/10, Mozambique 2009, Senegal 2005, Sierra Leone 2008, Swaziland 2006/07.
- Stage 1: Cameroon 2011, Cote d'Ivoire 2005, Democratic Republic Congo 2007, Ethiopia

[12] To capture the evolution of the HIV-Education gradient over the HIV epidemic we need to interact education with either some specific function (e.g., a quadratic or cubic polynomial) of $\omega(\tau,\zeta)$ or with a discretized version of $\omega(\tau,\zeta)$. We prefer to follow the latter approach and partition the continuous variable $\omega(\tau,\zeta)$ into the discrete pieces which delivers a cleaner interpretation of the estimates of the HIV-Education gradient across stages.

2005, Ghana 2003, Kenya 2003, Liberia 2006/07, Malawi 2004/05, Mali 2006, Niger 2006, Senegal 2010/11, Tanzania 2003/04, and Zambia 2007.

- Stage 2: Gabon 2012, Malawi 2010, Tanzania 2007/08, Zimbabwe 2005.
- Stage 3: Congo Brazaville 2009, Cote d'Ivoire 2011, Kenya 2008/09, Rwanda 2005, Tanzania 2011/12, Zimbabwe 2006.
- Stage $\geq$4: Burkina Faso 2003, Burkina Faso 2010, Burundi 2010, Cote d'Ivoire 2012, Ethiopia 2011, Niger 2012, Rwanda 2010/11, Uganda 2011 and Zimbabwe 2010/11.

Figure 2.3: Stages of the HIV Epidemic: DHS Sample




*Source*: The plot shows the location of SSA Countries (DHS Sample) on the 2D-normalized space at the time of DHS data collection. The vertical axis is the normalized HIV prevalence, and the horizontal axis is the normalized time (Subsection 2.2.2). The orange markers in the scatterplots represent a DHS dataset. The plotted trends are locally weighted polynomials with 95% confidence intervals.

Figure 2.3 depicts these allocations in two dimensions in the normalized space $T \times \Lambda$. It is identical to panel (d) in Figure 2.2 with the addition of the arrays initiating from the origin that define the breakdown of discrete stages of the HIV epidemic defined above. It is relevant to note that the DHS observations covers the entire evolution of the HIV epidemic except its initial rise, which is not surprising given that the first DHS surveys with HIV testing were conducted in 2003. In any case, the DHS observations provide a large degree of heterogeneity across country positions over the normalized HIV epidemic. The large heterogeneity across stages of the HIV epidemic is sufficient to provide reliable nonstationary estimates of the HIV-Education gradient.[13]

**Discussion**

The normalization procedure that we have proposed to define stages of the HIV epidemic is drawn upon ideas developed by macroeconomists to define the stages of the demographic transition and the stages of economic development.

As it is the case with the HIV epidemic, the demographic changes behind the demographic transition occur at different calendar years and at different speeds for different countries. To take this into account, the demographic model controls for calendar year and speed of stage completion with a time-normalization analogous to the one we propose, and in turn defines the demographic stages in a similar way to ours. Precisely, many countries have gone (or as still going) through a first demographic stage of high mortality and fertility, followed by a second demographic stage in which mortality drops and, with some period lag, fertility declines, and finally a third demographic stage of low mortality and low fertility (Lee, 2003). This description of the stylized joint behavior of mortality and fertility exemplifies how the calendar year, the speed at which each country completes each demographic stage, and the country-specific levels of the variables of interest (mortality and fertility) needs to be controlled for to recover stylized facts across stages of the demographic transition.

With regard to the aggregate stages of economic development (or structural transformation out from agriculture), the heterogeneity across time and across space depends on the fact that countries take off at different calendar years and move across stages of economic development at different speeds (Gollin et al., 2002b, 2007; Hansen and Prescott, 2002). For example, China's income per capita raised by a factor of 6 between 1989 and 2009, while it took the U.K. from 1820 to 1970 to generate such growth. That is, China has grown at roughly 7.5 times the speed of the first industrial revolution (Bolt and van Zanden, 2013). With a definition of the stages of

[13]For the country of Niger and Senegal, we lack of projected paths, and therefore we cannot implement the time-normalization. We allocate the DHS surveys of these countries across stages by visual inspection. We find reassuring that the exclusion of Niger and Senegal from our analysis does not alter our results.

development that nets out calendar years and speed, we can study stylized economic relationships along the process of economic development such as the skill wage premium (Buera et al., 2015).

In sum, the spirit of our exercise is similar to what has been done to describe the stages of demographic transitions and the stages of economic development. First, we provide an algorithm that defines aggregate stages of the HIV epidemic netting out the calendar year effects, the country-specific speeds of the HIV epidemic, and the HIV prevalence levels. Second, we study the stylized dynamic relationship between HIV status and education across stages of the HIV epidemic.

### 2.2.3 The HIV-Education Gradient

Our empirical analysis consists of posing a simple econometric specification suitable for documenting the potentially nonstationary behavior of the HIV-Education gradient.

#### Econometric Specification

We consider a linear probability model (LPM) where the HIV-Education gradient is allowed to change over the stages of the HIV epidemic ($j$) defined in Section 2.2.2. Since for a large set of countries there are at least two cross-sections in our sample, and individuals in different periods are not the same people for each country ($g$), we index the variables by a double subscript. Namely, $i(t) \in \{ 1, ..., N_t\}$ denotes the individuals in cross-section $t$. Let $s_{i(t),t,j}$ denote the educational attainment of an individual $i(t)$ that lives in stage $j$ of the HIV epidemic at time $t$. Also, let $y_{i(t),t,j}$ be the individual's HIV status, a dummy variable equal to one if the individual HIV testing result is positive and zero otherwise. We estimate a linear projection of the type,

$$y_{i(t),t,j} = \alpha_0 + \sum_{j>0} \alpha_j \mathbf{1}_j + \left( \gamma_0 + \sum_{j>0} \gamma_j \mathbf{1}_j \right) s_{i(t),t,j} + \beta\, x_{i(t),t} + \psi\, m_{g,t} + \theta_t \mathbf{1}_t + \theta_g \mathbf{1}_g + \varepsilon_{i(t),t}, \quad (2.6)$$

where $\mathbf{1}_j$ is an indicator function that is equal to one when the stage of the HIV epidemic is $j$ and zero otherwise. That is, if the stage of the epidemic is $j = 0$ then the intercept is $\alpha_0$ and the slope is $\gamma_0$. However, for each the stage of the epidemic is $j > 0$, the associated intercept is $(\alpha_0 + \alpha_j)$ and the slope is $(\gamma_0 + \gamma_j)$. Namely, $\gamma_j$ is the difference in the HIV-Education gradient between individuals that are in stage $j$ and stage $0$ of the epidemic. This implies that the HIV-Education gradient is $\gamma_0 + \gamma_j$ for each epidemiological stage $j$. We cluster the individual observations at the country level to account for any unobserved shock that correlates observations within a country. Given that the number of countries is 25, we use the wild cluster bootstrap from Cameron et al.

(2008) to get better approximations to asymptotically valid standard errors.[14]

The vector $x_{i(t),t}$ corresponds to a set of individual characteristics that are likely to be correlated with both education and HIV status. Hence, controlling for these characteristics reduces the impact of omitted variables bias. Precisely, we find it is important to control for the type of area in which agents live because the HIV prevalence is, on average, higher in urban than in rural areas (respectively, 6.73% and 4.41% in our whole sample), and it is in urban areas where adult education levels are also higher (the average number of years of schooling in rural area is 3.23, while in urban areas reaches 3.83). Thus, a positive association between education and HIV may be driven by the fact that people living in urban areas are both more likely to be HIV-positive and more educated. Similarly, we also control for age because HIV prevalence is increasing with age (the DHS age sample is 15-49), and education is negatively correlated with age as younger cohorts are more educated than older cohorts.

We also control for time varying country-specific economic variables, $m_{g,t}$, which correct for the stage of economic development in which each country is. To do so, we use measures of output per capita and share of agricultural output following the literature on structural transformation. We also include year dummies ($\theta_t$) and country dummies ($\theta_g$) to pick up any spurious correlation between the regressors and the dependent variable. To the extent that such contextual effects affect all individuals in a country in a similar manner, the country dummies will sweep them up. All our specifications are weighted least squares regressions, where the weights are proportional to the relative population size of each country. By doing so, when we pool a number of countries in the same stage of the HIV epidemic, the relative DHS sample size of a given country corresponds to the relative population size of the country. We then combine these weights with the individual weights provided by the DHS surveys.

### Results

We first consider a linear probability model where the dependent variable is the individual HIV status. The estimates of the HIV-Education gradient using the whole sample are reported in Table 2.3. We then re-conduct our analysis separately for women and men.

[14] The results are robust to clustering at the country-year level. While we believe it would be interesting to explore also the within-country variation (e.g., across regions), it is not feasible to recover the epidemiological stages using our algorithm proposed in section 2.2.2 due to data limitations about the evolution of the HIV epidemic at the regional level; recall that the algorithm would require complete time-series of HIV prevalence for each region within a country and these estimates are generally provided at the national level (UNAIDS, 2015).

**Stationary Specification** To study the stationary gradient we restrict the econometric model (2.6) with $\alpha_j = \gamma_j = 0$ for all $j > 0$. We find that the stationary HIV-Education gradient is highly significant and positive (column 1, Table 2.3). The probability of being HIV infected increases by 0.43% per year of schooling. This suggests that completing five additional years of schooling increases the probability of being HIV positive by 2.15%, which is not small if we consider that the HIV prevalence is 5.18% in our sample.[15] Further, the probability of being HIV positive is higher for women (by 2.24%), for urban areas (by 2.12%), and it increases significantly with age (0.25% per year of age).[16] Aggregate variables denoting the stage of development such as the agricultural share of output and output per capita are negatively related with the probability of being infected. Next we explore how much this gradient changes as the HIV epidemic evolves.

**Non-Stationary Specification** Our non-stationary specification follows the econometric model in (2.6). Our key finding is that the HIV-Education gradient is significantly nonstationary and displays a positive-zero-positive U-shaped pattern over the stages of the HIV epidemic.

Focusing on our benchmark specification (column 2, Table 2.3), we find that at Stage 0 an additional schooling year raises the probability of being infected by $\gamma_0 = 1.12\%$. That is, for individuals in an economy that is at early stages of the epidemic the HIV-Education gradient is significantly positive and remarkably high (roughly three times larger than that of the stationary specification). Interestingly, as the HIV epidemic evolves, the HIV-Education gradient rapidly declines. At Stage 1 the rise in the probability of being infected associated with one additional year of schooling is $\gamma_0 + \gamma_1 = 0.51\%$, i.e., less than one-half of its value at Stage 0, and it is significantly different from zero at 1% level (column 2, panel A of Table 2.4). The educational disparities in HIV then vanish as the epidemic reaches Stage 2, where we cannot reject the null that $\gamma_0 + \gamma_2$ = -.04% is different from 0 (column 2, panel A in Table 2.4). As we move away from Stage 2, the HIV-Education gradient becomes increasingly positive as the epidemic evolves with $\gamma_0 + \gamma_3 = 0.19\%$ and $\gamma_0 + \gamma_4 = 0.48\%$ in Stages 3 and 4, respectively. This way, the HIV-Education gradient bounces back reaching a significant gradient in Stage 4 that is almost half the size of the gradient in Stage 0. Note that both the initial decline of the HIV-Education gradient from Stage 0 to Stage 2 and its posterior rebound from Stage 2 to Stage 4 are both significant. The size of the rebound from Stage 2 to Stage 4 is a significant 0.52% (column 2, panel B in Table 2.4). We conclude that the HIV-Education gradient exhibits a positive-zero-positive U-shape pattern over stages of the HIV epidemic. To illustrate this pattern, Figure 2.4 shows the isomorphic representation of the estimated HIV-Educ gradient across stages of the

[15] These findings are consistent with those obtained by Fortson (2008), who specifies a similar stationary econometric model for five DHS countries.

[16] While we introduce age linearly, we do find that the estimated coefficients for the HIV-Education gradient are robust when age enters non-linearly.

Table 2.3: The HIV-Education Gradient

| (A) *HIV Status* | (1) | (2) | (3) | (4) | (5) |
|---|---|---|---|---|---|
| Education | 0.0043*** | 0.0112*** | 0.0098*** | 0.0040*** | 0.0037*** |
| | (0.0006) | (0.0007) | (0.0010) | (0.0003) | (0.0003) |
| Education * Stage1 | | -0.0059*** | -0.0046*** | -0.0010*** | -0.0008** |
| | | (0.0008) | (0.0010) | (0.0003) | (0.0003) |
| Education * Stage2 | | -0.0116*** | -0.0103*** | -0.0027*** | -0.0024*** |
| | | (0.0007) | (0.0011) | (0.0002) | (0.0003) |
| Education * Stage3 | | -0.0093*** | -0.0076*** | -0.0020* | -0.0018 |
| | | (0.0015) | (0.0011) | (0.0011) | (0.0011) |
| Education * Stage4 | | -0.0064*** | -0.0051*** | -0.0015*** | -0.0012*** |
| | | (0.0008) | (0.0012) | (0.0003) | (0.0003) |
| Male | -0.0224*** | -0.0229*** | -0.0228*** | -0.0223*** | -0.0224*** |
| | (0.0027) | (0.0027) | (0.0021) | (0.0020) | (0.0019) |
| Age | 0.0025*** | 0.0025*** | 0.0025*** | 0.0025*** | 0.0025*** |
| | (0.0004) | (0.0004) | (0.0003) | (0.0003) | (0.0002) |
| Urban Area | 0.0212*** | 0.0197*** | 0.0227*** | 0.0280*** | 0.0285*** |
| | (0.0044) | (0.0045) | (0.0040) | (0.0027) | (0.0027) |
| Stage 1 | -0.0023 | 0.0124*** | 0.0111*** | -0.0055*** | 0.0088*** |
| | (0.0048) | (0.0036) | (0.0041) | (0.0008) | (0.0019) |
| Stage 2 | 0.0103 | 0.0498*** | 0.0598*** | -0.0012 | 0.0200*** |
| | (0.0078) | (0.0089) | (0.0100) | (0.0025) | (0.0032) |
| Stage 3 | -0.0094 | 0.0197 | 0.0314*** | -0.0102** | 0.0110** |
| | (0.0128) | (0.0122) | (0.0096) | (0.0052) | (0.0043) |
| Stage 4 | -0.0032 | 0.0131*** | 0.0394*** | -0.0147*** | 0.0032 |
| | (0.0042) | (0.0030) | (0.0072) | (0.0019) | (0.0029) |
| Agricultural Share | -0.0029*** | -0.0029*** | -0.0031*** | 0.0021*** | -0.0008*** |
| | (0.0002) | (0.0002) | (0.0003) | (0.0004) | (0.0003) |
| Output per Capita | -0.0000*** | -0.0000*** | -0.0000*** | 0.0000** | 0.0001*** |
| | (0.0000) | (0.0000) | (0.0000) | (0.0000) | (0.0000) |
| Constant | 0.0806*** | 0.0615*** | 0.0307*** | -0.1363*** | -0.1832*** |
| | (0.0089) | (0.0072) | (0.0089) | (0.0344) | (0.0175) |
| Year-Country Dum. | No-No | No-No | Yes-No | No-Yes | Yes-Yes |
| Sample Size | 402,766 | 402,766 | 402,766 | 402,766 | 402,766 |

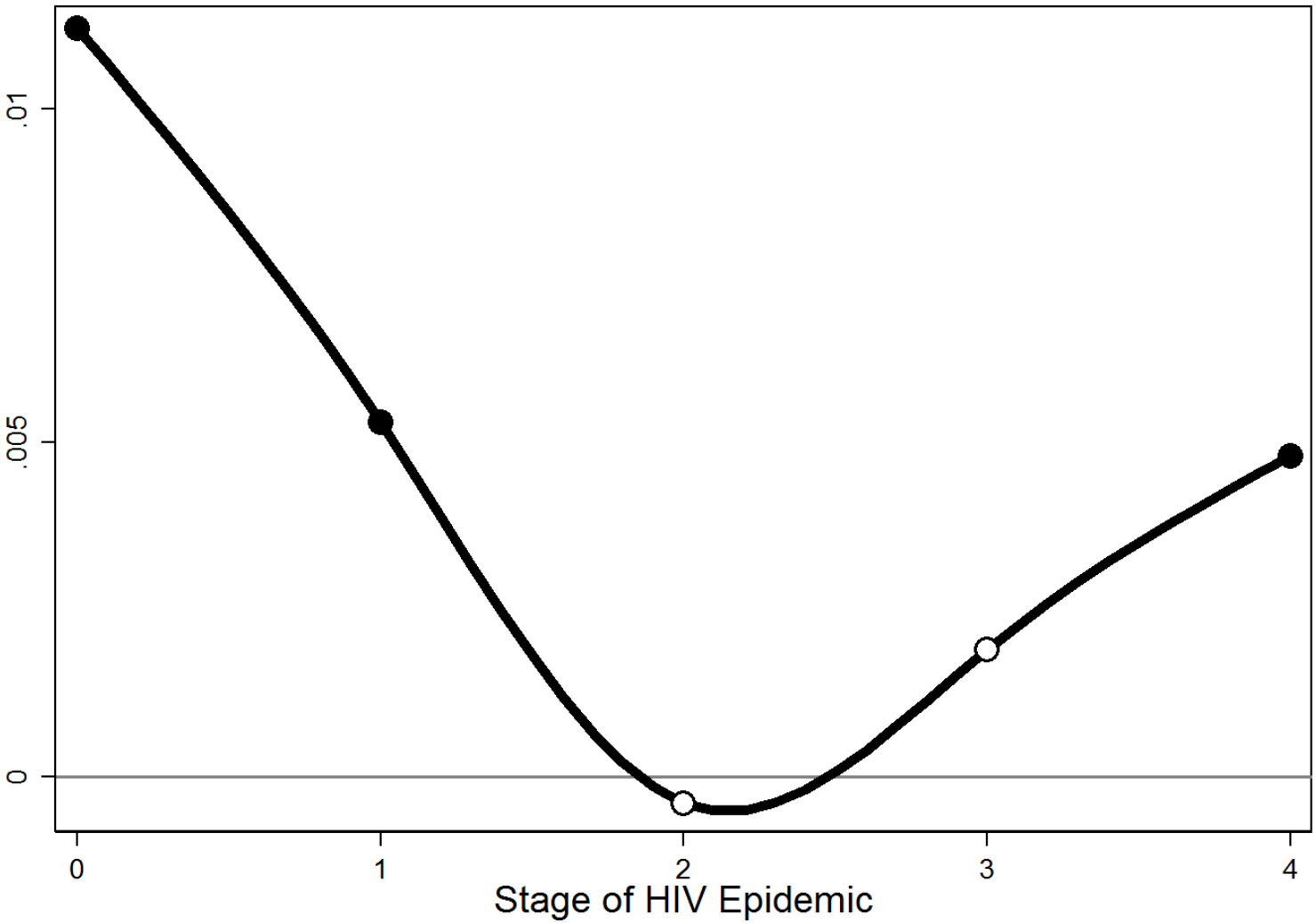
| (B) *HIV Status* | Stage 0 | Stage 1 | Stage 2 | Stage 3 | Stage 4 |
|---|---|---|---|---|---|
| Education | 0.0038*** | 0.0022*** | 0.0014*** | 0.0020* | 0.0025*** |
| | (0.000) | (0.000) | (0.000) | (0.0011) | (0.000) |
| Year-Country Dum. | Yes-Yes | Yes-Yes | Yes-Yes | Yes-Yes | Yes-Yes |
| Sample Size | 66,322 | 119,700 | 48,615 | 50,535 | 118,425 |

*Notes:* All specifications use the "Full Sample" described in Section 1.2 and the same set of controls. In Panel (A), Column (1) reports the results for the stationary specification, and columns (2) to (5) report the results for the non-stationary specification. We add year dummies in column (3), country dummies in column (4) and year-country dummies in column (5). Panel (B) reports the estimates of the HIV-Education gradient for each stage separately. Standard errors are clustered at the country level using the wild cluster bootstrap from Cameron et al. (2008), and reported in parenthesis. * significant at 10%; ** significant at 5%; *** significant at 1%.

epidemic, $\gamma_0 + \sum_{j>0} \gamma_j \mathbf{1}_j$ for each $j$ (panel A, Table 2.4).[17] Similar results are attained if we consider only the sexually active subsample (Appendix B.2 Table B.1), with an HIV-Education gradient of 1.23% at Stage 0, 0.59% at Stage 1, -0.06% at Stage 3, 0.22% at Stage 3 and 0.56% at Stage 4.

[17] Our results also hold under a Probit specification. The partial effects (and $p$-values) are as follows: $\gamma_0 =$ 0.691% (0.000), $\gamma_0 + \gamma_1 =$0.465% (0.000), $\gamma_0 + \gamma_2 =$0.018% (0.674), $\gamma_0 + \gamma_3 =$0.135% (0.133), and $\gamma_0 + \gamma_4 =$0.339% (0.004). Regarding the rebound, we also reject the null that $\gamma_4 - \gamma_2 =$0 (0.018).

Figure 2.4: The HIV-Education Gradient Across Stages of the Epidemic




*Notes*: This graph plots the benchmark estimates of the HIV-Education gradient using the full sample (with year controls). For each stage $j$ we plot $\left(\gamma_0 + \sum_{j>0} \gamma_j \mathbf{1}_j\right)$. We construct this estimates from column 3 of Table 2.3 (also reported in column 3, panel A, Table 2.4). Significance at 10%, 5%, and 1% is represented by, respectively, markers with open circles, markers with medium transparency fill, and markers with solid fill. We use a cubic spline for interpolation across stages.

The U-shape pattern of the HIV-Education gradient across stages of the HIV epidemic is robust to the addition of year dummies, country dummies, and year and country dummies (columns 3 to 5 in Table 2.3). When we control for year dummies, the HIV-Education gradient is significantly different from zero for all stages except Stage 2 showing the same positive-zero-positive behavior as our benchmark (column 3 in Table 2.4). When we control for country dummies the magnitude of the HIV-Education gradient is smaller (columns 4 and 5, panel A, Table 2.4) but the U-shape pattern is preserved and remains significant, that is, $\gamma_2$ and $\gamma_4$ are significantly different from each other (column 4 and 5, panel B, Table 2.4). While this specification is quite demanding, as it exploits only variation witin each country, the direction of the results discussed above remains unchanged. Finally, the same U-shape pattern is displayed in panel B of Table 2.3 where we re-estimate the model for each stage separately.

To summarize, the HIV-education gradient shows a U-shape positive-zero-positive pattern across stages of the epidemic. This stylized fact is twice more sizable for women than for men at early stages of the epidemic, the gradients across genders tend to equalize in later stages.

### 2.2.4 Further Evidence: Risky Sex and ARVs

Our previous theoretical interpretation suggests a parallel evolution between the HIV-Education gradient and education disparities in risky sexual behavior. We provide an empirical investigation of this phenomenon in this section.

**The Risky Sex-Education Gradient** The margins of risky sexual behavior that we study are: (i) the number of sex partners other than spouses (i.e., extramarital partners) during past 12 months, i.e., the extensive margin of sexual behavior,[18] and (ii) a dummy variable equal to 1 if the respondent used a condom during the last intercourse.We label the first Risky Sex-Education gradient as the Partners-Education gradient and the second as the Condom-Education gradient.

The results for the Partners-Education gradient are in panel A of Table 2.5. We report the results separately for women and for men, and we follow the same econometric specifications described for the HIV-Education gradient, results are shown in Figure 2.5. The more educated have significantly more sexual partners that the less educated. An additional year of schooling increases the chances of having an extramarital partners by 1.77% for women and by 2.85% for men.[19] The non-stationary specification uncovers an interesting inverted U-shaped pattern of

[18] For the extensive margin of risky sexual behavior we use the number of sex partners other than spouses (i.e., extramarital partners) in the past 12 months and note that for individuals who are single or do not cohabit, all sex partners are extramarital.

[19] Note that the females' Partners-Education gradient is smaller (about two thirds) than that of males but it shows a similar pattern over the stages of the epidemic. One potential caveat of this analysis is that women

Table 2.4: Additional Inference

| (A) *HIV-Education Gradient* | (1) | (2) | (3) | (4) | (5) |
|---|---|---|---|---|---|
| $\gamma_0$ | 0.0043*** | 0.0112*** | 0.0098*** | 0.0040*** | 0.0037*** |
| | (0.0006) | (0.0007) | (0.000) | (0.0003) | (0.0003) |
| $\gamma_0 + \gamma_1$ | | 0.0053*** | 0.0052*** | 0.0029*** | 0.0029*** |
| | | (0.0005) | (0.000) | (0.0003) | (0.0003) |
| $\gamma_0 + \gamma_2$ | | -0.0004 | -0.0005 | 0.0013*** | 0.0013*** |
| | | (0.0003) | (0.228) | (0.0001) | (0.0001) |
| $\gamma_0 + \gamma_3$ | | 0.0019 | 0.0022*** | 0.0020* | 0.0019* |
| | | (0.0014) | (0.005) | (0.0011) | (0.0011) |
| $\gamma_0 + \gamma_4$ | | 0.0048*** | 0.0047*** | 0.0025*** | 0.0025*** |
| | | (0.0005) | (0.000) | (0.0002) | (0.0001) |
| Year-Country Dum. | No-No | No-No | Yes-No | No-Yes | Yes-Yes |

| (B) *Rebound* | (1) | (2) | (3) | (4) | (5) |
|---|---|---|---|---|---|
| $\gamma_4 - \gamma_2$ | | 0.0052*** | 0.0051*** | 0.0011*** | 0.0012*** |
| | | (0.0006) | (0.0005) | (0.0001) | (0.0001) |
| Year-Country Dum. | No-No | No-No | Yes-No | No-Yes | Yes-Yes |

*Notes:* The underlying econometric models are as specified in the columns of Table 2.3. Column (1) reports the tests results for the stationary specification. Columns (2) to (5) report the tests results for the non-stationary specification. Standard errors are clustered at the country level using the wild cluster bootstrap from Cameron et al. (2008), and reported in parenthesis. * significant at 10%; ** significant at 5%; *** significant at 1%.

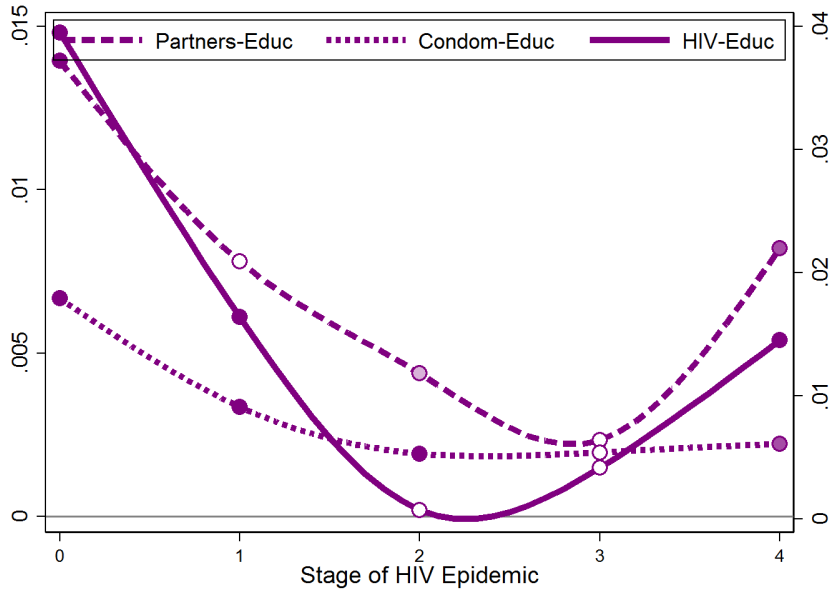
the Partners-Education gradient that represents our main finding in this section. The Partners-Education gradient first decreases (between aggregate stages 0 and 2) and then increases (between aggregate stages 2 and 4) for both women and men, and this dynamics are significant. The dynamics across stages of the epidemic show that the evolution of the Partners-Education gradient is remarkably consistent with the pattern of the HIV-Education gradient, as it is predicted by our theory in the previous section. To see this, Figure 2.5 shows separately for women (panel A) and for men (panel B) the isomorphic representation of the HIV-Education gradient and the Partners-education gradient (as in Figure 2.4, the significance of the gradients is denoted by the color of the marker). The sizes of the gradients are different, being larger for the Partners-Education gradient, which implies a positive elasticity of less than one from risky sex to HIV. In contrast, the Condom-Education gradient is reported in panel B of Table 2.5. In the stationary specification (columns 1 and 4) we find that the more educated women and men use, on average, more condoms than the less educated ones. Interestingly, we do not find a significant pattern across stages of development in the Condom-Education gradient. After Stage 0, the Condom-Education gradient remains positive but relatively constant across stages of the epidemic for both women and men. Finally, knowledge about the transmission mechanisms of HIV might affect sexual behavior (Dinkelman et al., 2006; Duflo et al., 2015a; Dupas, 2011a). We document that more-educated individuals acquire more information about HIV transmission than less-educated individuals at earlier stages of the epidemic, but these educational differences in knowledge remain constant as the epidemic evolves. This way, while knowledge might affect the HIV-Education gradient at early stages of the epidemic, this effect should rapidly vanish after the first stages.[20]

To sum up, the evolution of the Partners-Education gradient is consistent with the evolution of the HIV-Education gradient, as predicted by the theory. The evolution of educational disparities in the number of extramarital partners help explain both the decline and rebound of the HIV-Education gradient across stages of the epidemic.

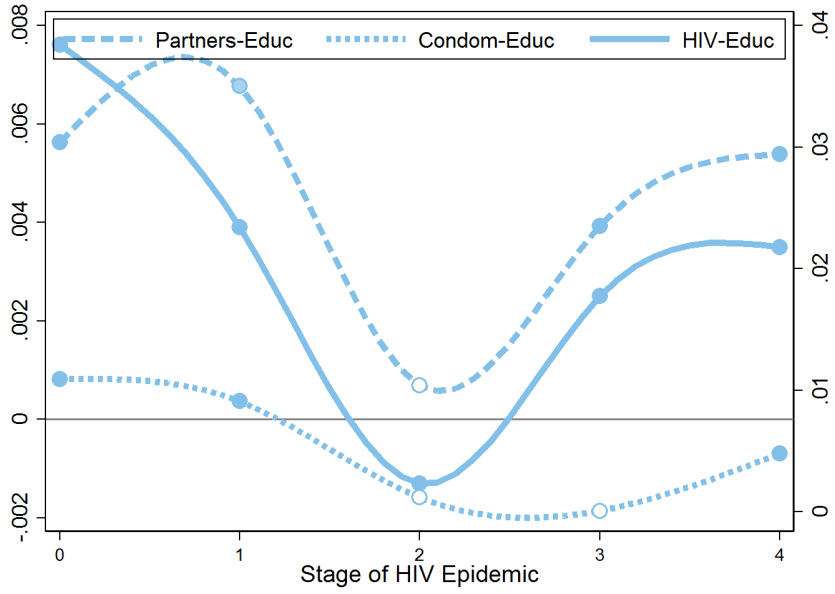
might under-report risky sexual behavior more than men, as it was pointed out by Smith (1992) and Gersovitz et al. (1998). However, more recently, Helleringer et al. (2009) find that men and women are equally likely to under-report risky sexual behavior when using sexual network data from Likoma Island, Malawi. In our analysis, as long as misreporting occurs systematically across all stages of the epidemic, the shape of the Partners-Education gradient for women over stages will not be biased.

[20] To this end, we consider two DHS questions regarding ways to avoid HIV infection that are directly related to the risky sex margins studied in the previous subsection. Specifically, respondents answer two questions: (i) *"Can you (the respondent) reduce the chances of getting HIV by having one sex partner who has no other partners?"* and (ii) *"Can you (the respondent) reduce the chances of getting HIV by always wearing a condom?"*. We estimate the education gradients in these knowledge variables. Our results are in Appendix B.2 Table B.2.

Figure 2.5: The Risky Sex-Education Gradient: Evolution Across Stages of the HIV Epidemic




(a) Women




(b) Men

*Notes*: The HIV-Education gradient is plotted on the left vertical axis. The Partners-Education gradient and the Condoms-Eduction gradient are plotted on the right vertical axis. For each stage $j$ we plot $\left(\gamma_0 + \sum_{j>0} \gamma_j \mathbf{1}_j\right)$. The specification we plot is with year controls. For the Partners-Education and Condoms-Education gradient we use column 3 (8) in Table 2.5 for respectively women (top panel) and men (bottom panel). Significance at 10%, 5%, and 1% is represented by, respectively, markers with open circles, markers with medium transparency fill, and markers with solid fill. We use a cubic spline for interpolation across stages.

Table 2.5: The Risky Sex-Education Gradient: Women and Men Separately

(A) Number of Extramarital Partners

| | Women | | | | | Men | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| *Risky Sex* | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) | (9) | (10) |
| Education | 0.0177** | 0.0372*** | 0.0348*** | 0.0294*** | 0.0296*** | 0.0285*** | 0.0304*** | 0.0269*** | 0.0290*** | 0.0295*** |
| | (0.029) | (0.000) | (0.000) | (0.001) | (0.001) | (0.000) | (0.001) | (0.002) | (0.000) | (0.000) |
| Education * Stage1 | | -0.0163** | -0.0222*** | -0.0204*** | -0.0199*** | | 0.0046 | -0.0071 | -0.0167** | -0.0167** |
| | | (0.025) | (0.000) | (0.006) | (0.008) | | (0.742) | (0.515) | (0.050) | (0.029) |
| Education * Stage2 | | -0.0254*** | -0.0267*** | -0.0243*** | -0.0249*** | | -0.0200 | -0.0238*** | -0.0290*** | -0.0294*** |
| | | (0.006) | (0.000) | (0.003) | (0.003) | | (0.125) | (0.007) | (0.000) | (0.000) |
| Education * Stage3 | | -0.0308*** | -0.0288*** | -0.0201** | -0.0213** | | -0.0069 | -0.0047 | 0.0010 | 0.0003 |
| | | (0.002) | (0.000) | (0.019) | (0.018) | | (0.550) | (0.588) | (0.932) | (0.980) |
| Education * Stage4 | | -0.0152 | -0.0134 | -0.0134* | -0.0138* | | -0.0010 | 0.0019 | 0.0021 | 0.0018 |
| | | (0.107) | (0.185) | (0.081) | (0.087) | | (0.928) | (0.845) | (0.840) | (0.866) |
| Year-Country Dum. | No-No | No-No | Yes-No | No-Yes | Yes-Yes | No-No | No-No | Yes-No | No-Yes | Yes-Yes |
| Sample Size | 227,935 | 227,935 | 227,9358 | 227,935 | 227,935 | 174,831 | 174,831 | 174,831 | 174,831 | 174,831 |

(B) Condom Use in Last Intercourse

| | Women | | | | | Men | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| *Risky Sex* | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) | (9) | (10) |
| Education | 0.0079*** | 0.0192*** | 0.0193*** | 0.0125*** | 0.0126*** | 0.0066*** | 0.0129*** | 0.0141*** | 0.0162*** | 0.0166*** |
| | (0.000) | (0.000) | (0.000) | (0.000) | (0.000) | (0.000) | (0.000) | (0.000) | (0.000) | (0.000) |
| Education * Stage1 | | -0.0102*** | -0.0114*** | -0.0054** | -0.0055*** | | -0.0038 | -0.0054 | -0.0064* | -0.0066* |
| | | (0.001) | (0.142) | (0.011) | (0.002) | | (0.0406) | (0.122) | (0.084) | (0.065) |
| Education * Stage2 | | -0.0135*** | -0.0132*** | -0.0075*** | -0.0075*** | | -0.0109*** | -0.0111** | -0.0149*** | -0.0156*** |
| | | (0.000) | (0.000) | (0.000) | (0.000) | | (0.010) | (0.028) | (0.000) | (0.000) |
| Education * Stage3 | | -0.0138 | -0.0133 | -0.0068 | -0.0068 | | -0.0101 | -0.0108 | -0.0131 | -0.0136 |
| | | (0.164) | (0.000) | (0.482) | (0.501) | | (0.611) | (0.640) | (0.592) | (0.585) |
| Education * Stage4 | | -0.0134** | -0.0133*** | -0.0073** | -0.0080** | | -0.0078** | -0.0088* | -0.0066*** | -0.0074*** |
| | | (0.000) | (0.000) | (0.024) | (0.007) | | (0.017) | (0.062) | (0.004) | (0.001) |
| Year-Country Dum. | No-No | No-No | Yes-No | No-Yes | Yes-Yes | No-No | No-No | Yes-No | No-Yes | Yes-Yes |
| Sample Size | 163,883 | 163,883 | 163,883 | 163,883 | 163,883 | 120,840 | 120,840 | 120,840 | 120,840 | 120,840 |

*Notes:* In panel (A) we report the marginal effects of the associated Tobit model where the endogenous variable is the number of extramarital partners in the past 12 months. In panel (B) we report the coefficients of a linear model where the endogenous variable is binary and refers to use of condom in last sexual intercourse. In both panels we include the same set of controls and fixed effects as in our benchmark specifications in Table **??**. Standard errors are clustered at the country level using the wild cluster bootstrap from Cameron et al. (2008), and reported in parenthesis.* significant at 10%; ** significant at 5%; *** significant at 1%.

**Antiretroviral Therapy (ART)** The effects of ART on the HIV-Education gradient are potentially ambiguous. On the one hand, ART increases survival probabilities (Greenwood et al., 2013) and decreases the degree of infectiousness of the HIV+ population that takes ART (Apondi et al., 2011). These effects, respectively, an increase in survival probabilities and a reduction in infectiousness, unambiguously increase sexual behavior by reducing the future marginal cost of today's risky sex. On the other hand, if agents have to pay for this treatment they will reduce today's risky sex because of the higher future marginal cost. As long as the monetary cost of ART does not offset the reductions in the marginal cost of sex through reduced mortality and infectiousness, ART will increase risky sex.

Treatment is indeed costly. While prices of the most common first-line ART regiments have declined over time, they remain relatively high for a vast majority of the population, with median prices in low and medium income countries of USD115 per patient per year (ppy) in 2013. The prices of USD330 (ppy) in 2013 for the second-line treatment, and more than USD1,500 for the third-line treatment, (WHO, 2014). This is prohibitive for a vast majority of SSA households.[21] In this context, if the more educated have more access to ART , then ART might help explain the rebound in the most advanced stages. The findings in Table 2.6 point in this direction. We estimate the HIV-Education gradient by epidemiological stage with and without ART controls in panel A and B, respectively. Note that when we include ART, the estimate of HIV-Education gradient in the most advanced stages of the epidemic decreases with respect to the counterpart in panel A. This suggests that the provision of ART partially accounts for the rebound in the gradient. This result should be taken with a grain of salt though, since we cannot directly test the impact of the education gradient on HIV because the DHS does not provide information of ART at the individual level.

[21] For example, income per capita in Malawi is on average USD250 in 2014, and the average income per capita in SSA is USD1638. We also expect more-educated individuals to have greater access to ART treatments for several obvious reasons—they (i) are more likely to live in the city (e.g., in Malawi, anyone who has a university degree is likely to live in the two largest cities, Lilongwe or Blantyre, where the ART drugs are available), (ii) have better transportation (do not have to walk several miles to refill prescriptions), or (iii) have access to someone in a hospital who can help them gain priority status when necessary to obtain ART .

Table 2.6: The HIV-Education Gradient: Specification by Stage

| (A) *HIV Status* | Stage 0 | Stage 1 | Stage 2 | Stage 3 | Stage 4 |
|---|---|---|---|---|---|
| Education | 0.0044*** | 0.0022*** | 0.0014*** | 0.0020* | 0.0025*** |
| | (0.000) | (0.000) | (0.000) | (0.081) | (0.000) |
| Year-Country Dum. | Yes-Yes | Yes-Yes | Yes-Yes | Yes-Yes | Yes-Yes |
| Sample Size | 58,560 | 112,024 | 48,615 | 50,535 | 118,425 |

| (B) *HIV Status with ART* | Stage 0 | Stage 1 | Stage 2 | Stage 3 | Stage 4 |
|---|---|---|---|---|---|
| Education | 0.0043*** | 0.0029*** | 0.0016*** | 0.0011 | 0.0015*** |
| | (0.000) | (0.0003) | (0.0002) | (0.0021) | (0.0002) |
| ART Coverage | 0.0003* | -0.0025*** | 0.0015*** | -0.0012*** | -0.0003*** |
| | (0.0002) | (0.0005) | (0.0000) | (0.0001) | (0.0000) |
| Education * ART Coverage | -0.0000 | -0.0000 | -0.0000*** | 0.0000*** | 0.0000*** |
| | (0.0000) | (0.0000) | (0.0000) | (0.0000) | (0.0000) |
| Year-Country Dum. | Yes-Yes | Yes-Yes | Yes-Yes | Yes-Yes | Yes-Yes |
| Sample Size | 58,560 | 112,024 | 48,615 | 50,535 | 118,425 |

*Notes:* We apply our stationary specification of the HIV-Education gradient separately for each stage of the epidemic. We include year and country fixed effects in all columns. In both panels we include the same set of controls as in our benchmark specifications in Table 2.3. We exclude Senegal (in Stage 0) and Niger (in Stage 1) since WPP does not provide information about ART coverage for these countries. Standard errors are clustered at the country level using the wild cluster bootstrap from Cameron et al. (2008), and reported in parentheses. * significant at 10%; ** significant at 5%; *** significant at 1%.

## 2.3 The Model

This is a perpetual youth economy with heterogeneous agents where the mortality rate is a function of the endogenously determined individual HIV status. There is an exogenous fertility rate that feeds newborns into the economy at a constant rate $f$. Agents differ in education level $e \in \mathcal{E}$, income shock $s \in \mathcal{S}$, sex type $i \in \mathcal{I}$, HIV status $h \in \mathcal{H}$ and whether the agent takes antiretroviral drugs(ARVs) against HIV $d \in \mathcal{D}$. The education level is permanent and exogenously given at the beginning of life. The sex type states whether agents are sex consumers or sex producers and is permanent and exogenously determined.[22]

[22] That is, we focus on endogenezing the intensive margin of sex and leave out from the model the endogenous decision on who becomes a sex consumer or a sex producer. This is clearly a caveat of this exercise. However, the

The economy transits across aggregate HIV stages, $g \in \mathcal{G}$ ={-1, 0, 1-2, 3-4}, through a sequence of unexpected shocks that reflect the evolution of the HIV epidemic. At stage -1, the economy lives in a stationary Pre-HIV epidemic era; at stage 0, the HIV epidemics starts, but agents are unaware of its workings; at stage 1-2, agents start to learn the sexual nature of the process of HIV infection and the speed of learning differs by education group; finally, at stage 3-4, ARVs are introduced.

Let us cast the household problem recursively within and across aggregate HIV stages and then explain it.

### [Stage -1] The Pre-HIV Era

At this stage there are no individuals infected with HIV. At any given period $t$, agents with education level, $e \in \mathcal{E}$ and income shock $s \in \mathcal{S}$, solve the following problem depending on their sex type, $i \in \mathcal{I}$:

**Risky sex-consumer household problem.** Sex consumers choose consumption $c$, and non-marital risky sex $x$ to solve the following dynamic problem:

$$V(e,i,s,\Phi) = \max_{c\geq 0, x\geq 0} \chi u(c,x) + \beta\gamma \sum_{s'|s} \pi(s'|s) V(e,i,s',\Phi') \tag{2.7}$$

s.t

$$c + p(\Phi)x = zy(e)s \tag{2.8}$$

$$s = (s_n) \text{ with } s_n \in \mathcal{S} \text{ and stochastic matrix } \pi \tag{2.9}$$

That is, sex consumers derive utility from $c$ and $x$ with preference parameter $\chi$ that depends on the individual' HIV status, and discount future at factor $\beta$ times a survival probability $\gamma$. The preference parameter $\chi$ controls the level of utility derived from $c$ and $x$. Moreover, $u(c,x)$ is concave, continuous and twice diferenciable.[23].We use consumption as numeraire and the relative price of sex is denoted by $p$. Labor income is the product of a permanent component $y(e)$ that depends on the level of education, a transitory component $s$ that follows a Markov process with

fact that we introduce a substantial degree of endogenous heterogeneity within each of the two groups implies that part of our sex consumers will consume a positive but negligible amount of sex, and part of our sex producers will produce a negligible amount of sex. The size of this population, for which sex transactions are small, which is endogenous, determines the size of the population that consumes/sells risky sex. Moreover, the joint distribution of education groups $e \in \mathcal{E}$ and sex types $i \in \mathcal{I}$ (with respective proportions $\vartheta_{e=1,i}, \vartheta_{e=1,-i}, \vartheta_{e=0,i}, \vartheta_{e=0,-i}$) is endogenous throughout the model. However, we are still required to provide an exogenous measure for $t = 0$, see Section 2.4 for more detail.

[23]We choose an additive separable CRRA functional form with common parameter $\xi$, at this stage the preference parameter $\chi$ is set to one for all agents.

given transition matrix $\pi$, and a permanent component $z$ that depends on the individual's health status. Since there is no HIV, $z$ is set equal to one for everyone. We assume that $y(e)s > y(e)\widetilde{s}$ for $s > \widetilde{s}$ and $\forall\, e \in \mathcal{E}$ [24]

**Risky sex-producer household problem.** Sex-producer households choose consumption $c$ and the fraction of time devoted to sex production $l$ to solve the following dynamic problem:

$$V(e,-i,s,\Phi) = \max_{c\geq 0, 1\geq l\geq 0} \chi u(c) + \beta\gamma \sum_{s'|s} \pi(s'|s) V(e,-i,s',\Phi') \tag{2.10}$$

s.t

$$c = z[p(\Phi)l^{\alpha} + y(e)s(1-l)], \tag{2.11}$$

Where $s$ follows the income shock process (2.9). Sex-producer households derive utility from the consumption good, but not from risky sex. The production of sex follows a technology $x = l^{\alpha}$ using time $l$ with decreasing returns to scale, $\alpha \in (0,1)$. This technology puts an upper bound to the amount of sex produced. Notice that labor is inelastically supplied, this way, $l$ denotes the fraction of labor allocated to the production of risky sex and the remaining labor $(1-l)$ is allocated to sex production. The permanent component $z$ affects both the ability to generate labor market income and sex production. This idea pursues the notion that HIV+ individuals do not necessarily increase the production of sex as a response to their lower labor income.

At any point in time in the pre-HIV stage -1, the economy is summarized by the joint distribution $\Phi$ of individual states $(e,i,s)$. The aggregate state variable evolves according to:

$$\Phi' = H(\Phi) \tag{2.12}$$

Where the function $H : \mathcal{M} \to \mathcal{M}$ is the aggregate law of motion, mapping distributions to distributions. $H$ summarizes the distribution of type and education evolves from one period to the next, however this is exactly what a transition function tell us. Define the transition function $\mathcal{Q} : \mathcal{Z} \times \mathcal{B}(\mathcal{Z}) \to [0,1]$ by:

$$Q((e,i,s)(\mathcal{E},\mathcal{I},\mathcal{S})) = \gamma \quad \forall \ (e,i,s) \in \mathcal{Z} \ \text{ and } \ (\mathcal{E},\mathcal{I},\mathcal{S}) \in \mathcal{B}(\mathcal{Z})$$

Where $\mathcal{Z}$ consists of all n-tuples of $E \times I \times S$. [25]

We now describe the recursive competitive equilibrium (RCE) for this Stage -1 economy. We

[24] This means that anyone at $s > \widetilde{s}$, will have a higher labour income regardless of his/her education

[25] Define $\mathcal{B}(\mathcal{Z})$ as the set of Borel sets on $\mathcal{Z}$, in particular $\mathcal{E},\mathcal{G},\mathcal{S} \in \mathcal{B}(\mathcal{Z})$ where $\mathcal{E},\mathcal{G},\mathcal{S}$ are projections of $\mathcal{Z}$ over the spaces $E, G$ and $S$ respectively. Let $\mathcal{P}$ be a probability measure on $\mathcal{B}(\mathcal{Z})$, then $\mathcal{P} : \mathcal{B}(\mathcal{Z}) \to [0,1]$.

focus on stationary solutions and its associated equilibrium.

**Definition of the Stage -1 (Stationary) Recursive Competitive Equilibrium**

A Stage-1 stationary RCE is a value function $V : \mathcal{Z} \to R$, policy functions $c : \mathcal{Z} \to R$, $x : \mathcal{Z} \to R$, and $l : \mathcal{Z} \to R$, price $p$, and a measure $\Phi \in \mathcal{M}$ such that:

1. Given $p$ the policy functions $c(e,i,s)$, $x(e,i,s)$ and $l(e,i,s)$ solve the sex-consumer household problem (2.7)-(2.8) and sex-producer households problem (2.10)-(2.11).

2. All markets clear.

$$\sum_{e,i,s} x(e,i,s) = \sum_{e,-i,s} x(e,-i,s),$$

   The sex markets clear and the consumption market clears by Walras law.

3. The stationary probability distribution,

$$\Phi = H(\Phi)$$

   is induced by the equilibrium policy functions.

Notice that value function, policy functions, and price are not any longer indexed by measures $\Phi$ because all conditions must be satisfied for the equilibrium stationary measure $\Phi$. The last requirement states that the measure $\Phi$ reproduces itself: starting with a measure of education, sex type, and income shocks today generates the same measure tomorrow.

### [Stage 0] The Myopic Onset of the HIV Epidemic

The HIV epidemics starts in this stage, but agents are neither aware of its presence nor its workings. Agents can be healthy or HIV infected $h \in \mathcal{H} = \{-,+\}$. Specifically, agents live with HIV myopia in two dimensions. First, agents are unaware of the fact that HIV infection

Then the evolution of the population distribution is,

$$\Phi'(\mathcal{E},\mathcal{I},\mathcal{S}) = F(\Phi)(\mathcal{E},\mathcal{I},\mathcal{S}) = \sum_{e,i,s} Q((e,i,s)(\mathcal{E},\mathcal{I},\mathcal{S})) + f\Phi((e,i,s')(\mathcal{E},\mathcal{I},\mathcal{S})), \tag{2.13}$$

which is the fraction of people with education $\mathcal{E}$, type $\mathcal{G}$ and states in $\mathcal{S}$ as measured by $\Phi$, that transit to $(\mathcal{E},\mathcal{G},\mathcal{S})$ as measured by $\mathcal{Q}$. The last term accounts for the new born. Population of each group increases according to respective fertility rate $f$.

is occurring and that its spread depends on the amount of risky sex transactioned $x$, (either consumed or produced) and the current prevalence rate, $\phi^+ \in [0,1]$. Specifically, HIV infection occurs at an endogenous rate:

$$\phi^+ \lambda(x;\rho) = \phi^+ \frac{e^x}{e^x + \rho e^{-x}}, \tag{2.14}$$

where $\rho \in [0,\infty)$ is a parameter that governs the mapping from the amount of sex transactioned to the probability of HIV infection. The lower is $\rho$ the higher is the probability of HIV infection per amount of sex transactioned.[26] Second, although agents are unaware of the nature of HIV infection (2.14), at every period, agents observe higher average mortality rates ($\gamma$), lower average labor market and sex production productivity ($z$) and a lower overall satisfaction ($\chi$). Since agents do no know that these changes in $\gamma$, $z$ and $\chi$ are due to HIV, at every period, the economy makes the mistake of taking these observations as unexpected one-time aggregate permanent shocks in mortality rates, productivity and preferences . We model this as a sequence of permanent unexpected aggregate shocks in $\gamma$, $z$ and $\chi$. That is, at every period, agents notice a change between $\gamma_t$ and $\gamma_{t-1}$, between $z_t$ and $z_{t-1}$, between $\chi_t$ and $\chi_{t-1}$ and agents assume that $\widetilde{\gamma}_\tau = \gamma_t$, $\widetilde{z}_\tau = z_t$, and $\widetilde{\chi}_\tau = \chi_t$ for all $\tau \geq t$.

In reality, however, this is not the case because the average survival rates, labor productivity and felicity depend on the distribution of HIV status across the population which is endogenous to risky sex. In particular, the true HIV distribution of the population evolves according to

$$\begin{bmatrix} \phi^-_{t+1} \\ \phi^+_{t+1} \end{bmatrix} = \begin{bmatrix} \gamma_- & 0 \\ 0 & \gamma_+ \end{bmatrix} \begin{bmatrix} 1-\phi^+_t \lambda_\rho(x_t) & 0 \\ \phi^+_t \lambda_\rho(x_t) & 1 \end{bmatrix} \begin{bmatrix} 1+f & f \\ 0 & 1 \end{bmatrix} \begin{bmatrix} \phi^-_t \\ \phi^+_t \end{bmatrix} \tag{2.15}$$

where $\phi^+_t$ and $\phi^-_t$ are the measures of HIV infected and HIV non-infected populations, $\gamma_+$ and $\gamma_-$ are survival rates for HIV infected and HIV non-infected populations, respectively, the odds of HIV infection $\phi^+_t \lambda_\rho(x)$ are defined by (2.14), and $f$ is the fertility rate for the aggregate economy,

[26] Notice that in order for the virus to start spreading we need to provide an initial HIV prevalence rate $\phi^+_{t=0} > 0$. The value for $\phi^+_{t=0}$ is exogenous, meaning that it is not directly linked to the risky sex practices between humans, which is the main mechanism of HIV spread in this model. The calibration of the initial prevalence is described in Section 2.4. We conduct some robustness on this assumption by setting the probability of infection equal to $\lambda_\rho(x)$, that is, the aggregate rate of HIV infection in the economy does not explicitly affect the infection probability. Our main results remained unchanged.

which is independent of HIV status.[27] Then, the evolution of the aggregate population is:

$$\phi_{t+1} = \phi^-_{t+1} + \phi^+_{t+1} \tag{2.16}$$

with:

$$\phi^-_{t+1} = \gamma_-(1 - \phi^+_t \lambda_\rho(x_t))[(1+f)\phi^-_t + f\phi^+_t]$$
$$\phi^+_{t+1} = \gamma_+ \left(\phi^+_t \lambda_\rho(x_t)[(1+f)\phi^-_t + f\phi^+_t] + \phi^+_t\right)$$

Since our agents are myopic in HIV they only see the current population $\phi_{t+1}$, the fertility rate, and the previous population $\phi_t$ so as to infer an aggregate survival rate, $\widetilde{\gamma}_t$,

$$\phi_{t+1} = \widetilde{\gamma}_t(1+f)\phi_t. \tag{2.17}$$

Notice that we can equate (2.16) and (2.17) to find the survival rate $\widetilde{\gamma}$ observed by myopic agents. We can proceed analogously to find the average labor productivity observed by agents,

$$\widetilde{z}_t = \frac{z_-\phi^-_t + z_+\phi^+_t}{\phi^-_t + \phi^+_t}. \tag{2.18}$$

And analogously for the preference parameter $\widetilde{\chi}_t$. We calibrate the preference parameter $\chi_+$ such that, conditional on infection and the state, the following inequality holds :

$$u(\cdot)\chi_+ < u(\cdot)\chi_- \tag{2.19}$$

Given the myopic updating formulas for $\gamma$ and $z$ in (2.17), (2.18) and the preference parameter calibration (2.19), we are now ready to formulate the risky-sex consumer and producer problems. Importantly, notice that $\widetilde{\gamma}_t$ and $\widetilde{z}_t$ get updated every period and, hence, the formulation of the households problem and the definition of equilibrium for the Stage 0 of the HIV epidemic needs to reflect this phenomenon.

[27] Note that we can develop (2.15) as:

$$\begin{bmatrix}\phi^-_t \\ \phi^+_t\end{bmatrix} = \begin{bmatrix}\gamma_- & 0 \\ 0 & \gamma_+\end{bmatrix}\begin{bmatrix}1-\phi^+_t\lambda_\rho(x_t) & 0 \\ \phi^+_t\lambda_\rho(x_t) & 1\end{bmatrix}\begin{bmatrix}(1+f)\phi^-_t + f\phi^+_t \\ \phi^+_t\end{bmatrix} = \begin{bmatrix}\gamma_- & 0 \\ 0 & \gamma_+\end{bmatrix}\begin{bmatrix}(1-\phi^+_t\lambda_\rho(x_t))[(1+f)\phi^-_t + f\phi^+_t] \\ \phi^+_t\lambda_\rho(x_t)[(1+f)\phi^-_t + f\phi^+_t] + \phi^+_t\end{bmatrix}$$

and hence,

$$\phi^-_{t+1} = \gamma_-(1-\phi^+_t\lambda_\rho(x_t))[(1+f)\phi^-_t + f\phi^+_t] \text{ and } \phi^+_{t+1} = \gamma_+\left(\phi^+_t\lambda_\rho(x_t)[(1+f)\phi^-_t + f\phi^+_t] + \phi^+_t\right).$$

**Risky-sex consumer household problem.** Risky-sex consumers choose $c$, and $x$ to solve:

$$V_t(e,i,s,\Phi) = \max_{c_t\geq 0, x_t\geq 0} \widetilde{\chi} u(c_t,x_t) + \beta\widetilde{\gamma}\sum_{s'|s}\pi(s'|s)V_{t+1}(e,i,s',\Phi') \tag{2.20}$$

s.t

$$c_t + p_t(\Phi)x_t = \widetilde{z}y(e)s \tag{2.21}$$

$$\widetilde{\gamma} = \widetilde{\gamma}_{t-1} = \frac{\phi_t}{(1+f)\phi_{t-1}} \tag{2.22}$$

an income shock process that follows (2.9)

**Risky sex-producer household problem.** Sex producers choose $c$ and $l$, to solve:

$$V_t(e,-i,s,\Phi) = \max_{c_t\geq 0, 1\geq l_t\geq 0} \widetilde{\chi} u(c_t) + \beta\widetilde{\gamma}\sum_{s'|s}\pi(s'|s)V_{t+1}(e,-i,s',\Phi') \tag{2.23}$$

s.t

$$c_t = \widetilde{z}\left[(p_t(\Phi)l_t^{\alpha} + y(e)s(1-l_t))\right] \tag{2.24}$$

$$\tag{2.25}$$

with income shock process (2.9), survival rates $\tilde{\gamma}$, productivity $\tilde{z}$, and preference parameter $\tilde{\chi}$.

At any point in time in the HIV Stage 0, the economy is summarized by the joint distribution $\Phi$ of individual states $(e,i,s)$. Importantly, notice that HIV status is not part of the individual states, as agents in our economy are unaware of HIV. The aggregate state variable of the economy evolves following:

$$\Phi_{t+1} = H_t(\Phi_t) \tag{2.26}$$

Notice that our objective functions and prices are indexed by time which captures the non-stationarity nature of the Stage 0 problem. This sequential formulation of the recursive problem is required to address the unexpected changes in $\gamma$, $z$ and $\chi$. On the top of that, notice that the myopia makes agents assume that last period's mortality, productivity and preferences will be permanent, that is, $\widetilde{\gamma}_\tau = \widetilde{\gamma}\ \forall\tau\geq t$, $\widetilde{z}_\tau = \widetilde{z}\ \forall\tau\geq t$ and $\widetilde{\chi}_\tau = \widetilde{\chi}\ \forall\tau\geq t$. Because of this myopia, the changes in average mortality, productivity and felicity are not only unexpected, but also occur at every period.

**Definition of the Stage 0 (Nonstationary) RCE**

Given a Stage-1 stationary joint distribution of $(e, i, s)$, $\Phi_{t=0}(g = -1)$, and a sequence of myopically unexpected and permanent changes in mortality rates $\{\widetilde{\gamma}_t\}_{t=0}^{\infty}$, labor productivity $\{\widetilde{z}_t\}_{t=0}^{\infty}$ and preference shocks $\{\widetilde{\chi}_t\}_{t=0}^{\infty}$, constructed from (2.17), (2.18) and (2.19), respectively, a competitive equilibrium is a sequence of individual household functions $\{V_t, c_t, x_t, l_t : Z \times M \to M\}_{t=0}^{\infty}$, sequence of prices $\{p_t\}_{t=0}^{\infty}$, and a sequence of measures $\{\Phi_t\}_{t=0}^{\infty}$ such that, $\forall t$:

1. The policy functions $c_t(e, i, s)$, $x_t(e, i, s)$ and $l_t(e, i, s)$ solve the sex-consumer household problem (2.20) and sex-producer households problem (2.23).

2. All markets clear.

$$\sum_{e,i,s} x_t(e, i, s) = \sum_{e,-i,s} x_t(e, -i, s),$$

   The sex markets clear and the consumption market clears by Walras law.

3. The aggregate law of motion is,

$$\Phi_{t+1} = H_t(\Phi_t)$$

   where $\Phi$ is the joint distribution of $(e, i, s)$ is induced by the equilibrium policy functions.

4. The true distribution of the HIV population, which is used to construct the sequences $\{\widetilde{\gamma}_t\}_{t=0}^{\infty}$, $\{\widetilde{z}_t\}_{t=0}^{\infty}$ and $\{\widetilde{\chi}_t\}_{t=0}^{\infty}$, endogenously evolves according to (2.15).

The main characteristic of this Stage 0 is that agents do not know that the HIV epidemic is unravelling. That is, agents in this Stage 0 do not internalize the evolution of the HIV (2.15) because they are unaware that their sexual behavior affects their chances of survival, labor productivity and felicity. We model this through a myopic updating of $\gamma$, $z$ and $\chi$. Where agents perceive the updates in $\gamma$, $z$ and $\chi$ as permanent changes. This implies that after one of these permanent changes, agents rationalize their current behavior by looking forward and solving the entire transition from today to a new steady state associated with the new triple $(\widetilde{\gamma}, \widetilde{z}, \widetilde{\chi})$. That is, we need to solve for a transition every time there is a perceived permanent change. Because this permanent changes occur every period, then at every period we need to compute the entire transition. The equilibrium value functions and policy functions are the sequence of first-period

solutions to the sequence of transitional problems. The sequence of myopic permanent changes do not go *ad infinitum* because Stage 1-2 (Maturity) arrives after the economy has been a finite amount of periods $T_0$in Stage 0.

### [Stage 1-2] Learning the HIV Epidemic

At this stage, agents are aware of the HIV epidemic and its consequences: higher mortality rates, lower productivity and lower felicity for HIV infected individuals. Agents are aware of their own HIV status and that of the rest of the population.[28] However, at the beginning of Stage 1-2, agents are not fully aware of the sexual nature of HIV infection in so far they do not accurately know $\rho$ in (2.14). Although agents do not know the odds of infection as function of risky sexual activity, agents learn about it through Bayesian updates on $\rho$ with some noise.

The speed in which agents learn about the actual odds of infection differs across education groups, this being faster for the more-educated agents. The learning speed is the only source of heterogeneity across education groups introduced in this stage. The initial degree of accuracy in which individuals know the odds of infection is the same across education groups, this follows from the fact that both groups were completely unaware of $\rho$ in the previous Stage.
More precisely, each educational group $e \in \mathcal{E}$ has a prior belief about the distribution of $\lambda(x;\rho)$, denoted by the p.d.f $\mathcal{P}_e(\lambda(x;\rho))$. Furthermore, at the beginning $\mathcal{P}_e(\lambda(x;\tilde{\rho}_o)) \sim N(\lambda(x;\tilde{\rho}_o),\sigma^2_\varepsilon)$ and $\mathcal{P}_{e=1}(\lambda(x;\tilde{\rho}_o)) = \mathcal{P}_{e=0}(\lambda(x;\tilde{\rho}_o))$. Afterwards, agents receive a signal $\widetilde{\lambda}_\rho(x)$ per period. This signal contains information about the actual probability of infection plus some noise $\varepsilon_t$ that is normally distributed with zero mean and variance $\sigma^2_\varepsilon(e)$. Explicitly:

$$\widetilde{\lambda}_\rho(x) = \lambda_\rho(x) + \varepsilon_t, \tag{2.27}$$

where the signal follows the following covariance stationary process:

$$\varepsilon_t = v_t + \mathbf{1}_{e=0} u_t \tag{2.28}$$

with $v \sim N(0,\sigma^2_v)$ and $u \sim N(0,\sigma^2_u)$. The dummy $\mathbf{1}_{e=0}$ equals one if an agent belongs to the less educated group, and zero otherwise. That is, the signal is noisier for the less educated individuals than for the more educated individuals. In particular, every period $t$ agents update their beliefs $\mathcal{P}_e(\lambda_\rho(x))$ given the information up to $t-1$ according to Bayes rule:

$$\mathcal{P}_e(\lambda_\rho(x)) = \mathcal{P}_e(\lambda_\rho(x)|\widetilde{\lambda}_\rho(x)) = \frac{\mathcal{P}_e(\widetilde{\lambda}_\rho(x)|\lambda_\rho(x))\mathcal{P}_e(\lambda_\rho(x))}{\mathcal{P}_e(\widetilde{\lambda}_\rho(x))} \tag{2.29}$$

[28] See Carli and Santaeulalia-Llopis (2019) for an economy in which individuals can hide their HIV status.

where the Bayesian updates will transit faster to the actual odds of HIV infection for the more educated individuals due to (2.28).[29]

Let us now write the nonstationary recursive problem and then explain it

**Risky-sex consumer household problem.** Risky-sex consumers choose $c$, and $x$ to solve:

$$V_t(e,i,s,h,\Phi) = \max_{c_t\geq 0, x_t\geq 0} \chi(h)u(c_t,x_t) + \beta \sum_{h'|h,s'|s} [\gamma(h')\widetilde{\lambda}_{\rho(e)}(h'|x_t,h)\pi(s'|s)V_{t+1}(e,i,s',h',\Phi')\,] \tag{2.30}$$

subject to,

$$c_t + p_t(\Phi)x_t = z(h)y(e)s \tag{2.31}$$

an income shock process that follows (2.9).

**Risky sex-producer household problem.** Sex producers choose $c$ and $l$, to solve:

$$V_t(e,-i,s,h,\Phi) = \max_{c_t\geq 0, 1\geq l_t\geq 0} \chi(h)u(c_t) + \beta \sum_{h'|h,s'|s} [\gamma(h')\widetilde{\lambda}_{\rho(e)}(h'|x_t,h)\pi(s'|s)V_{t+1}(e,-i,s',h',\Phi')\,] \tag{2.32}$$

subject to,

$$c_t = z(h)[p_t(\Phi)l_t^{\alpha} + y(e)s(1-l_t)], \tag{2.33}$$

with income shock process (2.9).

At any point in time in the HIV Stage 1-2, the economy is summarized by the joint distribution $\Phi$ of individual states $(e,i,s,h)$, which incorporates individual HIV status. In this Stage 1-2 of the epidemic, agents are aware of their HIV status, and that of the rest of the economy. The aggregate state variable of the economy evolves following $\Phi_{t+1} = H_t(\Phi_t)$. Notice that even though agents are aware of the average probability of infection of the rest of the economy they cannot infer the true value of $\rho$, moreover, even if agents are aware of the exact infection process (namely 2.14), having knowledge of the average probability of infection is not enough to retrieve the value of $\rho$.

[29] We assume normality of the prior belief to simplify the calculations, however this can be adapted to mimic more complex formulations.

An additional implication of the learning mechanism is that agents will also update their own mortality expectations and use them to make allocation decisions. This means agents keep subjective expectations in mortality since these expectations differ from their true individual probability of survival[30].

As it was the case of Stage 0, in Stage 1-2, the objective functions and prices are indexed by time which captures the nonstationarity nature the Stage. In the long-run, when both education groups have finalized their learning process of the odds of HIV infection a stationary RCE can be defined for Stage 1-2.

In this stage all agents in the economy learn about the risk of infection, a natural extension to this set up would be to make the proportion of people that learn increase gradually. This extension represents the introduction a new state variable since it will be necessary to keep track of the proportion of the population that is still miopic and the ones who are learning. This can be done in order to smooth out the transition from HIV Stage 0 to HIV Stage 1-2 and see a hump at the peak, as in the data.

[30] This is a consequence of the fact that agents learn about their risk of infection which is directly linked with their probability of survival. The lower people think their odds of infection are, (this happens at the beginning of the learning stage) the higher are their believed chances of survival.

Definition of the Stage 1-2 (Nonstationary) RCE

Given a Stage 0 joint distribution $\Phi_{t=0}(g = 0)$, and a simulated sequence of infection probabilities $\{\widetilde{\lambda}_\rho(e)\}_{t=0}^{\infty}$ by education group, a competitive equilibrium is a sequence of individual household functions $\{V_t, c_t, x_t, l_t : Z \times M \to M\}_{t=0}^{\infty}$, sequence of factor prices $\{p_t\}_{t=0}^{\infty}$, and a sequence of measures $\{\Phi_t\}_{t=0}^{\infty}$ such that, $\forall t$:

1. Given $\{p_t\}_{t=0}^{\infty}$ the policy functions $c_t(e,i,s,h)$, $x_t(e,i,s,h)$, and $l_t(e,i,s,h)$ solve the sex-consumer household problem (2.30) and sex-producer households problem (2.32).

2. All markets clear.

$$\sum_{e,i,s,h} x_t(e,i,s,h) = \sum_{e,-i,s,h} x_t(e,-i,s,h),$$

   The sex markets clear and the consumption market clears by Walras law.

3. The aggregate law of motion is,

$$\Phi_{t+1} = H_t(\Phi_t)$$

   where $\Phi$ is the joint distribution of $(e,i,s,h)$ is induced by the equilibrium policy functions.

4. The true distribution of the HIV population endogenously evolves according to (2.15).

5. The beliefs of on the odds of infection by education group evolve according to Bayes rule (2.29) and signal (2.28).

**Remark.** The Stage 1-2 stationary RCE is the limiting case of the nonstationary RCE in which beliefs of both education groups have converged to the actual odds of infection and the cross-sectional distribution $\Phi$ does not change over time. In that case, we can drop all time subscripts.

### [Stage 3-4] The Era of ARVs

We assume that ARV drugs entirely revert the negative effects of HIV on mortality and productivity, but not on felicity. This pursuits the argument that receiving treatment is not necessarily the same as being completely healthy, hence this is an implicit cost of ARV treatment that will have a positive effect on the equilibrium price of risky sex. For simplicity only those who are infected can receive treatment[31]. Let us now write and explain the nonstationary recursive problem.
Treatment is provided stochastically to the infected population, with the educated individuals having a higher probability to receive treatment $\eta_{t,e=1} > \eta_{t,e=0}$ at all $t$[32] . This means that ARV drugs now represent a new state variable $d \in \mathcal{D} = \{d^+, d^-\}$ as we now need to keep track of the portion of the infected population receiving treatment $(d^+)$ and that which doesn't $(d^-)$.
The aggregate proportion of the population that receives drugs at each $t$ is deterministic and represented by the monotonically increasing sequence $\{\eta\}_{t=0}^{\infty}$, where $0 \leq \eta_t \leq \tilde{\eta}\ \forall t$ with $\lim_{t\to\infty} \eta_t = \tilde{\eta}$ and $\tilde{\eta} \in (0, 1]$. In addition, ARV's affect the average probability of infection by decreasing the viral load and, hence, the infectiousness of those infected. In our formulation this would be translated into a proportional increase of $\rho$ with respect to the coverage rate.
In this stage agents keep learning about the value of $\lambda_\rho(x)$ according to (2.29).
Then the distribution of HIV positive population evolves according to:

$$\begin{bmatrix} \phi^-_{d^-t+1} \\ \phi^+_{d^+t+1} \\ \phi^+_{d^-t+1} \end{bmatrix} = \begin{bmatrix} \gamma_- & 0 & 0 \\ 0 & \gamma_- & 0 \\ 0 & 0 & \gamma_+ \end{bmatrix} \begin{bmatrix} 1-\lambda_{t,\rho} & 0 & 0 \\ \eta_t\lambda_{t,\rho} & \eta_t & \eta_t \\ (1-\eta_t)\lambda_{t,\rho} & 1-\eta_t & 1-\eta_t \end{bmatrix} \begin{bmatrix} 1+f & f & f \\ 0 & 1 & 0 \\ 0 & 0 & 1 \end{bmatrix} \begin{bmatrix} \phi^-_{d^-t} \\ \phi^+_{d^+t} \\ \phi^+_{d^-t} \end{bmatrix} \tag{2.34}$$

Let us now write the nonstationary recursive problem:

**Risky-sex consumer household problem.** Risky-sex consumers choose $c$, and $x$ to solve:

$$\begin{aligned} V_t(e,i,s,h,d,\Phi) = &\max_{c_t\geq 0, x_t \geq 0} \chi_d(h)u(c_t,x_t) \\ &+ \beta \sum_{\substack{d'|d,h'|h \\ s'|s}} [\eta_t(d'|e,d)\gamma_d(h')\lambda_{\rho(e)}(h'|x_t,h)\pi(s'|s)V_{t+1}(e,i,s',h',d,\Phi')\ ] \end{aligned} \tag{2.35}$$

[31] The Joint United Nations Programme on HIV/AIDS (UNAIDS) supplies ARVs coverage data only for that portion of the population that was infected and received treatment.

[32] The current data does not provide treatment composition by educational groups therefore we approximate these probabilities by $\eta_{t,e=1} = (1+\iota)\eta_t$ and $\eta_{t,e=0} = (1-\iota)\eta_t$, where $\eta_t$ is the aggregate coverage rate observed in the data at $t$, with $\iota$ controlling the odds of treatment with respect to the aggregate by education group.

subject to,

$$c_t + p_t(\Phi)x_t = z_d(h)y(e)s \tag{2.36}$$

an income shock process that follows (2.9).

**Risky sex-producer household problem.** Sex producers choose $c$ and $l$ to solve:

$$\begin{aligned} V_t(e,-i,s,h,d,\Phi) = &\max_{c_t\geq 0, 1\geq l_t\geq 0} \chi_d(h)u(c_t) \\ &+ \beta \sum_{\substack{d'|d,h'|h \\ s'|s}} [\eta_t(d'|e,d)\gamma_d(h')\lambda_{\rho(e)}(h'|x_t,h)\pi(s'|s)V_{t+1}(e,-i,s',h',d,\Phi') \,] \end{aligned} \tag{2.37}$$

subject to,

$$c_t = z_d(h)[p_t(\Phi)l_t^{\alpha} + y(e)s(1-l_t)], \tag{2.38}$$

with income shock process (2.9).

Notice that if ARVs fully revert the effects of HIV on mortality rates, productivity but not preferences then:

$$\begin{aligned} \gamma_{d^+}(+) &= \gamma_- = \gamma \\ z_{d^+}(+) &= z_- = z \\ \chi_{d^+}(+) &= \chi_- = \chi_+ \end{aligned}$$

However for those who are infected but not treated, survival rates, productivity and preferences go back to that of the maturity stage:

$$\begin{aligned} \gamma_{d^-}(+) &= \gamma_+ \\ z_{d^-}(+) &= z_+ \\ \chi_{d^-}(+) &= \chi_+ \end{aligned}$$

At any point in time in the HIV Stage 3-4, the economy is summarized by the joint distribution $\Phi$ of individual states $(e,i,s,h,d)$, which incorporates individual HIV status and treatment. In this Stage 3-4 of the epidemic, agents are know if they received drugs as well as their HIV status, and that of the rest of the economy. The aggregate state variable of the economy evolves following $\Phi_{t+1} = H_t(\Phi_t)$. Notice that, as it was the case of Stage 1-2, in Stage 3-4, the objective functions and prices are indexed by time which captures the nonstationarity nature of this stage.

In the long-run, we find a stationary RCE for Stage 3-4 that differs from Stage -1 in that some proportion of the population will be infected with HIV and a deterministic proportion of the population will receive treatment.

**Definition of the Stage 3-4 (Nonstationary) RCE**

Given a Stage 1-2 distribution $\Phi_0(g = 1-2)$ and a deterministic sequence for the treated proportion of the population $\{\eta_t\}_{t=0}^{\infty}$ , a competitive equilibrium is a sequence of individual household functions $\{V_t, c_t, x_t, l_t : Z \times M \to M\}_{t=0}^{\infty}$, sequence of factor prices $\{p_t\}_{t=0}^{\infty}$, and a sequence of measures $\{\Phi_t\}_{t=0}^{\infty}$ such that, $\forall t$:

1. Given $\{p_t\}_{t=0}^{\infty}$ the policy functions $c_t(e,i,s,h,d)$, $x_t(e,i,s,h,d)$ and $l_t(e,i,s,h,d)$ solve the sex-consumer household problem (2.35) and sex-producer households problem (2.37).

2. All markets clear.

$$\sum_{e,i,s,h,d} x_t(e,i,s,h,d) = \sum_{e,-i,s,h,d} x_t(e,-i,s,h,d),$$

   The sex markets clear and the consumption market clears by Walras law.

3. The aggregate law of motion is,

$$\Phi_{t+1} = H_t(\Phi_t)$$

   where $\Phi$ is the joint distribution of $(e,i,s,h,d)$ is induced by the equilibrium policy functions.

4. The true distribution of the HIV population endogenously evolves according to (2.34).

**Remark.** The Stage 3-4 stationary RCE is the limiting case of the nonstationary RCE in which the cross-sectional distribution $\Phi$ does not change over time. In that case, we can drop all time subscripts.

### 2.3.1 Pseudo-Algorithm Solution

We take the following steps to find a solution to our quantitative model for a given parametrization $\theta$:

1. Find the stationary RCE for Stage -1 (Pre- HIV Era).

2. The HIV epidemic starts in Stage 0 (HIV Myopia); remember that at every period of this stage arrives an unexpected permanent shock to mortality $\widetilde{\gamma}$, productivity $\widetilde{z}$ and felicity $\widetilde{\chi}$. Then, for every period, we need to compute both the new stationary RCE associated with the new $\widetilde{\gamma}$, $\widetilde{z}$, $\widetilde{\chi}$ and the corresponding nonstationary RCE that captures the equilibrium transition from the current period (when the permanent shock occurs) to the period when the economy reaches the stationary RCE (we compute this transition backwards). Note that we are only interested in the first value function of each of those transitions, because every next period a new set unexpected permanent shocks ($\widetilde{\gamma}$, $\widetilde{z}$, $\widetilde{\chi}$) occur, which requires us to recompute again the associated stationary and nonstationary RCE.

3. Stage 1-2 (HIV Maturity) arrives as an unexpected shock that hits Stage 0 at $T_0$. Starting with a set of common prior beliefs $\widetilde{\rho_0}$, simulate a series of sequentially updated beliefs by education group until both convergence to the actual $\rho$. Solve the stationary RCE using $\rho$, and then solve the transition backwards using the simulated series for beliefs $\{\rho_t(e)\}_{t=T_0}^{\mathscr{T}}$. Where $\mathscr{T}$ is an arbitrary large number representing the time needed to converge to the stationary RCE.

4. Stage 3-4 (ARV Era) arrives as an unexpected shock that hits the transition in Stage 1-2 after $T_1$ periods. We compute the stationary RCE with full ARV coverage. Then solve the transition backwards for a given monotonically increasing sequence of coverage levels $\{\eta_t(e)\}_{t=T_1}^{\mathscr{T}}$ by education.

## 2.4 Calibration

In this Section, we discuss our calibration strategy. Some parameters which have direct observable data analogs were assigned its respective values, or values that are commonly used in the literature ($\beta, \alpha, \xi, \gamma_-, \gamma_+, f, y_{e=0}/y_{e=1}, s, z_+, \pi, \vartheta_{e=1,i}, \vartheta_{e=1,-i}, \vartheta_{e=0,i}, \tilde{\rho_o}, \phi^+_{t=0}, T_1$ ). Most of these parameters are calibrated to match the pre-HIV Era. The rest of the parameters ($T_0, \rho, \vartheta_{e=0,-i}, \sigma^2_\epsilon(e), \chi_+, \iota$) were picked to match several targeted moments in the data. We choose the same number of moments as of free parameters (i.e. exactly identified moment estimation). This procedure involves solving the model many times as to minimize the distance between the model generated moments and the moments observed in the data [33].
We explicitly target the cross country educational gradient coefficients documented in Santaeulalia-Llopis and Iorio (2016), and the evolution of the Malawi HIV prevalence from year $1990$ to $2010$. [34] The parameter governing the risk infection probability $\rho$, the time to reach maturity $T_0$ and the proportion of sex producers in the economy $\vartheta_{e=0,-i}$ are calibrated to match features of the HIV prevalence across education groups at the peak of the epidemic. The speed of learning across education groups $\sigma^2_{\epsilon,e=1}, \sigma^2_{\epsilon,e=0}$, as well as the preference shock $\chi_+$, are calibrated to match the behavior of a mature HIV epidemic. Finally, treatment coverage by education $\iota$ and a new value of $\rho$, are calibrated to reflect the effects of the introduction of ARVs at the last stage of the epidemic. We now discuss our calibration strategy in detail by stage of the epidemic.

### Stage -1: Pre-HIV Era

We calibrate $\beta$ to $0.98$ for all stages, this reflects a subjective discount rate of $2\%$ of the economy. The relative coefficient of risk aversion $\xi$ is set to $3$ as to reflect a high risk averse country like Malawi. Survival rates $\gamma = 97.7\%$ for the pre-epidemic stage are calibrated in such a a way that the individuals have an average life expectancy of $64$ years. Remember that agents enter the model when they are $18$ years old. The fertility rates $f = 4\%$ reflect the average fertility rate in Malawi in $2016$ according to the World Bank Sustainable Development Indicators.

Labor income is normalized to one for educated individuals, i.e., $y_{e=1} = 1$. The National Statistical Office of Malawi (2013) shows that the educational premium for someone who has completed secondary education is at least of $55\%$ compared to someone who has not completed primary school, so we set $y_{e=0} = .6452$. Households are subject to income shocks, $s$, that take

[33] Our objective function is based on the SMM method (Simulated Method of Moments). Specifically, let the targeted moments be $\mathcal{M}(\theta) = [\mathbf{m} - \widehat{\mathbf{m}}(\theta)]$ where $\mathbf{m}$ is a vector of observed moments and $\widehat{\mathbf{m}}(\theta)$ is the vector of model generated moments given parametrization $\theta$. Then, we construct the objective function $\min_\theta \ \mathcal{M}(\theta)^T W \mathcal{M}(\theta)$, where the weighting matrix $W$ is the diagonal matrix.

[34] Although the model is not explicitly calibrated to a particular country, we use data from Malawi to discipline most of the parameters of the model.

two possible values: $s$ equals to one in good times and $s$ equals to $.4$ in bad times which mimics a $60\%$ loss of household income during a period of unemployment Magalhaes and Santaeulalia-Llopis (2018). The income shock follows a Markov process with a transition matrix $\pi$ from high (i.e employment) to low (i.e unemployment) that is calibrated so that at all times $5.4\%$ of the population is under low income[35]. Since there is no HIV in at this point, labor and sex productivity $z$ and preference parameter $\chi$ are equal to one. We choose $\alpha$ such that the proportion of sex income in the aggregate economy is $7\%$ Sulaimon et al. (2018)[36]; this in turn, implies a proportion of sex income in the total income of sex producers, $\omega$ of $12\%$.

We also need to choose the proportion of educated and non educated individuals. We set the proportion of educated to be $13\%$[37] as observed in the DHS surveys for Malawi; we classify as educated those with completed primary education, this anchors the estimation of the model-generated HIV-education gradient. To see this, notice that the individuals who didn't complete primary education average $3.35$ schooling years, and those with at least primary education average $8.32$ schooling years. We use use this difference in schooling years across education for the estimation of the HIV-education gradient with the model-generated data.

We assume that all sex producers are non educated ($\vartheta_{e=1,-i} = 0$ i.e $\vartheta_{e=1,i} = 0.13$ from (37)). This is reasonable either because prostitutes are less educated or because teenagers that engage in sexual activity (STDs, pregnancy, etc.) are less likely to finish school Duflo et al. (2015b), Dupas (2011b). This implies that all educated individuals are sex consumers. Consequently, we need to choose the proportion of sex producers in the economy ($\vartheta_{e=0,-i}$), which in turn delivers the proportion of sex consumers among the uneducated people ($\vartheta_{e=0,i}$). We choose this proportion to match the HIV prevalence rate at the peak of the epidemic in the next stage as we discuss next. Finally, we set the initial prevalence $\phi^+_{t=0}$ to $0.5\%$, this is number is directly linked with the calibration of the duration of the next stage (Stage 0 HIV Myopia) $T_0$ because the larger is $\phi^+_{t=0}$, the less periods necessary to reach the peak.

### Stage 0: Myopic Onset of the HIV Epidemic

At this stage the ability of infected individuals to produce both sex and the consumption good at a given scale $z$ is reduced by $65\%$. We choose the value of $z_+$ such that the proportion of sex income among sex producers at the peak is the same $\omega$ as in the pre-HIV stage. Additionally the survival probability of someone who is infected with HIV reduces to $\gamma_+ = 90\%$, which is

[35] This is calibrated to the average unemployment rate in Malawi in 2019, Source: ILOSTAT database

[36] Sulaimon et al. (2018) mention that in Indonesia, Malaysia, the Philippines and Thailand, the sex sector (prostitution) accounts for between $2\%$ and $14\%$ of the Gross Domestic Product.

[37] In the model this is the sum of the proportion of educated consumers plus educated producers $\vartheta_{e=1,i} + \vartheta_{e=1,-i} = 0.13$

translated to a life expectancy of $11$ years after the moment of infection.

There are three additional parameters to calibrate. The first parameter carries from the previous stage, which is the proportion of sex producers in the economy $\vartheta_{e=0,-i}$. The second is a parameter, $\rho$, that governs the true rate of infection as a function of risky sex, $\lambda(x;\rho)$. The third is the time until the epidemic reaches maturity $T_0$. We choose these three parameters such that we match the HIV prevalence by education group at the peak of the epidemic as well as the number of years needed to reach the peak[38].

Once we have the proportion of individuals that are producers in the economy $\vartheta_{e=0,-i}$, we use the joint distribution of education groups and sex types (producers vs. consumers) at the pre-HIV stage ($\vartheta_{e=1,i}, \vartheta_{e=1,-i}, \vartheta_{e=0,i}, \vartheta_{e=0,-i}$) to feed the economy at each and every period (and stage) with a fertility that maintains these proportions at birth.

We finally need to choose the magnitude of the preference shock $\chi_+$. This parameter is chosen in the next stage to match the HIV education gradient just before the introduction of ARVs.

### Stage 1-2: HIV Maturity, learning the HIV Epidemic

Recall that in this stage agents are aware of the existence of HIV, but their knowledge of the degree of infection risk through sex $\lambda(x;\rho)$ is imperfect. They learn about $\lambda(x;\rho)$ (i.e., $\rho$) through Bayesian updates. The speed at which agents learn about the true risk of infection depends on two factors. First, the noise of the updating signal $\sigma^2_\varepsilon(e)$ which differs across education groups. Second, how far their initial prior of the infection probability $\tilde{\lambda}_o$ (i.e., $\tilde{\rho}_o$), is from the true value $\lambda$ (i.e., $\rho$). This initial prior belief is common across education groups. [39]
The initial common prior belief $\tilde{\rho}_o$ is set to an arbitrary high number following from the fact that agents were myopic in the previous stage and their initial belief of the risk of infection through sex $\widetilde{\lambda_o} = \lambda(x, \tilde{\rho}_o)$ is approximately zero.
We choose the $\sigma^2_\varepsilon(e)$ by education group and the magnitude of the preference shock $\chi_+$ that carries from the previous stage; such that we match the HIV-education gradient (i.e., HIV prevalence by education group) and the average time that it takes to transit from the peak of the HIV epidemic to the end of Stage 1-2 (HIV Maturity). Finally we select the duration of this stage ($T_1$) such that $year(T_0) + T_1 = 2005$ which is the year in when ARVs were introduced in Malawi.

[38] The first HIV positive patient in Malawi was detected in 1985. In 1986 the government of Malawi started implementing preventive measures against the spread of the virus Mwale (2002). After calibration, the agents in our model become aware of the existence of the virus in year 1986. In section 2.6 we explore alternative scenarios in which the population starts learning about the virus in earlier stages of the epidemic.

[39] We tried a version having a common noise for the signal updates $\sigma^2_\varepsilon$ and different initial prior beliefs $\tilde{\rho}_o(e)$, however this set up did not guarantee different convergence times across education groups.

### Stage 3-4: The Era of ARVs

In this stage, the HIV/AIDS effects disappear from the budget constraint of those who are treated, therefore they now have the same survival rate as if healthy. To inform the model about the evolution across time of the proportion of the infected population receiving ARV treatment, we use aggregate treatment data from Malawi starting in 2005 until 2018.[40] We calibrate the parameter governing the share of educated and non educated individuals receiving ARV's $\iota$ as to match the HIV gradient of this stage.

In addition, the introduction of ARV treatment reduces the overall degree of infectiousness in the economy, this translates into a reduction of the true infection rate conditional on sex consumption $\lambda(x;\rho)$(i.e an increase in $\rho$). We calibrate the new value of $\rho$ as to match Malawi's average prevalence rate in 2018. Table 2.7 summarizes the calibration of the parameters of the model.

**Table 2.7: List of parameters**

| Description | Symbol | Perceived | | | True | Stage |
|---|---|---|---|---|---|---|
| | | Miopic | Onset | ART | Value | Dependent |
| Discount factor | $\beta$ | | 0.98 | | 0.98 | No |
| Labor share of sex income | $\alpha$ | | 0.01 | | 0.01 | No |
| Risk aversion | $\xi$ | | 3.00 | | 3.00 | No |
| Survival rate healthy (%) | $\gamma(-)$ | $\widetilde{\gamma}$ | 97.7 | 97.7 | 97.7 | Yes |
| Survival rate infected (%) | $\gamma(+)$ | $\widetilde{\gamma}$ | 90.0 | 90.0 | 90.0 | Yes |
| Survival rate infected but treated (%) | $\gamma_{d+}(+)$ | - | - | 97.0 | 97.0 | Yes |
| Preference parameter healthy | $\chi(-)$ | $\widetilde{\chi}$ | 1.0 | 1.0 | 1.0 | Yes |
| Preference parameter infected | $\chi(+)$ | $\widetilde{\chi}$ | 230 | 230 | 230 | Yes |
| Preference parameter infected but treated | $\chi_{d+}(+)$ | - | - | 230 | 230 | Yes |
| Fertility rate (%) | $f$ | | 4.0 | | 4.0 | No |
| Understanding of epidemic $e=1$ | $\rho_{e=1}$ | - | $\widetilde{\rho}_{e=1}$ | $\widetilde{\rho}_{e=1}$ | $\rho$[41] | Yes |
| Understanding of epidemic $e=0$ | $\rho_{e=0}$ | - | $\widetilde{\rho}_{e=0}$ | $\widetilde{\rho}_{e=0}$ | $\rho$ | Yes |
| Education premium (%) | $y_{e=0}/y_{e=1}$ | | 45.0 | | 45.0 | No |
| Productivity if infected (%) | $z(+)$ | $\widetilde{z}$ | 65.0 | 65.0 | 65.0 | Yes |
| Productivity if treated (%) | $z_{d+}(+)$ | - | - | 100.0 | 100.0 | Yes |
| Income shock (%) | $s$ | | 60.0 | | 60 | No |

[40] Recent DHS data does not provide micro level information to distinguish if ARV treatment is higher among educated individuals, therefore we are unable to compute any gradient related to ARV treatment.

[41] $\rho = 19$ for the Miopic and Onset stages, however once ARVs are introduced the overall infectiousness reduces; this is translated into an increase of to $\rho = 44$ for the ARV stage.

Table 2.7 – Continued

| Description | Symbol | Perceived | | | True | Stage |
|---|---|---|---|---|---|---|
| | | Miopic | Onset | ART | Value | Dependent |
| Transit probability from $s_g$ to $s_g$ | $p_{gg}$ | | 0.95 | | 0.95 | No |
| Transit probability from $s_g$ to $s_b$ | $p_{gb}$ | | 0.05 | | 0.05 | No |
| Transit probability from $s_b$ to $s_g$ | $p_{bg}$ | | 0.90 | | 0.90 | No |
| Transit probability from $s_b$ to $s_b$ | $p_{bb}$ | | 0.10 | | 0.10 | No |
| Initial proportion of type $e = 1, i$ | $\vartheta_{e=1,i}$ | | 13.0 | | 13.0 | No |
| Initial proportion of type $e = 1, -i$ | $\vartheta_{e=1,-i}$ | | 0.0 | | 0.0 | No |
| Initial proportion of type $e = 0, i$ | $\vartheta_{e=0,i}$ | | 57.6 | | 57.6 | No |
| Initial proportion of type $e = 0, -i$ | $\vartheta_{e=0,-i}$ | | 29.4 | | 29.4 | No |
| Variance of the signal's noise $\epsilon$ for $e = 1$ | $\sigma^2_{\epsilon,e=1}$ | - | 7.9 | 7.9 | - | No |
| Variance of the signal's noise $\epsilon$ for $e = 0$ | $\sigma^2_{\epsilon,e=0}$ | - | 84 | 84 | - | No |
| Initial mean of prior $\mathcal{P}_{e=1}(\lambda(x;\tilde{\rho}_o(e=1)))$ | $\tilde{\rho}_o$ | - | 15700 | - | - | No |
| Initial mean of prior $\mathcal{P}_{e=0}(\lambda(x;\tilde{\rho}_o(e=0)))$ | $\tilde{\rho}_o$ | - | 15700 | - | - | No |
| Odds of treatment parameter | $\iota$ | - | - | 0.074 | - | No |
| Initial Prevalence (%) | $\phi^+_{t=0}$ | 0.5 | - | - | 0.5 | No |
| Duration of HIV Myopia (g=0) (Years) | $T_0$ | $\infty$ | - | - | 17 | No |
| Duration of HIV Maturity (g=1-2) (Years) | $T_1$ | - | $\infty$ | - | 16 | No |

### 2.4.1 Model Fit

Table 2.8, presents the data moments and shows how well the benchmark model matches them. The benchmark calibration matches the HIV-Education Gradient very well. Figure 2.4 shows the isomorphic representation of the estimated HIV-Educ gradient. The model also gets pretty close to the actual values of the prevalence all along the evolution of HIV epidemic; Figure 2.7 compares the prevalence generated by the model with data for Malawi. Note that the Model's prevalence's is always within the $95\%$ confidence interval. Keep in mind however, that the model was calibrated using cross country data for the gradient, and not exclusively prevalence data for Malawi, therefore we consider this a very good approximation for the prevalence.

We have explicitly modelled the direct link between risky sexual behaviour and the probability of infection across education groups through $\lambda_\rho(x)$, as to capture the parallel evolution between the HIV-Education Gradient and disparities in risky sexual behaviour by education that is found the

data, therefore the model is able to generate a Risky sex Education gradient that is consistent with the U-shaped pattern of the HIV education gradient and the U-shape of Risky sex Education gradient in the data. Figure 2.6 Panel (a) illustrates this pattern.

Additionally, the model performs remarkably well mimicking other moments relevant for the HIV epidemic. In the model the average HIV incidence at the peak is $1.66\%$, close to $1.6\%$ that is reported in the data[42] for the population between 14-49 years in Malawi. The model also captures the slow down of population growth before the peak of the HIV epidemic and the increased population growth afterwards.

The Model also performs well in other non-targeted dimentions. Panels (a) and (b) of Figure 2.9 illustrate the behaviour of the flow of (HIV) infections and the flow of (HIV) deaths compared to the data.

**Table 2.8: Targeted Moments**

| Observation | Data | Model |
|---|---|---|
| HIV Education gradient at the peak (1999) | 0.0099 | 0.0112 |
| HIV Education gradient at the end of Maturity (2005) | -0.0005 | -0.0015 |
| HIV Education gradient ARV Stage (2018) | 0.0046 | 0.0047 |
| Prevalence at the peak (1999) | 14.6% | 14.1% |
| Prevalence at the end of Maturity (2005) | 12.2% | 13.1% |
| Prevalence ARV Stage (2018) (Stage 3-4 ARV Era) | 9.2% | 9.4% |
| Time to reach from bottom to peak (in years) | 29 | 28 |
| Time to reach from peak to end of Maturity (in years) | 6 | 6 |

[42] Source: UNAIDS Estimates 2019.

Figure 2.6: HIV Education Gradient, comparison between model and data

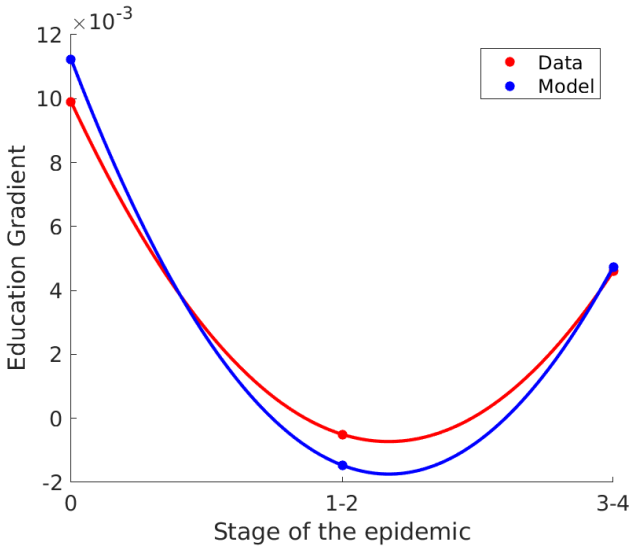
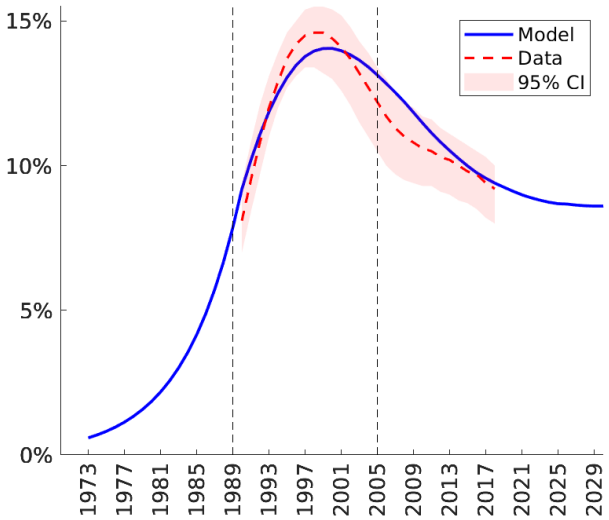



Figure 2.7: Evolution of the HIV Prevalence rates




Notes: The figure compares prevalence generated by the model and data for Malawi. Data source: UNAIDS estimates 2019.

### 2.4.2 Comparative Statics

This section performs comparative statics concerning the parameters estimated in Section 2.4. Figure 2.8 shows contour-maps for some of the moments of interest. These moments were computed using model generated data for given combination of parameter values. The red contour line represents the value observed in the data that is to be matched. The intersection between the red lines was used as the initial guess for the SMM estimation, this considerable speeds up the estimation. Looking at the contour plots and the red lines, we can easily see the parameters that identify each the moment. We now explore identification in some detail.

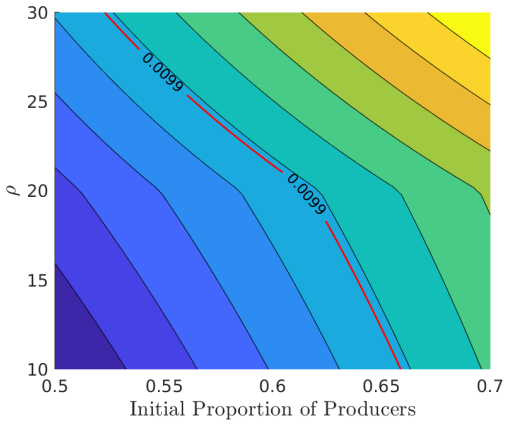
Panel (a) and (b) show that the HIV education gradient at the peak is more sensitive to variations of the initial proportion of less educated Risky-sex consumers $\vartheta_{-i,e=0}$, whereas the parameter governing the infection rates $\rho$ has a larger impact on the prevalence's at the peak.

In Panels (c) and (d) we see that both the standard deviation of the signal's noise for less-educated $\sigma^2_{\epsilon,e=0}$ and the preference parameter $\chi$, play a role in determining the HIV education gradient. However, Panel (d) shows that the time to reach the prevalence at the bottom is mostly sensitive to the preference parameter $\chi$.

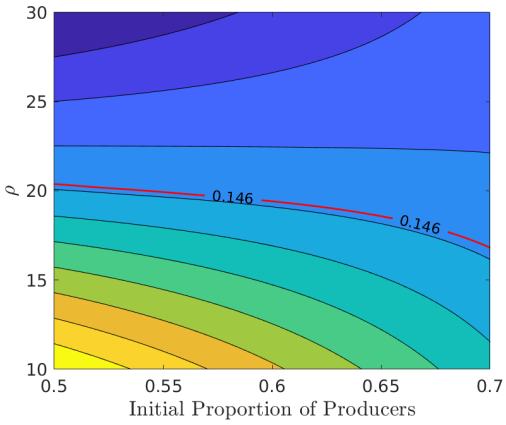
Finally, Panels (e) and (f) show that the HIV education gradient responds strongly to changes in the coverage differences by education $\iota(e)$ and that the level of the prevalence moves along with the new value of the infection rate parameter $\rho$.

Figure 2.8: Comparative Statics

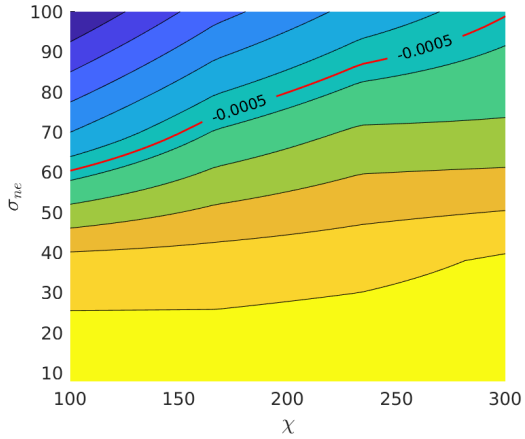
(a) Education Gradient at the peak



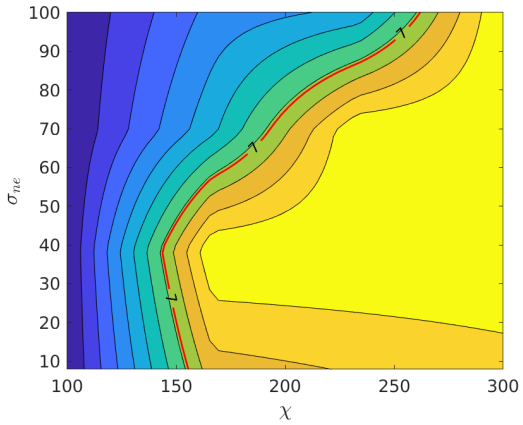

(b) Prevalence at the peak



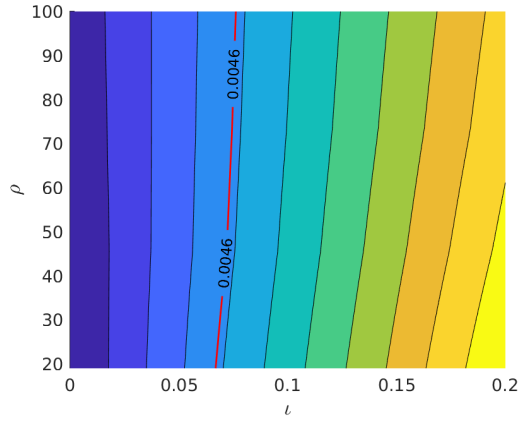

(c) Education Gradient at the end of Maturity



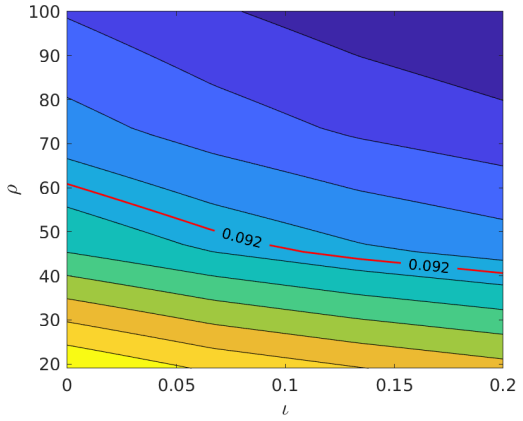

(d) Years to reach from peak to end of Maturity




(e) Education Gradient ARV stage

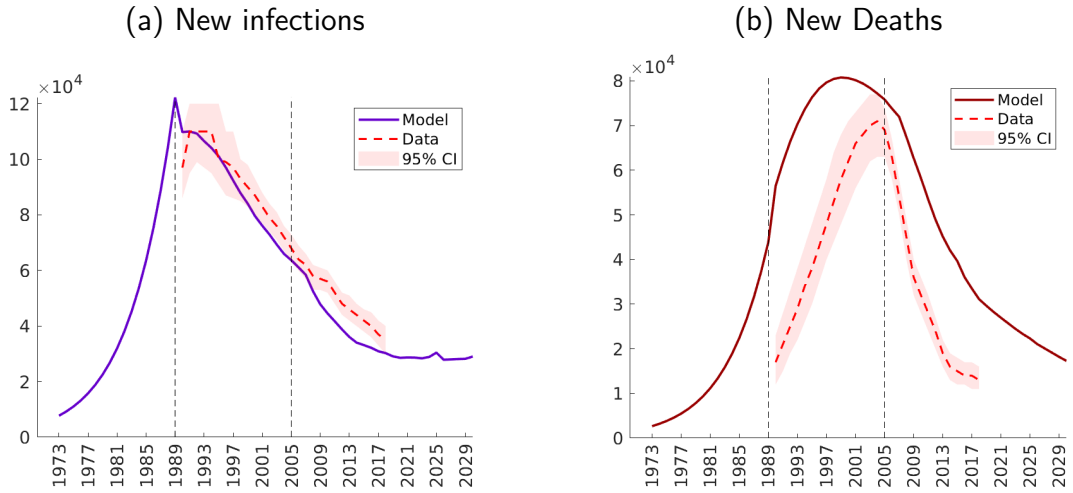



(f) Prevalence ARV stage




Notes: Panels (a) and (b) show sensitivity of the HIV Education Gradient at the peak and the prevalence at the peak with respect to the infection parameter $\rho$ and the initial proportion of sex producers $\vartheta_{-i,e=0}$. Panels (c) and (d) display the sensitivity of the HIV Education Gradient at the end of Maturity with respect to the preference parameter $\chi$ and the variance of the signal for less educated individuals $\sigma^2_{\epsilon,e=0}$. Finally panels (e) and (f) show the sensitivity of the the gradient and prevalence after the introduction of ARV's, this time with respect to the coverage difference parameter $\iota$ and the new value of $\rho$.

Figure 2.9: Non Targeted Moments




Notes: Panels (a) and (b) show the evolution of the new infections and new deaths respectively. Panel (a) shows that the model does are very good job matching infections although the series was not directly targeted.

## 2.5 What drives the epidemic?

Education affects the risky sex behaviour of individuals through three different channels along the evolution of the HIV epidemic: First, higher current income increases the amount of risky sex consumed because risky sex is a normal good. This channel is present throughout the complete duration of the HIV epidemic. Second, during Stage 1-2 more-educated individuals acquire information about the negative effects of HIV faster than their less-educated counterparts, which in turn reduces their risky sex consumption. Third, at stage 3-4, educated individuals have better access to (costly) ARV's that mitigate the effects of the disease, therefore increasing incentives for risky sex consumption. In this section we explore counterfactual experiments neutralizing the second and third channels, while keeping the first present. Note that neutralizing the first channel would be analogous to having a starting HIV Education-gradient equal to zero.

Depending on their magnitude, changes of the education composition of the population can also affect the dynamics of the HIV epidemic [43]. To account for this, we experiment by exogenously varying the education composition of the population across stages to resemble that of Malawi.

### 2.5.1 Removing Learning Asymmetry

**Experiments 1-2: Removing learning differences between education groups**

We explore two different ways of removing learning differences between the educated and the

[43] Note that throughout the different stages of the benchmark calibration we indirectly maintained the joint distribution of education groups and sex types constant across time.

less-educated:

1. Making the less-educated individuals learn at the same (fast) speed as their more-educated counterparts. This means setting $\sigma^2_{\epsilon,e=0} = \sigma^2_{\epsilon,e=1} = 7.9$
2. Making the more-educated individuals learn at the same (slow) speed as their less-educated counterparts. This means setting $\sigma^2_{\epsilon,e=1} = \sigma^2_{\epsilon,e=0} = 84$

Columns 3-4 on Table 2.9 show the results for these two experiments. If the less-educated learn as fast as the educated (Column 3), we find that $52\%$ of the HIV Education gradient and $53\%$ of the Partners Education gradient is explained by the information differences, as before this effect is carried out to the next stage, where $36\%$ of the HIV Education gradient is explained by information differences coming from the previous stage.
Similarly, if the more-educated learn at the same slow rate as the less educated (Column 4 ), we see that the gradient keeps increasing, to reach a peak at $0.08$. In this experiment gents learn to slow therefore delaying the maturity of the epidemic.
Figures 2.10 illustrate these counterfactual experiments against the benchmark.

### 2.5.2 Removing Differences in Access to ARVs

**Experiment 3: Removing coverage access differences between education groups**
This experiment involves setting $\iota = 0$. This means that educated individuals will get the same chance of treatment as everyone else in the population. Column 5 on Table 2.9 show the results for this experiment. We can see that the gradient continues to be negative, consistently the HIV prevalence at after ARV introduction is higher.

### 2.5.3 Accounting for the evolution of the education composition

**Experiment 4: Accounting for the evolution of the education composition in Malawi**
In this experiment we exogenously modify the education composition of the population to mimic that of Malawi. We achieve this by modifying the proportion of new born educated such that along the equilibrium path the resulting (endogenous) proportion of educated vs less educated resembles the evolution path observed in the data. In 1977 only $7.3\%$ of the population above 25 years old had completed primary education. By 2000 this number increased to $19.8\%$ (WorldBank, 2020).

**Table 2.9: Counterfactual Experiments**

| Observation | Benchmark | Experiments | | | |
|---|---|---|---|---|---|
| | | 1 Fast Learning | 2 Slow Learning | 3 $\iota = 0$ | 4 Increasing Education |
| HIV Education gradient at end of HIV Myopia (1989) | 0.0595 | 0.0595 | 0.0595 | 0.0595 | 0.0533 |
| HIV Education gradient at the peak (1999) | 0.0112 | 0.0527 | 0.0796 | 0.0112 | -0.0177 |
| HIV Education gradient at the end of Maturity (2005) | -0.0015 | 0.0310 | 0.0720 | -0.0015 | -0.0294 |
| HIV Education gradient ARV stage (2018) | 0.0047 | 0.0246 | 0.0531 | -0.0027 | -0.0183 |
| Prevalence at the peak (1999) | 14.1% | 10.9% | 16.8% | 14.1% | 14.1% |
| Prevalence at the end of Maturity (2005) | 13.1% | 7.1% | 16.4% | 13.1% | 13.6% |
| Prevalence ARV stage (2018) | 9.4% | 5.4% | 12.2% | 9.8% | 10.4% |
| Time to reach from bottom to peak (in years) | 28 | 21 | 30 | 28 | 26 |
| Time to reach from peak to end of Maturity (in years) | 6 | 13 | 4 | 6 | 6 |

## 2.6 Policy Experiments

In this section we explore a set counterfactual experiments concerning the evolution of the HIV epidemic and policy interventions. We ask ourselves how would heave the HIV epidemic evolved in the following cases:

1. Early acknowledgement of the presence of the virus and its workings.
2. Improving the education level of the population.
3. Early and universal adoption of ARVs.
4. HIV prevention through Pre-exposure prophylaxis (PrEP).

**Early acknowledgement of the presence of the virus and its workings**

In Figure 2.14 panels (a) to (c) we show how the epidemic would have evolved had the learning stage started 5 years earlier and 10 years earlier than in the benchmark calibration. Panel (a) shows that the peak reaches a lower level the earlier the learning starts. Along the same line, the HIV education gradient and the sexual partners gradient start dropping at earlier stages.

**Improving the education level of the population**

In 2000 the UN set out a series of time-bound targets, with a deadline of 2015, that were aimed to reduce extreme poverty around the globe. These targets have later become known as the Millennium Development Goals (MDGs) and were succeeded by what we currently know as the UN Sustainable Development Goals (SDGs) to be achieved by 2030. One of the goals in the agenda (Goal 4) aims to ensure that all girls and boys complete free, equitable and quality primary and secondary education.

In our experiment we explore the possibility of achieving this goal by 2030. We do this by gradually increasing the proportion of educated new born individuals over time $\{\vartheta_{e=1,i,t}\}_{t=2000}^{\infty}$ such that $\lim_{t\to\infty} \vartheta_{e=1,i,t} = 85\%$ (the current level attained by South Africa) [44]. We start in 2000 but we also explore an alternative scenario where the starting point is 2018 instead, in this case the goal is achieved by 2048.

Figure 2.13 panels (d) to (f) show the results of these experiments. We see that there is no significant change in the prevalence levels however, once a larger share of the population educated, the HIV education gradient and Risky sex education gradient turn negative for the rest of the path.

**Early and universal adoption of ARVs**

In Figure 2.15 panels (d) to (f) we show how the epidemic would have evolved if ARV's were introduced at an earlier stage. If ARV's would have been introduced 6 years earlier (that is at the peak of the epidemic in 1999 ) universal coverage would have been attained by 2019 however the prevalence rate does not show a significant reduction. Moreover, if ARV's were introduced 16 years earlier (that is 1989 which is also the year when agents start learning about HIV) universal coverage would have been attained by 2009 and the prevalence rate would have been significantly reduced.

**HIV prevention through Pre-exposure prophylaxis (PrEP)**

Pre-exposure prophylaxis (PrEP) is taken on a daily basis by HIV-negative people as protection from HIV infection. Evidence shows that PrEP reduces the chances of HIV infection to near-zero ($99\%$ effectiveness) when taken consistently and correctly (Avert, 2020). PrEP is not widely available in Malawi, although a clinical trial among HIV-positive pregnant adolescents and young women (ages 16-24) and an implementation study for at-risk adults and adolescents are underway (Avert, 2020).

We construct an experiment where we analyze the evolution of the epidemic as if PrEP was

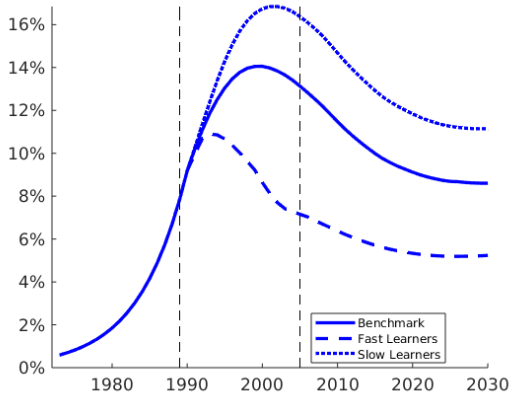
[44] The education composition for South Africa was chosen as a more realistic alternative to fully achieving primary education attainment as per the SDG's, that is $\lim_{t\to\infty} \vartheta_{e=1,i,t} > 85\%$. However, we explored by setting the limit to $90\%$ and the main conclusions remained unaltered.

implemented nationwide starting in 2018, [45] with coverage gradually increasing and reaching full coverage by 2040; we further assume PrEP is administered at no costs and that there is no differences on PrEP take-up by education. In the model, PrEP implies a higher value of $\rho$ for those taking the drug, we calibrate this value such that the current average probability of infection is reduced by $99\%$. Moreover, we ask ourselves: What if PrEP would only reduce the infection probability by $30\%$, $50\%$ and $70\%$? would it still be worth scaling up its use?
Panels (a) to (c) of Figure 2.16 show that in all four cases the prevalence levels reduce considerably as well as the HIV incidence and the numbers of HIV deaths. From the model perspective we can conclude that PrEP implementation is highly recommended at any degree of effectiveness.

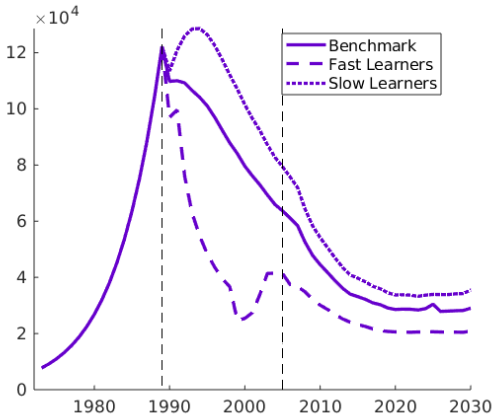
[45] This involves the introduction of a new state variable: those who are healthy can now be taking PrEP. We introduce transitions to PrEP in the same spirit as previously done with ARV's.

Figure 2.10: Experiment 1-2, Removing Learning differences

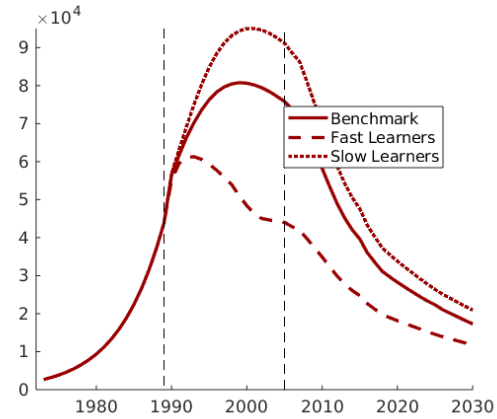
(a) Prevalence



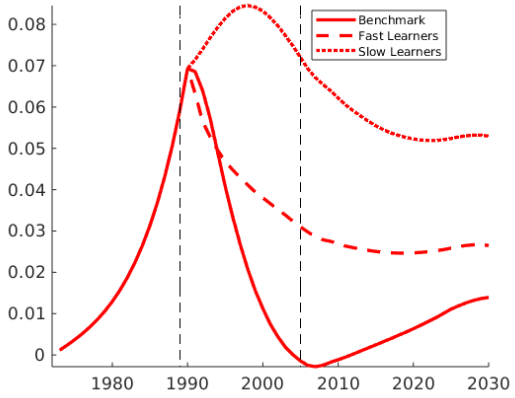

(b) New Infections



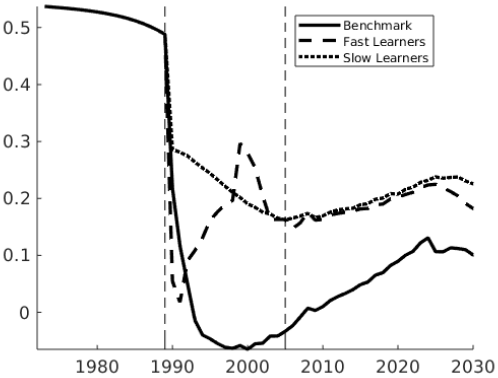

(c) New Deaths




(d) HIV Education Gradient

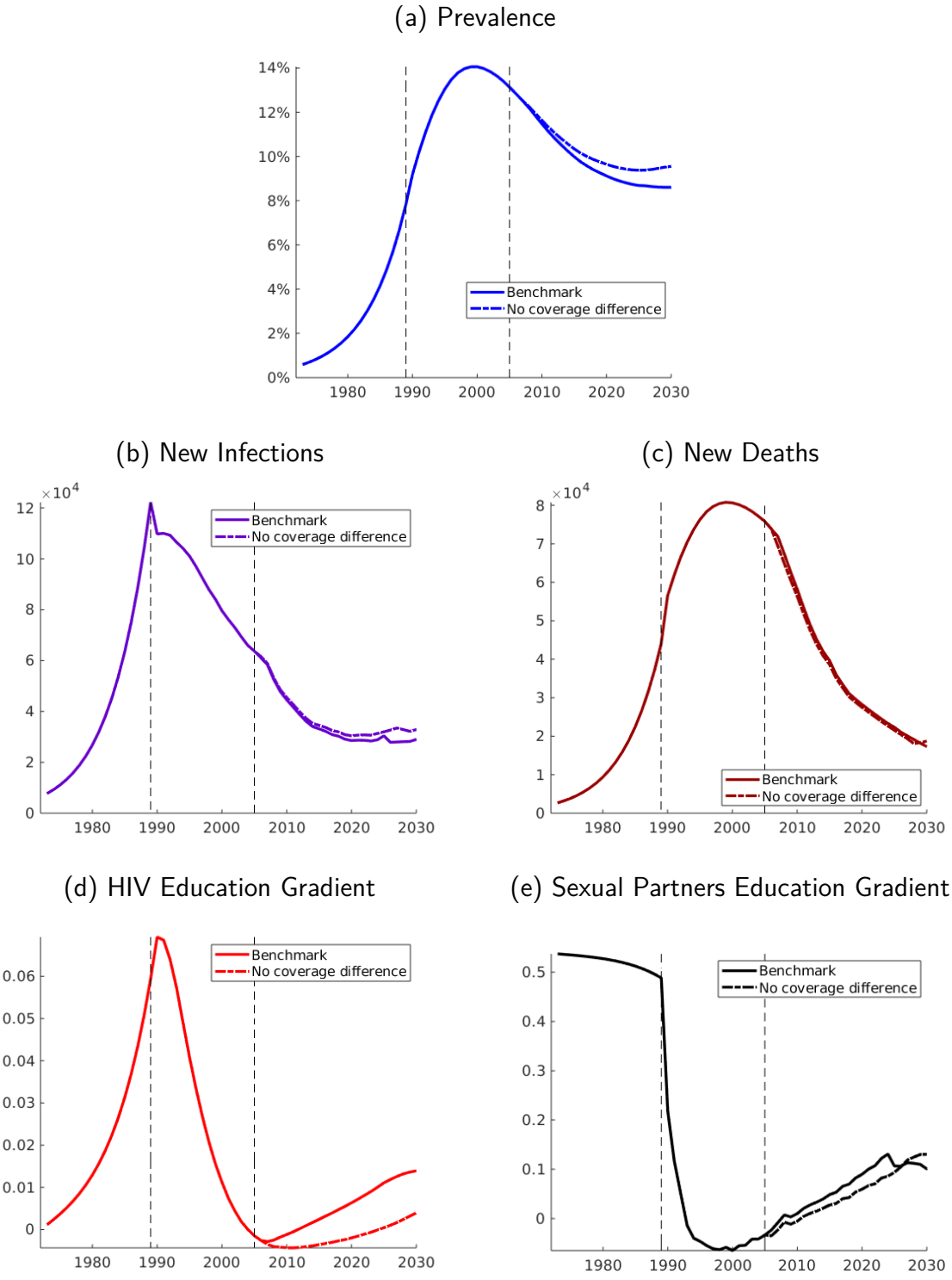



(e) Sexual Partners Education Gradient



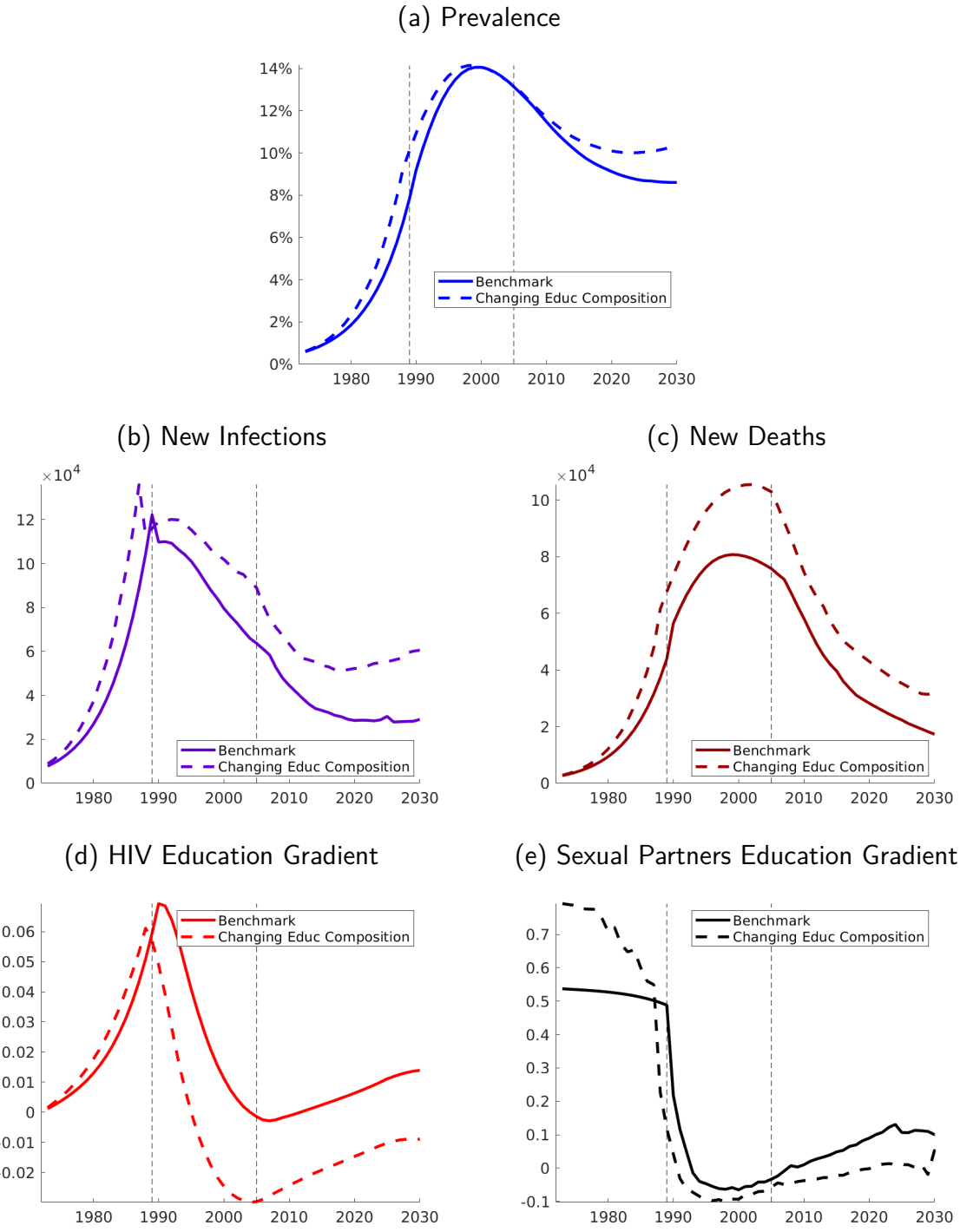

Notes: The figure shows the results of different counterfactual experiments; each row shows the effects on the time series of the prevalence rate, HIV education gradient panel and Sexual partners education gradient. Panels (a) to (c) show experiments 1-3 and panels (d) to (f) illustrate a gradual increase of the proportion educated agents. The figures also show the effects for different starting dates.

Figure 2.11: Experiment 3, Removing ARV accessibility differences



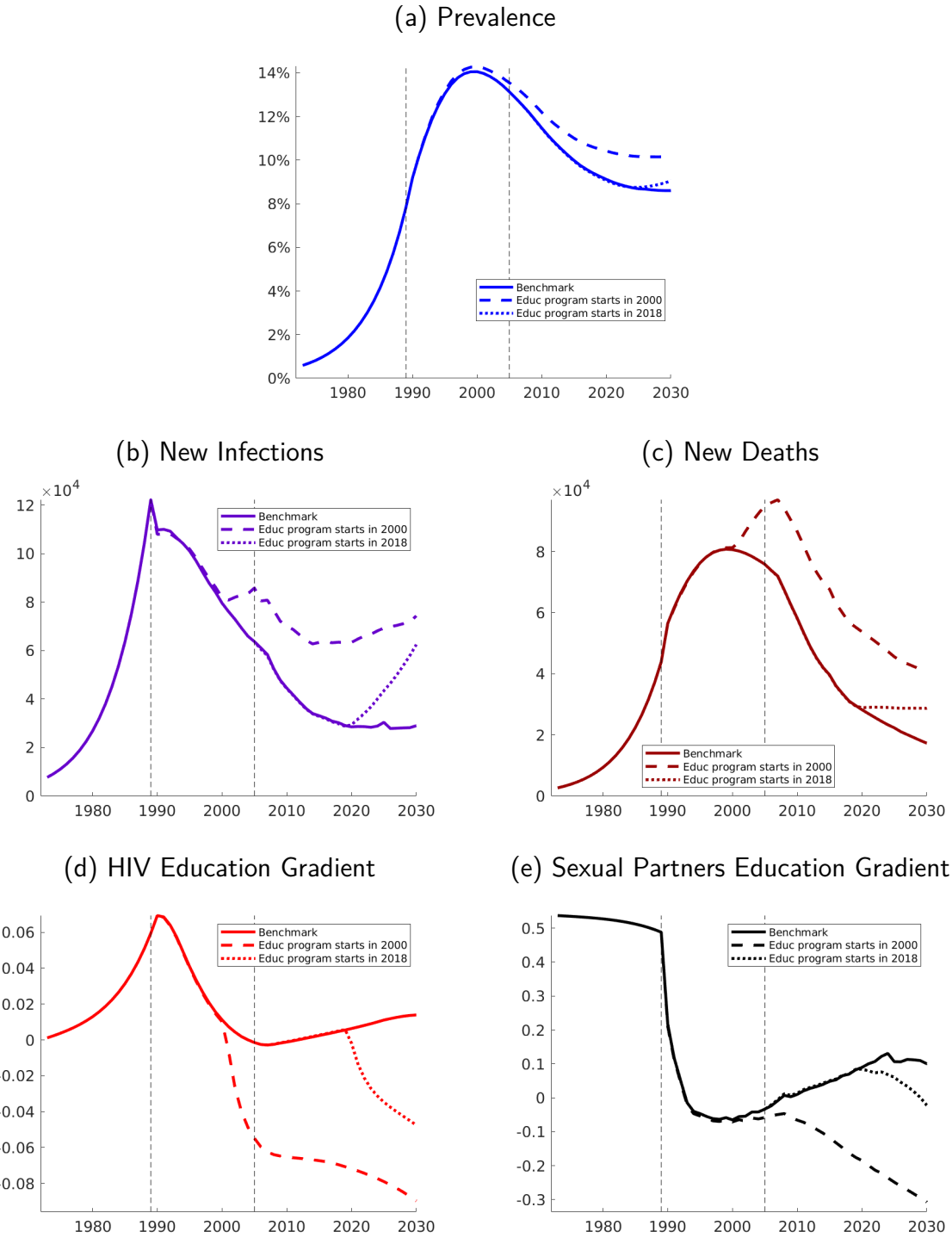

Notes: The figure shows the results of different counterfactual experiments; each row shows the effects on the time series of the prevalence rate, HIV education gradient panel and Sexual partners education gradient. Panels (a) to (c) show experiments 1-3 and panels (d) to (f) illustrate a gradual increase of the proportion educated agents. The figures also show the effects for different starting dates.

Figure 2.12: Experiment 4, Accounting for the evolution of the education composition



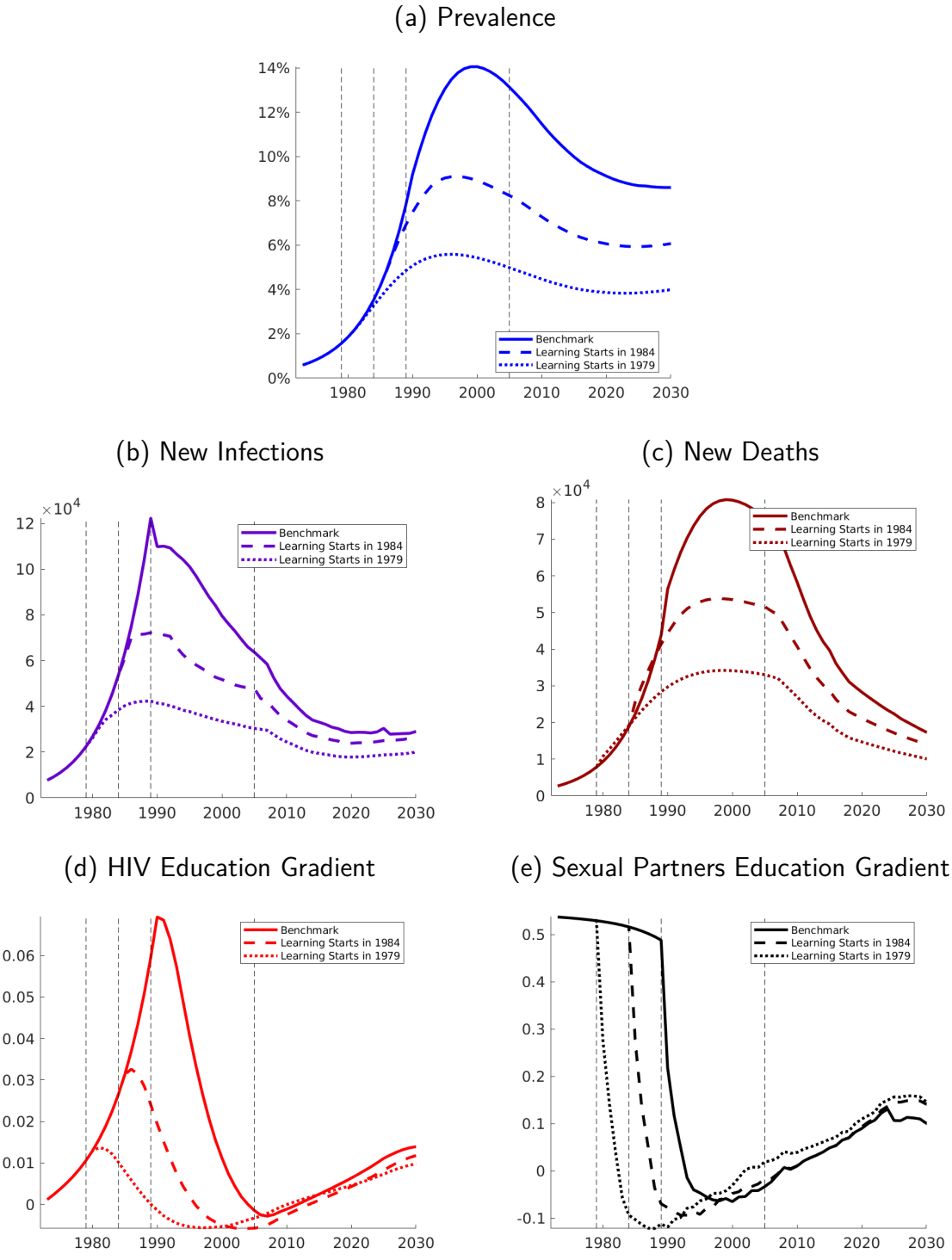

Notes: The figure shows the results of different counterfactual experiments; each row shows the effects on the time series of the prevalence rate, HIV education gradient panel and Sexual partners education gradient. Panels (a) to (c) show experiments 1-3 and panels (d) to (f) illustrate a gradual increase of the proportion educated agents. The figures also show the effects for different starting dates.

Figure 2.13: Policy Experiment 1, Increasing the education level of the population



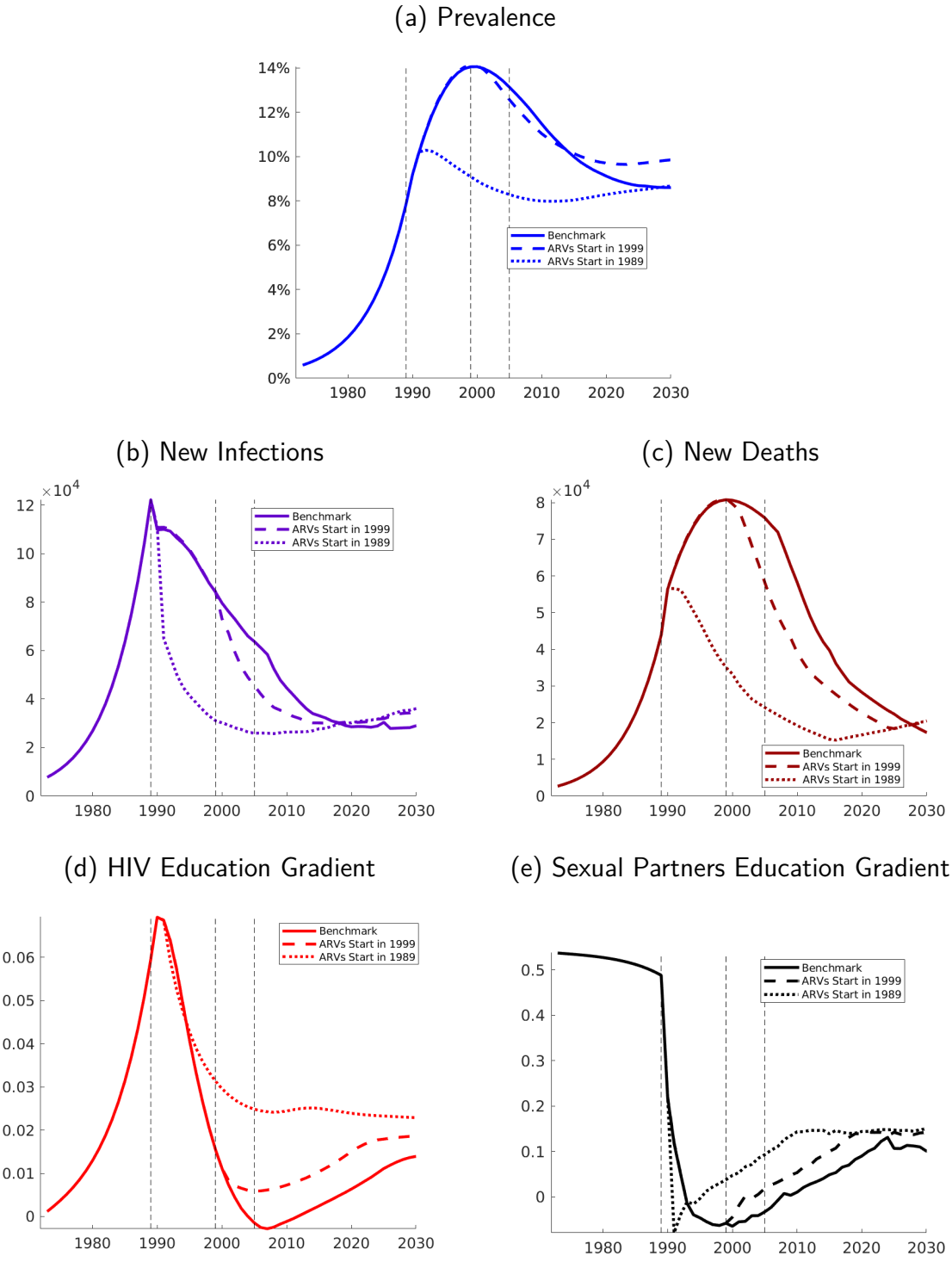

Notes: The figure shows the results of different counterfactual experiments; each row shows the effects on the time series of the prevalence rate, HIV education gradient panel and Sexual partners education gradient. Panels (a) to (c) show experiments 1-3 and panels (d) to (f) illustrate a gradual increase of the proportion educated agents. The figures also show the effects for different starting dates.

Figure 2.14: Policy Experiment 2, Early Learning



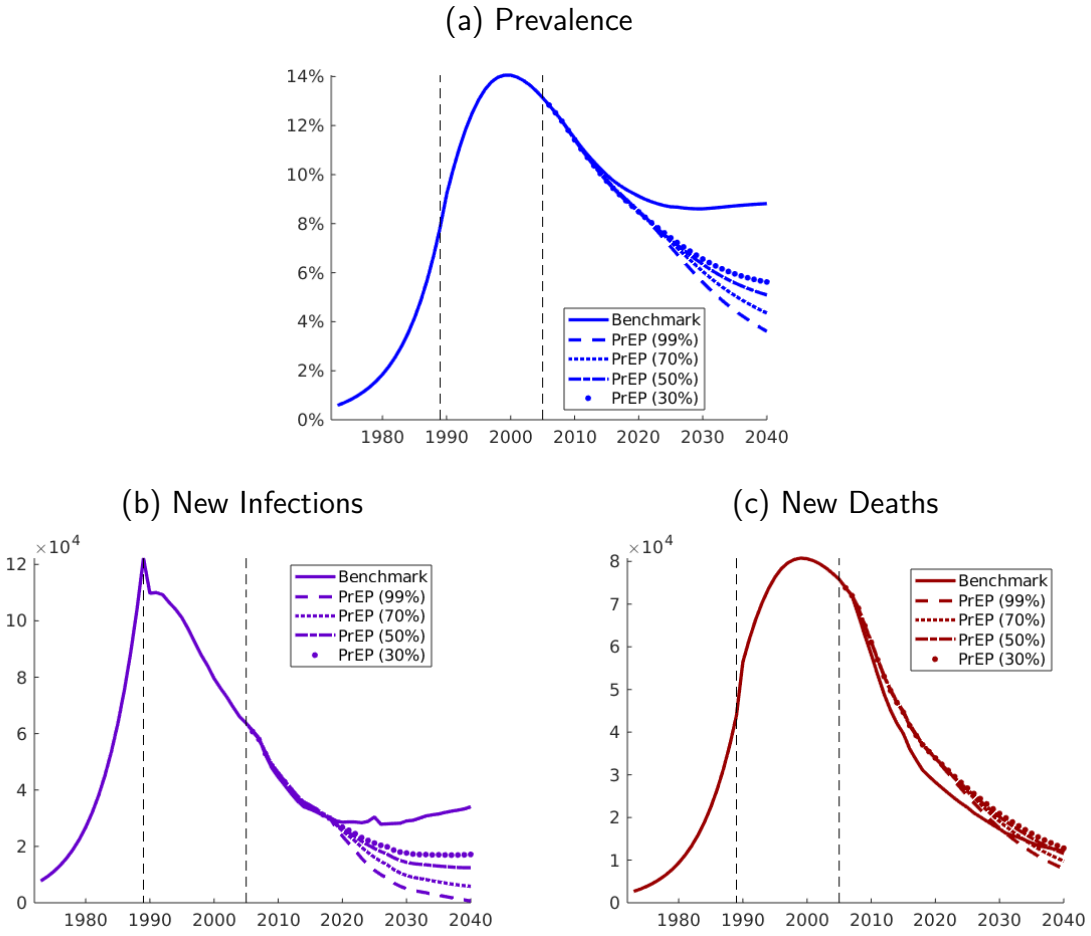

Notes: The figure shows the results of different counterfactual experiments, panels (a) to (c) show the effects of different (earlier) starting dates of the learning stage. Panels (d) to (f) simulate different dates for the introduction of ARVs.

Figure 2.15: Policy Experiment 3, Early Adoption of ARVs




Notes: The figure shows the results of different counterfactual experiments, panels (a) to (c) show the effects of different (earlier) starting dates of the learning stage. Panels (d) to (f) simulate different dates for the introduction of ARVs.

Figure 2.16: Policy Experiment 4, Preventing HIV infection with PrEP

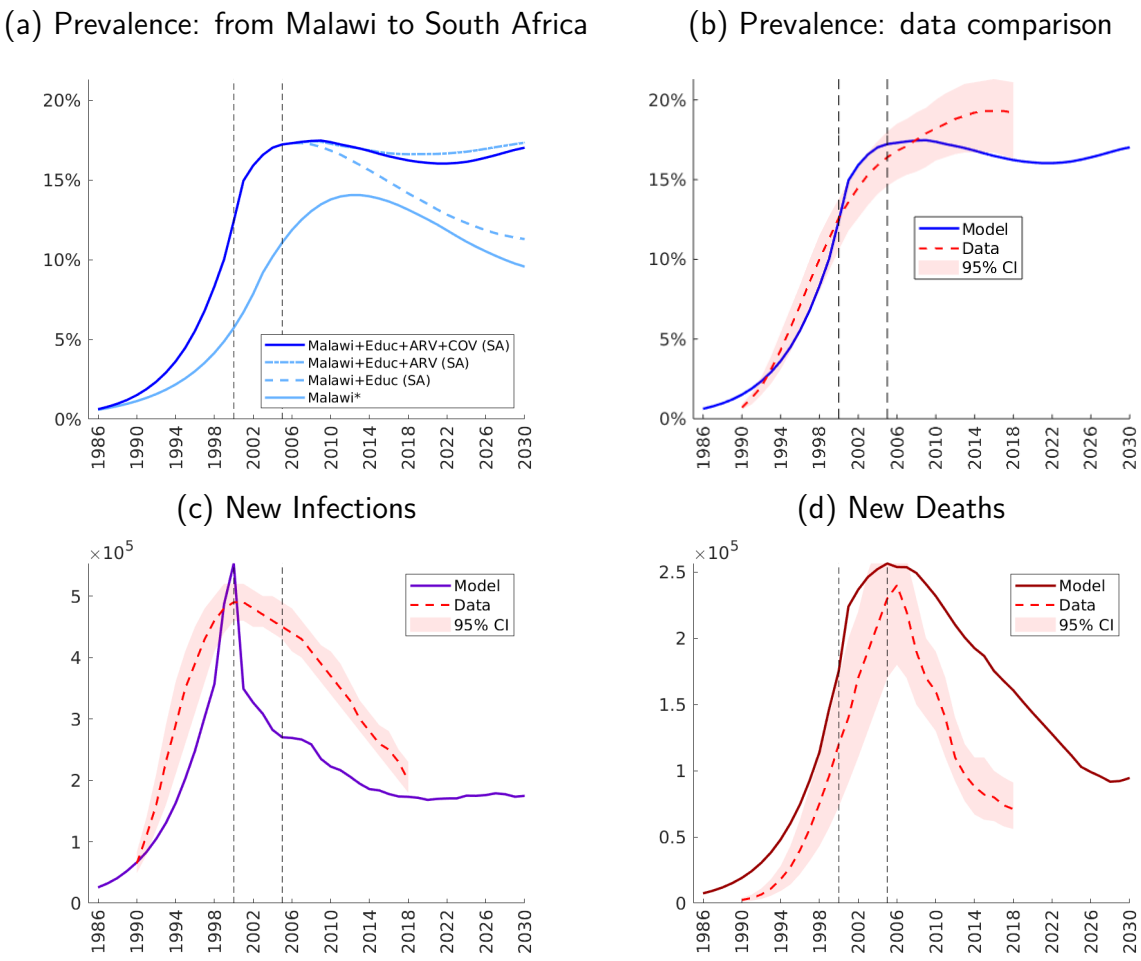



Notes: The figure shows the results of different counterfactual experiments, panels (a) to (c) show the effects of different (earlier) starting dates of the learning stage. Panels (d) to (f) simulate different dates for the introduction of ARVs.

### 2.6.1 Can this theory explain epidemic heterogeneity across countries?

We argue that with the adequate information, the model is certainly capable of characterizing the evolution of the HIV epidemic for individual countries. To illustrate this, we show in Figure 2.17 the model results characterizing the evolution of the HIV epidemic in South Africa (SA). To obtain these results the benchmark parametrization was modified to account for the South African education composition [46], the year of introduction of ARV's (2004) and the respective evolution of ARV coverage. According to AIDSinfo (2020), the ARV coverage in South Africa reached $59\%$ in 2018, this compares to a $70\%$ coverage in Malawi the same year.

Figure 2.17: Characterizing the South African (SA) HIV Epidemic




*The starting year of the epidemic in Malawi was normalized to that of South Africa(SA), that is 1986.

[46] In 1985 only $33\%$ of the South African population had finished primary school, by 2018 it increased to $85\%$ of the population (WorldBank, 2020).

## 2.7 Conclusion

The mixed evidence in the literature investigating the relationship between education and the probability of being HIV-positive in SSA suggests that finding which type of individuals are at greater risk of HIV infection is not an easy task. We proposed a fresh look to this question that consists of explicitly introducing the stages of the HIV epidemic into the analysis. Using nationally representative data from 39 DHS surveys to exploit variation across stages of the HIV epidemic, we showed that the relationship between completed educational attainment and individual HIV status (i.e., the HIV-Education gradient) is dynamic, and significantly evolves with the epidemic. At early stages of the epidemic more-educated individuals are more likely to be infected; however, this relationship strongly decreases as the epidemic evolves, and eventually reaches a stage where education and the probability of being HIV-positive are no longer significantly correlated. Interestingly, in the most advanced stages of the epidemic, the education gradient of HIV returns to being high and positive. We showed theoretically and empirically that the educational disparities in risky sexual behavior (in terms of extramarital partners) closely resemble the U-shaped pattern of the education gradient in HIV. In light of our findings, we proposed a quantitative frameworks of policy evaluation that incorporate the stylized dynamic relationship between education, HIV and risky sexual behavior that we document along the course of the HIV epidemic. We have found that asymmetric learning about the process of infections across education groups goes a long way in explaining the patterns in the data.

# Chapter 3

# A Stage-Based Identification of Policy Effects

*Written jointly with Christopher Busch, Alexander Ludwig Raül Santaeulàlia-Llopis.*

## 3.1 Introduction

Consider the path of an aggregate outcome variable in some region before and after some policy is introduced (or changed). The biggest challenge for the evaluation of the policy effect on the aggregate dynamics is the construction of a credible counterfactual that is informative about the evolution of the outcome variable had it not been for the policy. One possibility is to construct such a counterfactual based on the empirically observed path of the outcome variable in another region not subject to the policy intervention. For this to be reasonable, the regions should display similar, ideally identical, pre-policy dynamics of the outcome variable.

We develop a new empirical method to construct the counterfactual, which builds on the notion that the dynamics of an outcome variable are best tracked over *stages* and not over time: at a given point in time there is potential heterogeneity across regions in terms of how far they moved along the path of the outcome variable. Two reasons lie behind this: regions differ both in the start date of the dynamics and in the speed at which the outcome variable evolves. Furthermore, there is heterogeneity in the overall level or magnitude of the outcome variable. For example, consider the stages of economic development (Lucas, 2004b), stages of the demographic transition (Cervellati and Sunde, 2015; Delventhal et al., 2019; Galor and Weil, 2000b; Greenwood et al., 2005b), and stages of an epidemic (Iorio and Santaeulàlia-Llopis, 2016). For merely expositional reasons we speak of regions as the unit of observation throughout, since our illustrative applications consider region-level outcomes. More generally, our method is further suited for outcomes of any unit of observation with panel data.

Given a set of regions for which the outcome variable is observed, our method can be applied in

three different empirically relevant settings: (i.) policy is introduced in one region only, (ii.) policy is introduced in all regions at different calendar dates (staggered policy implementation), (iii.) policy is introduced in all regions at the same date. For simplicity think of two regions. An ideal scenario is one where both regions are exactly identical before the policy change is implemented in one of them: the observed path in the region not subject to policy provides the counterfactual. The first step of our two-step-method can unveil such an ideal scenario as sketched in each of the three settings above—given that dynamics over stages are relevant, heterogeneity in the stage at a given point in time yields an empirical counterfactual for the post-policy path even when policy is implemented at the same calendar time in all regions, e.g., through a nation-wide policy affecting all (sub-national) regions simultaneously. This counterfactual comes from the region that is more advanced, and thus at a later stage at the policy date: this region went through earlier stages without being subject to the policy (change). The second step of the method then simply uses the counterfactual to identify the policy effect by comparing it to the path in the region which experiences the policy (first).

In a nutshell, our method proceeds as follows. First, we select one region as the benchmark region and normalize stage as time. The goal is to uncover the other region's stage relative to this benchmark region, thus is has no relevance for the results which region is chosen as benchmark. For the other region, we estimate the stage as a parametric function of time (together with a region-specific level shifter that controls for differences in the magnitude of the outcome variable across regions). This estimation is done on pre-policy data only and the objective is to minimize the difference between the regional paths. Second, we apply the estimated stage-time mapping to post-policy data. This delivers paths that are comparable across regions, because they are stated over stages, not over time. The identifying assumption behind using those paths to estimate the policy effect is that the stage-time mapping estimated on pre-policy data continues to be correct and any difference between the paths is therefore due to policy. This is the equivalent within our method to the parallel trends in classical Difference in Differences approaches.

We establish that our method works successfully in different Monte Carlo studies that are designed to resemble empirical applications. First, we consider a public health policy that aims at containing the spread of a virus in the population. We do this in a micro-founded general equilibrium model featuring an epidemic component with viral spread through economic interactions, in which utility maximizing individuals do not internalize the impact of their own interactions on the population infection rate. In multiple experiments, we let two (not connected) regions differ in various aspects and then introduce a policy change. The data generating process of infections is known and thus is the true effect of policy. Our method successfully identifies this true effect in various scenarios. Importantly, the method works well even under regional differences

in the behavioral reaction to the epidemic. Second, we consider the effect of introducing the oral contraception (the pill) on female education. Parents derive utility from children, however, children represent a cost to human capital accumulation, therefore any a technology that reduces fertility can enhance human capital. In plausibly calibrated versions of the model, the method is able to recover true policy effect. Third, we consider two regions, which are each described by a two-sector growth model of structural transformation. The policy experiment removes an institutional barrier to the transformation process. The outcome variable of interest is GDP per capita. Again, our method successfully uncovers the policy effect.

We then illustrate the method in practical applications. First, we show how it can be applied to estimate the effectiveness of a strict stay-at-home lockdown policy that was enacted nationwide in Spain in response to the first wave of the Covid-19 pandemic in the Spring of 2020. This application showcases how our method uses the fact that across Spanish regions the epidemic dynamics were at different stages at the time of policy implementation, and establishes a scenario in which one region (Madrid) is subject to the policy intervention later than the rest of Spain. Thus, the path in Madrid is used to construct a counterfactual for the rest of Spain.

Second, we study the effects of the 1960 nationwide introduction of oral contraceptives for adult women in the United States. We focus on its effects on women's careers choice and fertility. For the latter, the algorithm identifies that 4 states (Washington D.C., Massachusetts, Colorado and Connecticut) lead the rest of the United States, thus we use the path of an artificial region Top 4 to construct the counterfactual for the rest of the United States. The opposite occurs for the case of fertility, thus we use region Bottom 4 (composed of Idaho, West Virginia, Nevada and Arkansas) as counterfactual.

Third, we study the effect of the German reunification in 1990 on GDP per capita in West Germany. We use our method to normalize the path of GDP per capita in Hessen, which turns out to be the regional leader, onto the path in the rest of West Germany. Application of the obtained normalization parameters to the rest of the time series then delivers a scenario in which Hessen is subject to reunification later than the rest of West Germany. The path in Hessen is used to construct a counterfactual for the rest of West Germany.

**Related literature** Our method belongs to the group of empirical control-treatment analyses that are widely used to assess the impact of some event or policy in settings resembling natural experiments, like canonical Difference-in-Differences (DiD) (e.g., Card, 1990; Card and Krueger, 2000), Event Studies or DiD with staggered policy adoption (e.g., Athey and Imbens, 2021; Borusyak et al., 2021), or the Synthetic Control Group approach (SC) (Abadie et al., 2010; Abadie and Gardeazabal, 2003). For a useful common framework for DiD and SC see Doudchenko and

Imbens (2017). Our approach is particularly suited for the analysis of policies implemented in the context of potentially non-linear dynamics of the outcome variable. In the following we briefly emphasize how key aspects of our method relate to benchmark alternatives.

The fact that observed paths are weighted in the process of constructing the counterfactual makes our method a close sibling to SC. However, instead of assuming that treatment and control are on the same stage, we acknowledge that regions differ in their *stage* at the time of policy implementation and explicitly exploit this variation to provide identification.

In the context of a more traditional DiD setting one can partly address the latter point by considering time since start of the dynamics of interest instead of time itself. This requires a choice by the researcher to fix the region-specific start dates. For the analysis of a Covid-19 containment policy this has been suggested in an event study design by Glogowsky et al. (2021). Importantly, counting time since some region-specific start date does not address the problem of regional differences in the speed of the dynamics. In contrast, our method endogenously finds the appropriate time shifter together with a speed adjustment.

The rest of the paper is structured as follows. Section 3.2 outlines the method and discusses how identification is achieved. Section 3.3 illustrates in various Monte Carlo experiments that the method identifies the policy effect correctly. Section 3.4 presents empirical applications. Section 3.5 concludes.

## 3.2 Identification of Policy Effects: A Stage-Based Method

**Policy Effect in an Ideal Scenario** To fix ideas, consider a scenario in which two regions $r \in \{\mathcal{C}, \mathcal{T}\}$ are identical in every aspect relevant for the dynamics of some outcome variable $y_{t,r}$ over time $t$ such that absent any policy intervention $y_{t,\mathcal{C}}$ and $y_{t,\mathcal{T}}$ evolve identically.[1] Reflecting that data would typically be observed at discrete dates, time $t$ evolves discretely. Now assume that region $\mathcal{T}$ implements a policy at some date $t_p$ that affects the path in $\mathcal{T}$ from $t_p + 1$ onwards without altering the path in $\mathcal{C}$. Assume further that region $\mathcal{C}$ introduces the same policy at some later date $t_p + \Delta$.

This scenario is ideal for two reasons. First, absent any policy intervention $y_{t,r}$ evolves identically in the two regions. Second, at any given point in time before policy implementation at $t_p$, the two regions are at the same stage, which is identical to the calendar time itself, i.e., $s_{\mathcal{C}}(t) = s_{\mathcal{T}}(t) = t$. This implies that $y_{t,\mathcal{C}}$ in the interval $[s_{\mathcal{T}}(t_p+1), s_{\mathcal{C}}(t_p+\Delta)] = [t_p+1, t_p+\Delta]$

[1] We use *regions* in the description of the method due to the applications presented below. *Region* can be used interchangeably with *group* or *unit* throughout.

gives a counterfactual for region $\mathcal{T}$.

We capture the difference by a function $\omega(s;\gamma^p)$ that minimizes the distance between the post-policy path in region $\mathcal{T}$ and the corresponding path (still pre-policy) in region $\mathcal{C}$ adjusted by some $\omega(\cdot)$:

$$\min_{\gamma_p} \left\| \ln(y_{s\mathcal{T}}) - \ln(y_{s\mathcal{C}}) - \ln(1+\omega(s;\gamma^p)) \right\|_{s=s_{\mathcal{T}}(t_p+1)}^{s_{\mathcal{C}}(t_p+\Delta)}, \tag{3.1}$$

where $y_{sr}$ is the outcome at stage $s$ in region $r$, and $\|\cdot\|$ is a distance norm. The distance function $\omega(s;\gamma^p)$ satisfies $\omega(s \leq s_{\mathcal{T}}(t_p),\gamma^p)=0$, and $\frac{\partial\omega(s;\gamma^p)}{\partial\gamma^p} > 0$ for $s > s_{\mathcal{T}}(t_p)$ so that when the policy is effective, a larger value of parameter $\gamma^p$ means a stronger positive effect of the policy on $y_{s,\mathcal{T}}$. A negative $\gamma^p$ captures a negative effect of the policy on the outcome variable.

**Normalization of Regional Dynamics** The previous description immediately puts at the center the challenges that are to overcome before being able to identify the policy effect based on the dynamics of $y_{t,r}$ over stages. First, if the regional dynamics in $\mathcal{C}$ and $\mathcal{T}$ start at *different dates*, then the regions are at *different stages* at a given date $t$. Second, $y_{t,r}$ evolves at *different speed* if the regions are not identical in every aspect relevant to the dynamics. Third, and directly related, the dynamics will be of *different magnitude*, i.e., exhibit a different regional level of the flow. The first step of our method is thus to find normalization parameters based on pre-policy data: the goal is to obtain a mapping of the paths in $\mathcal{T}$ and $\mathcal{C}$ that controls for all three pre-policy differences.

In the following discussion we treat region $\mathcal{C}$ as the benchmark region and define stages as

$$s_r(t;\boldsymbol{\psi}) = \begin{cases} s_{\mathcal{T}}(t;\boldsymbol{\psi}) = \sum_{k=1}^{n} \psi_k t^{k-1} & \text{if } r=\mathcal{T} \\ s_{\mathcal{C}} = t & \text{if } r=\mathcal{C} \end{cases} \tag{3.2}$$

where $\boldsymbol{\psi} = \{\psi_1, \psi_2, \dots\}$ is a vector collecting the $n$ polynomial coefficients. There is no "natural choice" for which region is the benchmark, and choosing $\mathcal{T}$ as benchmark region gives identical results for the policy effect. $\psi_1$ moves the whole path forward or backwards in time, adjusting for different start dates, and the higher degree coefficients adjust the speed at which regions move through the path. While in principle the mapping can be of a higher degree, we consider linear or quadratic mapping to be two plausible options. In a linear stage-time mapping, the parameter $\psi_2$ adjusts the speed in a constant way: if $\psi_2 > 1$, the treatmet region is permanently faster than the control region, i.e., in one time-period region $\mathcal{T}$ advances by more than one stage, and vice versa for $\psi_2 < 1$. Allowing for the stage-time mapping to be quadratic captures the notion that the relative speed across the regions can change over time: for example, region $\mathcal{T}$'s dynamics

might initially be slower than region $\mathcal{C}$'s, then catch up, and eventually move faster.

We pin down the stage-time mapping parameters together with a scaling parameter $\psi_0$ such that the log-difference between the path in $\mathcal{C}$ and the normalized path in $\mathcal{T}$ is minimized. The scaling parameter adjusts for differences in magnitude between $y_{t,\mathcal{T}}$ and $y_{t,\mathcal{C}}$, and as such captures region-specific fixed effects that permanently affect the flow of the outcome variable. Importantly, the normalization step only uses the paths up to $s_{\mathcal{T}}(t_p;\boldsymbol{\psi})$, the stage of policy implementation in the treatment region:

$$\min_{\{\psi_0,\boldsymbol{\psi}\}} \left\| \phi(s) \left( \ln(\hat{y}_{t=s,\mathcal{C}}) - \ln(\psi_0 \hat{y}_{t=s_{\mathcal{T}}^{-1}(s;\boldsymbol{\psi}),\mathcal{T}}) \right) \right\|_{s \leq s_{\mathcal{T}}(t_p;\boldsymbol{\psi})}, \tag{3.3}$$

where $\phi(s)$ is a weight function, which is chosen exogenously. The time index for region $\mathcal{T}$ in equation (3.3) is the inverse of the stage-time mapping, $t = s_{\mathcal{T}}^{-1}(s;\boldsymbol{\psi})$, and thus assigns the date $t$ to a stage $s$, given the parameters $\boldsymbol{\psi}$.

We consider two possibilities for the way the outcome variable $y_{t,r}$ is used in the normalization step, denoted here by $\hat{y}_{t,r}$. The first is to work directly with the data. In this case, to obtain $\hat{y}_{t=s_{\mathcal{T}}^{-1}(s;\cdot)),\mathcal{T}}$, we interpolate between $y_{t=fl(s_{\mathcal{T}}^{-1}(s;\cdot)),\mathcal{T}}$ and $y_{t=cl(s_{\mathcal{T}}^{-1}(s;\cdot)),\mathcal{T}}$, where $fl(\cdot)$ and $cl(\cdot)$ denote the integer floor or integer ceiling, respectively. The reason for the interpolation is that $s_{\mathcal{T}}^{-1}(s;\psi_1,\psi_2,\psi_3)$ delivers non-discrete dates, while the outcome variable is only observed on discrete dates.

If classical measurement error is a concern, the second option is to use smoothed data. In this case, denote by $g(t;\boldsymbol{\beta}_r)$ some continuous function with parameters $\boldsymbol{\beta}_r$ fitted to the flow in region $r$, and by $\varepsilon_{t,r}$ a multiplicative error term, giving $y_{t,r} = g(t;\boldsymbol{\beta}_r)\varepsilon_{t,r}$. Importantly, the normalization procedure does not hinge on the exact form of $g(t;\boldsymbol{\beta}_r)$: we simply need *some* continuous function estimated to capture the dynamics. When chosing this option, $\hat{y}_{t,r}$ in equation (3.3) is replaced by $g(t;\hat{\boldsymbol{\beta}}_r)$.

**No Policy in Control Region** In the above description of the normalization, data up to the policy date is used for both regions $\mathcal{T}$ and $\mathcal{C}$ to find the mapping parameters $\{\psi_0,\boldsymbol{\psi}\}$. If there is no policy in region $\mathcal{C}$, we need to determine up to which date the underlying time series should be used in the normalization. While the normalization procedure endogenously determines the stage at policy implementation, this mapping can in principle be sensitive to the length of the time series used for region $\mathcal{C}$. In such cases, we use data up to $t = t_p$ for region $\mathcal{T}$, and use a simple iterative procedure to determine the end of the mapping time series for region $\mathcal{C}$. We start with some $T_{\mathcal{C}} > t_p$, where $T_{\mathcal{C}}$ denotes the last period of the (raw) time series. We then normalize $s_{\mathcal{T}}(t) = t$ and estimate $s_{\mathcal{C}}(t)$, which implies that some period $t_{\mathcal{C}}$ of the region $\mathcal{C}$ time

series will be mapped to period $t_p$ of the region $\mathcal{T}$ time series. If $t_{\mathcal{C}} < T_{\mathcal{C}}$, we let $T_{\mathcal{C}} = T_{\mathcal{C}} - 1$ and repeat the estimation. The procedure is repeated until $t_{\mathcal{C}} \geq T_{\mathcal{C}}$, in which case we set $T_{\mathcal{C}} = T_{\mathcal{C}} + 1$ (the previous iteration) and end the procedure.

**Estimation of Policy Effect** Equipped with the mapping parameters $\{\hat{\psi}_0, \hat{\boldsymbol{\psi}}\}$ we are now in the position to establish the post-policy counterfactual for region $\mathcal{T}$ and identify the policy effect. To this end, we apply the mapping parameters (obtained for pre-policy data) to the rest of the time series, which gives a path of region $\mathcal{T}$ that is adjusted for differences in start date, speed, and magnitude. We ascribe the remaining difference between this normalized post-policy $\mathcal{T}$-path and the path in $\mathcal{C}$ to the policy.

If the regional dynamics in $\mathcal{C}$ lead the dynamics in $\mathcal{T}$, then there is an interval $D$ in terms of stages during which the policy is already implemented in $\mathcal{T}$, but not yet in $\mathcal{C}$, even if the policy is implemented at the same $t$. We refer to $D$ as the overlap interval:

$$D = [s_{\mathcal{T}}(t_p + 1; \hat{\boldsymbol{\psi}}), \ldots, s_{\mathcal{C}}(t_p)].$$

As the data is observed on discrete calendar dates, we need to translate the overlap interval into discrete steps. Thus, consider

$$\begin{aligned} \bar{D} &= \{cl(s_{\mathcal{C}}(\min\{D\})), \ldots, fl(s_{\mathcal{C}}(\max\{D\}))\} \\ &= \{cl(s_{\mathcal{T}}(t_p + 1; \hat{\boldsymbol{\psi}})), \ldots, t_p\} \end{aligned} \tag{3.4}$$

Note that the time $t$ assigned to any stage $s \in \bar{D}$ for region $\mathcal{T}$ according to $t_{\mathcal{T}}(s; \hat{\boldsymbol{\psi}}) = s_{\mathcal{T}}^{-1}(s; \hat{\boldsymbol{\psi}})$ is not discrete. Thus, we interpolate between $\hat{\psi}_0 y_{t=fl(t_{\mathcal{T}}(s;\hat{\boldsymbol{\psi}})),\mathcal{T}}$ and $\hat{\psi}_0 y_{t=cl(t_{\mathcal{T}}(s;\hat{\boldsymbol{\psi}})),\mathcal{T}}$ to obtain $\hat{\psi}_0 \hat{y}_{t=t_{\mathcal{T}}(s)),\mathcal{T}}$.

The distance function (3.1) to be minimized becomes

$$\left\| \ln\left(\hat{\psi}_0 \hat{y}_{t=t_{\mathcal{T}}(s;\hat{\boldsymbol{\psi}})=s_{\mathcal{T}}^{-1}(s;\hat{\boldsymbol{\psi}}),\mathcal{T}}\right) - \ln(y_{t=s,\mathcal{C}}) - \ln\left(1 + \omega(s;\gamma^p)\right) \right\|_{s\in\bar{D}}. \tag{3.5}$$

In order to treat the data observations for both regions symmetrically, we also consider the following alternative representation of the interval $D$, which gives discrete dates for the normalized region $\mathcal{T}$:

$$\begin{aligned} \tilde{D} &= \{cl(s_{\mathcal{T}}^{-1}(\min\{D\}; \hat{\boldsymbol{\psi}})), \ldots, fl(s_{\mathcal{T}}^{-1}(\max\{D\}; \hat{\boldsymbol{\psi}}))\} \\ &= \{t_p + 1, \ldots, fl(s_{\mathcal{T}}^{-1}(t_p; \hat{\boldsymbol{\psi}}))\}. \end{aligned} \tag{3.6}$$

For any $t \in \tilde{D}$, for region $\mathcal{T}$, the raw data scaled to the magnitude of region $\mathcal{C}$ are observed on date $t$ as $\hat{\psi}_0 y_{t,\mathcal{T}}$. In the same fashion as above, we now obtain $s(t) = s_{\mathcal{T}}(t; \hat{\boldsymbol{\psi}})$. We then interpolate between $y_{t=s=fl(s(t)),\mathcal{C}}$ and $y_{t=s=cl(s(t)),\mathcal{C}}$ to obtain $\hat{y}_{t=s=s_{\mathcal{T}}(t;\hat{\boldsymbol{\psi}}),\mathcal{C}}$. The distance function (3.1) to be minimized becomes

$$\left\| \ln(\hat{\psi}_0 y_{t=t,\mathcal{T}}) - \ln(\hat{y}_{t=s=s_{\mathcal{T}}(t;\hat{\boldsymbol{\psi}}),\mathcal{C}}) - \ln(1 + \omega(s = s_{\mathcal{T}}(t; \hat{\boldsymbol{\psi}}); \gamma^p)) \right\|_{t \in \tilde{D}} \tag{3.7}$$

We then stack (3.5) and (3.7) and minimize over $\gamma_p$, which gives our estimate of the policy effect in region $\mathcal{T}$.

**Restrictions** Stated as above, the normalization procedure delivers mapping parameters $\boldsymbol{\psi}$ even if the two regions are arguably too different to render the resulting counterfactual dynamics reasonable. Whether this is a potential concern or not is application-specific, as is the judgement about what is *too different*. In the application to the reunification effect on West German GDP per capita we truncate the time series of West Germany (the treatment region) on the left before applying the normalization procedure. The motivation behind is that the West German time series should reach the minimally observed GDP per capita of the rest of the OECD (the control region). More generally, the regional time series should cover the same domain before we apply the normalization procedure. In the application to the policy effect of the Covid-19 pandemic in Spain this is not a concern, as all regions are observed from the onset of the regional pandemics.

## 3.3 Monte Carlo Analyses

Here, we implement our newly proposed SBI strategy on model-generated policy effects in order to assess whether the SBI correctly recovers the true policy effects generated by a simulated model. In particular, we are interested in testing our methodology in instances where regional variation is affected by unobserved heterogeneity (or traits) that gives rise to differences in endogenous behavior across regions. We use three policy contexts that include: (1) public health policy against a pandemic using an econ-epi model where economic activity shapes and is shaped by the pandemic; (2) the effects of the pill in a model of women career and fertility choices. and (3) economic growth policy using a model of structural transformation.

### 3.3.1 Public Health Policy Against a Pandemic

**A theoretical framework** Consider an economy with many individuals that is unexpectedly hit by an epidemic at time $t = 1$ with an initial number of infections $I_1 > 0$. We normalize the pre-pandemic population, $N_0$, to one. Given a set of beliefs on how economic activity affects the

probability of infection in a manner that we define below, a planner solves:

$$\max_{\{c_t\geq 0, h_t\in[0,1]\}_{t=0}^{\infty}} \sum_{t=0}^{\infty}\delta^t \Pi_{\tau=0}^{t}\phi_{\mathcal{P}}(h_{\tau-1})u(c_t,h_t;\omega) \tag{3.8}$$

where the felicity function is strictly concave in both arguments consumption $c_t$ and labor $l_t \in [0,1]$. The parameter $\omega$ measures the individual value of life.[2] Our planner is subject to an aggregate resource constraint $N_t c_t = w_t h_t N_t$ where $w_t$ is the implicit price (marginal product) of labor using technology $Y_t = z h_t N_t$, i.e. $w_t = z$. The timing of the model is such that individuals work and consume and, when working, there is infection risk. After working and consuming, the infected face death or recovery. In this manner, individuals are either susceptible $S_t$, infected $I_t$, recovered $R_t$ or dead $D_t$. The total population alive is $N_t = S_t + I_t + R_t$. Using $X_{G,t} = G_{t+1} - G_t$ for $G = \{S, I, R, D\}$, the planner's belief is that law of motion of the population structure is:

$$X_{S,t} = -\lambda_{\mathcal{P}}(h_t)\beta\frac{I_t}{N_t}S_t \tag{3.9}$$

$$X_{I,t} = (1-\gamma)\lambda_{\mathcal{P}}(h_t)\beta\frac{I_t}{N_t}S_t - \gamma I_t \tag{3.10}$$

$$X_{R,t} = (1-\zeta)\gamma\widetilde{I}_t \tag{3.11}$$

$$X_{D,t} = \zeta\gamma\widetilde{I}_t \tag{3.12}$$

where $\widetilde{I}_t = \lambda_{\mathcal{P}}(h_t)\beta\frac{I_t}{N_t}S_t + I_t$.[3]. The parameter $\beta$ captures features like density, occupation-industry composition, age-health structure of the population or pollution (among others) which can differ across locations. Then, conditional on randomly meeting an infected individual at rate $\frac{I_t}{N_t}$, the planner's belief is that individuals get infected with probability $\lambda_{\mathcal{P}}(h_t) = \xi_{\mathcal{P}} h_t^{\alpha}$ which depends on the choice of $h_t$. Hence, the parameter $\xi_{\mathcal{P}}$ captures the planner's beliefs on how much economic activity affects the probability of infection. However, the actual infections arise from the true probability function $\lambda(h_t) = \xi h_t^{\alpha}$ where the true effects $\xi$ can differ from the planner's beliefs. If $\xi_{\mathcal{P}} < \xi$, then the planner underestimates the actual effects of $h_t$ on infections and deaths. In contrast, if $\xi_{\mathcal{P}} > \xi$ the planner overestimates these effects.

Then, given $N_t$, $S_t$ and $I_t$ and the population law of motion (3.9)-(3.12), the planner's belief is that the survival rate between $t$ and $t+1$ is $\phi_{\mathcal{P}}(h_t) = 1 - \frac{X_{D,t}}{N_t}$, which can differ from the actual survival rate, $\phi(h_t)$. Note that since the planner chooses $h_t$ based on her beliefs on the

[2]We assume the period utility takes the form $u(c,h;\omega) = log(c) - \kappa\frac{h^{1+\frac{1}{\nu}}}{1+\frac{1}{\nu}} + \omega$

[3]Note that we allow for new infections to transit to death in the same period $t$—in this manner, $h_t$ has an immediate effect on the survival rate between $t$ and $t+1$. This assumption is innocuous and we use it to ease the exposition of the trade-off between economic activity and public health in the model. We can easily accommodate a lagged effect of $h_t$ on survival rates.

infection process, she is not able to forecast the true population dynamics.[4] In this context, we introduce an unexpected population shock that fully corrects for the forecast error *ex-post*. Precisely, whereas at period $t$ the planner's belief is that population between $t$ and $t+1$ changes following the survival probability function $\phi_{\mathcal{P}}(h_t)$—and chooses $h_t$ accordingly, at period $t+1$ we allow the planner to learn the actual survival rate without being aware that $\phi(h_t)$ determines it. That is, we define the unexpected shock at $t+1$—or equivalently the one-period ahead forecast error at $t$—as the difference $\phi(h_t) - \phi_{\mathcal{P}}(h_t)$, which occurs at every period $t$. Note that this setting implies that looking backwards the planner observes the actual evolution of the population $N_t = \Pi_{\tau=0}^{t}\phi(h_{\tau-1})$,[5] whereas looking forward the planner always commits a forecast error. Analogously to $N_t$, $I_t$ and $S_t$ are also updated by the actual infections with an unexpected shock equal to $\lambda(h_t) - \lambda_{\mathcal{P}}(h_t)$.

The amount of economic activity $h_t$ is determined by the following Euler condition

$$\underbrace{\frac{\partial u(c_t, h_t; \omega)}{\partial c_t} w}_{\substack{\text{Marginal Benefit of Working:} \\ \text{Consumption Gain}}} - \underbrace{\frac{\partial u(c_t, h_t; \omega)}{\partial h_t}}_{\substack{\text{Marginal Cost of Working:} \\ \text{Loss of Leisure}}} = \underbrace{\delta \frac{\partial \phi_{\mathcal{P}}(h_t)}{\partial h_t} u(c_{t+1}, h_{t+1}; \omega)}_{\substack{\text{Marginal Cost of Working:} \\ \text{Loss of Lives}}} \qquad \forall t, \tag{3.13}$$

which states that the marginal benefit of working (more consumption) needs to equate its marginal costs consisting of an intratemporal component (disutility from working) and an intertemporal component (loss of lives). Note the Euler equation is a first order difference equation in $h_t$. We need a terminal condition to solve for the path of $h_t$. Note that we can separately solve for the pre-pandemic $t = 0$ equilibrium before the unexpected arrival of the pandemic at $t = 1$. In this pre-pandemic era there are no infections and, hence, $\phi(h_0) = 1$. That is, the equilibrium $h_0$ sets the right hand side of the Euler equation (3.13) to zero in which case $h_0$ simply solves an intratemporal trade-off. The same equilibrium emerges after the pandemic at some large $t = T$ which delivers a terminal condition $h_T = h_0$ that we use to solve for $\{h_t\}$. Given $h_T$ (or $h_0$), we can easily solve for the optimal labor path $\{h_t\}_{t=1}^{T-1}$ during the epidemic using standard techniques. Last, since there is an unexpected population shock every period $t$, the planner reoptimizes the entire sequence $\{h_t\}_{t=1}^{T-1}$ every period after the realization of the shock; see our Appendix for our complete solution algorithm.

In what follows, we focus on a Montecarlo analysis with two modeled regions that follow different epidemic paths but are subject to the same nationwide policy against a pandemic. In particular, we create two regional epidemic paths using our econ-epi the model. The epidemic

[4] Precisely, given $N_t$, $S_t$ and $I_t$, the actual survival rate between $t$ and $t+1$ is the result of plugging the $h_t$ that the planner's chooses—under her beliefs on the infection process $\lambda_{\mathcal{P}}(h_t)$—into the population law of motion (3.9)-(3.12) after replacing the planner's belief by the true probability of infection $\lambda(h_t)$.

[5] There is no fertility in this economy. That is, the evolution of the population is solely determined by survival.

paths across these two regions are heterogeneous in that we assume that a region ($\mathcal{C}$) is characterized by a different $\beta, \gamma,$; see panel (a1) of Figure 3.1.

**True model-generated policy effects** There are different public health policies that can be used against a pandemic. We focus on the stay-home policies widely implemented across the globe against the Covid-19 pandemic. In the context of our model, this implies imposing a constraint $h < \overline{h}$ for a given interval of time in which the policy is in place from period (day) $t_p$ to $t_f$.

We now discuss the results of imposing a constraint of $\overline{h} = 0.4$ for a (long) interval of time from period $t_p = 36$ to $t_f = 250$ (dashed line). There are effects of such policy on hours because the policy is binding[6] in the sense that without the stay-home policy the equilibrium path of hours (solid line) shows higher economic activity than what the policy dictates for the entire interval of time in which the policy is in place; see panel (a) of Figure 3.1. The lower economic activity has consequences for the flow of deaths that now peaks by a lower magnitude (and earlier) relative to a model economy without policy; see panel (b) of Figure 3.1. The difference between the flow of deaths without policy (solid line) and the flow of deaths with policy (dashed line) are the true policy effects, as generated from the model. [7]

**Stage-based identification of policy effects** Panel (c) and (d) of Figure 3.1 shows the results from using the stage-based identification taking the model simulated data with policy as the only observable data to the policy evaluator interested in assessing the effects of policy. A policy evaluator that faces a scenario in which she/he has information on a outcome (statistic) of interest, e.g., $X_D(t)$. Say this outcome is available for the two regions $\mathcal{C}$ and $\mathcal{T}$. The evaluator also knows the dates of policy implementation in each region. The policy evaluator knows nothing else. In particular, she/he does not know the model that generates the data and, hence, cannot use the model to identify the true policy effects following the steps that we described above. This implies that the policy evaluator has the following information: Available time-series data on a statistic of interest $X_D^{\mathcal{C}} = \mathcal{C}_p$ (dashed blue line) and $X_D^{\mathcal{T}} = \mathcal{C}_p$ dashed red line). In both cases the policy is implemented at $t_p = 38$ (until $t_p = 250$) depicted as the vertical (dashed gray) lineThe evaluator is not provided with the true model that generates the data.

The mapping from $\mathcal{C}$ to $\mathcal{T}$ normalizes the pre-policy epidemic path of the control region to that of the treatment region (dashed blue with crosses).[8] The normalization implies an overlap interval

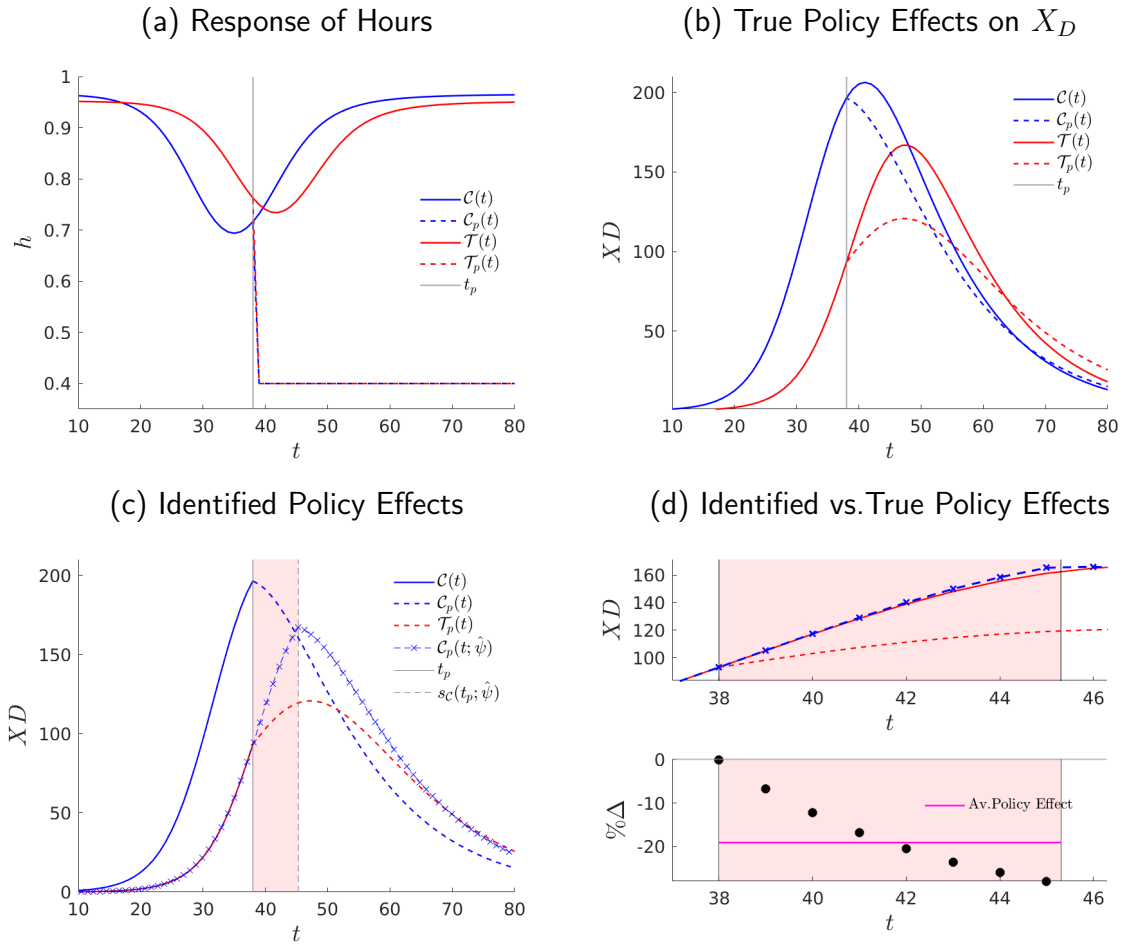
[6] At the period the policy is implemented the equilibrium $h_{t_p}$ is 0.8, whereas the policy sets $h$ to a lower value of 0.5.

[7] In Appendix C.3 we explicitly discuss the case of latent time varying unobserved heterogeneity.

[8] The mapping $\mathcal{C}$ to $\mathcal{T}$ delivers identical results to mapping $\mathcal{T}$ to $\mathcal{C}$, see reduced for simulation in Appendix C.

of one week between $t_p = 38$ and $t = 44$ in which the treatment region is subject to the policy whereas the normalized control regions is not. Comparing the actual model-based percentage of lives saved in that overlap interval and the one estimated from the stage-based empirical identification we find, respectively, 9.53% and 10.33%. That is, the stage-based identification provides a good estimate of the actual effects of policy.

Figure 3.1: Stage-Based Identification of Policy Effects: Nationwide Public Health Policy




*Notes:* Where $\bar{h} = 0.4, t_p = 38, t_f = 250$ $\Theta_{\mathcal{C}} = \{\delta = 0.95, \omega = 560400, z = 64, \beta = 0.509, \zeta = 0.001, \kappa = 1.05, \xi = 0.2, \alpha = 0.65\}$ and $\Theta_{\mathcal{T}} = \{\delta = 0.95, \omega = 560400z = 64, \beta = 0.501, \zeta = 0.0008, \kappa = 1.07, \xi = 0.19, \alpha = 0.65\}$. The exogenously chosen policy parameters are $\bar{h} = 0.4, t_p = 38, t_f = 250$.

### 3.3.2 The Pill and Women's Choice

**A theoretical framework** Motivated by the important work in Goldin and Katz (2002), we now turn to assess the effects of the pill—oral contraceptives—on women's lifetime choices. We pose a model that captures—in a purposefully oversimplified manner—how the pill potentially affects women's trade-off between career choice and fertility. Our goal is to provide a framework in which access to the pill can impact career choice, which can occur using the notion that the pill reduces unwanted pregnancies at the time (ages) where education decisions are taken.[9] In particular, we assume that each cohort-$t$ of women maximizes utility derived from consumption and children by choosing the amount of human capital, $h$, and pill use, $o$, that solves:

$$\max_{\{h,o\}} c + \kappa n \tag{3.14}$$

where $c$ is consumption and children $n \in [0,1]$ generate joy by a relative factor $\kappa$. Women are subject to a resource constraint,

$$c + q(1+\tau(n))h = w(1+z_t e(h)) \tag{3.15}$$

where $q$ is the relative price of human capital (e.g. tuition fees or job training), $\tau(n)$ captures an additional cost of accumulating human capital associated with the presence of children with $\tau_n(n) > 0$ and $\tau_{nn}(n) < 0$. In terms of earnings, $w$ is a constant base wage and $z_t e(h)$ is an endogenous human capital wage premium with two components. First, there is skill-biased technical change (SBTC), $z_t = z_0 \Pi_{\tau=1}^{\tau=t}(1+\gamma_\tau)$, where $z_0 > 0$ and $\gamma_t > 0$ captures cohort-$t$ growth in SBTC. Second, there is a mapping from human capital $h$ to a rate $e(h) \in [0,1]$ with $e_h(h) > 0$ and $e_{hh}(h) < 0$. We interpret $e(h)$ as the fraction of educated (e.g. college completed) women in the economy that benefit from the SBTC.[10] Finally, children production is given by,

$$n = \phi[1 - \mathbf{1}_{t_p} g(o)], \tag{3.16}$$

where access to the pill is captured by the policy dummy $\mathbf{1}_{t_p}$ that is equal to zero if a cohort $t$ does not have access to the pill, and equal to one otherwise. Hence, if women do not have access to the pill, then the amount of children is solely determined by the probability of pregnancy $\phi \in [0,1]$ where we assume that all pregnancies end in a child, that is $n = \phi$. If women have access to the

[9] The analysis in Goldin and Katz (2002) encompasses career choice and marriage delays. In their case, they model the increase in a counterpart of our $h$ as the utility lost from abstinence and/or forgone home production.

[10] The mapping of the outcome variable from $h$ to $e(h)$ is innocuous for our analysis and we use it merely for exposition convenience. In particular, if the outcome $e(h)$ is a rate we can interpret it as the fraction of educated women (e.g. college degree completion) in the population.

pill, then the probability of pregnancy is adjusted downward by the pill effectiveness in preventing pregnancy, $g(o) \in [0,1]$. We assume that larger use of the pill—e.g. higher adherence to follow protocol, increases the effectiveness of the pill. That is, $g_o(o) < 0$ with $g_{oo}(o) > 0$ and, hence, $n_o(o) < 0$ and $n_{oo}(o) > 0$.[11]

Note that we can plug the resource constraint (3.15) and child production (3.16) into women's objective function (3.14). The first order condition of $h$ implies,

$$FOC(h): \quad \underbrace{q(1+\tau(n))}_{\text{Marginal Cost of Human Capital}} = \underbrace{wz_t e_h(h)}_{\text{Marginal Benefit of Human Capital}}, \tag{3.17}$$

where the marginal cost of human capital is the price of education qualified by the cost of children, and the marginal benefit of human capital are the premium returns from increases in human capital. Note that the marginal benefit of human capital is convex in $h$ whereas the marginal cost of human capital is concave in $n$, hence, an increase in the number of children results in a decrease in human capital. Therefore, a technology that reduces $n$ can enhance human capital. The first order condition for pill use is:

$$FOC(o): \quad \underbrace{q\tau_n(n)h}_{\text{Marginal Benefit of Pill}} = \underbrace{\kappa}_{\text{Marginal Cost of Pill}} \tag{3.18}$$

where the marginal cost of the pill is a reduction utility derived from children and the marginal benefit of the pill is a reduction in the price of human capital.

Clearly, without access to the pill equation (3.18) is not present and the marginal cost of human capital (left-hand-side of (3.17)) is constant. In panel (b) of Figure 3.2 we show the associated fraction of educated women within each region where we allow for $q$, $w$ and the path for $z_t$ to be different across these regions. [12]

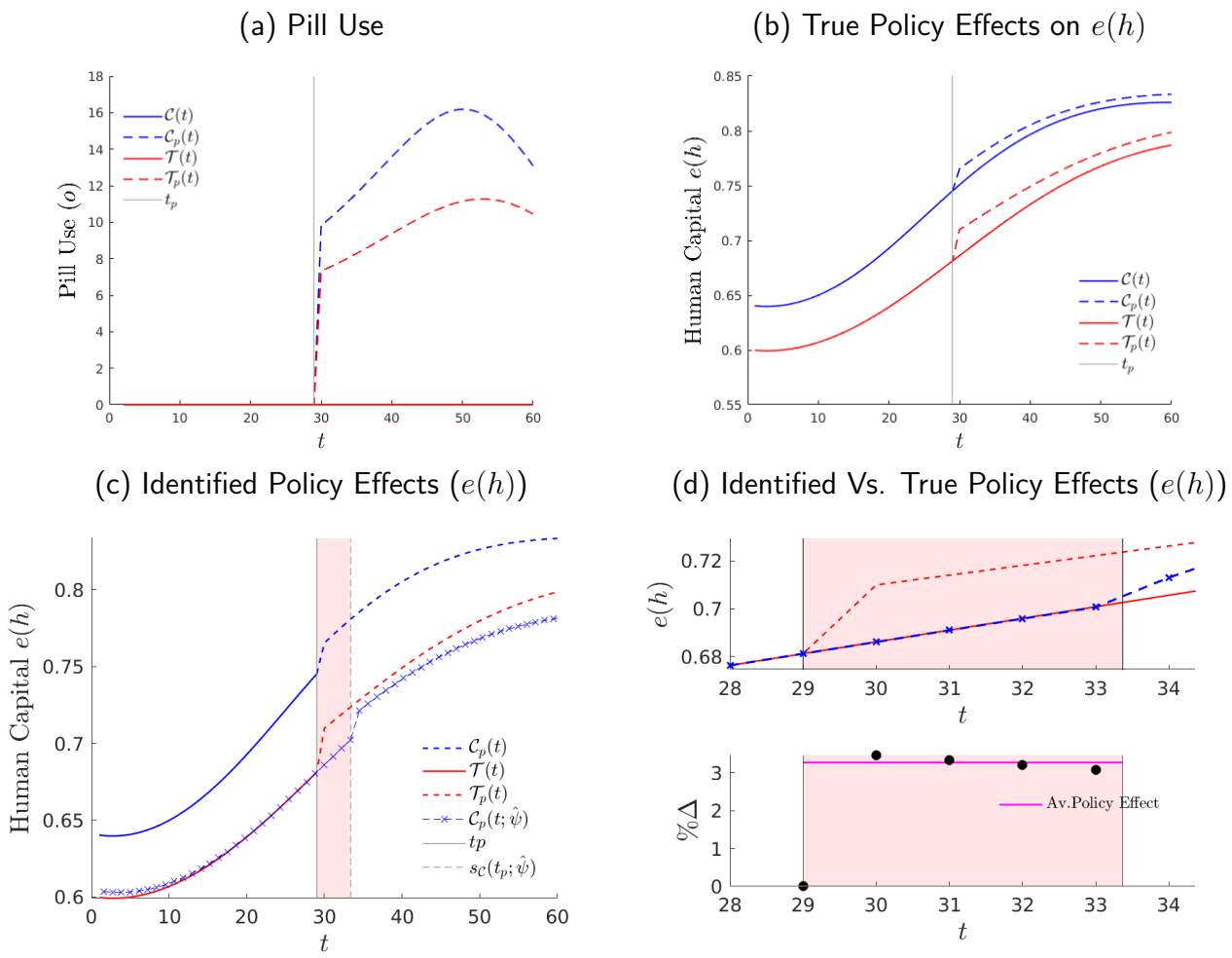
**Model-generated policy effects** Introducing access to the pill for these same two regions allows the pill trade-off in (3.18) to work affecting $h$ and therefore $e(h)$ as we show in panel (b) of Figure 3.2.

**Stage-based identification of policy effects** Panels (c) and (d) of Figure 3.2 show the results from applying the stage-based identification. The normalization implies an overlap interval of 5.3 years . In that overlap interval, the policy generates an average 3.25% increase in the share of

[11] We note that lawful access to the pill does not necessarily fully determine use which is also likely to be affected by social norms—i.e. the shame before the game. Interestingly, access to the pill can—at the same time—shape social norms (Fernández-Villaverde et al., 2014).

[12] We assume a path that shares the same qualitative behavior of the (college) wage premium in the data.

Figure 3.2: Model-Generated Policy Effects: Introduction of the Pill




*Notes:* $\Theta_{\mathcal{C}} = \{w = 1, q = 0.035, \kappa = 0.07, \alpha = 0.1, , \theta_h = 0.43, \theta_o = -0.07\}$ and $\Theta_{\mathcal{T}} = \{w = 0.98, q = 0.045, \kappa = 0.09, \alpha = 0.1, , \theta_h = 0.5, \theta_o = -0.07\}$. Policy Parameters are $t_p = 30$

college women whereas the stage-based identification measure a policy effect of 3.27%.

### 3.3.3 Growth policy and structural transformation

**A theoretical framework** We consider an economy with structural transformation from an agricultural to a manufacturing sector, respectively, $i = \{a, m\}$. An infinitely-lived representative agent chooses sectoral allocations of consumption $\{c_{at}, c_{mt}\}_{t=0}^{\infty}$, labor $\{n_{at}, n_{mt}\}_{t=0}^{\infty}$ and next period capital $\{k_{t+1}\}_{t=0}^{\infty}$:

$$\max_{\{c_{at}, c_{mt}, n_{at}, n_{mt}, k_{t+1}\}_{t=0}^{\infty}} \sum_{t=0}^{\infty} \beta^t \left(\ln(c_{at} - \bar{c}_a) + \kappa \ln c_{mt}\right)$$

subject to a budget constraint,

$$p_a c_{at} + c_{mt} + k_{t+1} = \sum_{i \in \{a,m\}} w_{it} n_{it} + r_t k_t + (1-\delta) k_t + \pi_t(\ell)$$

where $\beta \in (0,1)$ is the discount factor, $\kappa$ is the utility weight of manufacturing consumption relative to agriculture and $\bar{c}_a$ a subsistence level. The non-homothetic preferences are a force behind the structural transformation of the economy. The hosuehold is endowed with one unit of time, i.e. $n_{at} + n_{mt} = 1 \ \forall t$ that is allocated to either agriculture or manufacturing and receive, respectively, wage rates $\{w_{at}, w_{mt}\}$. The capital's return is $r_t$ and capital depreciates at rate $\delta$. The manufacturing good is the numeraire and $p_a$ is the price of agriculture relative to manufacturing, exogenously given. Land $\ell$ is fixed and inelastically supplied by the household that receives pure rents from renting it to agricultural firms.

There is one representative firm per sector and we assume competitive markets. The agricultural firm produces output $y_a$ with labor $n_a$ and land. Agricultural firms solve:

$$\max_{n_{at}} \pi_t(l) = p_a y_{at} - w_{at} n_{at} \quad \text{s.t} \quad y_{at} = z_{at} n_{at}^{\phi} \ell^{1-\phi},$$

where $\phi$ is the labor share in agriculture. Since land is fixed, the agricultural technology exhibits decreasing returns to scale which is an additional force shifting resources out of agriculture as the economy grows.[13] Manufacturing firms produce output $y_{mt}$ with labor $n_{mt}$ and capital $k_t$ solving:

$$\max_{n_{mt}, k_{t+1}} \quad y_{mt} - w_{mt} n_{mt} - r_t k_t \quad \text{s.t} \quad y_{mt} = z_{mt} n_{mt}^{\alpha} k_t^{1-\alpha},$$

where $\alpha$ is the labor share in manufacturing. We assume that total factor productivity (TFP) differs by sector following $z_{it} = z_{i,0}(1+\gamma_i)^t$ for $i = \{a, m\}$ with $\gamma_a < \gamma_m$. That is, productivity in the manufacturing sector grows at a faster rate than that of the agricultural sector.

We solve the economy by guessing the sequences of factor prices $\{w_a, w_m, r\}_{t=0}^{\infty}$. Given these prices, we find the allocations $c_{at}, k_{t+1}$ and $n_{at}$ that solve the following set of first order conditions.

[13] The ability of non-homothetic preferences to generate structural change is studied in, for example, Gollin et al. (2002a). The role of technological choice in generating structural change—from a decreasing returns to scale technology to a constant returns to scale, is studied in Hansen and Prescott (2002); in the context of a one-good economy. Our identification of policy effects is innocuous as to which force drives structural change.

First, an intratemporal condition governing the substitution across consumption goods:

$$FOC(c_{at}): \qquad u_{c_{at}}(c_{at}) \underbrace{\frac{1}{p_a}(-1)}_{\frac{\partial c_{at}}{\partial c_{mt}}} + \kappa u_{c_{mt}}(c_{mt}) = 0 \tag{3.19}$$

Second, an intertemporal Euler condition for $k_{t+1}$ governing the trade off between one additional unit of consumption today versus tomorrow's consumption,

$$FOC(k'): \qquad u_{c_a}(c_{at}) \underbrace{\frac{1}{p_a}(-1)}_{\frac{\partial c_{at}}{\partial k_{t+1}}} + \beta u_{c_a}(c_{at+1}) \underbrace{\frac{1}{p_a}(1 + r_{t+1} - \delta)}_{\frac{\partial c_{at+1}}{\partial k_{t+1}}} = 0 \tag{3.20}$$

Third, an intratemporal condition for $n_a$ equating wages across sectors,

$$FOC(n_{at}): \qquad u_{c_a}(c_{at})(\underbrace{w_{at} - w_{mt}}_{\frac{\partial c_{at}}{\partial n_{at}}}) = 0 \tag{3.21}$$

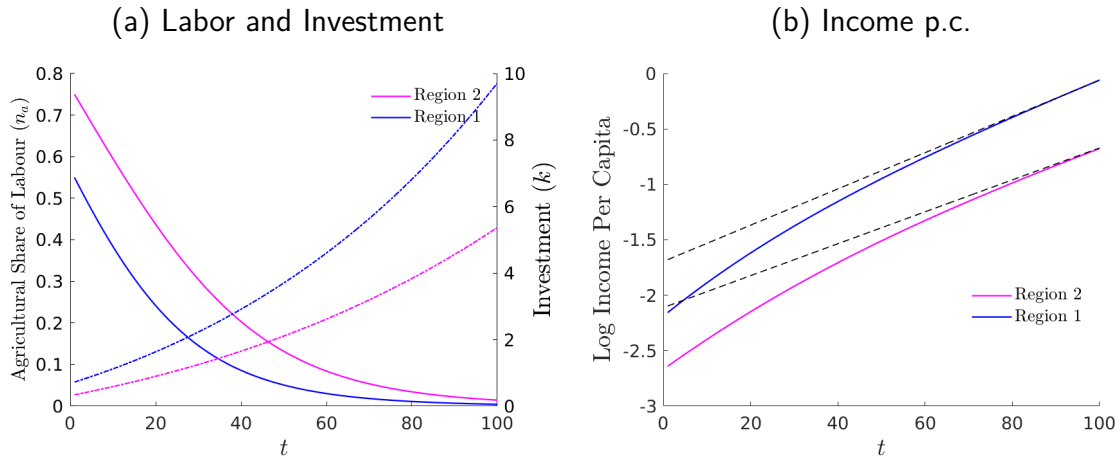
These allocations need to satisfy the marginal product conditions arising from the firms' problems in competitive markets, that is, $w_{at} = \phi \frac{y_{at}}{n_{at}}$, $w_{mt} = \alpha \frac{y_{mt}}{n_{mt}}$ and $r_t = (1-\alpha)\frac{y_{mt}}{k_t}$. There is market clearing in labor and capital, and aggregate consistency. Note that the interemporal Euler condition (3.20) is a second order different equation in $\{k_t, k_{t+1}, k_{t+2}\}$ at every period $t$. We use as initial and terminal conditions the corresponding steady states of the economy at $t = 0$—without TFP growth—and at a large $T$ with negligible agricultural share of labor. We consider the case of two regions which are at different development paths. In Figure 3.3 panel (a) and (b) and we show the equilibrium path for their agricultural share and income per capita respectively. The model is able to generate an agricultural share that declines over time whereas, at the same time, income per capita increases asymptotically reaching a balanced growth path with a trifling agricultural share; a phenomenon well documented by the macro-development literature.[14]

**Model-generated policy effects** Consider an exogenous positive TFP shock to the manufacturing sector of magnitude $\tau > 1 + \gamma_m$, at period $t = 39$ this could be interpreted as removing an institutional constraint which in turn accelerates the structural transformation of the economy.The effects of the policy can be seen in Figure 3.4 panels (a) and (b).

**Stage-based identification of policy effects** Panels (c) and (d) of Figure 3.4 show the results from applying the stage-based identification. Again the evaluator is not provided with the

[14] See a comprehensive description in Herrendorf et al. (2014a).

Figure 3.3: Model-Generated Structural Transformation

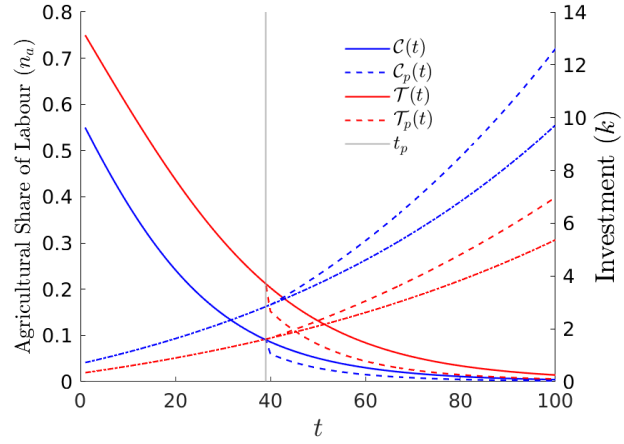



Notes: The decreasing solid lines dotted dashed lines (left axis) represent the agricultural labor share of output. The increasing dash-doted lines (right axis) are capital investment. For region $\mathcal{T}$, we choose, $n_{a,0} = 0.45, z_{a,0} = 0.15, z_{m,0} = 0.17, \gamma_a = 0.007, \gamma_m = 0.0073$. For region $\mathcal{C}$, we choose, $n_{a,0} = 0.65, z_{a,0} = 0.145, z_{m,0} = 0.145, \gamma_a = 0.007, \gamma_m = 0.0072$. Common parameters between both regions are $\beta = 0.98, \alpha = 0.6, \phi = 0.8, \kappa = 2, \delta = 0.02$.

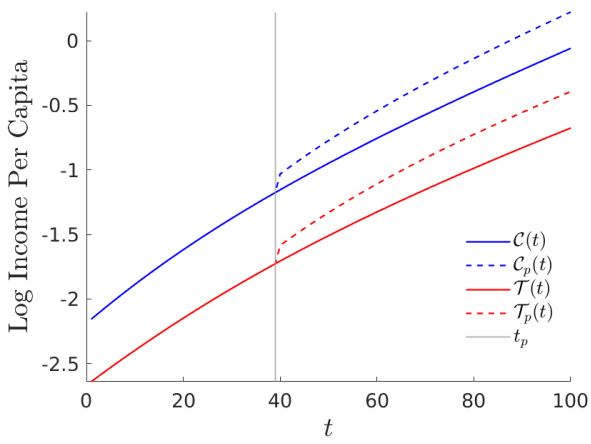
true model generating the data. The normalization implies an overlap interval of 7 years . In that overlap interval, the policy generates an average 8.6% increase in log income percapita whereas the stage-based identification measure a policy effect of 8.5%.

Figure 3.4: A Stage-Based Identification of Model-Generated Policy Effects: Structural Transformation

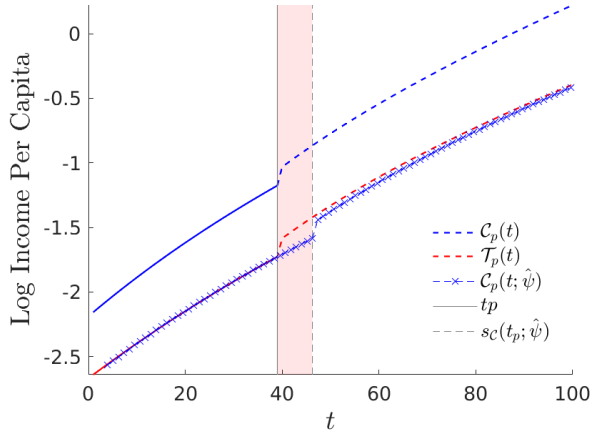
(a) Response of Labor and Investment



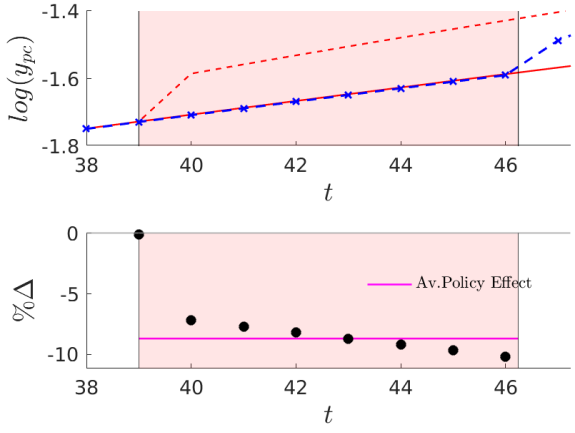

(b) True Policy Effects on Income p.c.




(c) Identified Policy Effects




(d) Identified Vs. True Policy Effects



## 3.4 Applications

### 3.4.1 Spanish *Confinamiento* Against Covid-19

Like many countries, Spain pursued non-pharmaceutical public health policies in response to the Covid-19 pandemic. On March 14, 2020, the Spanish government announced a nationwide stay-at-home policy—enacted the following day—which locked down all non-essential workers in all regions of Spain. Indicative of its strictness, the public debate referred to the policy as confinement. The strictest measures were lifted on May 2 when the first wave of the epidemic flattened out. We apply our stage-based method to identify the effect of this policy intervention on the course of the pandemic. The outcome variable of interest is the daily flow of deaths attributed to Covid-19 (reflecting that daily case data is not a good measure of infections, especially during the onset of the pandemic, when testing was only gradually introduced). The epidemic in Madrid leads the other Spanish regions and thus provides the basis for the counterfactual dynamics in an artificial region Rest of Spain, which is comprised of all Spanish regions without Madrid. In the next paragraphs we illustrate how to construct the counterfactual and document the average daily policy effect during the first week (the length of the overlap interval) of the *confinamiento*.

**Step 1: Normalization to Stage-Dynamics** Given notorious measurement error in the daily number of deaths, we smooth data and represent the daily stock of deaths as a generalized logistic function $G(t;\boldsymbol{\beta}) = \beta_0 \left(1 + \beta_3 e^{(-\beta_1(t-\beta_2))}\right)^{-\frac{1}{\beta_3}}$, with $\boldsymbol{\beta} = \{\beta_0, \beta_1, \beta_2, \beta_3\}$. The flow of deaths $g(\cdot)$ follows as the first analytical derivative of $G(t;\boldsymbol{\beta})$ with respect to time. We fit $g(\cdot)$ to data starting on February 20, 2020, which in our notation is equal to $t = 1$. Period $t_0(r)$ is the region-specific start period in the estimation, which we determine as the day on which we observe the first death in the respective region. We add a lag parameter $\tau$ to the policy date, reflecting that a policy that aims at reduction of infections will show an effect on the flow of deaths with a delay. Here, we document results for a benchmark choice of $\tau = 12$.[15] Similarly, to determine the end date of the sample, we take the date of the end of the policy intervention (May 2, 2020), period $t_e$, and add the policy lag parameter, thus $T(\tau) = t_e + \tau$. It is crucial for our normalization that $g(\cdot)$ matches well the flow of death time series before date $t_p + \tau$. Panel (a) of Figure 3.5 shows that this is the case. We then use a linear stage-time mapping, thus $\boldsymbol{\psi}$ in Equation (3.2) is a $(2 \times 1)$ -vector. We specify the weight function $\phi(s)$ as a quadratic in $s$, such that increasingly more weight is assigned to the observations closer to the policy date.

[15] In Aleman et al. (2021) we extend the stay-home application of this paper and conduct robustness with respect to the $\tau$. There, we also pursue a region-specific application of our method. We document heterogeneous policy effectiveness across Spanish regions, which systematically varies with the stage of a region at policy implementation.

Figure 3.5(b) shows the epidemic paths after the normalization. The normalization unveils an overlap interval in terms of *stages*, in which the region of Madrid is not yet under the effect of policy whereas the rest of Spain is.

The parameter estimates for the relative stage-time mapping are $\hat{\boldsymbol{\psi}} = \{-1.15, 0.85\}$. These estimates imply that the artificial region Rest-of-Spain goes through the epidemic stages somewhat slower than Madrid: it takes Rest-of-Spain $35.3$ $(= 30/0.85)$ days to go through Madrid's epidemic path of one month. Thus, only adjusting for different start dates (as in Glogowsky et al., 2021) would imply wrong estimates. Even stronger differences in the epidemic speed could imply that regional epidemics peaked at different times since first case, and therefore, one can easily end up in a situation where one region is already in the decreasing part of the daily flows, while another is still in the increasing part—creating an even stronger bias in the estimates.

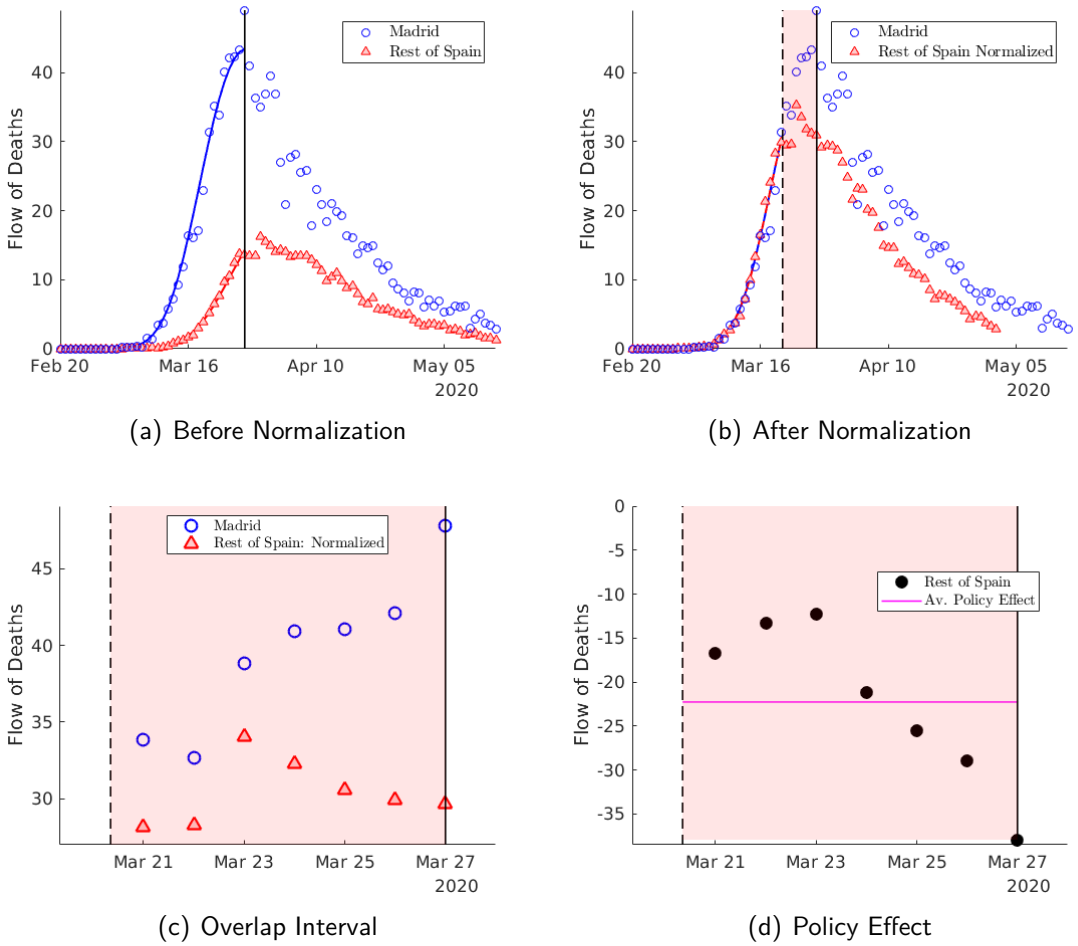
**Step 2: Policy Effect** We compute the average effect of policy as the average difference between the deaths in Madrid and the normalized rest of Spain within the overlap interval. Panel (c) of Figure 3.5 shows the normalized data on the overlap interval, which turns out to be seven days long. During this interval, the normalized rest of Spain faces the stay-at-home policy while the region of Madrid does not. The distance between the time paths of the flow of deaths in Madrid and in the normalized rest of Spain gives the effect of policy in terms of the amount of lives saved.

As distance function $\omega(t;\gamma)$ we choose a simple dummy variable that captures the average effect during the overlap interval

$$\omega(t;\gamma^p) = \begin{cases} 0 & \text{for } t \leq t_p + \tau \\ \gamma^p & \text{for } t \geq t_p + \tau + 1, \end{cases} \tag{3.22}$$

and thus (minus) the estimate, $-\hat{\gamma}^p$, captures the average reduction of deaths in the overlap interval. We find that over the course of the first week of the policy $20.1\%$ of lives were saved in the rest of Spain—relative to the counterfactual number of deaths that would have happened without the policy. We conduct a bootstrapping procedure for statistical inference. The estimate is significantly different from zero (the 90% bootstrap confidence band ranges from $16.6\%$ to $36.2\%$). The corresponding number of lives saved is about 1,176.

Figure 3.5: Normalization of the Epidemics: Madrid ($\mathcal{C}$) vs. Rest of Spain ($\mathcal{T}$)




(a) Before Normalization

(b) After Normalization

(c) Overlap Interval

(d) Policy Effect

*Notes:* Panel (a) shows the epidemics in Madrid ($\mathcal{C}$) and the rest of Spain ($\mathcal{T}$)), panel (b) shows the normalized epidemics. The fitted lines for $t < t_p + \tau$ show the smooth epidemics pre-policy that are used in the normalization procedure. Panel (c) zooms in on the overlap interval.

### 3.4.2 The Effects of the Pill

In 1960 the first oral contraceptive is approved in the U.S. by the Food and Drug Administration(FDA). The use of pill was approved for use by women above the age of majority.[16] Access to the pill has presumable benefits to women's education attainment and fertility decision. For this application we select two outcome variables: first, the share of women of age 25[17] with college

[16]Nevertheless, its full availability for sale at the national level was not until 1965, when the U.S. Supreme Court's 1965 Griswold v. Connecticut struck down Connecticut's ban on the use of contraceptives for married women. Until then many states still had anti-obscenity statutes in place, which banned contraception sales Bailey et al. (2011).

[17]Robustness on this number is done in the appendix.

attainment, second the share of women below age 26 that have ever given birth. We then apply our stage-based method to identify the effect of the pill on these two outcomes.

To back out the share of women of a certain age with college attainment we use decennial CENSUS data from IPUMS starting in 1940 up to 1980. In the absence of information on the year of graduation, we construct the historical series by using cohort information by CENSUS year. For example when using CENSUS data for 1960, the share of college women of age 25 in 1959 will be the share of a woman age 26 who reported (already) having attained college by 1960.[18] After computing the historical series per CENSUS year we compute the average across CENSUS series, the result is depicted in Figure 3.6. In a similar way, we compute the share of women below age 26 who have given birth. After computing the series of education attainment, our algorithm identifies that 4 states (Washington D.C., Massachussets, Colorado and Connecticut) lead the rest of the United states. We create an artificial region Top 4, constructed as the population weighted average of these four states, in the same spirit we compute region Rest of U.S.(RoUSA) as the population weighted average of the remaining stares.[19]

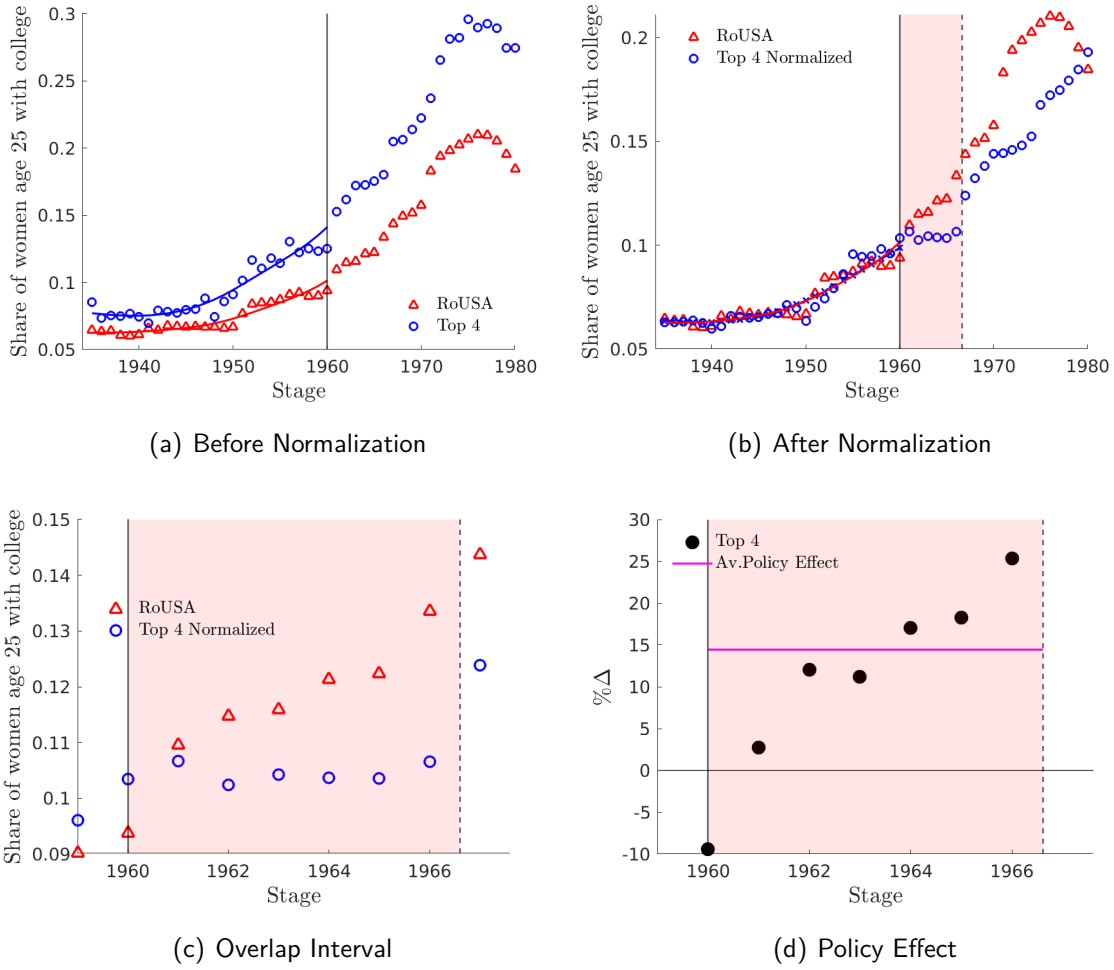
**Step 1: Normalization to Stage-Dynamics** Panel (b) in Figure 3.6 shows the result of normalizing the Control region on to the treatment region. For case of education attainment, the estimates of the mapping are $\hat{\psi} = \{0.8, 2.14, 0.75\}$, they show that the Top 4 started earlier and evolved faster than the rest of the U.S.

**Step 2: Policy Effects** For the college attainment the length of the overlap interval is 7.5 years, with an average policy effect of $11.65\%$ increase in college attainment of women age 25 had the pill not been implemented.

[18] Later completion and death could hinder the precision of our measure, however after comparing the historical series from various census years, the measure doesn't seem to be suffering from these problems

[19] See Appendix C, Section C.2 for results on fertility outcomes.

Figure 3.6: Normalization of time paths: Top 4 ($\mathcal{C}$) vs. RoUSA ($\mathcal{T}$)




(a) Before Normalization

(b) After Normalization

(c) Overlap Interval

(d) Policy Effect

*Notes:* Panel (a) shows the outcome variable for the aggregate of the 4 leading states in the U.S. ($\mathcal{C}$) and the rest of the U.S. ($\mathcal{T}$)), panel (b) shows the normalized epidemics. The fitted lines for $t < \tau$ show the smooth epidemics pre-policy that are used in the normalization procedure. Panel (c) zooms in on the overlap interval. Panel (d) Shows the policy effect, computed as the percentage difference had the policy not been implemented

### 3.4.3 German Reunification

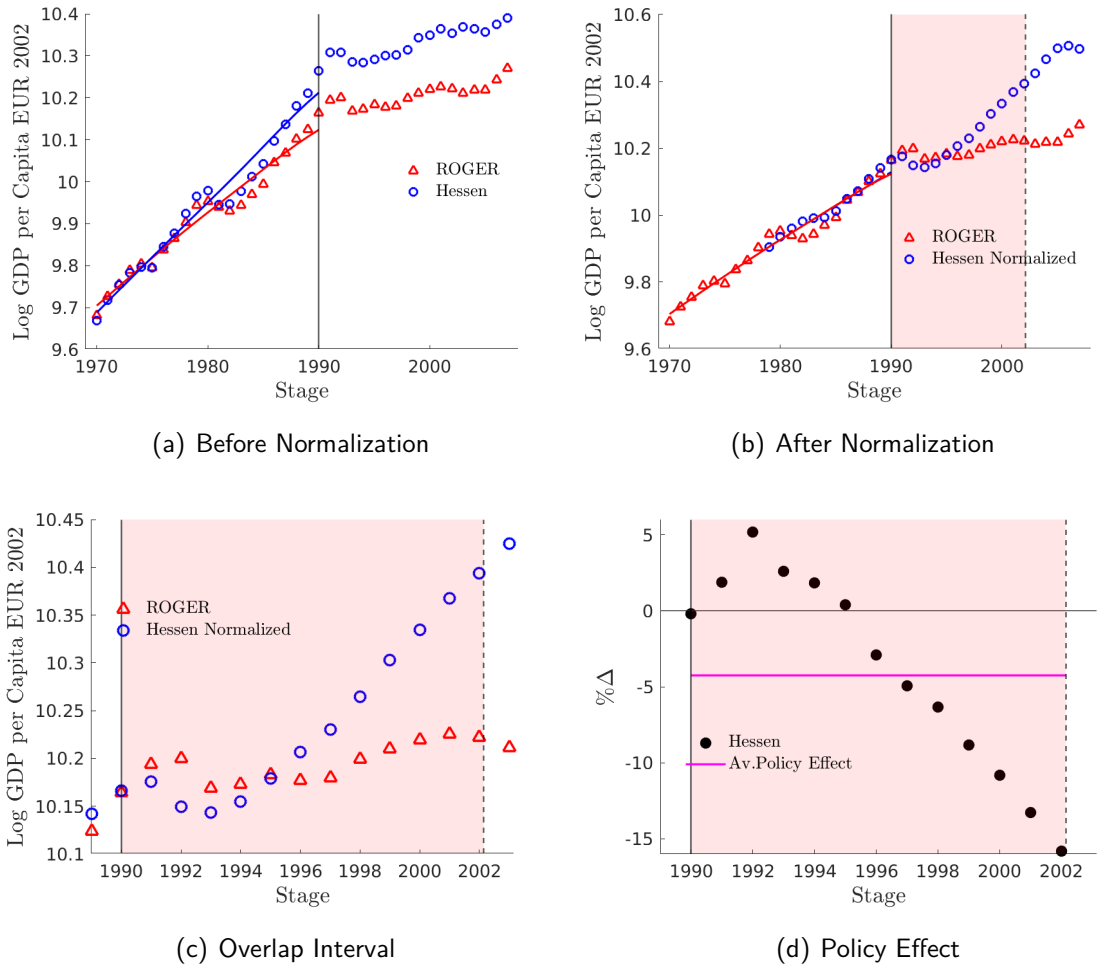
In 1990, after the fall of the Berlin wall in 1989, the German Democratic Republic was abolished and integrated fully into the Federal Republic of Germany. Given large differences between the West German states and the East German states, the political and economic integration came at some cost—the size of which is subject to debate. Abadie et al. (2014) study the consequences for West German GDP per capita, and form a counterfactual path using their Synthetic Control Group approach. Inspired by their analysis, we apply our method to the same context, and

construct a counterfactual for the evolution of GDP per capita in West Germany had it not been for the reunification. Different to Abadie et al. (2014) we can base the analysis on GDP data from Germany only—the key is the heterogeneity of regional GDP across West German states.

**Step 1: Normalization to Stage-Dynamics** Panel (a) of Figure 3.7 shows the time series of real GDP per capita in Hessen, which is the most advanced (in terms of GDP per capita) state of West Germany at the time of reunification, and ROGER, which is the population weighted average of GDP per capita in the remaining West German states. Panel (b) shows the normalized path of Hessen—the logic of the underlying mapping procedure is the same as what we apply in the previous application. The normalization unveils that Hessen delivers a counterfactual path for ROGER from 1991–94, in other words, Hessen receives the "reunification treatment" at the stage corresponding to ROGER's 1994.

**Step 2: Policy Effect** Figure 3.7 shows the percentage difference of ROGER' GDP per capita from the normalized Hessen GDP per capita. The identified reduction of GDP per capita ascribed to reunification is about 13% after ten years.

Figure 3.7: Normalization of GDP Time-Series: Hessen ($\mathcal{C}$) vs. Rest of Germany ($\mathcal{T}$)




(a) Before Normalization

(b) After Normalization

(c) Overlap Interval

(d) Policy Effect

*Notes:* Panel (a) shows the time-series of GDP per capita in Hessen ($\mathcal{C}$) and the rest of Germany ($\mathcal{T}$)), panel (b) shows the normalized paths. Panel (c) shows percentage difference of GDP per capita in rest of Germany relative to normalized Hessen. The solid vertical line indicates reunification, the dashed vertical line indicates the year after which the normalized Hessen time series is also subject to reunification.

## 3.5 Conclusion

We develop a novel empirical approach to estimate the effectiveness of public policies. Given a set of regions for which the outcome variable of interest is observed, our method can be applied in three different empirically relevant settings: (i.) policy is introduced in one region only, (ii.) policy is introduced in all regions at different calendar dates, (iii.) policy is introduced in all regions at the same date. The key of our method is the distinction between calendar time, at which outcomes are observed, and stages, over which dynamics evolve. With one region serving

as benchmark, we estimate the regional stage-time mapping using pre-policy data. We then apply this mapping to post-policy data, and the resulting normalized time series are the basis for a clean identification of the effects of policy. We show that our method works successfully in various Monte Carlo studies, and we illustrate its use in different applications. In the applications we look at aggregate outcomes at the regional level, and therefore focus the exposition on differences across regions. However, the method is more generally applicable to any group-level outcomes.

# Appendix A

# Appendix Chapter 1

## A.1 Parametrization of the two period model

We need to assign values to for $\sigma, \xi, \rho, \sigma^2_{\epsilon_w}, \varphi, \eta, r$ and $\beta$. Since this is a qualitative example most of the parameters set apriori. I choose a coefficient of relative risk aversion $\sigma = 2$ following Ortigueira and Siassi (2013). The Frish Elasticity of labor supply $\xi$ is set to $0.5$ following Blundell et al. (2016). The parameters for the income process are borrowed from Greenwood et al. (2016), with $\rho = 0.98$ and $\sigma^2_{\epsilon_w} = 0.011$. I select a risk free interest rate of $r = 2\%$ and a discount factor of $\beta = 0.98$ that are standard in the literature. The value of the public good $\eta$ is adhoc set to one. Finally fertility is set to $\varphi = 0.57$ which is the share of married households with children in 1970.

The rest of the parameters $\mu_g, \kappa_g, \nu_g, \phi_g, Ł_g, \sigma_{\epsilon_q}$ are calibrated depending on the following scenarios:

1. **Reference Model**: This is a gender equality scenario, that is, there are no differences between men and women. I set $\bar{w}_f/\bar{w}_m = 1, \kappa_m = 0.5, \nu_g = 0.5, \mu_g = 0.5$ and $\phi_m = \phi_f, Ł_m = Ł_f$. I calibrate the values for $\phi_g$ such that the average number of hours worked is equal to 1. I set $\sigma_{\epsilon_q}$ such that everyone gets married in period 1, finally, $Ł_g$ is set to have a divorce rate larger than $1\%$ in the second period.

2. **Alternative Model 1**: Sets some more realistic gender differences, as found in the literature and in the data, I set $\bar{w}_f/\bar{w}_m = 0.55, \kappa_m = 0.6, \mu_m = 0.6$. I calibrate $\phi_m < \phi_f$ such that the ratio of hours worked by women with respect to men is equal to $0.7$. This scenario keeps the mean of individual the match quality $Ł_g$ symmetric between men and women $Ł_m = Ł_f = 4$ and keeps the same probability of getting custody of the children across gender $\nu_g = 0.5$.

3. **Alternative Model 2**: Keeps the parameter values of Alternative Model 1, but sets Ł$_m$ > Ł$_f$, explicitly $L_m = 4$and $L_f = 3$, that is men an women enjoy married life differently. With women having lower match quality than men.

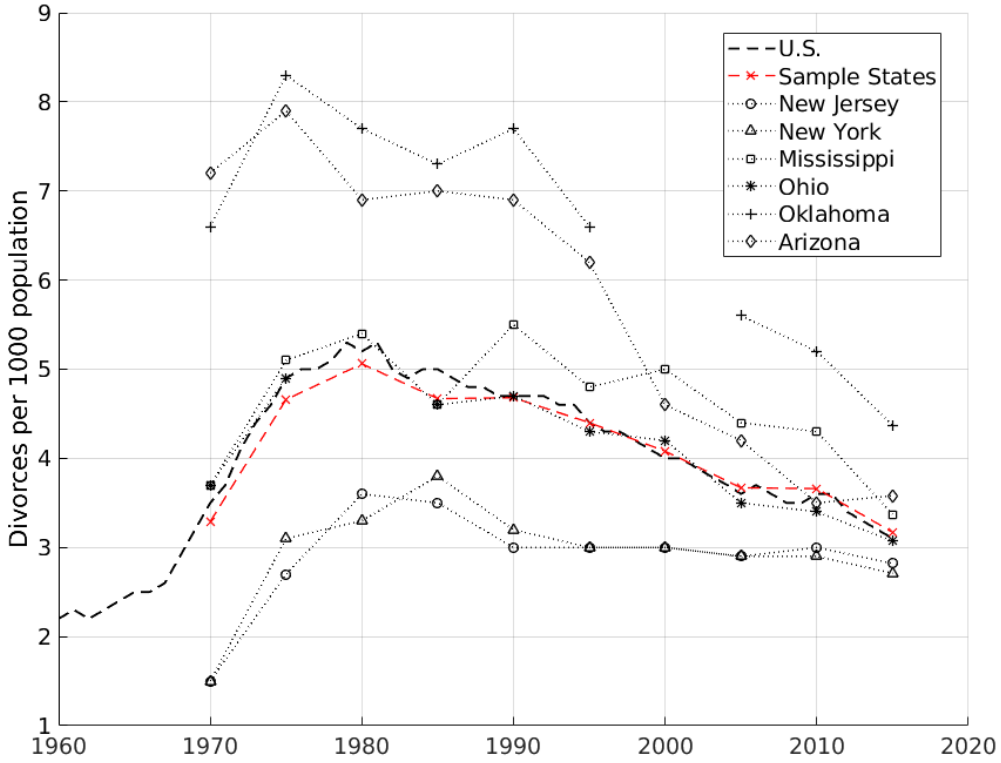
4. **Alternative Model 3**: Keeps the parameter values of Alternative Model 2, but sets $\nu_m = 0.1$, that is women have a higher probability of getting custody of the children upon divorce.

## A.2 List of States with reported divorce filing records

The list of sample countries is the following: Alabama, Alaska, Arkansas, Connecticut, Delaware, Georgia, Hawaii, Idaho, Illinois, Iowa, Kansas, Kentucky, Maryland, Massachusetts, Michigan, Missouri, Montana, Nebraska, New Hampshire, New York, Ohio, Oregon, Pennsylvania, Rhode Island, South Carolina, South Dakota, Tennessee, Utah, Vermont, Virginia, Wisconsin, Wyoming. A total of 31 states.

Figure A.1 compares the divorce rates for the sample states vs. aggregate divorce rates. Additionally, It shows divorce rates for selected states: Oklahoma and Arizona which are the two states with the two highest divorce rates 1970. Mississippi and Ohio, with divorce rates at the median. Finally, New Jersey and New York with the lowest divorce rates in 1970. We see that the divorce rates for the sample states and the aggregate divorce rates move closely following the same trends and reaching the peat at the same year. In addition we see that all states exhibit a declining trend since the mid 80's.

Figure A.1: Divorces per 1000 population




Notes: Divorce rates are taken from the CDC/NCHS National Vital Statistics System reports.

# Appendix B

# Appendix Chapter 2

## B.1 Solution Algorithm

**Solution Algorithm**

**Computing the recursive stationary equilibrium for Stage -1 (Pre-HIV Era)**

***Algorithm No.1*: Computation of the recursive stationary equilibrium of an Aggregate Epidemic Stage:**

Step 1: Make initial guesses of price $p$ (and prevalence $\phi^+$ if not in Pre-HIV Stage).

Step 2: Compute the agents decision rules.

Step 3: Compute the stationary distribution of the population across states (follow Algorithm No.2).

Step 4: Compute aggregate sex demand and aggregate sex supply. Check the aggregate consistency conditions.

Step 5: If conditions are not met, update $p$ and $\phi^+$ and return to Step 2.

In the absence of continuous state variables, the decision rules are reduced to single values conditional on the different states
This algorithm is generic enough that can be used to compute the recursive stationary equilibrium of any aggregate stage of the epidemic.

***Algorithm No.2*: Computation of the invariant distribution of the population:**

Step 1: Make an initial guess for the (discrete) mass function $\phi_0$ over the respective stage state space[1].

Step 2: For all individual states in $\mathbf{\Phi}$ compute the following expression:

if Stage: -1 (Pre-epidemic)

$$\phi_{t+1}(\mathbf{\Phi}') = \sum_{\mathbf{\Phi}} \sum_{s'|s} \gamma \pi(s'|s) \phi_t(\mathbf{\Phi}) + f \phi_t(\mathbf{\Phi}') \tag{B.1}$$

if Stage: 0 (HIV Myopia) or in Stage:1-2 (HIV Maturity)

$$\phi_{t+1}(\mathbf{\Phi}') = \sum_{\mathbf{\Phi}} \sum_{s'|s,h'|h} \gamma(h) \pi(s'|s) \phi_t^+ \lambda_{t,\rho}(h'|h) \phi_t(\mathbf{\Phi}) + f \phi_t(\mathbf{\Phi}') \tag{B.2}$$

if Stage:3-4 (ARV Era)

$$\phi_{t+1}(\mathbf{\Phi}') = \sum_{\mathbf{\Phi}} \sum_{\substack{d'|d,h'|h \\ s'|s}} \gamma(h,d) \pi(s'|s) \phi_t^+ \lambda_{t,\rho}(h'|h) \nu(d'|d) \phi_t(\mathbf{\Phi}) + f \phi_t(\mathbf{\Phi}') \tag{B.3}$$

Step 3: If $|\phi_{t+1} - \phi_t|$ is close to zero stop, otherwise set $\phi_t = \phi_{t+1}$ and return to Step 2.

**Computing the recursive stationary equilibrium for Stage 0 (HIV Myopia)**

***Algorithm No.3*: Computation of the recursive stationary equilibrium of the Myopic stage:**

Same as Algorithm No.1

**Computing the Non-stationary equilibrium for Stage 0 (HIV Myopia)**

***Algorithm No.4*: Computation of the solution of the Myopic stage:**
We are after a sequence of $\{\Phi_\tau\}_{\tau=T_{-1}}^{T_0}(g=0)$ where at each period $\tau \in \{T_{-1}+1, ..., T_0\}$, agents get a permanent unexpected shock to $\widetilde{\gamma}$, $\widetilde{z}$ and $\widetilde{\chi}$ following (2.17) and (2.18). $T_0$ being the period in which Stage 1-2 quick's in. To get each of the elements of the sequence we need to solve for an entire transition.

[1]We choose the uniform distribution as the initial values of the distribution. The algorithm should converge regardless of the choice of the initial distribution.

Step 0: Set $\tau = T_{-1} + 1$,

Step 1: Following (2.17) and (2.18) compute new values for $\widetilde{\gamma}_\tau$, $\widetilde{z}_\tau$ and $\widetilde{\chi}_\tau$ (Remember agents believe these values will be permanent).

Step 2: Compute the recursive stationary equilibrium of the Myopic stage associated with the new $\widetilde{\gamma}_\tau$, $\widetilde{z}_\tau$ and $\widetilde{\chi}_\tau$, (follow Algorithm No.2).

Step 3: Choose a very large number of transition periods $(\mathscr{T} - \tau)$.

Step 4: Guess a time path for the prices $\{p_t\}_{t=\tau}^{\mathscr{T}}$ and prevalences $\{\phi_t^+\}_{t=\tau}^{\mathscr{T}}$.

Step 5: Compute the equilibrium policy (and value) functions iterating backwards in time, $t = \mathscr{T} - 1, ..., \tau$.

Step 6: Simulate the evolution of the population distribution from $t = \tau$ to $t = \mathscr{T}$ with the help of the optimal policy functions and the initial distribution $\Phi_\tau(g = 0)$.

Step 7: Compare the simulated distribution at $\mathscr{T}$ with the stationary distribution function from Step 2. If they are not the same try increasing the horizon $\mathscr{T}$ and go back to Step 4.

Step 8: Compute the time path of excess demand for sex, and the path of prevalence's $\{\hat{\phi}_t^+\}_{t=\tau}^{\mathscr{T}}$. If markets don't clear along the path, or $\{\hat{\phi}_t^+\}_{t=\tau}^{\mathscr{T}} \neq \{\phi_t^+\}_{t=\tau}^{\mathscr{T}}$, then update $\{p_t\}_{t=\tau}^{\mathscr{T}}$ and $\{\phi_t^+\}_{t=\tau}^{\mathscr{T}}$ return to Step 5.

Step 9: Record the first elements of the transition decision rules $(c_{\tau+1}, x_{\tau+1})$, prices $(p_{\tau+1})$, prevalence $(\phi_{\tau+1}^+)$ and joint distribution $(\Phi_{\tau+1})$.

Step 10: Stop if $\tau = T_0$

Step 11: Replace $\tau = \tau + 1$ and go back to Step 1.

**Computing the recursive stationary equilibrium for Stage 1-2 (HIV Maturity)**

***Algorithm No.5*: Computation of the recursive stationary equilibrium of the Pre-Epidemic stage:**

Same as Algorithm No.1

**Computing the Non-stationary equilibrium for Stage 1-2 (HIV Maturity)**

***Algorithm No.6*: Computation of the solution of the Maturity Stage:**
We are after a sequence of $\{\Phi_t\}_{t=T_0}^{\mathscr{T}}(g = 1-2)$ that goes from the last period in Stage 0 (HIV Myopia $t = T_0$) to the recursive stationary equilibrium of Stage 1-2 of the epidemic (HIV Maturity).

Step 1: Choose a large number of transition periods $(\mathscr{T} - T_0)$

Step 2: Simulate a sequence of $\{\rho_{e,t}\}_{t=T_0}^{\mathscr{T}}$ by education group, following (2.29).

Step 3: Compute the recursive stationary equilibrium of Stage 1-2 of the epidemic. This stationary equilibrium is associated with $\lim_{t\to\mathscr{T}} \rho_{e,t} = \rho$. That is, in the stationary equilibrium both education groups have completed learning of the true probability HIV infection risk as a function of sex.

Step 4: Guess a time path for the prices $\{p_t\}_{t=T_0}^{\mathscr{T}}$ and prevalences $\{\phi_t^+\}_{t=T_0}^{\mathscr{T}}$.

Step 5: Compute the equilibrium policy (and value) functions iterating backwards in time, $t = \mathscr{T} - 1, ..., T_0$.

Step 6: Simulate the evolution of the population distribution from $t = T_0$ to $t = \mathscr{T}$ with the help of the optimal policy functions and the initial distribution $\Phi_{T_0}(g = 1)$.

Step 7: Compare the simulated distribution at $T$ with the stationary distribution function from Step 3. If they are not the same try increasing the horizon $\mathscr{T}$ and go back to Step 2.

Step 8: Compute the time path of excess demand for sex, and the path of prevalence's $\{\hat{\phi}_t^+\}_{T_0}^{\mathscr{T}}$. If markets don't clear along the path, or $\{\hat{\phi}_t^+\}_{T_0}^{\mathscr{T}} \neq \{\phi_t^+\}_{T_0}^{\mathscr{T}}$, then update $\{p_t\}_{T_0}^{\mathscr{T}}$ and $\{\phi_t^+\}_{T_0}^{\mathscr{T}}$ return to Step 5.

**Computing the recursive stationary equilibrium for Stage 3-4 (ARV Era)**

***Algorithm No.7*: Computation of the recursive stationary equilibrium of the ARV stage:**

Same as Algorithm No.1

**Computing the Non-stationary equilibrium for Stage 3-4 (ARV Era)**

***Algorithm No.8*: Computation of the solution of the ARV Stage:**
After $T_1$ periods in Stage 1-2 (HIV Maturity), ARV's are introduced unexpectedly. We are after

a sequence of $\{\Phi_t\}_{t=T_1}^{\mathscr{T}}(g = 3-4)$ that go from $T_1$ to the recursive stationary equilibrium of Stage 3-4 of the epidemic (ARV Era).

Step 1: Choose a large number of transition periods $(\mathscr{T} - T_1)$

Step 2: Compute the recursive stationary equilibrium of Stage 3-4 of the epidemic. This stationary equilibrium is associated with $\lim_{t\to\infty} \eta_t = \widetilde{\eta} = 1$. That is, there is full coverage for all HIV infected individuals.

Step 3: Guess a time path for the prices $\{p_t\}_{t=T_1}^{\mathscr{T}}$ and prevalences $\{\phi^+\}_{t=T_1}^{\mathscr{T}}$.

Step 4: Given the sequences for ARV coverage by education group $\{\eta_t(e)\}_{t=T_1}^{\mathscr{T}}$, compute the equilibrium policy (and value) functions iterating backwards in time, $t = \mathscr{T} - 1, ..., T_1$.

Step 5: Simulate the evolution of the population distribution from $t = T_1$ to $t = \mathscr{T}$ with the help of the optimal policy functions and the initial distribution $\Phi_{t=T_1}(g = 2)$.

Step 6: Compare the simulated distribution at $\mathscr{T}$ with the stationary distribution function from Step 2. If they are not the same try increasing the horizon $\mathscr{T}$ and go back to Step 3.

Step 7: Compute the time path of excess demand for sex. and the path of prevalence's $\{\hat{\phi}_t^+\}_{t=T_1}^{\mathscr{T}}$. If markets don't clear along the path, or $\{\hat{\phi}_t^+\}_{t=T_1}^{\mathscr{T}} \neq \{\phi_t^+\}_{t=T_1}^{\mathscr{T}}$, then update $\{p_t\}_{t=T_1}^{\mathscr{T}}$ and $\{\phi_t^+\}_{t=T_1}^{\mathscr{T}}$ return to Step 4.

## B.2 Further Results

Table B.1: The HIV-Education Gradient, Sexually Active Sample

| *HIV Status* | (1) | (2) | (3) | (4) | (5) |
|---|---|---|---|---|---|
| Education | 0.0048*** | 0.0123*** | 0.0107*** | 0.0042*** | 0.0039*** |
| | (0.0009) | (0.0008) | (0.0013) | (0.0003) | (0.0004) |
| Education * Stage1 | | -0.0064*** | -0.0047*** | -0.0007 | -0.0004 |
| | | (0.0010) | (0.0013) | (0.0004) | (0.0005) |
| Education * Stage2 | | -0.0129*** | -0.0115*** | -0.0029*** | -0.0027*** |
| | | (0.0009) | (0.0014) | (0.0003) | (0.0004) |
| Education * Stage3 | | -0.0101*** | -0.0081*** | -0.0018 | -0.0016 |
| | | (0.0020) | (0.0015) | (0.0015) | (0.0014) |
| Education * Stage4 | | -0.0067*** | -0.0054*** | -0.0019*** | -0.0014*** |
| | | (0.0013) | (0.0016) | (0.0004) | (0.0004) |
| Male | -0.0267*** | -0.0273*** | -0.0272*** | -0.0268*** | -0.0269*** |
| | (0.0039) | (0.0038) | (0.0025) | (0.0022) | (0.0021) |
| Age | 0.0021*** | 0.0021*** | 0.0021*** | 0.0022*** | 0.0022*** |
| | (0.0004) | (0.0004) | (0.0003) | (0.0002) | (0.0002) |
| Urban Area | 0.0260*** | 0.0243*** | 0.0276*** | 0.0337*** | 0.0344*** |
| | (0.0059) | (0.0059) | (0.0053) | (0.0038) | (0.0038) |
| Stage 1 | -0.0018 | 0.0137*** | 0.0097 | -0.0082*** | 0.0099*** |
| | (0.0062) | (0.0047) | (0.0060) | (0.0012) | (0.0023) |
| Stage 2 | 0.0132 | 0.0573*** | 0.0676*** | 0.0010 | 0.0289*** |
| | (0.0105) | (0.0122) | (0.0136) | (0.0029) | (0.0045) |
| Stage 3 | -0.0114 | 0.0198 | 0.0330** | -0.0129** | 0.0138** |
| | (0.0164) | (0.0149) | (0.0128) | (0.0061) | (0.0060) |
| Stage 4 | -0.0027 | 0.0143*** | 0.0436*** | -0.0183*** | 0.0056 |
| | (0.0052) | (0.0040) | (0.0097) | (0.0025) | (0.0036) |
| Agricultural Share | -0.0034*** | -0.0034*** | -0.0035*** | 0.0027*** | -0.0006 |
| | (0.0003) | (0.0003) | (0.0004) | (0.0005) | (0.0004) |
| Output per Capita | -0.0000*** | -0.0000*** | -0.0000*** | 0.0001** | 0.0001*** |
| | (0.0000) | (0.0000) | (0.0000) | (0.0000) | (0.0000) |
| Constant | 0.1128*** | 0.0918*** | 0.0554*** | -0.1603*** | -0.1992*** |
| | (0.0101) | (0.0075) | (0.0098) | (0.0493) | (0.0282) |
| Year-Country Dum. | No-No | No-No | Yes-No | No-Yes | Yes-Yes |
| Sample Size | 329,205 | 329,205 | 329,205 | 329,205 | 329,205 |

*Notes:* All specifications use the "Sexually Active" subsample. The underlying econometric models are as specified in the columns of Table 2.3. Column (1) reports the tests results for the stationary specification. Columns (2) to (5) report the tests results for the non-stationary specification. We include the same set of controls and fixed effects as in our benchmark specifications in Table 2.3. Standard errors are clustered at the country level using the wild cluster bootstrap from Cameron et al. (2008), and reported in parenthesis. * significant at 10%; ** significant at 5%; *** significant at 1%.

Table B.2: The Knowledge-Education Gradient: Women and Men Separately

(A) One Sexual Partner without Other Partners

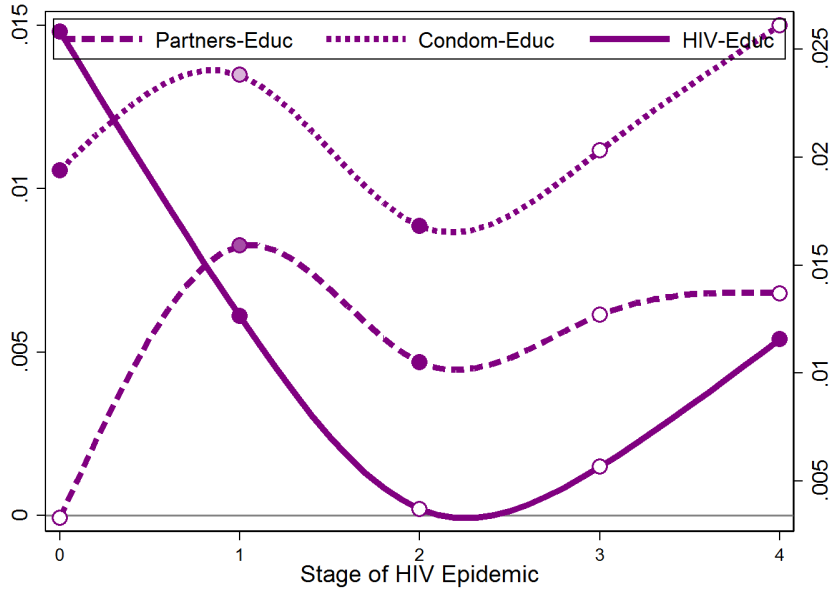
| | Women | | | | | Men | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| *HIV Knowledge* | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) | (9) | (10) |
| Education | 0.0135 | 0.0033 | 0.0005 | 0.0074*** | 0.0076*** | 0.0076** | 0.0010 | 0.0013 | 0.0078*** | 0.0087*** |
| | (0.111) | (0.110) | (0.866) | (0.000) | (0.000) | (0.001) | (0.326) | (0.580) | (0.000) | (0.000) |
| Education * Stage1 | | 0.0126 | 0.0120** | 0.0047 | 0.0046 | | 0.0078*** | 0.0059** | 0.0005 | -0.0001 |
| | | (0.156) | (0.043) | (0.309) | (0.292) | | (0.000) | (0.039) | (0.840) | (0.952) |
| Education * Stage2 | | 0.0072** | 0.0093** | 0.0038** | 0.0033 | | 0.0091*** | 0.0086*** | 0.0028* | 0.0017 |
| | | (0.046) | (0.049) | (0.045) | (0.210) | | (0.000) | (0.001) | (0.088) | (0.285) |
| Education * Stage3 | | 0.0094 | 0.0120 | 0.0018 | 0.0020 | | 0.0057 | 0.0053 | -0.0034 | -0.0042 |
| | | (0.612) | (0.412) | (0.912) | (0.911) | | (0.386) | (0.337) | (0.444) | (0.304) |
| Education * Stage4 | | 0.0104 | 0.0134 | 0.0071 | 0.0062 | | 0.0069** | 0.0075* | 0.0019 | 0.0007 |
| | | (0.732) | (0.648) | (0.812) | (0.830) | | (0.044) | (0.066) | (0.424) | (0.759) |
| Year-Country Dum. | No-No | No-No | Yes-No | No-Yes | Yes-Yes | No-No | No-No | Yes-No | No-Yes | Yes-Yes |
| Sample Size | 213,907 | 213,907 | 213,907 | 213,907 | 213,907 | 167,894 | 167,894 | 167,894 | 167,894 | 167,894 |

(B) Always Use Condom During Sex

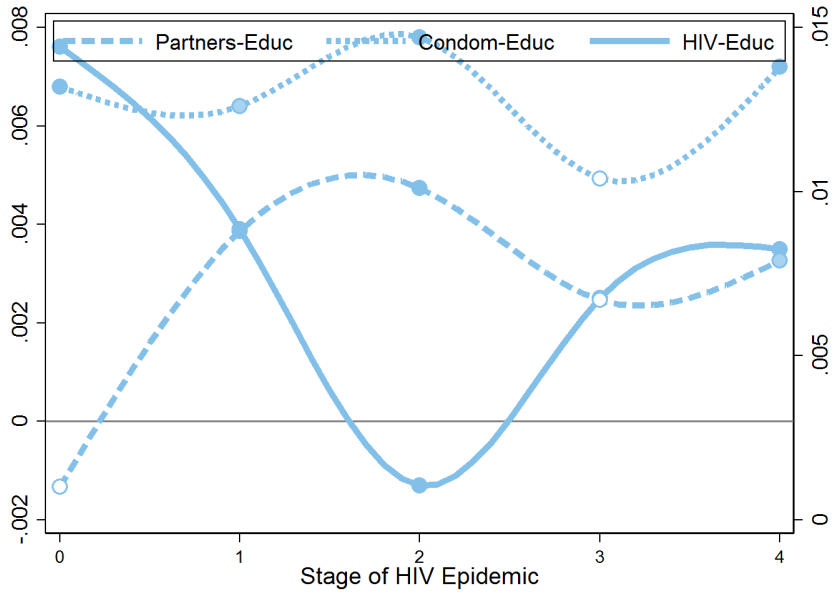
| | Women | | | | | Men | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| *HIV Knowledge* | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) | (9) | (10) |
| Education | 0.0221 | 0.0194*** | 0.0169*** | 0.0196*** | 0.0196*** | 0.0126*** | 0.0132*** | 0.0141*** | 0.0187*** | 0.0181*** |
| | (0.133) | (0.000) | (0.000) | (0.000) | (0.000) | (0.002) | (0.000) | (0.000) | (0.000) | (0.000) |
| Education * Stage1 | | 0.0044 | 0.0073 | 0.0034 | 0.0039 | | -0.0006 | -0.0007 | -0.0036 | -0.0029 |
| | | (0.801) | (0.593) | (0.809) | (0.754) | | (0.935) | (0.929) | (0.638) | (0.668) |
| Education * Stage2 | | -0.0026 | 0.0003 | -0.0017 | -0.0025 | | 0.0015 | 0.0017 | -0.0036 | -0.0035 |
| | | (0.616) | (0.953) | (0.648) | (0.572) | | (0.639) | (0.637) | (0.236) | (0.246) |
| Education * Stage3 | | 0.0009 | 0.0039 | 0.0008 | 0.0014 | | -0.0028 | -0.0024 | -0.0073 | -0.0064 |
| | | (0.956) | (0.804) | (0.952) | (0.920) | | (0.770) | (0.789) | (0.415) | (0.496) |
| Education * Stage4 | | 0.0067 | 0.0089 | 0.0047 | 0.0033 | | 0.0006 | -0.0004 | -0.0046* | -0.0047** |
| | | (0.946) | (0.929) | (0.963) | (0.974) | | (0.828) | (0.910) | (0.065) | (0.029) |
| Year-Country Dum. | No-No | No-No | Yes-No | No-Yes | Yes-Yes | No-No | No-No | Yes-No | No-Yes | Yes-Yes |
| Sample Size | 213,763 | 213,763 | 213,763 | 213,763 | 213,763 | 167,800 | 167,800 | 167,800 | 167,800 | 167,800 |

*Notes:* In panel (A) we report the coefficients of a linear model where the endogenous variable is binary for *"Can you (the respondent) reduce the chances of getting HIV by having one sex partner who has no other partners?"*. In panel (B) we report the coefficients of a linear model where the endogenous variable is binary for *"Can you (the respondent) reduce the chances of getting HIV by always wearing a condom?"*. In both panels we include the same set of controls and fixed effects as in our benchmark specifications in Table **??**. Standard errors are clustered at the country level using the wild cluster bootstrap from Cameron et al. (2008), and reported in parenthesis.* significant at 10%; ** significant at 5%; *** significant at 1%.

Figure B.1: The Knowledge-Education Gradient: Evolution Across Stages of the HIV Epidemic




(a) Women

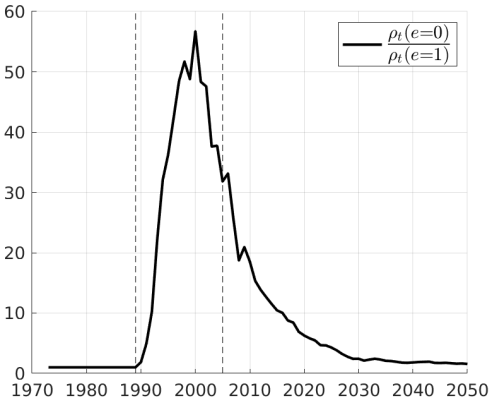



(b) Men

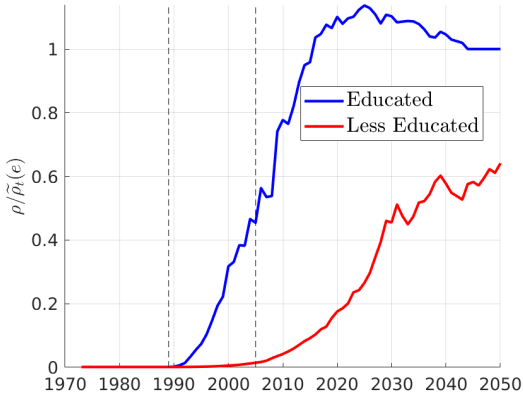
*Notes*: The HIV-Education gradient is plotted on the left vertical axis. The HIV knowledge in terms of the Partners-Education gradient and the Condoms-Eduction gradient are plotted on the right vertical axis. For each stage $j$ we plot $\left(\gamma_0 + \sum_{j>0} \gamma_j \mathbf{1}_j\right)$. The specification we plot is with year controls. For the HIV knowledge in terms of Partners-Education and Condoms-Education gradient we use column 3 (8) in Table B.2 for respectively women (top panel) and men (bottom panel). Significance at 10%, 5%, and 1% is represented by, respectively, markers with open circles, markers with medium transparency fill, and markers with solid fill. We use a cubic spline for interpolation across stages.

Figure B.2: Comparing beliefs ($\rho$) between education groups.

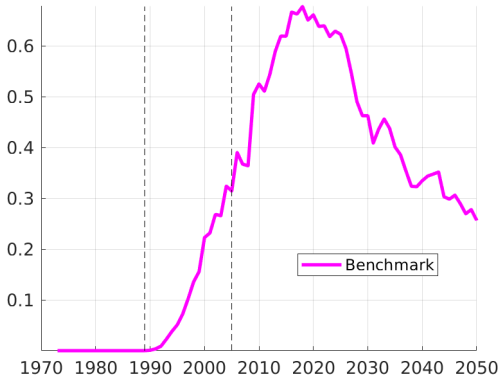
(a) Beliefs ratio $\rho_t(e=0)/\rho_t(e=1)$




(b) Relative belief convergence by education group




(c) Knowledge of $\rho$ Education Gradient



# Appendix C

# Appendix Chapter 3

## C.1 Reduced-Form Montecarlo

### The SIRD Model

Consider a SIRD model $\mathcal{M}(\Theta, \mathcal{P})$ summarized by the following laws of motion:

$$X_{S,t+1} = -\alpha^p \beta \frac{I_t}{N_t} S_t \tag{C.1}$$

$$X_{I,t+1} = \alpha^p \beta \frac{I_t}{N_t} S_t - \gamma I_t \tag{C.2}$$

$$X_{R,t+1} = (1-\zeta)\gamma I_t \tag{C.3}$$

$$X_{D,t+1} = \zeta \gamma I_t \tag{C.4}$$

The triplet $\mathcal{P} = (\alpha^p, t_p, t_f)$ defines a policy with effects against new infections determined by $p \in [0,1]$ implemented in a subinterval $[t_p, t_f]$. All model parameters $\Theta = (t_0, \beta, \zeta, \gamma)$ and the policy effects $p$ could be time variant.
Different values of $p$ imply alternative counterfactual scenarios that pin down the effect of policy on an outcome variable (i.e. $X_{D,t}$).

### Simulating artificial data

1. Simulate a SIRD economy $\mathcal{C}$ without policy: $\mathcal{M}(\Theta_{\mathcal{C}}) = \mathcal{M}(\Theta_{\mathcal{C}}; p = 1, t_p, t_f)$ (solid blue).
2. Add a policy to $\mathcal{C}$: Simulate $\mathcal{M}(\Theta_{\mathcal{C}}; p < 1, t_p, t_f)$ (dashed blue)
3. Simulate a SIRD economy $\mathcal{T}$ without policy: $\mathcal{M}(\Theta_{\mathcal{T}})$ with $\Theta_{\mathcal{T}} \neq \Theta_{\mathcal{C}}$ (solid red).

4. Add the same policy as in (2) to $\mathcal{T}$: Simulate $\mathcal{M}(\Theta_{\mathcal{T}}; p < 1, t_p, t_f)$ (dashed red)

### Policy effects

We are interested in uncovering the true policy effects on model statistics that can have an observable counterpart in the data, e.g., $X_D(\mathcal{M})$.
We can calculate the true policy effect for any economy $j \in \{\mathcal{C}, \mathcal{T}\}$ that adopts a given policy $p < 1$. The total number of lives saved is given by:[1]

$$LS = D_{t_p}^{t_f}(p = 1) - D_{t_p}^{t_f}(p < 1) \tag{C.5}$$

where where $D_{t_p}^{t_f}(p) = \sum_{t=t_p}^{t_f} X_{D,t}^{j}(p)$ is the accumulated number of deaths from time $t_p$ until $t_f$ given policy $p$.The percentage of lives saved is defined as

$$LS(\%) = \frac{LS}{D_{t_p}^{t_f}(p = 1)} \tag{C.6}$$

### Policy effects for the overlap interval between $X_D^{\mathcal{T}}(p < 1)$ and $X_D^{\mathcal{C}}(p < 1)$

1. Map $X_D^{\mathcal{T}}(p = 1)$ to $X_D^{\mathcal{C}}(p = 1)$[2]; that is, solve for $\boldsymbol{\psi} = \{\psi_0, \psi_1, \psi_2\}$ that minimizes the log difference of the regions flow of deaths.
2. Translate $tp$ into time of $\mathcal{T}$ by applying: $t_{tp} = floor((tp - \psi_2)/\psi_3)$.
3. Compute the number of lives saved over the interval $[tp, t_{tp}]$ as: $LS = \sum_{t=tp}^{t_{tp}} X_{D,t}^{\mathcal{T}}(p = 1) - \sum_{t=tp}^{t_{tp}} X_{D,t}^{\mathcal{T}}(p < 1)$
4. Compute the estimated $\%$ of lives saved as: $\%LS = \frac{LS}{\sum_{t=tp}^{t_{tp}} X_{D,t}^{\mathcal{T}}(p<1)}$.

### Estimating the Policy effects

Consider a policy evaluator that faces a scenario in which she/he has information on a outcome (statistic) of interest, e.g., $X_D(t)$. Say this outcome is available for the two regions $\mathcal{C}$ and $\mathcal{T}$. The evaluator also knows the dates of policy implementation in each region.
The policy evaluator knows nothing else. In particular, she/he does not know the model that

[1] Note that we can also calculate the number of lives saved for any time interval.
[2] This could also be $X_D^{\mathcal{T}}(p < 1)$ to $X_D^{\mathcal{C}}(p < 1)$ using the series up to the policy

generates the data and, hence, cannot use the model to identify the true policy effects following the steps that we described above. This implies that the policy evaluator has the following information set:

1. Available time-series data on a statistic of interest $X_D^{\mathcal{C}}$ and $X_D^{\mathcal{T}}$. In particular, what is available to the evaluator is the left hand side of:

$$X_D^{\mathcal{C}}(t) = X_D\left(\mathcal{M}(\Theta_{\mathcal{C}}; p < 1, t_p, t_f)\right) \quad \text{(blue dashed)}$$
$$X_D^{\mathcal{T}}(t) = X_D\left(\mathcal{M}(\Theta_{\mathcal{T}}; p < 1, t_p, t_f)\right) \quad \text{(red dashed)}$$

   The evaluator is not provided with the true model that generates the data (i.e., RHS), which implies that the evaluator does not know $p$ ex-ante.

2. This includes the period of policy implementation that could differ across regions $(t_{\mathcal{C},p}, t_{\mathcal{C},f})$ and $(t_{\mathcal{T},p}, t_{\mathcal{T},f})$.

In our benchmark scenario we assume $t_{\mathcal{C},p} = t_{\mathcal{T},p} = t_p$ and $t_{\mathcal{C},f} = t_{\mathcal{C},f} = t_f \to \infty$

**Cases:**

Now we consider the performance of the method in estimating the policy effects when region $\mathcal{T}$ differs from $\mathcal{C}$ in the following dimensions:

1. $t_{\mathcal{C},0} < t_{\mathcal{T},0}$, Region $\mathcal{T}$ starts with $5$ periods later than $\mathcal{C}$.
2. Previous case $+\beta_{\mathcal{C}} > \beta_{\mathcal{T}}$. We choose $\beta_{\mathcal{T}}$ to be 95% of $\beta_{\mathcal{C}}$
3. Previous case $+\zeta_{\mathcal{C}} > \zeta_{\mathcal{T}}$. We choose $\zeta_{\mathcal{T}}$ to be 85% of $\zeta_{\mathcal{C}}$
4. Previous case $+\gamma_{\mathcal{C}} > \gamma_{\mathcal{T}}$. We choose $\gamma_{\mathcal{T}}$ to be 85% of $\gamma_{\mathcal{C}}$
5. Exogenous (observed or unobserved) Pre-Policy Behavioural change:
   Starting at $t_{bc} < t_p$ region $\mathcal{T}$ experiences an exogenous *time varying* change in the infection rate. That is $\beta_{t \geq bc}^{\mathcal{T}} = \alpha_t^{be} \beta_{t=0}^{\mathcal{T}}$ with $\alpha_t^{be} \in [0, 1]$. This is change could be observed or unobserved by the evaluator. At $t_p$ a policy is applied in $\mathcal{T}$, that sets the $\beta_{t>=tp}^{\mathcal{T}} = \alpha^p \beta_{t=0}^{\mathcal{T}}$, such that $0 \leq \alpha^p \leq \min(\alpha_t^{be})$. We choose $t_{bc}$ to be $13$ periods before policy, $\min(\alpha_t^{be}) = 0.95$ and $\alpha^p = 0.7$.

We also consider a more extreme set of cases in which the lenght of the overlap period is around 11 periods. This is achieved by setting $t_{\mathcal{T},0} = 9$.

**Results**

Table C.1 shows the experiment results. The first three columns correspond to the true percentage of lives saved (PLS), number of lives saved (LS) and length of the overlap interval (OLP). The last three, are the corresponding estimates. The last five columns also show that the results are valid regardless of whether it has been chosen to map T to C or C to T.

Table C.1: MC Results

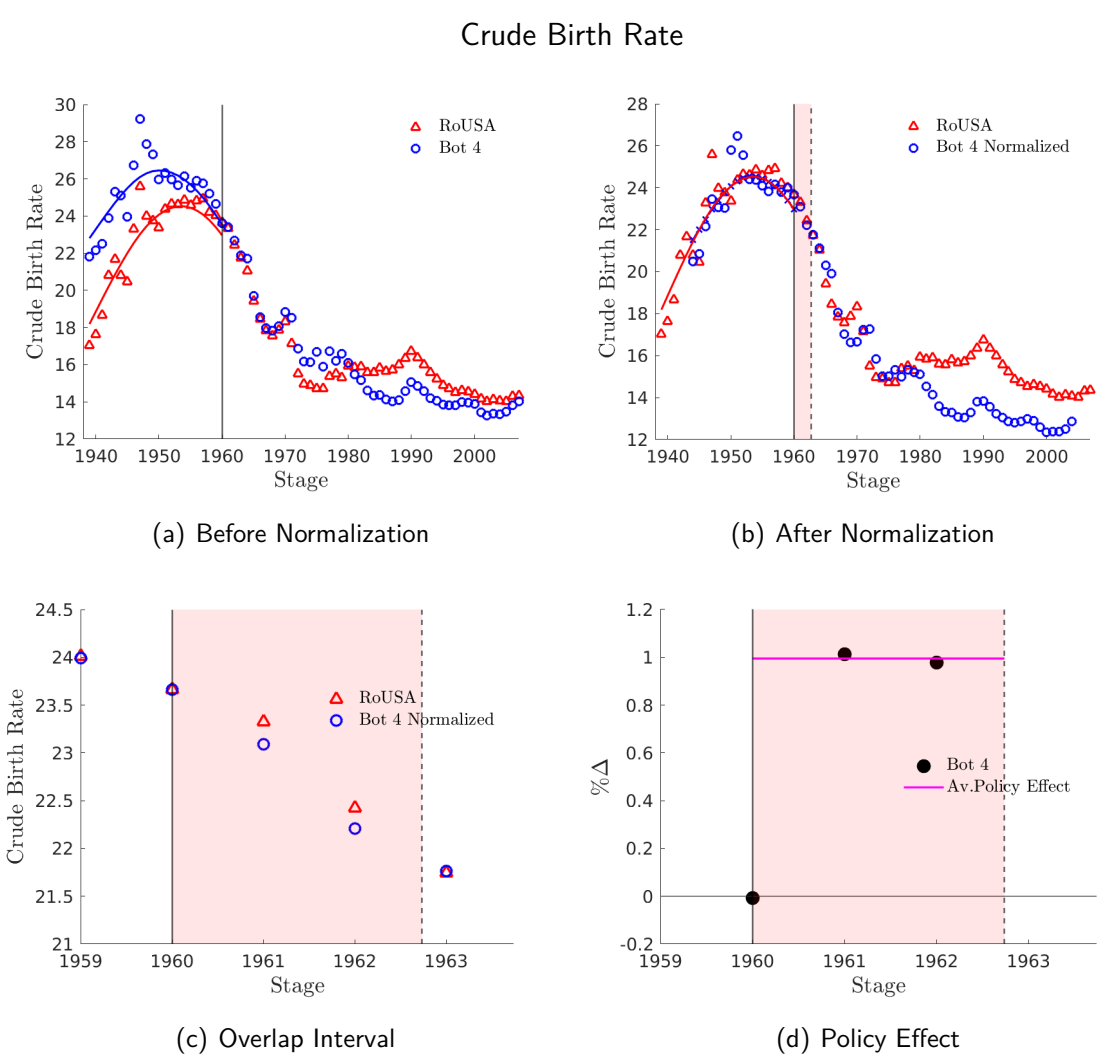
| | | | OLP$\sim 7$ | | | |
|---|---|---|---|---|---|---|
| | True | | | Mapping T to C | | |
| | PLS | LS | OLP(days) | PLS | LS | OLP(days) |
| 1.Late | 8,5754 | 221,34 | 5 | 8,5758 | 221,35 | 5 |
| 2.Late+Slow | 14,33 | 453,86 | 7,1787 | 14,242 | 451,17 | 7,2546 |
| 3.Late+Slow+Small | 14,342 | 420,65 | 7,1747 | 14,236 | 417,59 | 7,2385 |
| 4.Late+Slow+Small+Fast recovery | 9,1513 | 238,71 | 6,1153 | 9,6619 | 218,52 | 5,8801 |
| 5+Time Varying Behaviour Change | 14,122 | 401,72 | 7,2823 | 14,202 | 404,51 | 7,3869 |
| | True | | | Mapping C to T | | |
| 1.Late | 8,5754 | 221,34 | 5 | 8,5743 | 221,31 | 5 |
| 2.Late+Slow | 14,321 | 453,58 | 7,4886 | 14,236 | 450,99 | 7,7745 |
| 3.Late+Slow+Small | 14,334 | 420,41 | 7,5002 | 14,232 | 417,44 | 7,7582 |
| 4.Late+Slow+Small+Fast recovery | 9,1026 | 237,37 | 6,6159 | 9,67 | 218,71 | 5,9819 |
| 5+Time Varying Behaviour Change | 14,115 | 401,5 | 7,6134 | 14,183 | 403,91 | 7,9507 |
| | | | OLP$\sim 11$ | | | |
| | True | | | Mapping T to C | | |
| 1.Late | 16,349 | 654,25 | 9 | 16,349 | 654,25 | 9 |
| 2.Late+Slow | 22,524 | 979,24 | 11,055 | 22,525 | 980,42 | 11,063 |
| 3.Late+Slow+Small | 22,52 | 906,65 | 11,045 | 22,487 | 906,1 | 11,045 |
| 4.Late+Slow+Small+Fast recovery | 17,232 | 632,82 | 9,8887 | 17,132 | 596,38 | 9,8023 |
| 5+Time Varying Behaviour Change | 21,806 | 848,82 | 11,152 | 22,888 | 945,98 | 11,275 |
| | True | | | Mapping C to T | | |
| 1.Late | 16,349 | 654,25 | 9 | 16,349 | 654,24 | 9 |
| 2.Late+Slow | 22,518 | 978,93 | 11,532 | 22,516 | 979,98 | 11,86 |
| 3.Late+Slow+Small | 22,514 | 906,38 | 11,546 | 22,479 | 905,72 | 11,841 |
| 4.Late+Slow+Small+Fast recovery | 17,193 | 631,26 | 10,698 | 17,139 | 596,65 | 9,9586 |
| 5+Time Varying Behaviour Change | 21,801 | 848,59 | 11,659 | 22,577 | 931,02 | 12,108 |

*Notes:* For region $\mathcal{C}$ we choose parameters
$N = 6M, I_0 = 1000, R_0 = 0, D_0 = 0, S_0 = N - I_0, \beta = 0.32, \gamma = 1/12, \zeta = 0.0011, t_0 = 1, p = 0.7$

## C.2 The effect of the pill on crude birth rates

When analyzing fertility outcomes, Idaho, West Virginia, Nevada, Arkansas lead the rest of the U.S. therefore we use artificial region Bottom 4 to construct the counterfactual. Normalization results are shown in Figure C.1. The average policy effect is very small suggesting that crude birth rates were not affected by the pill.

Figure C.1: Normalization of time paths: Bot 4 ($\mathcal{C}$) vs. RoUSA ($\mathcal{T}$)



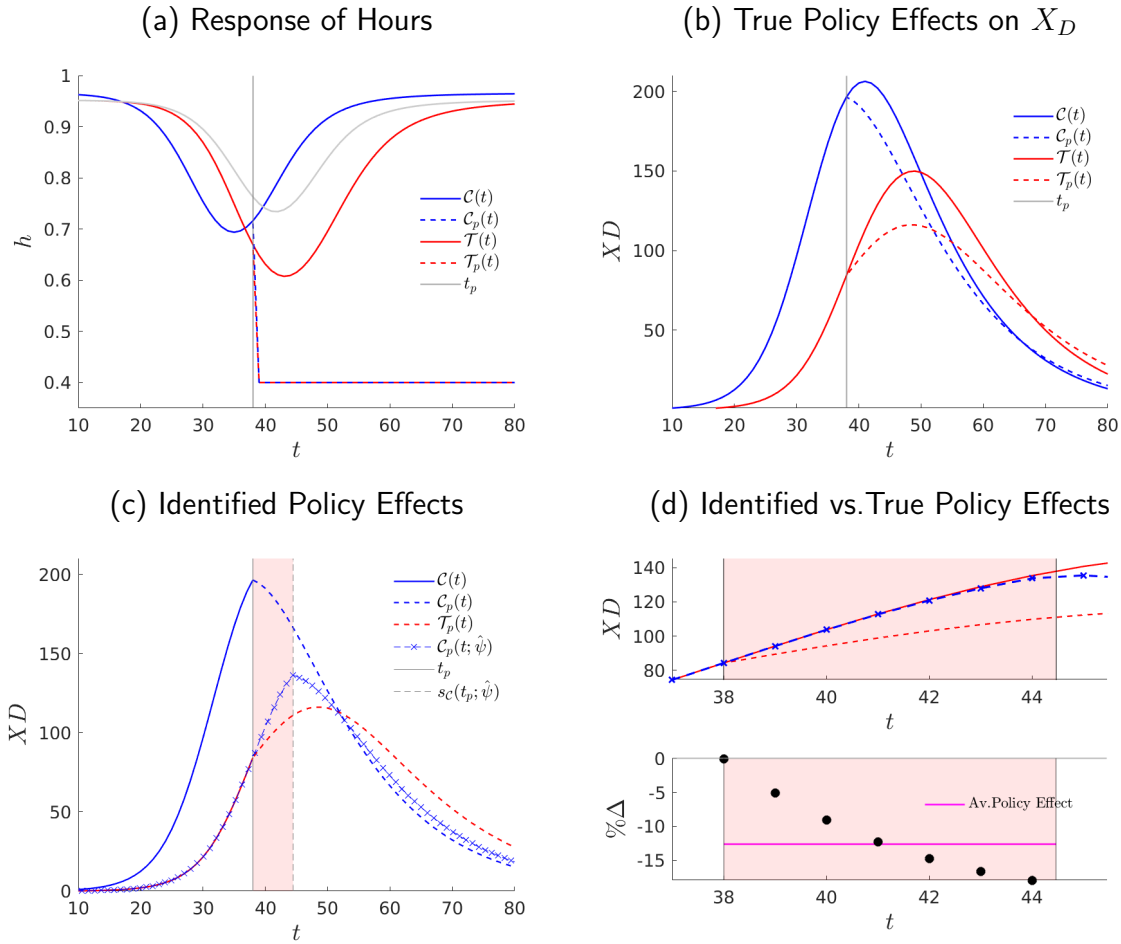

*Notes:* Panel (a) shows the outcome variable for the aggregate of the 4 leading states (Idaho, West Virginia, Nevada, Arkansas)in the U.S. ($\mathcal{C}$) and the rest of the U.S. ($\mathcal{T}$)), panel (b) shows the normalized epidemics. The fitted lines for $t < \tau$ show the smooth epidemics pre-policy that are used in the normalization procedure. Panel (c) zooms in on the overlap interval. Panel (d) Shows the policy effect, computed as the percentage difference had the policy not been implemented

## C.3 Addressing Latent Time Varying Heterogeneity

In panel (a) of Figure 3.1 we re-conduct the same exercise but imposing latent time varying heterogeneity in the treatment region $\mathcal{T}$. This time varying heterogeneity It is induced by changes in $\xi_{\mathcal{P}}$ across time, gradually moving closer to the actual $\xi$. [3](See Figure C.2) This implies a pre-policy reduction in the equilibrium hours. From the perspective of an empirical strategy aimed to assess policy effects, the change in the model $\xi_{\mathcal{P}}$ generates time-varying unobserved heterogeneity in the treatment region. Note that we purposefully left the actual effects of policy on region $\mathcal{C}$ unchanged. We find that our stage-based identification is robust to this type of heterogeneity; results are shown bellow.

[3]For example, in the context of the economy that we postulated, a government that knows $\xi$ could nudge the economy's households to learn about the actual effects of economic activity. This would imply that after the nudging households privately decide to change their prevention behavior providing less labor in equilibrium.

Figure C.2: Stage-Based Identification of Policy Effects: Case with Latent Time Varying Heterogeneity




*Notes:* *Where* $\bar{h} = 0.4, t_p = 38, t_f = 250$.

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