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

BSE Working Paper 1418| December 2023

Christian Alemán-Pericón, Daniela Iorio, Raül Santaeulàlia-Llopis

bse.eu/research

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

Christian Aleman-Pericon
New York University Abu Dhabi

Daniela Iorio
University of Bologna

Raül Santaeulàlia-Llopis
New York University Abu Dhabi
UAB, BSE and CEPR

December 31, 2023

Abstract

We explore learning about HIV infection odds from risky sex as a new mechanism explaining the Sub-Saharan Africa HIV epidemic. Our novel empirical evidence reveals a U-shaped relationship between education and being HIV positive across epidemic stages, which prompts the idea of asymmetric learning: more educated individuals potentially learn faster and update their (latent) beliefs about infection odds more accurately than less educated individuals, inducing earlier sexual behavioral change among the more educated. Our *nonstationary* model incorporates three HIV epidemic stages, chronologically: a myopic stage where agents are unaware of how risky sex causes infections, a learning stage where agents update beliefs on infection odds, and an ARV stage reflecting treatment introduction. Anchored in the micro evidence—explaining the HIV-education gradient—we find that our learning mechanism is powerful: a 5-year earlier learning reduces new AIDS deaths by almost 45%, and a 10-year earlier learning results in a 60% drop.

Keywords: Quantitative, Macroeconomics, Equilibrium, HIV, Epidemic, Stages, Risky Sex, Asymmetric Learning
JEL Classification: E00

*We would like to thank Luiz Brotherhood, Cezar Santos, Karen Kopecky, Michele Tertilt, Nezih Guner, Federico Curci, Maia Güell, Alex Ludwig, Vahagn Jerbashian, Hans-Peter Kohler, Ludo Visschers, Luis Rojas and José-Víctor Rios-Rull for their valuable comments and seminar participants in multiple venues. Raül Santaeulàlia-Llopis acknowledges financial support from the AGAUR 2020PANDE00036 "Pandemies" Grant 2020-2022, from the Spanish Ministry of Economy and Competitiveness, through the Proyectos I+D+i 2019 Retos Investigacion PID2019-110684RB-I00 Grant and the Severo Ochoa Programme for Centres of Excellence in R&D (CEX2019-000915-S).

1 Introduction

Motivation In Sub-Saharan Africa (SSA), most HIV infections are acquired through heterosexual intercourse, making risky sexual behavior a critical factor in understanding the HIV epidemic. As a result, the evaluation of policy interventions to mitigate the epidemic places significant importance on the dynamics of sexual behavior change (Behrman and Kohler, 2012). In this context, assessing policies requires a thorough consideration of endogenous sexual behavior responses. A compelling illustration of this complexity comes from Greenwood et al. (2019), who show in an equilibrium model of HIV that the introduction of anti-retroviral (ARV) treatment can have unintended consequences, leading to riskier sexual practices and ultimately contributing to an increase in HIV prevalence.[1]

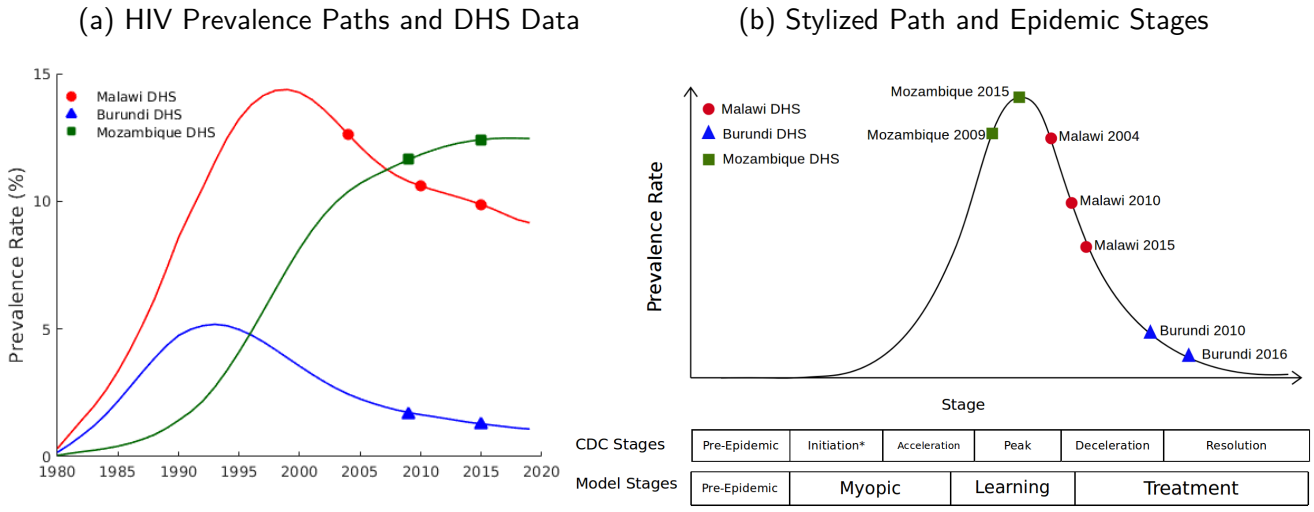
Idea In this paper, we propose learning about the odds of HIV infection due to risky sexual practices as a new mechanism to explain the evolution of the HIV epidemic in SSA. The concept is straightforward. In the early stages of the epidemic, individuals are simply myopic (unaware) of the consequences that risky sexual behavior has on the probability of HIV infection and, consequently, mortality risk. However, as the epidemic advances, individuals start learning about the modes of infection, particularly how risky sexual behavior generates HIV infection. We argue that a natural consequence of this learning is a potential shift in agents' sexual behavior to minimize the risk of infection. This, in turn, can shape the overall trajectory of the HIV epidemic. In particular, an equilibrium sexual behavior response to learning (e.g., engaging in less risky sex) has the potential to decelerate the epidemic. Further, if some individuals, like the more educated, learn faster, we should observe different sexual behavioral responses and HIV prevalence across individuals over the course of the epidemic. Of course, these responses potentially undergo another transformation once antiretroviral drugs (ARVs) are introduced at later stages of the epidemic.

Empirical Evidence To provide evidence of the proposed mechanism, we leverage variation in HIV status and sexual behavior across education groups over the course of the epidemic. Specifically, operating on the premise that more educated individuals learn earlier about risky sex as a mode of infection, it follows that the more educated should respond sooner by engaging in less risky activities.[2] We empirically investigate this proposition in two steps. First, as HIV infection

[1] For equilibrium models of other recent epidemics such as Covid-19 and opiods, see also Brotherhood et al. (2020) and Greenwood et al. (2022), respectively.

[2] Education can also act as a shield against misinformation, a sadly common occurrence in SSA. A poignant example is South Africa's former president, Thabo Mbeki, who succeeded Nelson Mandela in 1999 and served until 2008. He publicly insisted that HIV was not the cause of AIDS. Adding to this, when antiretroviral drugs were developed, Mbeki's Minister of Health, Manto Tshabalala-Msimang, encouraged those with the virus to continue traditional remedies and adopt "*a diet of lemons, garlic, beetroot, and olive oil*." She believed that the new drugs were tailored for Western populations. Source: The Conversation.

Figure 1: Demographic Health Surveys (DHS) and Epidemic Stages: An Illustration



Notes: Panel (a) shows the HIV prevalence paths from UNAIDS and corresponding HIV prevalence from the DHS country-year observations for three countries. Panel (b) shows the stylized path of an epidemic. The bottom of that panel shows different intervals defined by the Center for Disease Control (CDC) as a way to characterize the complete evolution of an epidemic. It also includes the endogenous stages of the HIV epidemic that result from the calibration of our model described in Section 3 of this paper. The information of the CDC epidemic intervals was taken from the Loudoun Health District pandemic response plan, published in march 2020. www.loudoun.gov.

is the ultimate consequence of sexual behavior, our focus is on documenting the behavior of the HIV-education gradient—the difference in the probability of being HIV positive between more and less educated individuals—over the course of the epidemic. Second, we directly examine the behavior of the risky sex-education gradient—the difference in engagement in risky sex activities between more and less educated individuals—throughout the epidemic. Following our rationale that sexual behavior change occurs earlier for the more educated than the less educated, our hypothesis is that we should observe a decrease in the probability of being HIV positive for the more educated relative to the less educated, and a contemporaneous decrease in the engagement in risky sex by the more educated relative to the less educated over the course of the epidemic.

This empirical assessment faces the challenge that nationally representative microdata with available information about HIV status, risky sexual behavior, and education at the individual level—mainly from the Demographic Health Surveys (DHS)—is rather scattered across time and space, with limited yearly observations per country (at most three years per country). We illustrate this phenomenon showing the HIV prevalence time path for three countries and the associated DHS country-year observations in panel (a) of Figure 1. Consequently, it is difficult to trace the relationship between HIV status, sexual behavior, and education over the course of the epidemic within countries. To address this issue, we propose using variation in *stages* of the epidemic across DHS country-year observations, where we define *stages* as the location

of a country-year HIV prevalence on the support of a reference (stylized) HIV prevalence path. Precisely, we conduct a normalization of coordinates (namely, prevalence rate and time) in order to map the country-specific HIV prevalence time paths onto a reference (stylized) path. In panel (b) of Figure 1, we show that through this normalization, the DHS country-year observations are assigned a stage (i.e. a location) along a reference epidemic path, enabling us to conduct our analysis of the HIV-education and risky sex-education gradients over the course of the epidemic. Although the *stage* is a continuous variable, it can be discretized to characterize specific intervals along the epidemic. E.g., at the bottom of Figure 1, we show the discretization of epidemic stages as defined by the Center for Disease Control and Prevention (CDC) guidelines.[3]

Our main finding is that the HIV-education gradient follows a stylized U-shape pattern (positive-zero-positive) over the course of the epidemic. In the early stages of the epidemic, one additional year of education is associated with a 1.12 percentage point increase in the probability of being HIV-positive. These education disparities in HIV status gradually vanish (become not significantly different from zero) as the epidemic reaches mid-stages. At later stages, it reverts back to a positive education gradient in HIV, where one additional year of education results in a 0.48 percentage point increase in the probability of being HIV-positive. This U-shape pattern is robust to a large set of individual controls and country and year fixed effects. We interpret our methodology—the conditioning of the HIV-education gradient (a micro object) on the stage of the epidemic (a macro object)—and our results as one potential solution to the previous lack of consensus regarding the HIV-education gradient (Beegle and de Walque, 2009).[4]

Further, we explore education disparities in risky sexual behavior, measured by the number of extramarital partners. We find that the education disparities in risky sexual behavior closely mirrors the U-shape pattern of the HIV-education gradient over the course of the epidemic. Overall, these results highlight the potential role of education disparities in sexual behavior changes in determining the evolution of the HIV-education gradient over the course of the epidemic. Lastly, if the monetary costs of ARVs do not outweigh its benefits, such as increasing survival probabilities and decreasing virus spread, the provision of ARV treatment should theoretically lead to an increase in risky sex practices Greenwood et al. (2019). In a scenario where the educated have

[3]More broadly, the definition of the *stages* of the epidemic is analogous to the definition of *stages* of economic development (e.g., Galor and Weil, 2000; Hansen and Prescott, 2002; Lucas, 2004; Herrendorf et al., 2014) or the *stages* of the demographic transition (e.g., Lee, 2003; Greenwood et al., 2005).

[4]While mixed evidence on the HIV-education gradient may arise from differences in methodology, sampling strategies, and measures of education and HIV status, these factors may not entirely explain differing conclusions in prior studies. For example, using data from rural areas of Manicaland province in eastern Zimbabwe, Gregson et al. (2001) suggest that the relationship between education and HIV status could be dynamic and potentially explained by differential exposure to risky sexual practices. Similarly, de Walque (2007) drew parallels in his analysis of a cluster of villages in rural Uganda between 1989 and 2001. The findings in these prior micro studies align with our results emerging from a more systematic cross-country analysis of the epidemic's evolution.

greater access to ARVs, the introduction of ARV treatment might help explain the rebound of the HIV-education gradient in later stages of the epidemic. We empirically demonstrate this possibility by showing that the HIV-education gradient decreases when we include ARV controls for the most advanced stages of the epidemic.

Equilibrium Model with Asymmetric Learning We develop a *nonstationary* quantitative model that endogenizes HIV infections through risky sex consumption (and production) exchanged on a centralized market. Precisely, higher risky sex consumption increases the probability of contracting HIV. In turn, HIV infection reduces life expectancy, productivity and life satisfaction (enjoyment of utility flows). Our main theoretical contribution is the introduction of a new mechanism that allows individuals to learn about their probability of infection due to risky sex as time goes by. Individuals are heterogeneous in education, income shocks, sex type (consumer or producer), HIV status, and ARV treatment status. Further, motivated by our empirical findings we allow for asymmetric learning: more educated individuals potentially learn faster and update their latent beliefs about infection odds more accurately than less educated individuals, inducing earlier sexual behavioral change among the more educated. Our model incorporates these mechanisms through three distinct stages of the HIV epidemic: a myopic stage where agents are unaware of how risky sex causes infections; a learning stage where understanding how risky sex generates infections leads to significant behavioral changes; and an ARV stage reflecting the introduction of treatment that extends life expectancy and mitigates productivity losses.

We anchor the theory to the evolution of the micro HIV-education gradient. Specifically, our internal calibration successfully targets the empirical U-shape pattern of the HIV-education gradient over the course of the epidemic. During the myopic stage, the HIV prevalence rises along with the HIV-education gradient as the more educated engage in more risky sex because they can afford it, as their income is higher. This happens because agents do not understand the link between infections and risky sex consumption, therefore they do not adjust their risky sexual behaviour. This trend continues until the learning stage starts. During the learning stage, educated individuals learn about the negative effects of infection and reduce their risky sexual behaviour. They do so faster than the less educated, which results in a decrease in the HIV-education gradient eventually approximating zero. That is, asymmetric learning is key to reproduce the decline of the gradient from early stages of the epidemic. Finally, the introduction of ARV's brings the HIV-education gradient back to showing positive values. Since the educated have better access to costly ARV treatment, they now face lower risk per marginal unit of risky sex consumed, thus triggering a rebound in the gradient.

What Explains the HIV-education gradient? We perform three counterfactual experiments to quantify the factors influencing the HIV-education gradient. First, we find that equalizing income between education groups reduces the HIV-education gradient by approximately 65% at early stages of the epidemic, turning it negative during the latter learning and ARV stages. That is, income differences by education groups are relevant for the level of the HIV-education gradient. Second, addressing learning differences by making everyone learn as fast as the educated substantially decelerates the decline of the HIV-education gradient in the learning stage by approximately 50% leading to a less pronounced U-shape gradient overall. Additionally, making everyone learn as slow as the less educated entirely reverts the decline in the HIV-education gradient. That is, asymmetric learning is clearly the main driver of the decline the HIV-education gradient during the learning stage. In the final experiment, equalizing ARV coverage across education groups reveals that coverage differences explain approximately 60% of the gradient rebound.

Drivers of the HIV Epidemic and Policy Experiments We conduct a set of counterfactual experiments with the aim of understanding the drivers of the HIV epidemic and inform policy. The first policy experiment imposes an earlier start date for the learning stage. We find that if learning would have started 5 years earlier the number of AIDS deaths would have reduced by approximately 45%. Further, the initiation of the learning stage by 10 implies a 60% drop in the numer of AIDS deaths. In the second policy experiment, we increase the share of educated, such that it reaches 85% by 2030; this represents a significant improvement of the education level of the population. In this case, we find that risky sex consumption experiences a boost, but not enough for the prevalence to be too far from the benchmark scenario. In our third policy experiment we simulate an earlier adoption of ARV treatment. We find that if ARV's would have been introduced as early as 1989 (16 years earlier than the actual year), the prevalence rate would have been reduced by 40%. Our last experiment, consists of the introduction of pre-exposure treatment (PrEP) to prevent further spread of the HIV virus. In our set up, PrEPs reduce the probability of infection by a certain percentage. We find that if PrEPs reduce the probability of infection by 30%, the prevalence would decrease by 33%.

External Validation Can our theory explain the epidemic heterogeneity across SSA countries? To address this question we modify our benchmark calibration set for Malawi to cumulatively include country-specific features of South Africa such as the specific education structure, the year of arrival of ARVs and the ARVs coverage rates. After imposing these country-specific features of South Africa, we find that our simulated path for prevalence rate overlaps with that observed for South Africa which implies, for example, elevating the prevalence rate from approximately 10% in benchmark Malawi to 16% in South Africa in year 2005.

The remainder of our paper is structured as follows. Section 2 proposes a unified framework assigning DHS country-year observations to HIV epidemic stages and documents the behavior of the HIV-education and risky sex-education gradients over the epidemic. Section 3 introduces the model endogenizing the entire HIV epidemic across myopic, learning, and ARV stages. Section 4 covers the model calibration. Section 5 decomposes the sources of the HIV-education gradient. Section 6 discusses the HIV epidemic drivers and provides policy experiments. Section 7 explores the model's ability to account for other country-specific epidemics. Section 8 concludes.

2 Empirical Evidence

Our empirical strategy focuses on examining the relationship between education and HIV status over the stages of the epidemic. The empirical challenge lies in the scarcity and dispersion of nationally representative microdata, primarily sourced from the Demographic Health Surveys (DHS), offering limited yearly observations per country (often just three years); see Section 2.1. To overcome this, we propose utilizing variation in *stages* across DHS country-year observations, where *stages* represent the position of a country-year HIV prevalence on a reference path; see Section 2.2. We discuss our econometric specification that explicitly conditions on the stage of the epidemic and discuss our results in Section 2.3.

2.1 Data

The demographic and health surveys, DHS. The DHS are based on nationally representative samples and are available for a large set of SSA countries. Several aspects make DHS data sets appealing for our exercise. Specifically, these data provide measures of individual HIV status,[5] education and risky sexual behavior (e.g., number of extramarital relationships and condom use) in harmonized manner across SSA countries. Further, since the appearance of the DHS, the WHO and UNAIDS use DHS estimates as official HIV prevalence rates.[6] We consider the full sample of SSA DHS surveys for which individual HIV testing has been conducted and were available as of January 2021. We thus use a total of 60 DHS surveys corresponding to 30 SSA countries.[7] Descriptive statistics for the sample are provided in Table 1. The whole sample consists of a total of 735,455 individuals. The average cross country HIV prevalence is 6.1%. Notice, there is substantial degree of heterogeneity in HIV prevalence across countries, the prevalence rate ranges

[5] A large portion of the adult DHS respondents have been tested for HIV, the proportion of respondents who did not take the HIV test is .318 in the original whole sample. However, we find that the association between the likelihood of taking the HIV test and the educational attainment is virtually zero in the DHS.

[6] Before the DHS, the majority of HIV prevalence estimates came from antenatal clinics. Indeed, the DHS dropped by approximately 20% the previous official estimates of HIV prevalence in SSA which were subject to substantial selection driven by young women in antenatal clinics.

[7] See our Online Appendix A for more details.

Table 1: The DHS Sample Characteristics (across Countries)

	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

Notes: The computation of these statistics is performed using individual HIV weights provided by the DHS survey. Country-specific population weights (i.e., the population size of each country provided by the World Bank) are used to compute the statistics across countries. The number of extramarital partners refers to those in the last 12 months. The frequency of condom use refers to the last sexual intercourse. Our sample is based on 60 DHS with 30 countries and a total of 735,455 individuals.

between 0.5 to 31.2. The Gini index of the HIV prevalence across countries is high 0.54. For the core analysis we restrict our attention to HIV-tested adults 15-49 years old who reported their schooling achievement. Approximately, 56.5% of the total population has completed primary education and 33% live in urban areas.

Our preferred choice for measuring education is: years of schooling.[8] In our sample, the cross-country mean schooling years is 3.2 with a median of 2.8, a minimum of 0.6 and a maximum of 4.8. 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). In our sample, the number of extramarital partners is on average 0.15. The share of people who do not have extramarital partners is 87.4%. The frequency of condom use is 9.5% on average. These figures are conditional on being sexually active.

UNAIDS and World Bank data We track the evolution of the HIV epidemic in SSA using country level prevalence rate time paths published by UNAIDS. UNAIDS provides estimates of

[8]The reasoning for our choice—rather than using other socioeconomic status variables such as 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.

the HIV prevalence rates from 1980 until 2023 for all 30 countries in our sample.[9] Finally, for all our SSA sampled countries we use data on real output per capita and on the agricultural share of output, from the World Bank Development Indicators (WDI). We use these data in our empirical analysis to control for country-specific stages of aggregate economic development.

2.2 Normalizing the Country-Specific Epidemic Time Paths to a Reference Path

This section presents an algorithm that normalizes the coordinates (the level and time) of each country-specific HIV prevalence path to a reference path. This normalization assigns each (country-year) DHS dataset to an epidemic stage, where a stage (normalized time) is defined as a location on the support of the reference path.

Algorithm. A Normalization of the Evolution of the HIV epidemic *Given the country-specific time paths of the HIV prevalence rate $\lambda_{i,t}$ and a reference path λ_t,[10] we define the interpolands as $g_i : t \to [0, \max_t \lambda_{i,t}]$. Let $g(t)$ be the interpoland of the reference path.*

1. *Level normalization*

   Normalize the country-specific and reference interpolands by their respective peak prevalence $g_i(t_*^i) = \max_t g_i(t)$ and $g(t_*) = \max_t g(t)$, to obtain $\widetilde{g}_i, \widetilde{g} : t \to \Lambda = [0, 1]$: $\widetilde{g}_i(t) = g_i(t)/g_i(t_*^i)$ and $\widetilde{g}(t) = g(t)/g(t_*)$. Note now that $\widetilde{g}_i(t_*^i) = \widetilde{g}(t_*) = 1 \; \forall i$.

2. *Time normalization*

   Normalize the time input of the country-specific interpolands by constants α_i^L and α_i^R, to obtain $\widetilde{g}_i : s \to \Lambda$ and $\widetilde{g} : s \to \Lambda$, hence comparable in the normalized space $\Omega = \{S \times \Lambda\}$, using:

$$s = \alpha_i^L (t - t_*^i) \quad \text{for} \quad t \leq t_*^i \quad \text{where} \quad \alpha_i^L = \frac{t_* - t_0}{t_*^i - t_0^i}$$
$$s = \alpha_i^R (t - t_*^i) \quad \text{for} \quad t > t_*^i \quad \text{where} \quad \alpha_i^R(\gamma) = \frac{t_\gamma - t_*}{t_\gamma^i - t_*^i}$$

   where $s \in S$ are the normalized units of time, t_0 is the initial year and t_γ^i is the year when country i's relative prevalence reaches threshold γ [11]. Constants α_i^L and α_i^R compress/stretch the interpolant[12] to ensure that the number of normalized periods s that it takes each country to move to and away from the peak are the same across countries. In

[9] We use UNAIDS data available in 2021. The HIV prevalence levels from our DHS samples and the UNAIDS counterparts are very similar. See also a detailed discussion of these data in Bongaarts et al. (2008).

[10] The choice of the reference path does not alter the results of the normalization. For simplicity we chose reference to be the region's weighted average $\lambda_t = \sum_i^I \lambda_{i,t} \mu_{i,t}$, where $\mu_{i,t}$ is the country population weight.

[11] The choice of γ does not alter the ranking of countries across stages of the epidemic

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

other words, the stage s is formally the location of each country's year prevalence on the support of the reference path.

To implement the algorithm we need to make two choices: (1) the form of the interpolant and (2) the (prevalence) threshold γ for the time normalization after the peak. First, we specify $\widetilde{g}(s)$ as a B-spline with cubic pieces $\widetilde{g}_i(s) = \sum_{j=1}^{n} \theta_j \psi_j(s)$. [13] Second, we set γ =0.8, which maximizes the number of countries having surpassed that threshold. Our results are robust to alternative choices of γ.

We next apply our algorithm to the 30 SSA countries in our sample. The results are depicted in Figure 2. Panel (a) shows the HIV prevalence level across time for each and all countries. Panel (b) shows the result of the HIV prevalence level normalization, which does not correct for countries having HIV peaks at different years. Panel (c) shows the result of the time normalization, which does not correct for differences in the HIV prevalence level. Finally, panel (d) jointly applies the level and the time normalization. It is clear from panel (d) that after the normalization of both the level and time the epidemic paths are comparable across countries, i.e., the stages of the epidemic (normalized time) are common across countries. In particular, the stage for each DHS country-year observation can be computed by solving for,

$$s_i^{DHS} = \widetilde{g}_i^{-1}\left(\frac{\lambda_{i,t_{DHS}}}{g_i(t_*^i)}\right) \in \mathbb{R}.$$

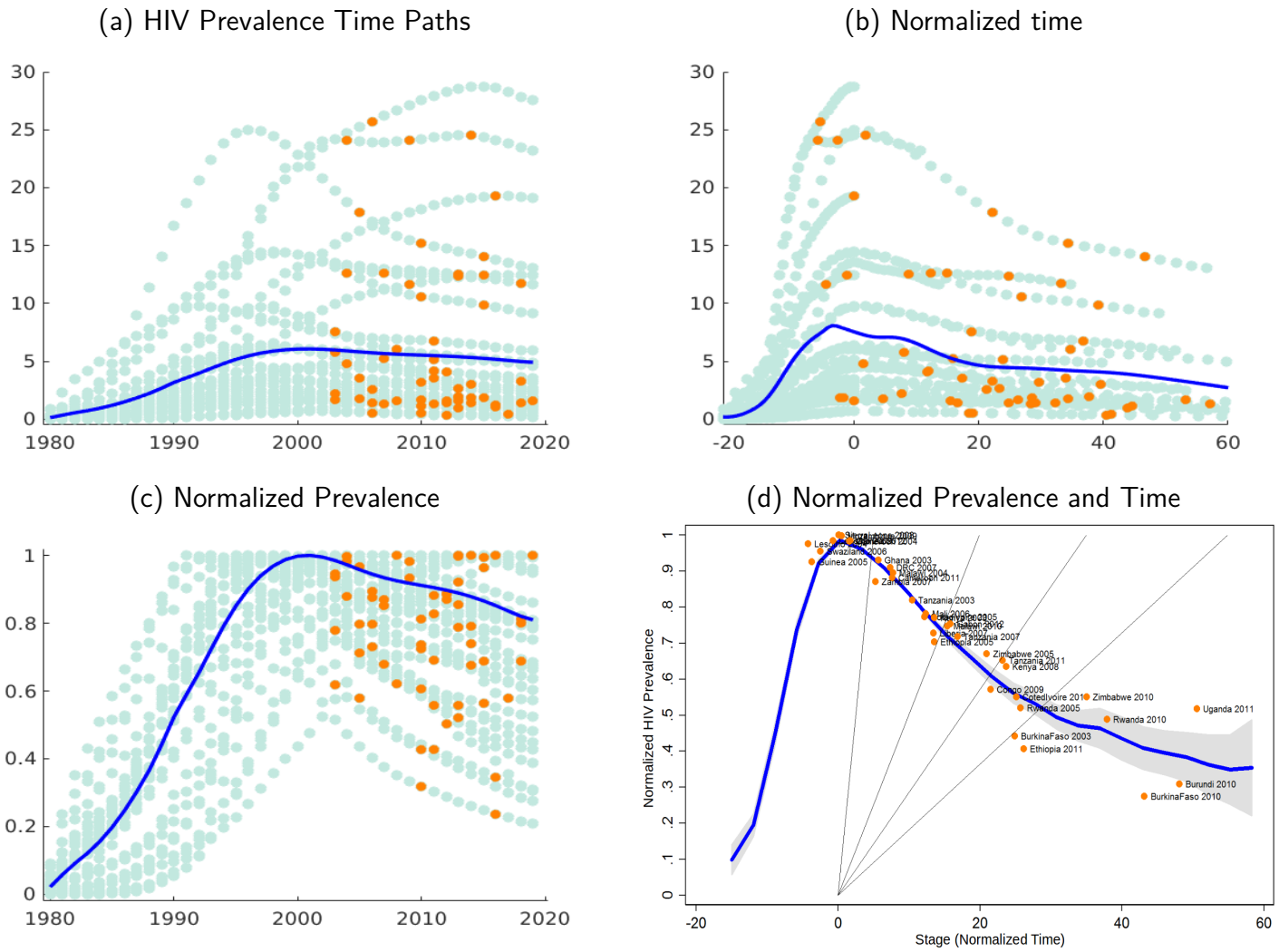
Although the stage is a continuous variable, for our estimation purposes it is useful to consider the discretization of the stages of the epidemic.[14] One way to do that is to consider some ad-hoc definition of the stages of the epidemic as the one provided by the CDC (see Figure 1), but these particular discretization ends up with rather wide intervals for stages in that all our available DHS datasets fall into two of the intervals defined by the CDC, either "peak" or "decceleration." Here, we pursue a thinner (more granular) discretization in order to characterize the potential nonstationary behavior of the HIV-education gradient by making use of the entire stage variation of our full sample of sixty DHS country-year observations.

To this end, note that on the normalized space Ω, each pair $(s, \zeta) \in \Omega$ is unique, thus we can define $\omega(s, \zeta) = \zeta/s \to \mathbb{R}$ which geometrically represents the slope of the arrays from the

[13] Splines are the most common finite element method of interpolation. We find our results are robust to the use of linear or quadratic pieces.

[14] 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 the stages or with a discretized version of them. We prefer to follow the latter approach and partition the continuous variable into the discrete pieces which delivers a cleaner interpretation of the estimates of the HIV-Education gradient across stages.

Figure 2: Normalization of Country-Specific Epidemic Time Paths: Stages of the Epidemic



Notes: We use our full sample of 30 SSA countries. 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. In each panel, the orange markers in the scatter plots represent a DHS country-year datset, whereas the light blue markers represent a UNAIDS country-year observation of prevalence rate. The solid blue is the reference path, a locally weighted polynomial averaging our sample of SSA countries (with 95% confidence intervals). In panel (d), the arrays from the origin discretize the stages of the epidemic as described in Section 2.2.

origin in the (S, Λ) space with the following limiting properties:

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

Definition 1. [A Discretization of the Stages of the HIV Epidemic] *Given a set of thresholds of the normalized prevalence rate* $\{\zeta_0, ..., \zeta_j, ..., \zeta_n\}$ *with* $\zeta_j < \zeta_{j+1}$ $\forall j \geq 0$, *the discrete stages of the HIV epidemic consist of the stages* s *such that the pairs* $(s, \zeta) \in \Omega$ *satisfy*

$\omega(\widetilde{g}^{-1}(\zeta_{j+1}), \zeta_{j+1}) \leq \omega(s, \zeta) < \omega(\widetilde{g}^{-1}(\zeta_j), \zeta_j)$, *where* $\widetilde{g}(s)$ *is the normalized reference path of the HIV epidemic.*

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 $\zeta_j = \zeta_0 - .05j \ \forall j \geq 0$. The discretization implies an assignment of DHS datasets to a set of five discrete stages of the epidemic with an average of twelve country-year DHS observations per stage; see panel (d) of Figure 2. The detailed groupings are in the Online Appendix B.

2.3 The HIV-education gradient

We pose a simple econometric specification suitable to document the non-stationary behavior of the HIV-Education gradient.

2.3.1 Econometric Specification

We consider a linear probability model (LPM) where the HIV-Education gradient is allowed to change over the discrete stages of the HIV epidemic (s) from the previous section:

$$y_{i,r,t,s} = \alpha_0 + \underbrace{\left(\gamma_0 + \sum_{s>0} \gamma_s \mathbf{1}_s\right) e_{i,r,t,s}}_{\text{education} \times \text{stages}} + \beta \, x_{i,r,t,s} + \psi \, m_{r,t} + \theta_r \mathbf{1}_r + \theta_t \mathbf{1}_t + \theta_s \mathbf{1}_s + \varepsilon_{i,r,t,s}, \quad (1)$$

For individual i, in country r, at time t and stage of the epidemic s. The outcomes $y_{i,r,t,s}$ of interest are the individual's HIV status, measures of risky sex behavior and knowledge about HIV transmission. $e_{i,r,t,s}$ denote the educational attainment. $\mathbf{1}_s$ is an indicator function equal to one when the stage of the HIV epidemic is s, zero otherwise. That is, if the stage of the epidemic is $s = 0$ then the intercept is α_0 and the slope is γ_0. However, if $s > 0$, the associated intercept is $(\alpha_0 + \theta_s)$ and the slope is $(\gamma_0 + \gamma_s)$. Namely, γ_s is the difference in the HIV-Education gradient between individuals that are in stage s and stage 0. Then, the HIV-Education gradient is $\gamma_0 + \gamma_s$ for each epidemiological stage s. The vector $x_{i,r,t,s}$ includes individual characteristics such as age, gender and residential area. The vector $m_{r,t}$, includes measures of output per capita and share of agricultural output to correct for the stage of economic development in which each country is in. We include country-fixed effects θ_r, time-fixed effects θ_t and stage-fixed effects θ_s. Our specifications are weighted least squares regressions, where the weights a combination of the relative population size of each country with the individual weights provided by the DHS surveys. We cluster the individual observations at the country level to account for any unobserved shock that correlates observations within a country.

2.3.2 Results

Consider a model where the dependent variable is the individual HIV status. Our benchmark results reported in Table 2, column 2. Figure 3 plots the benchmark estimates of the HIV-Education gradient ($\gamma_0 + \gamma_s$) across discrete stages of the epidemic.

From non-stationary benchmark specification (column 2, Table 2), 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. 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 $\gamma_0 + \gamma_1 = 0.53\%$, i.e., less than one-half of its value at Stage 0, and it is significantly different from zero at 1% level (see Online Appendix C). 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 (see Online Appendix C). 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. We conclude that the HIV-Education gradient exhibits a positive-zero-positive U-shape pattern over stages of the HIV epidemic.[15]

The Risky Sex-Education Gradient We study risky sexual behavior by considering as independent variables: (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,[16] and (ii) whether 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 and Condom-Education gradient are plotted in panel (b) of Figure 3; also reported the see Online Appendix C. The Partners-Education gradient first decreases (between aggregate stages 0 and 2) and then increases (between aggregate stages 2

[15] 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). Similar results are attained if we consider only the sexually active subsample (see Online Appendix C), 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. 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).

[16] For individuals who are single or do not cohabit, all sex partners are extramarital.

Table 2: The HIV-Education Gradient

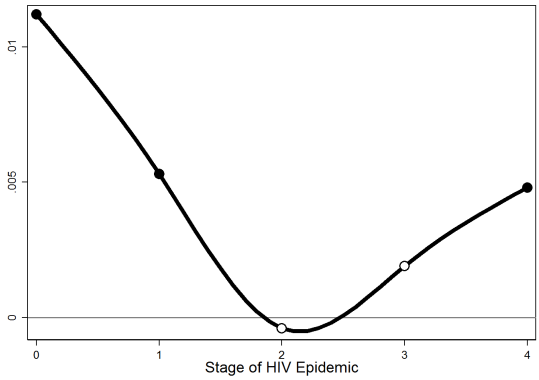
(A) *HIV Status*	(1)	(2)	(3)	(4)	(5)
Education	0.0043***	0.0112***	0.0098***	0.0040***	0.0037***
	(0.0006)	(0.0007)	(0.0010)	(0.0003)	(0.0003)
Education * Stage1		-0.0059***	-0.0046***	-0.0010***	-0.0008**
		(0.0008)	(0.0010)	(0.0003)	(0.0003)
Education * Stage2		-0.0116***	-0.0103***	-0.0027***	-0.0024***
		(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

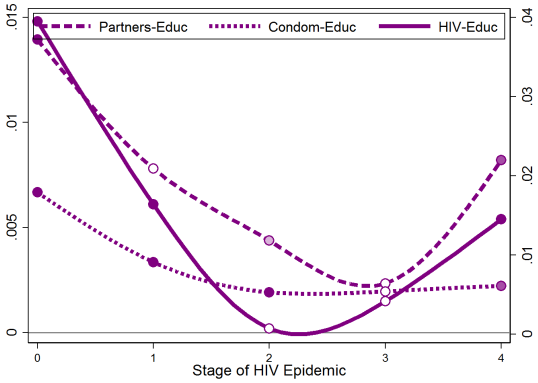
Notes: All specifications use the "Full Sample" described in Section 2 and the same set of controls. In Panel (A), Column (1) reports the results for the stationary specification, and columns (2) to (5) report the results for the non-stationary specification. We add year dummies in column (3), country 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%.

Figure 3: HIV-Education and Risky Sex-Education Gradients across Stages of the Epidemic

(a) HIV-Education Gradient



(b) Risky Sex-Education Gradient



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 (also reported in column 3, panel A, Table 6). 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.

and 4), 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, potentially explaining both its decline and rebound. Regarding, the Condom-Education gradient we do not find a significant pattern across stages. After Stage 0, the Condom-Education gradient remains positive but relatively constant across stages of the epidemic.

Knowledge about HIV Transmission Respondents knowledge about the transmission mechanisms of HIV is measured by asking if the chances of getting HIV reduce by (1) having only one sex partner, (2) always wearing a condom. We estimate the education gradients in these knowledge variables. Our results are in the Online Appendix C. We see that overall more-educated individuals understand better than the less educated that having fewer partners is a plausible prevention strategy against HIV transmission. In terms of knowledge about the usefulness of condoms, we find that more-educated are consistently better informed than the less-educated women (see Online Appendix C). The above evidence implies a positive effect of education on knowledge.

Antiretroviral Therapy (ART) Treatment is costly and prohibitive for a vast majority of SSA households.[17] In this context, if the more educated have more access to ART, then ART might

[17]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

help explain the rebound in the most advanced stages. The findings in the Online Appendix C point in this direction. We estimate the HIV-Education gradient with and without ART controls in panel A and B, respectively. When we include ART, the HIV-Education gradient in the most advanced stages decreases with respect to its 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.

3 The model

This is a perpetual youth economy with exogenous fertility rate f and endogenous mortality rates that depend on individual, HIV status and ARV treatment. There is a continuum of agents that are heterogeneous in: education level $e \in E$ which is permanent and exogenously given at the beginning of life; income shocks $\varepsilon \in \mathcal{E} = \{\varepsilon_1, ..., \varepsilon_n\}$, which follow a Markov process where $\pi(\varepsilon'|\varepsilon)$ denotes the probability of transiting from ε to ε';[18] sex type which is defined exogenously and permanently as either risky-sex consumers (i) or risky-sex producers $(-i)$ with $\{i, -i\} \in I$; HIV status $h \in H = \{-, +\}$, which is endogenously determined through risky-sex choices; and whether the agent is under antiretroviral treatment (ARVs) against HIV $d \in D = \{-, +\}$, which follows a Markov process if HIV infected.[19] Further, let Φ be the joint distribution of education, income shocks, sex type, hiv status and ARV treatment.

In this economy, HIV is endogenously transmitted through risky-sex choices: risky sex (either consumed or produced) has an attached positive probability of HIV infection. The endogenous risky sex choices determine HIV status the next period, and hence, mortality rates. Risky-sex consumers and producers meet in a centralized market to transact risky-sex services at the competitive price $p(\Phi)$. Our modeling choice, using a centralized market for risky sex, is a compromise between numerical tractability and our goal to explore the asymmetric learning mechanism about the odds of infection that we put forward in a highly nonstationary framework.[20]

In particular, the aggregate economy is nonstationary, evolving across four stages of the HIV epidemic, with $s \in \mathcal{S} = \{0, 1, 2, 3\}$. In Stage 0, the economy exists in a stationary pre-HIV

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.

[18] We denote with primes the one-period-ahead variables, i.e. $\varepsilon = \varepsilon_t$ and $\varepsilon' = \varepsilon_{t+1}$ for period t.

[19] We focus on endogenizing the intensive margin of sex, excluding the endogenous decision of who becomes a sex consumer or producer. Introducing significant endogenous heterogeneity within each group we find that some sex consumers engage in negligible transactions, and some sex producers generate negligible amounts, resembling corner solutions. The size of this population, exhibiting small sex transactions, is endogenous.

[20] Alternatively, the clearing could occur in a decentralized matching framework as in Greenwood et al. (2019).

epidemic era without HIV. In Stage 1, the HIV epidemic begins, but agents are myopic, unaware of the epidemic. In Stage 2, agents begin to learn the sexual nature of the HIV infection process, and the speed of learning differs by education group. Finally, in Stage 3, antiretroviral drugs (ARVs) are introduced. The economy transitions over time through these four stages of the HIV epidemic via a sequence of unexpected shocks: the pre-HIV era (Stage 0) concludes with the exogenous arrival of patient zero, marking the first period of the myopic stage (Stage 1); the myopic stage concludes when agents begin to recognize the epidemic and start learning about the infection process, defining the first period of the learning stage (Stage 2); and the exogenous arrival of ARV treatment marks the beginning of the ARV stage (Stage 3), during which agents continue learning.

Let us cast the household problem recursively separately by stage of the epidemic from Sections 3.1 to 3.4. We define the model equilibrium for the entire economy across all the stages of the HIV epidemic in Section 3.5 and provide a pseudo solution algorithm in Section 3.6

3.1 The Pre-HIV Era [Stage 0]

In the pre-HIV era, there is no HIV. During this stage, at any given period t, agents with education level, e, and income shock s, solve the following problem depending on their sex type, i.

Risky-sex consumers problem. Risky-sex consumers choose consumption $c \in \mathbb{R}^+$ and the amount of risky sex $x \in \mathbb{R}^+$ to solve the following dynamic program:

$$V(e,i,\varepsilon,\Phi) = \max_{c\geq 0, x\geq 0} \chi u(c,x;\omega) + \beta\gamma \sum_{\varepsilon'|\varepsilon} \pi(\varepsilon'|\varepsilon) V(e,i,\varepsilon',\Phi') \tag{2}$$

subject to

$$c + p(\Phi)x = zy(e)\varepsilon \tag{3}$$

where loss of utility due to death ω (an isolated constant) and the discount factor β are common across all agents and stages. The derived utility flow from c and x is captured through the continuous, concave and differentiable instantaneous felicity function $u(c,x)$. We choose consumption as numeraire and the relative price of risky sex is denoted by $p(\Phi)$. Prices are determined in competitive equilibrium in the sex market, and agents are price takers. Market income is the product of a permanent component $y(e)$ that depends on the level of education and an income shock ε that follows a Markov process with given transition matrix $\pi(\varepsilon'|\varepsilon)$. We separately highlight three parameters that at later stages can depend on HIV status: the preference shifter parameter χ,

the survival rate γ and a permanent income component z. Accordingly, in the pre-HIV era we set χ, γ and z equal across all agents, including both risky-sex consumers and producers.[21]

Plugging the isolated consumption from the budget constraint (3) into the objective function (2), agents face the following intratemporal condition:

$$FOC(x): \qquad \underbrace{\chi u_c(c,x;\omega)(-p(\Phi))}_{MC(x)} + \underbrace{\chi u_x(c,x;\omega)}_{MB(x)} = 0, \tag{4}$$

weighing the benefit of enjoying higher risky-sex against the cost of consuming less goods.

Risky-sex producers problem. Sex-producer households choose consumption $c \in \mathbb{R}^+$ and the fraction of time devoted to sex production $l \in [0,1]$ to solve the problem:

$$V(e,-i,\varepsilon,\Phi) = \max_{c\geq 0, 1\geq l\geq 0} \chi u(c;\omega) + \beta\gamma \sum_{\varepsilon'|\varepsilon} \pi(\varepsilon'|\varepsilon) V(e,-i,\varepsilon',\Phi') \tag{5}$$

subject to

$$c = z[p(\Phi) l^{\alpha} + y(e)\varepsilon(1-l)]. \tag{6}$$

Sex-producers derive utility from the consumption good, but not from risky sex. One unit of labor is inelastically supplied and l denotes the fraction of labor allocated to the production of risky sex and the remaining labor $(1-l)$ is allocated to the production of goods. The production of sex uses time l as input following a decreasing returns technology $x = l^{\alpha}$ with $\alpha \in (0,1)$, and is valued at the competitive price $p(\Phi)$. The decreasing returns bound risky sex production.

Plugging the isolated consumption from the budget constraint (6) into the objective function (5), agents face the following intratemporal condition:

$$FOC(l): \qquad \chi u_c(c;\omega) z \left(\underbrace{p(\Phi)\alpha l^{\alpha-1}}_{MB(l)} - \underbrace{y(e)\varepsilon}_{MC(l)} \right) = 0 \tag{7}$$

weighing the time allocated to production of sex and goods by equating the marginal (revenue) product across technologies. For a given price $p(\Phi)$, the marginal product of sex is decreasing in l, whereas that of the goods is linear, which allows for interior solutions for suitable prices.

[21] In particular, χ and z are equal to one, and γ is the survival rate before the pre-HIV era.

At any point in time in the pre-HIV era, the economy is summarized by the joint distribution Φ of individual states (e, i, ε). The aggregate state variable evolves according to:

$$\Phi' = H(\Phi), \tag{8}$$

where the function $H : \mathcal{M} \to \mathcal{M}$ is the aggregate law of motion, mapping distributions to distributions. That is, H summarizes how the joint distribution of individual states evolves from one period to the next.[22]

[Stage 0 Equilibrium] Definition of the Pre-HIV *Stationary* Recursive Competitive Equilibrium

The Pre-HIV *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 $c(e, i, \varepsilon)$ and $x(e, i, \varepsilon)$ solve the risky-sex consumers problem (2)-(3) and $c(e, -i, \varepsilon)$ and $l(e, -i, \varepsilon)$ solve the risky-sex producers problem (5)-(6).

2. The riksy-sex markets clear,

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

and the consumption market clears by Walras law.

3. The stationary probability distribution,

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

is induced by the equilibrium policy functions.

[22] Precisely, define the transition function $\mathcal{Q} : \mathcal{Z} \times \mathcal{B}(\mathcal{Z}) \to [0, 1]$ by:

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

where $\mathcal{Z}$ consists of all n-tuples of $E \times I \times \mathcal{E}$ and $\mathcal{B}(\mathcal{Z})$ is the set of Borel sets on $\mathcal{Z}$, in particular $(E, I, \mathcal{E}) \in \mathcal{B}(\mathcal{Z})$. Let $\mathcal{P}$ be a probability measure on $\mathcal{B}(\mathcal{Z})$, then $\mathcal{P} : \mathcal{B}(\mathcal{Z}) \to [0, 1]$. Then the evolution of the population distribution is,

$$\Phi'(E, I, \mathcal{E}) = F(\Phi)(E, I, \mathcal{E}) = \sum_{e,i,s} Q((e, i, s)(E, I, \mathcal{E}) + f\Phi((e, i, s')(E, I, \mathcal{E})), \tag{9}$$

which is the fraction of people with education E, sex type I and income shocks in $\mathcal{E}$ as measured by Φ, that transit to $(E, I, \mathcal{E})$ as measured by $\mathcal{Q}$. The last term in equation (9) accounts for the new born. Population of each group increases according to respective fertility rate f.

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

3.2 The Myopic Onset of the HIV Epidemic [Stage 1]

The HIV epidemics starts in this stage as an unexpected shock to the pre-HIV era at some period T_0+1, where T_0 reflects the last period of the pre-HIV era. From then on, agents can be healthy or HIV infected $h \in \mathcal{H} = \{-,+\}$. We assume agents at this stage are myopic about the epidemic. In particular, agents are unaware of the fact that HIV infection is occurring through the amount of risky sex x (either consumed or produced). Specifically, the unconditional probability that an HIV negative agent is infected with HIV next period is given by the transmission function:

$$\lambda(x,\rho,\phi^+) = \underbrace{\phi^+}_{\text{Meeting Prob.}} \quad \underbrace{\frac{e^x}{e^x+\rho e^{-x}}}_{\text{Conditional Transmission}}, \tag{10}$$

which depends on the probability of meeting an HIV infected individual in the sex market, i.e. the current prevalence rate $\phi^+ \in [0,1]$, the amount of individual risky sex x that is endogenously chosen, and a parameter $\rho \in [0,\infty)$ that governs the mapping from the amount of individual risky sex to the probability of HIV infection.[23] The lower is ρ the higher is the probability of HIV infection per amount of sex transactioned; see Figure 4.[24,25]

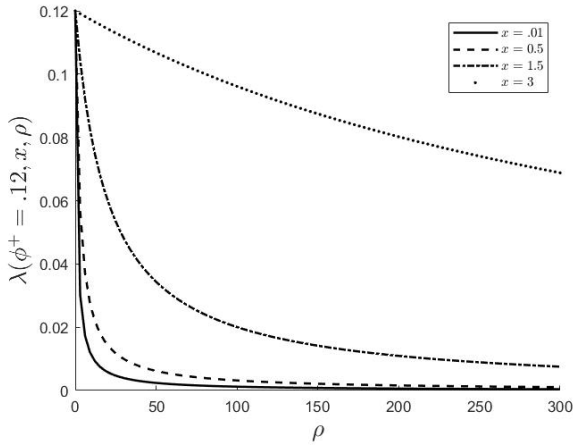
Although agents are unaware of the nature of HIV infection (10), at every period, agents observe higher average mortality rates (γ), lower average labor market and sex production productivity (z) and a lower overall ability to enjoy utility flows (χ). Since agents do no know that these changes in γ, z and χ 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 γ, z and χ. That is, at every period, agents notice a change between γ_t and γ_{t-1},

[23] The conditional component in (10) is analogous to that in Bethencourt and Ríos-Rull (2009) modeling effort.

[24] Note that we need to initiate the epidemic with some $\phi^+_{t=0} > 0$. 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 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.

[25] Alternatively instead of the meeting probability, we have the density (simply the total number) of individuals that are HIV positive which brings a different dimension for cross-country (or space) heterogeneity.

Figure 4: Objective Probability of Infection, $\lambda(x, \rho, \phi^+)$



Notes: Here we plot $\lambda(x, \rho, \phi^+)$, i.e. the probability that an HIV negative agent is infected with HIV next period as a function of the amount of individual risky sex x, the probability of meeting and HIV+ individual in the sex market, and a parameter ρ that governs the mapping from the amount of individual risky sex to the probability of HIV infection as described in equation (10). For this illustration we set the HIV prevalence rate to $\phi^+ = 1.2$.

between z_t and z_{t-1}, between χ_t and χ_{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, productivity and felicity depend on the actual distribution of HIV status across the population which is endogenous to risky sex. In particular, the true distribution of the population evolves according to

$$\begin{bmatrix} \phi^-_{t+1} \\ \phi^+_{t+1} \end{bmatrix} = \underbrace{\begin{bmatrix} \gamma(-) & 0 \\ 0 & \gamma(+) \end{bmatrix}}_{\text{Survival Matrix}} \underbrace{\begin{bmatrix} 1-\lambda(x_t, \rho, \phi^+_t) & 0 \\ \lambda(x_t, \rho, \phi^+_t) & 1 \end{bmatrix}}_{\text{Infection Matrix}} \underbrace{\begin{bmatrix} 1+f & f \\ 0 & 1 \end{bmatrix}}_{\text{Fertility Matrix}} \begin{bmatrix} \phi^-_t \\ \phi^+_t \end{bmatrix} \tag{11}$$

where ϕ^+_t and ϕ^-_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 $\lambda(x_t, \rho, \phi^+_t)$ are defined by (10), and f is the fertility rate for the aggregate economy, which is independent of HIV status and all newborns are not infected with HIV.[26] Then, note that we can develop (11) to find that tomorrow's aggregate population is:

$$\phi_{t+1} = \phi^-_{t+1} + \phi^+_{t+1} \tag{12}$$

where the measure of HIV negative population is $\phi^-_{t+1} = \gamma(-)(1-\lambda(x_t, \rho, \phi^+_t))[(1+f)\phi^-_t + f\phi^+_t]$ and the measure of HIV positive population is $\phi^+_{t+1} = \gamma(+)\left(\lambda(x_t, \rho, \phi^+_t)[(1+f)\phi^-_t + f\phi^+_t] + \phi^+_t\right)$.

[26] That is, in our benchmark model, we assume away mother-to-child transmission which represents less than 5% of total HIV infections in Sub-Saharan Africa. This can be easily modified by allowing some HIV positive agents to bear HIV positive children replacing the top right element of the fertility matrix in (11) with $(1+fm)$ and the bottom right element with fm where m indicates the proportion of newborn that are HIV $+$.

In this manner, the actual evolution of the population in (11) defines the mortality rates

$$\widetilde{\gamma}_t = \frac{\phi_{t+1}}{(1+f)\phi_t}. \tag{13}$$

Our agents take the time changes in $\widetilde{\gamma}_t$ as unexpected permanent shocks to mortality during this myopic stage of the epidemic. Further, in the same vein, agents myopically update every period their idiosyncratic productivity and ability to enjoy utility, respectively, $\widetilde{z}_t$ and $\widetilde{\chi}_t$. These updates actually follow idiosyncratic changes in HIV status, i.e.

$$\widetilde{z}_t = z(h) \;\text{ and }\; \widetilde{\chi}_t = \chi(h), \tag{14}$$

Again, our agents are unaware of the rationale behind the changes in $\widetilde{z}_t$ and $\widetilde{\chi}_t$—that is, they are not aware that their HIV status has changed—and they take the new productivity and preference shifters as unexpected permanent shocks. Given the myopic updates for γ, z and χ in (13) and (14), we are now ready to formulate the risky-sex consumer and producer problems in this stage. Importantly, since $\widetilde{\gamma}_t$, $\widetilde{z}_t$ and $\widetilde{\chi}_t$ get actually updated every period, the formulation of the households problem and the definition of equilibrium for this myopic stage needs to reflect this phenomenon which will take the form of unexpected permanent shocks: $(\widetilde{\gamma}, \widetilde{z}, \widetilde{\chi})$.

Risky-sex consumer problem. At every period t, our looking forward risky-sex consumers choose c_t and x_t taking $(\widetilde{\gamma}, \widetilde{z}, \widetilde{\chi})$ as permanently given to solve:

$$V_t(e,i,\varepsilon,\Phi) = \max_{c_t\geq 0, x_t \geq 0} \widetilde{\chi} u(c_t, x_t; \omega) + \beta \widetilde{\gamma} \sum_{\varepsilon'|\varepsilon} \pi(\varepsilon'|\varepsilon) V_{t+1}(e,i,\varepsilon',\Phi') \tag{15}$$

subject to

$$c_t + p_t(\Phi) x_t = \widetilde{z} y(e) \varepsilon \tag{16}$$

Then, plugging the isolated consumption from the budget constraint (16) into the objective function (15), agents face an intratemporal condition for x identical to that of (4). This highlights the main characteristic of the myopic stage by which agents do not internalize the evolution of the HIV (11) unaware that their sexual behavior affects their chances of survival γ, their future productivity z and their future ability to enjoy utility flows χ.

Risky sex-producer problem. At every period t, our looking forward risky-sex producers choose c_t and l_t taking $(\widetilde{\gamma}, \widetilde{z}, \widetilde{\chi})$ as permanently given to solve:

$$V_t(e, -i, \varepsilon, \Phi) = \max_{c_t \geq 0, 1 \geq l_t \geq 0} \widetilde{\chi} u(c_t; \omega) + \beta \widetilde{\gamma} \sum_{\varepsilon'|\varepsilon} \pi(\varepsilon'|\varepsilon) V_{t+1}(e, -i, \varepsilon', \Phi') \tag{17}$$

subject to

$$c_t = \widetilde{z} \left[(p_t(\Phi) l_t^{\alpha} + y(e)\varepsilon(1 - l_t)) \right] \tag{18}$$

Then, agents face an intratemporal condition for l identical that of (7).

For both the risky-sex consumer and producer problems, the value functions, policy functions, and the price function are indexed by t. The sequential formulation of the recursive problem is necessary to address unexpected changes in survival rates γ, productivity shifter z, and preference shifter χ. Specifically, in each period, agents myopically observe an unexpected change in $\widetilde{\gamma}, \widetilde{z}$, and $\widetilde{\chi}$, which follows the updating formulas in (13) and (14). Agents are myopic in that they assume that these unexpected changes in the environment are permanent. That is, agents assume that the survival rates, productivity shifter, and preference shifter updated at period $\tau = t$ will remain constant in future periods: $\widetilde{\gamma}_\tau = \widetilde{\gamma}\ \forall \tau > t$, $\widetilde{z}_\tau = \widetilde{z}\ \forall \tau > t$ and $\widetilde{\chi}_\tau = \widetilde{\chi}\ \forall \tau > t$.

[Stage 1 Equilibrium] Definition of the Myopic *Nonstationary* Recursive Competitive Equilibrium

Given a Pre-HIV *stationary* joint distribution, $\Phi_{T_0}(s=0)$ (i.e. $\Phi(s=0)$ for all $t < T_0$), and a sequence of unexpected changes in mortality rates $\{\widetilde{\gamma}_t\}_{t\geq T_0}^{\infty}$, productivity shifters $\{\widetilde{z}_t\}_{t\geq T_0}^{\infty}$ and preference shifters $\{\widetilde{\chi}_t\}_{t\geq T_0}^{\infty}$, constructed from the updates in (13) and (14), myopically assuming that the changes occuring at $\tau = t$ are permenant, i.e. $\widetilde{\gamma}_\tau = \widetilde{\gamma}_t = \widetilde{\gamma}$, $\widetilde{z}_\tau = \widetilde{z}_t = \widetilde{z}$ and $\widetilde{\chi}_\tau = \widetilde{\chi}_t = \widetilde{\chi}$ for all $\tau > t \geq T_0$, a *nonstationary* RCE is a sequence of individual household functions $\{V_\tau, c_\tau, x_\tau, l_\tau : Z \times M \to M\}_{\tau\geq t\geq T_0}^{\infty}$, prices $\{p_\tau\}_{\tau\geq t\geq T_0}^{\infty}$, and measures $\{\Phi_\tau\}_{\tau\geq t\geq T_0}^{\infty}$ such that, $\forall \tau \geq t \geq T_0$:

1. Given p, the policy functions $c_\tau(e,i,\varepsilon)$ and $x_\tau(e,i,\varepsilon)$ for risky-sex consumers and $c_\tau(e,i,\varepsilon)$ and $l_\tau(e,i,\varepsilon)$ for risky-sex producers solve their respective problems (15)-(16) and (17)-(18).

2. All markets clear.

$$\sum_{e,i,\varepsilon} x_\tau(e,i,\varepsilon) = \sum_{e,-i,\varepsilon} x_\tau(e,-i,\varepsilon),$$

   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 Φ is the joint distribution of (e,i,ε) 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 (11).

Remark Since at each period $\tau = t$, a new triplet $(\widetilde{\gamma}, \widetilde{z}, \widetilde{\chi})$ emerges, agents solve for an entirely new transition (from $\tau = t$ onward) toward the new steady state associated to the updated triplet $(\widetilde{\gamma}, \widetilde{z}, \widetilde{\chi})$. Within the myopic stage, the equilibrium path collects the value functions, policy functions, and the price function associated solely with the current period in which a new triplet $(\widetilde{\gamma}, \widetilde{z}, \widetilde{\chi})$ emerges, i.e. $\tau = t$. Note that this still requires solving for the entire transition at each period t after the permanent shock occurs.

Finally, the sequence of myopic updates does not run *ad infinitum* because at some period T_1 (last period of the myopic stage), the economy unexpectedly moves to a new stage of the epidemic where agents initiate the learning about the sexual nature of the mode of HIV infection.

3.3 Learning the HIV Epidemic [Stage 2]

Agents become unexpectedly aware of their own HIV status and that of the rest of the population entering this stage at period $T_1 + 1$, where T_1 represents the last period of the myopic stage.[27] However, agents are not fully aware about the the process of HIV infection. Specifically, agents know that risky sex translates into HIV infections (following (10)), but they are not fully aware of that mapping insofar they do not accurately know the true value of ρ in (10) and, hence, they do not know how risky sex actually maps onto HIV infections.

We allow for agents to learn about ρ through Bayesian updates with some noise. We allow for the speed in which agents learn about the actual odds of infection to potentially differ across education groups. Specifically, each educational group $e \in E$ has a prior belief about the distribution of $\lambda(x, \rho)$, denoted by the probability density function $\mathcal{P}_e(\lambda(x, \rho))$. We assume that the initial accuracy in which individuals know the odds of infection is the same across education groups, $\mathcal{P}_e(\lambda(x, \tilde{\rho}_o)) \sim N(\lambda(x, \tilde{\rho}_o), \sigma^2_\varepsilon)$ with $\mathcal{P}_{e=1}(\lambda(x, \tilde{\rho}_o)) = \mathcal{P}_{e=0}(\lambda(x, \tilde{\rho}_o))$, which follows from the fact that both groups were completely unaware of ρ in the previous stage. From then onward, agents receive a signal $\lambda(x, \tilde{\rho})$ per period. This signal contains information about the actual probability of infection plus some noise ϵ_t that is normally distributed with zero mean and a variance $\sigma^2_\epsilon(e)$ that depends on education. Explicitly:

$$\lambda(x, \tilde{\rho}) = \lambda(x, \rho) + \epsilon, \tag{19}$$

where the signal follows the following covariance stationary process:

$$\epsilon_t = v_t + \mathbf{1}_{e=0} u_t \tag{20}$$

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, if $\sigma^2_u = 0$, then the signal is equally noisy across education groups. Whereas if $\sigma^2_u > 0$ then the signal is noisier for the less educated group than for the more educated group.

[27] We assume agents are able to know their own HIV status and others', as in Greenwood et al. (2019).

Every period t agents update their beliefs $\mathcal{P}(\lambda(x,\rho(e))$ given the information up to $t-1$ according to Bayes rule:

$$\mathcal{P}(\lambda(x,\rho(e))) = \mathcal{P}(\lambda(x,\rho)|\lambda(x,\widetilde{\rho}(e))) = \frac{\mathcal{P}(\lambda(x,\widetilde{\rho}(e))|\lambda(x,\rho))\mathcal{P}(\lambda(x,\rho))}{\mathcal{P}(\lambda(x,\widetilde{\rho}(e)))} \tag{21}$$

where the Bayesian updates will converge faster to the actual odds of HIV infection for the more educated individuals if $\sigma_u^2 > 0$.[28]

In particular, the believed transition probabilities to a new HIV status h' given the amount of risky sex x, the believed $\widetilde{\rho}$, the prevalence rate ϕ^+, and the current HIV status h is,

$$q(h'|x_t,\widetilde{\rho}(e),\phi^+,h) = \begin{pmatrix} q(+|x_t,\widetilde{\rho}(e),\phi^+,-) = \lambda(x,\widetilde{\rho}(e),\phi^+) \\ q(-|x_t,\widetilde{\rho}(e),\phi^+,-) = 1-\lambda(x,\widetilde{\rho}(e),\phi^+) \\ q(+|x_t,\widetilde{\rho}(e),\phi^+,+) = 1 \\ q(-|x_t,\widetilde{\rho}(e),\phi^+,+) = 0 \end{pmatrix} \tag{22}$$

That is, the perceived probability of getting infected with HIV is $\lambda(x,\widetilde{\rho}(e),\phi^+)$, which differs by education groups. We further assume that once infected, always infected ($q(+|.,+) = 1$) and that there is no ability to revert a positive HIV status ($q(-|.,+) = 0$).[29]. Finally, although individuals make their economic decisions based on their believed probabilities of infection (22), the true evolution of the distribution of the population is given by (11)—as in the myopic stage.

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

$$\begin{aligned} V_t(e,i,\varepsilon,h,\Phi) = \max_{c_t\geq 0, x_t\geq 0} & \chi(h)u(c_t,x_t;\omega) \\ & + \beta \sum_{h'|h,\varepsilon'|\varepsilon} [\gamma(h')q(h'|x_t,\widetilde{\rho}(e),\phi^+,h)\pi(\varepsilon'|\varepsilon)V_{t+1}(e,i,\varepsilon',h',\Phi')\ ] \end{aligned} \tag{23}$$

subject to,

$$c_t + p_t(\Phi)x_t = z(h)y(e)\varepsilon \tag{24}$$

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

[29] We relax this assumptions later on in our quantitative experiments in Section 5

Then, consumers choose risky sex according to (assuming HIV negative status):

$$FOC(x): \quad \underbrace{\chi(h)u_c(c_t,x_t;\omega)(-p_t(\Phi))}_{MC(x)}+\underbrace{\chi(h)u_x(c_t,x_t;\omega)}_{MB(x)} + \underbrace{\beta\sum_{s'|s}\pi(s'|s)\lambda_x(x_t,\widetilde{\rho}(e),\phi^+,-)(\gamma(+)V^+_{t+1}-\gamma(-)V^-_{t+1})}_{MC'(x)}=0 \tag{25}$$

where the intratemporal marginal benefits of risky sex (higher utility flow from these services) and costs (lower utility flow fom lower consumption of goods) are analogous to those in previous stages of the epidemic, but now HIV negative consumers take into account that higher risky-sex further implies higher odds of getting infected ($\lambda_x(.)>0$) by as much as their beliefs of $\widetilde{\rho}$ state, and hence less chances of survival to the next period. This is reflected in an additional intertemporal (future) cost of consuming risky-sex today captured by the last term in (25) where note that we have explicitly substituted out $q(h'|x_t,\widetilde{\rho}(e),\phi^+,h)$ following (22). V^+_{t+1} and V^-_{t+1} are the agent's future value functions associated with being, respectively, HIV positive tomorrow or HIV negative tomorrow. Note that for agents that are currently HIV positive, the intertemporal term drops from (25) as their sexual behavior does not further impact survival rates.

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

$$V_t(e,-i,\varepsilon,h,\Phi)=\max_{c_t\geq 0,1\geq l_t\geq 0}\chi(h)u(c_t;\omega) + \beta\sum_{h'|h,\varepsilon'|\varepsilon,d'}[\gamma(h')q(h'|x_t,\widetilde{\rho}(e),\phi^+,h)\pi(\varepsilon'|\varepsilon)V_{t+1}(e,-i,\varepsilon',h',\Phi')\,] \tag{26}$$

subject to,

$$c_t=z(h)[p_t(\Phi)l_t^{\alpha}+y(e)\varepsilon(1-l_t)], \tag{27}$$

Then, producers choose across production technologies as follows,

$$FOC(l): \quad \chi(h)u_c(c_t;\omega)\left(\underbrace{p_t(\Phi)\alpha z(h)l_t^{\alpha-1}}_{MB(l)}-\underbrace{y(e)s}_{MC(l)}\right) + \underbrace{\beta\sum_{s'|s}\pi(s'|s)\lambda_l(x_t,\widetilde{\rho}(e),\phi^+,-)(\gamma(+)V^+_{t+1}-\gamma(-)V^-_{t+1})}_{MC'(l)}=0 \tag{28}$$

where $\lambda_l = \lambda_x \frac{\partial x}{\partial l}$. The intratemporal tradeoff between producing risky sex versus consumption goods is analogous to that in previous stages of the epidemic, but now HIV negative producers take into account that the production of risky-sex implies higher odds of getting infected (note that $\lambda_l(.) > 0$ with $x = l^\alpha$) and hence less chances of survival to the next period. This is reflected in an additional intertemporal (future) cost of producing risky-sex today captured by the last term in (28).

[Stage 2 Equilibrium] Definition of the Learning *Nonstationary* Recursive Competitive Equilibrium

Given a Stage 1 joint distribution $\Phi_{T_1}(s = 1)$, and a sequence of beliefs $\{\widetilde{\rho}(e)\}_{t \geq T_1}^{\infty}$ by education group, a *nonstastionary* RCE is a sequence of individual household functions $\{V_t, c_t, x_t, l_t : Z \times M \to M\}_{t \geq T_1}^{\infty}$, prices $\{p_t\}_{t \geq T_1}^{\infty}$, and measures $\{\Phi_t\}_{t \geq T_1}^{\infty}$ such that, $\forall t \geq T_1$:

1. Given p the policy functions $c_t(e, i, \varepsilon, h)$ and $x_t(e, i, \varepsilon, h)$ solve the risky sex-consumer problem (23)-(24) and $c_t(e, -i, \varepsilon, h)$ and $l_t(e, -i, \varepsilon, h)$ solve the risky sex-producer problem (26)-(27).

2. All markets clear.

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

   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 Φ is the joint distribution of (e, i, ε, h) is induced by the equilibrium policy functions.

4. The true distribution of the HIV population endogenously evolves according to (11).

5. The beliefs of on the odds of infection by education group evolve according to Bayes rule (21) and signals arrive according to (19) and (20).

Remark. The stationary Stage 2 equilibrium is the limiting case of the nonstationary RCE defined above in which beliefs of both education groups have converged to the actual odds of

infection and the cross-sectional distribution Φ does not change over time. In that case, we can drop all time subscripts.

3.4 The Era of ARVs [Stage 3]

ARV drugs arrive unexpectedly to the economy at period $T_2 + 1$, where T_2 represents the last period of the prior stage (learning without ARVs). In this stage, agents keep learning about the process of infection $\lambda(x, \widetilde{\rho}(e))$ according to (21).

ARV 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.[30] This means that ARV treatment is a new state variable $d \in D = \{+, -\}$ that keeps track of the portion of the infected population receiving treatment $(+)$ versus not $(-)$. The aggregate proportion of the population that receives drugs at each t is deterministic and represented by the monotonically increasing sequence $\{\eta_t\}_{t=0}^{\infty}$, where $0 \leq \eta_t \leq \tilde{\eta}\ \forall t$ with $\lim_{t\to\infty} \eta_t = \tilde{\eta}$ and $\tilde{\eta} \in (0, 1]$. Further, we assume that ARV drugs partly (or entirely) revert the negative effects of HIV on mortality and productivity. Only those who are infected can receive treatment.[31] In addition, ARVs 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 ρ with respect to the coverage rate. Let $\phi_t^{h,d}$ be the measure of the population with HIV status h and ARV treatment status d. Then the true distribution of the population evolves as follows:

$$\begin{bmatrix} \phi_{t+1}^{-,-} \\ \phi_{t+1}^{+,+} \\ \phi_{t+1}^{+,-} \end{bmatrix} = \underbrace{\begin{bmatrix} \gamma(-,-) & 0 & 0 \\ 0 & \gamma(+,+) & 0 \\ 0 & 0 & \gamma(+,-) \end{bmatrix}}_{\text{Survival Matrix}} \underbrace{\begin{bmatrix} 1-\lambda(x_t,\rho(e),\phi_t^+) & 0 & 0 \\ \eta_t(e)\lambda(x_t,\rho(e),\phi_t^+) & \eta_t(e) & \eta_t(e) \\ (1-\eta_t(e))\lambda(x_t,\rho(e),\phi_t^+) & 1-\eta_t(e) & 1-\eta_t(e) \end{bmatrix}}_{\text{Infection and Treatment Matrix}} \underbrace{\begin{bmatrix} 1+f & f & f \\ 0 & 1 & 0 \\ 0 & 0 & 1 \end{bmatrix}}_{\text{Fertility Matrix}} \begin{bmatrix} \phi_t^{-,-} \\ \phi_t^{+,+} \\ \phi_t^{+,-} \end{bmatrix} \tag{29}$$

where $\gamma(h, d)$ denotes the survival rate for individuals with HIV status h and ARV treatment status d. Then, (29) replaces the law of motion of the population in prior stages (11). Let us now write the nonstationary recursive problem and then explain it.

[30] 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 η_t is the aggregate coverage rate observed in the data at t, with ι controlling the odds of treatment with respect to the aggregate by education group.

[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.

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

$$V_t(e,i,\varepsilon,h,d,\Phi) = \max_{c_t\geq 0, x_t\geq 0} \chi(h,d)u(c_t,x_t;\omega) + \beta \sum_{h'|h,\ \varepsilon'|\varepsilon,\ d'} [\gamma(h',d')\eta_t(d'|e)q(h'|x_t,\tilde{\rho}(e),\phi^+,h)\pi(\varepsilon'|\varepsilon)V_{t+1}(e,i,\varepsilon',h',d',\Phi')\ ] \tag{30}$$

subject to,

$$c_t + p_t(\Phi)x_t = z(h,d)y(e)\varepsilon \tag{31}$$

Then, consumers choose risky sex according to (assuming HIV negative status):

$$FOC(x): \underbrace{\chi(h,d)u_c(c_t,x_t;\omega)(-p_t(\Phi))}_{MC(x)} + \underbrace{\chi(h,d)u_x(c_t,x_t;\omega)}_{MB(x)} + \underbrace{\beta \sum_{h'|h,\varepsilon'|\varepsilon,d'} \pi(\varepsilon'|\varepsilon)\lambda_x(x_t,\tilde{\rho}(e),\phi^+,-)(\gamma(+,-)(1-\eta(+|e))V_{t+1}^+ + \gamma(+,+)\eta(+|e)V_{t+1}^+ - \gamma(-)V_{t+1}^-)}_{MC'(x)}$$

Thus, the intertemporal margin here implies that risky-sex consumers take into account that risky sex affects their HIV infection and hence future mortality rates and, at the same time, take into account that there is a probability of ARV treatment that partly reverts the effects of HIV.

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

$$V_t(e,-i,\varepsilon,h,d,\Phi) = \max_{c_t\geq 0, 0\leq l_t\leq 1} \chi(h,d)u(c_t;\omega) + \beta \sum_{h'|h,\ \varepsilon'|\varepsilon} [\gamma(h',d')\eta_t(d'|e)q(h'|x_t,\tilde{\rho}(e),\phi^+,h)\pi(\varepsilon'|\varepsilon)V_{t+1}(e,-i,\varepsilon',h',d',\Phi')\ ] \tag{32}$$

subject to,

$$c_t = z(h,d)[p_t(\Phi)l_t^\alpha + y(e)\varepsilon(1-l_t)], \tag{33}$$

Then, producers choose risky sex according to (assuming HIV negative status):

$$FOC(l): \chi(h,d)u_c(c_t;\omega)\left(\underbrace{p_t(\Phi)\alpha z(h)l_t^{\alpha-1}}_{MB(l)} - \underbrace{y(e)s}_{MC(l)}\right) + \underbrace{\beta \sum_{h'|h,\varepsilon'|\varepsilon,d'} \pi(\varepsilon'|\varepsilon)\lambda_l(x_t,\tilde{\rho}(e),\phi^+,-)(\gamma(+,-)(1-\eta(+|e))V_{t+1}^+ + \gamma(+,+)\eta(+|e)V_{t+1}^+ - \gamma(-)V_{t+1}^-)}_{MC'(l)} =$$

where $\lambda_l = \lambda_x \frac{\partial x}{\partial l}$.

We assume that ARVs fully revert the effects of HIV on mortality rates and productivity then $\gamma(h,d) = \gamma(+,+) = \gamma(-)$ whereas $\gamma(h,d) = \gamma(+,-) = \gamma(+)$, and $z(h,d) = z(+,+) = z(-)$ whereas $z(h,d) = z(+,-) = z(+)$. At the same time, we assume that ARVs do not alter the ability to enjoy consumption and risky sex.

[Stage 3 Equilibrium] Definition of the ARV *Nonstationary* Recursive Competitive Equilibrium

Given a Stage 2 distribution $\Phi_{T_2}(s=2)$ and a sequence of beleifs $\{\widetilde{\rho}(e)\}_{t\geq T_2}^{\infty}$ by education group and a deterministic sequence for the treated proportion of the population $\{\eta_t(e)\}_{t=0}^{\infty}$ by education, a competitive equilibrium is a sequence of individual household functions $\{V_t, c_t, x_t, l_t : Z \times M \to M\}_{t\geq T_2}^{\infty}$, prices $\{p_t\}_{t\geq T_2}^{\infty}$, and a sequence of measures $\{\Phi_t\}_{t\geq T_2}^{\infty}$ such that, $\forall t \geq T_2$:

1. Given p the policy functions $c_t(e,i,\varepsilon,h,d)$ and $x_t(e,i,s,h,d)$ solve the risky-sex consumers problem (30)-(31) and $c_t(e,-i,\varepsilon,h,d)$ and $l_t(e,-i,s,h,d)$ solve the risky-sex producer problem (32)-(33).

2. All markets clear.

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

   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 Φ is the joint distribution of (e,i,ε,h,d) is induced by the equilibrium policy functions.

4. The true distribution of the HIV population endogenously evolves according to (29).

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

3.5 Model Equilibrium

Here we define the fully-fledged model equilibrium *across* all the stages of the epidemic.

Definition of Model Equilibrium:

The model equilibrium consists of the collection of equilibria *across* stages of the epidemic:

(1) the pre-HIV *stationary* equilibrium up to period T_0 (Stage 0);

(2) the myopic *nonstationary* equilibrium between T_0 and T_1 (Stage 1);

(3) the learning *nonstationary* equilibrium between T_1 and T_2 (Stage 2);

(4) and ARV *nonstationary* equilibrium between T_2 and T_3 (Stage 3), where at T_3 the economy has completed converged to te ARV stationary equilibrium.

The transition *across* stages occurs exogenously at periods T_0, T_1 and T_2.

3.6 Pseudo-Algorithm to Solve the Model

Given the model parameters, including the timing of the unexpected transitions across stages of the epidemic (i.e. T_0, T_1 and T_2), the following algorithm solves the model (Online Appendix D):

1. Find the *stationary* RCE for the Pre- HIV Era (Stage 0).

2. After the unexpected arrival of T_0 and until the unexpected arrival of T_1, solve the myopic *nonstationary* RCE for $t \in [T_0 + 1, T_1]$ (Stage 1). Remember that at every period of this stage, i.e. $t \in [T_0 + 1, T_1]$, an unexpected shocks to mortality $\widetilde{\gamma}$, productivity $\widetilde{z}$ and felicity $\widetilde{\chi}$ arrives and agents take them as permanent. Thus, for every period, we need to solve both the new stationary RCE associated with the new triplet $(\widetilde{\gamma}, \widetilde{z}, \widetilde{\chi})$ 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.

3. After the unexpected arrival of T_1 and until the unexpected arrival of T_2, solve the learning *nonstationary* RCE for $t \in [T_1+1, T_2]$ (Stage 2). Starting with a set of common prior beliefs $\widetilde{\rho_0}$, simulate a series of sequentially updated beliefs by education group until both converge to the actual ρ. Solve the stationary RCE using ρ, 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. Note that if $T_2 \leq \mathscr{T}$, then the arrival of ARVs ends Stage 2.

4. After the unexpected arrival of T_2, solve the ARV *nonstationary* RCE for $t \in [T_2+1, T_3]$ (Stage 3). This stage ends when it reaches stationary RCE at some period T_3. Thus, we proceed to compute this stationary RCE analogously to Stage 2 (recall that agents keep learning in Stage 3) and solve the transition backwards for a given monotonically increasing sequence of coverage levels $\{\eta_t(e)\}_{t=T_2+1}^{\mathscr{T}}$ by education between T_3 and T_2.

4 Calibration Strategy

Our benchmark calibration largely focuses on a specific country, Malawi, for which a large set of moments is readily available.[32] Our strategy to pin down the model parameters includes externally calibrated parameters and an internal calibration of some parameters through the simulated moments method (SMM). We highlight that our internal calibration strategy is anchored to the micro evidence as we explicitly target in our SMM estimation the U-shape evolution of the HIV-education gradient across stages of the epidemic that we documented in Section 2.

The parameters $\beta, \omega, \alpha, \xi, \gamma_-, \gamma_+, f, y_{e=0}/y_{e=1}, s, z_+, \pi, \vartheta_{e=1,i}, \vartheta_{e=1,-i}, \vartheta_{e=0,i}, \tilde{\rho_o}$ and $\phi_{t=0}^+$ are externally calibrated to observable data analogs for or values that are commonly used in the literature. The rest of the parameters $T_1 - T_0, \rho, \vartheta_{e=0,-i}, \sigma_\epsilon^2(e), \chi_+$ and ι are internally calibrated to match several targeted moments in the data through a simulated methods of moments (SMM) procedure.[33] This involves solving the model many times to minimize the distance between the model generated moments and the data counterparts.

We find that the parameters governing the risk infection probability ρ, the time to reach the stage in which agents learn, at T_1+1 and the proportion of sex producers in the economy $\vartheta_{e=0,-i}$ are relevant 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 χ_+, are relevant to pin down some features of the stage of the epidemic where agents learn. Finally, treatment coverage by education ι and a new value of ρ associated with ARVs, become relevent after the introduction of ARVs in the last stage of the epidemic.

[32] In our final Section we discuss the ability of the model to generate alternative country-specific epidemics.

[33] 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 θ. 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. We choose the same number of moments as of parameters.

Note that some parameters are common across stages of the epidemic, and some others are not. Table 3 summarizes the calibration of all the parameters of the model. We now discuss our calibration strategy in detail by epidemic stage.

Stage 0: Pre-HIV Era. We calibrate β to 0.98 for all stages, this reflects a discount rate of 2% of the economy. The relative coefficient of risk aversion ξ 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; agents enter the model when they are 18 years old. We set fertility rates $f = 4\%$ as per the WDI in Malawi, 2016. Additionally, we set the loss of utility due to death, ω, to a large negative number but with no apparent effect because the decreasing returns to sex production put a bound to how much HIV affects mortality.

Labor income is normalized to one for educated individuals, i.e., $y_{e=1} = 1$, and set $y_{e=0} = .6452$ following a 55% educational premium for someone who has completed secondary education according to the National Statistical Office of Malawi. Households are subject to income shocks (ε) that take two possible values: one in good times and 0.4 in bad times which mimics a 60% loss of household income during a period of unemployment in Malawi De Magalhaes and Santaeulàlia-Llopis (2018). These shocks follow a Markov process with a transition matrix π that is calibrated so that at all times 5.4% of the population is under low income[34]. Since there is no HIV in at this point, labor and sex productivity z and preference parameter χ are equal to one. We set α such that the proportion of sex income in the aggregate economy is 7%;[35] implying a share of sex income for sex producers of 12%.

We set the proportion of educated to be 13%[36] 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 did not 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 ($\vartheta_{e=1,-i} = 0$). This is reasonable either because prostitutes are less educated or because teenagers that engage in sexual activity are less likely to finish school (Dupas, 2011; Duflo et al., 2015). This implies that all educated

[34] Interpreting the bad income shock as unemployment, the transition matrix ensures that the average unemployment rate matches that in Malawi in 2019, Source: ILOSTAT database.

[35] In Indonesia, Malaysia, the Philippines and Thailand, estimates of the sex sector (prostitution) range from between 2% and 14% of the Gross Domestic Product (Sulaimon et al., 2018).

[36] This is the sum of the proportion of educated consumers plus educated producers $\vartheta_{e=1,i} + \vartheta_{e=1,-i} = 0.13$.

Table 3: List of parameters

Description	Parameter	Myopic	Perceived Learning	ARV	True Value	Stage Dependent
Discount factor	β		0.98		0.98	No
Utility loss to death	ω		$-\infty$		$-\infty$	No
Labor share of sex income	α		0.01		0.01	No
Risk aversion	ξ		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 $e=1$	$\rho_{e=1}$	-	$\widetilde{\rho}_{e=1}$	$\widetilde{\rho}_{e=1}$	ρ	Yes
Understanding of epidemic $e=0$	$\rho_{e=0}$	-	$\widetilde{\rho}_{e=0}$	$\widetilde{\rho}_{e=0}$	ρ	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
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 ϵ for $e=1$	$\sigma^2_{\epsilon,e=1}$	-	7.9	7.9	-	No
Variance of the signal's noise ϵ 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	ι	-	-	0.074	-	No
Initial Prevalence (%)	$\phi^+_{t=T_0}$	0.5	-	-	0.5	No
Duration of Myopic Stage (s=1) (Years)	T_1-T_0+1	∞	-	-	17	No
Duration of Learning Stage (s=2) (Years)	T_2-T_1+1	-	∞	-	16	No

Notes: This Table shows the externally and internally calibrated values of the parameters of the model. Importantly, note that $\rho = 19$ for the myopic and learning stages, however once ARVs are introduced the overall infectiousness decreases; this translates into an increase to $\rho = 44$ for the ARV stage.

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 epidemic stage, as we discuss next. Finally, we set the initial prevalence $\phi^+_{t=T_0}$ to 0.5%, which is directly linked with the calibration of the duration of the next stage $T_1 - T_0 + 1$ because the larger is $\phi^+_{t=T_0}$, the less periods are needed to reach the peak.

Stage 1: 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 as in the pre-HIV stage. Additionally the survival probability of someone who is infected with HIV decreases to $\gamma_+ = 90\%$, which translates to a life expectancy of 11 years after infection; a typical estimate without ARV treatment.

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, ρ, 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 the learning stage $T_1 - T_0 + 1$. 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.[37] 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 preference shock χ_+. This parameter is chosen in the next stage to match the HIV education gradient just before the introduction of ARVs.

Stage 2: Learning. In this stage agents are aware of the existence of HIV, but their knowledge of the odds infection from risky-sex sex $\lambda(x, \rho)$ is imperfect. They learn about ρ 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 $\sigma^2_\epsilon(e)$ which differs across education groups. Second, how far their initial prior of the infection probability $\tilde{\rho}_o$ is from the true value ρ. This initial prior belief is common across education groups.[38] 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. Further, we choose the $\sigma^2_\epsilon(e)$ by education group and the magnitude of the preference shock χ_+ 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 the learning stage. Finally we select the duration of this stage (i.e. $T_2 - T_1 + 1$) such that $T_2 + 1 = 2005$ which is the year in when ARVs were introduced in Malawi.

[37] 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 $T_1 + 1 = 1989$. In section 6 we explore alternative scenarios in which the population starts learning about the virus earlier in the epidemic.

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

Table 4: Internal Calibration: (SMM) Targeted Moments

Moment	Data	Model
HIV-Education gradient at the peak (1999)	0.0099	0.0112
HIV-Education gradient, end of learning stage (year before ARVs) (2005)	-0.0005	-0.0015
HIV-Education gradient, during ARV stage (2018)	0.0046	0.0047
Prevalence at the peak (1999)	14.6%	14.1%
Prevalence, end of learning stage (year before ARVs) (2005)	12.2%	13.1%
Prevalence, during ARV stage (2018)	9.2%	9.4%
Time to reach the peak (in years)	29	28
Time from peak to ARVs arrival (in years)	6	6

Notes: These are the set of targeted moments in the SMM loop for the internally calibrated parameters. The calendar years are specific to Malawi.

Stage 3: 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.[39] We calibrate the parameter governing the share of educated and non educated individuals receiving ARV's ι 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 ρ). We calibrate the new value of ρ as to match Malawi's average prevalence rate in 2018.

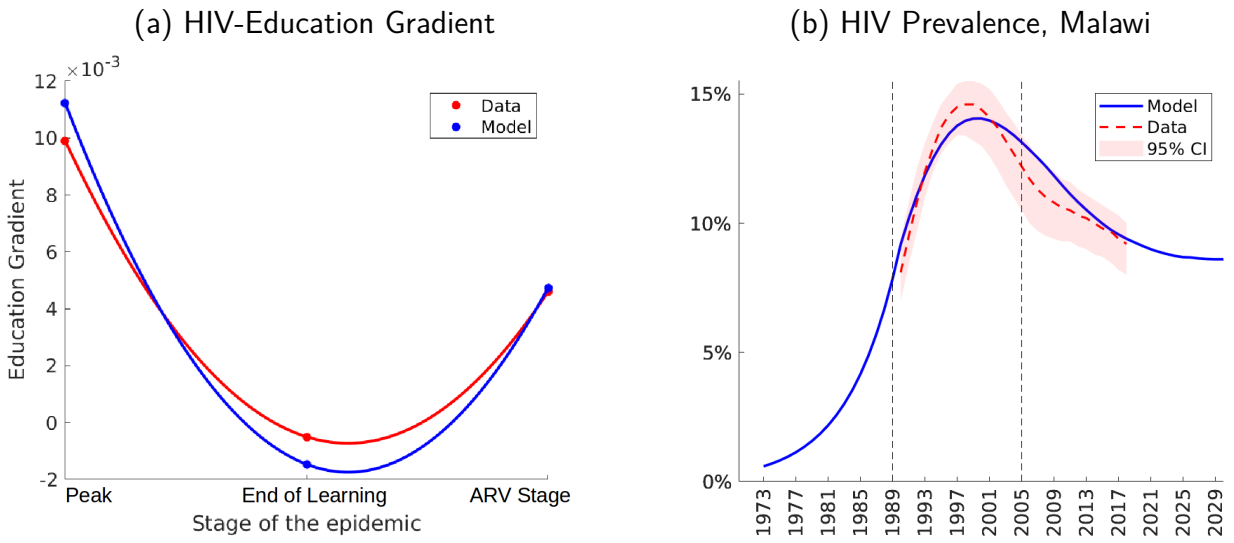
4.1 Model Fit

The benchmark calibration matches the HIV-Education gradient very well, see Table 4 and panel (a) in Figure 5. The model also captures the HIV prevalence rate at the peak, end of learning stage (year before ARVs) and during the ARV stage; see Table 4. The model time to reach the peak of HIV prevalence and time from peak to ARVs arrival is also consistent with the data, see Table 4. Indeed, the entire time path of the HIV prevalence in the model fits well its data counterpart (as reported by UNAIDS), see panel (b) of Figure 5. The model's prevalence's is always within the UNAIDS 95% confidence interval.

Additionally, the model performs remarkably well mimicking other moments relevant for the HIV epidemic that are not targetd. Specifically, the average HIV incidence at the peak is 1.66%, close to 1.60% that is reported in by the UNAIDS for the population between 14-49 years in Malawi; panel (a), Figure 6. The model generates slightly larger AIDS deaths than those reported by UNAIDS, e.g. overstating the peak of deaths by approximately 9%; panel (b), Figure 6.

[39] 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.

Figure 5: Model Fit: HIV-Education Gradient and HIV Prevalence

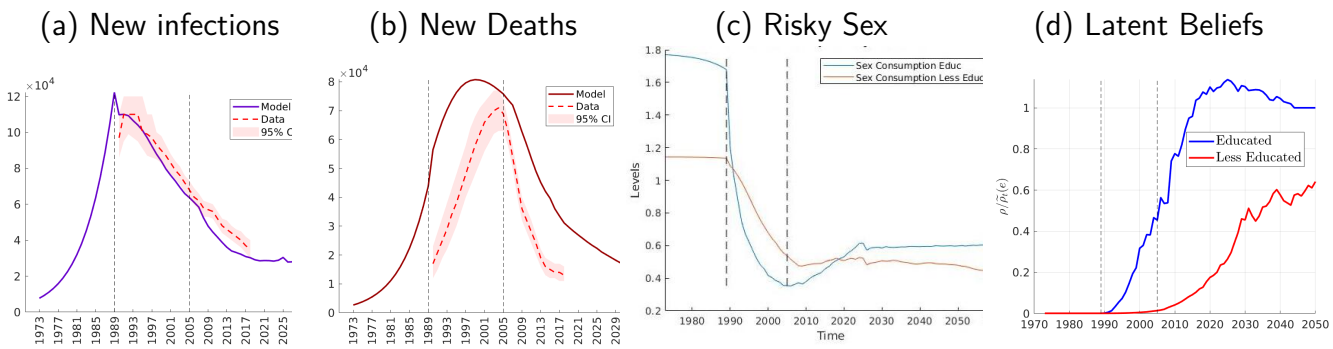


Notes: Panel (a) shows the model-generated education gradient against the data counterpart that we document in Section 2 across stages of the epidemic. Panel (b) compares the HIV prevalence generated by the model and the counterpart data (model) from UNAIDS for Malawi. Data source: UNAIDS estimates 2019.

Further, we have explicitly modelled the direct link between risky sexual behaviour and the probability of infection across education groups through $\lambda(x, \rho)$, 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; see panel (c) in Figure 6. Finally, the latent beliefs that we uncover regarding the odds of infection due to risky sex imply that agents tend to be optimistic in that the belief in $\widetilde{\rho}$ is such that $\widetilde{\rho} < \rho$—implying that the expect less infections per unit of risky sex than that actual amount of infections. However, learning occurs in a manner that ρ is approximated from below and hence agents become less optimistic over time; see panel (d) in Figure 6. In addition, for the educated sample we find that $\widetilde{\rho}$ overshoots ρ in later stages of the epidemic. This phenomenon, $\widetilde{\rho} > \rho$ in which $\widetilde{\rho}$ approaches ρ from above resembles the pessimistic scenario described in Delavande and Kohler (2016) in which after information campaigns on HIV transmission risk, which in our setting could be interpreted as $\widetilde{\rho}$ approaches ρ from above, there is an increase in risky sex (see panel (c) for the same period).

Last, it is worth mentioning that even though we do not target it, for lack of direct measurement of ARV coverage at the individual leve in DHS data, we find that the obtained relative coverage of the more educated compared to those less educated turns out to be $(\eta_{t,e=1} - \eta_{t,e=0})/\eta_{t,e=0} =$

Figure 6: Non Targeted Moments and Latent Beliefs



Notes: Panels (a) and (b) show the evolution of the new infections and new deaths respectively in our benchmark model and in the data. Regarding with panels (a) and (b) note that the 'observed' data refers to the UNAIDS model-generated data for the HIV epidemic. Panel (c) shows the model evolution of risky sex across education groups. Panel (d) shows the latent beliefs on the odds of infection due to risky sex by education group.

15.98%, which is close to that reported in Lulseged et al. (2022) from PHIA survey data;[40] which is the value of the likelihood that the educated achieve viral suppression relative to the less educated in Malawi 2015-2016.

4.2 SMM Identification at Work: How Parameters Affect Targeted Moments

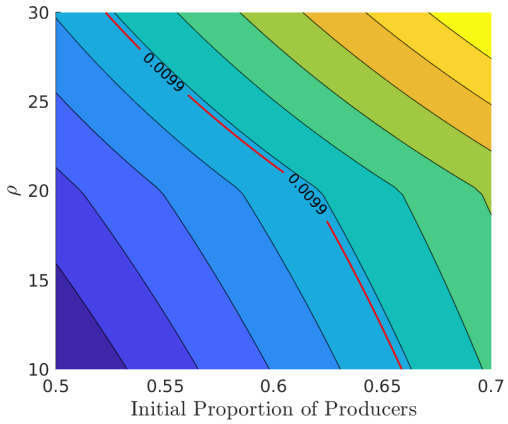
Here, we explicitly show how some of the internally calibrated model parameters are identified through SMM in order to match targeted moments. To this end, Figure 7 displays a set of contour-maps showing how some of the moments of interest are shaped by different parameters. In each panel, the red contour line represents the target that is to be matched.[41] For example, panels (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 ρ has a larger impact on the prevalence's at the peak. Further, 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 χ, 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 χ. 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 ρ.

[40] This value is computed using the PHIA survey data (Population-Based HIV impact assessment) for Malawi 2015-2016. Lulseged et al. (2022) find that individuals with secondary education and above were 8.2% more likely to have achieved viral suppression compared to their less educated counterparts.

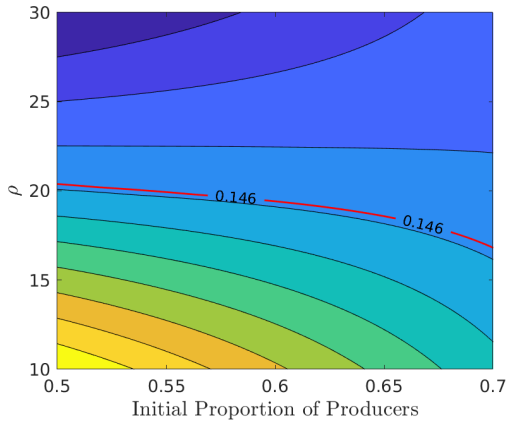
[41] The parameter range where the target moments are found is ex-ante unknown, however based on intuition it is possible to guess the range until a visible intersection of two jointly identified moments (red lines) appears. To the extent possible, we use the intersection between the red lines as the initial guess for the SMM estimation, this considerable speeds up the estimation.

Figure 7: SMM Identification at Work: Parameters and Targeted Moments

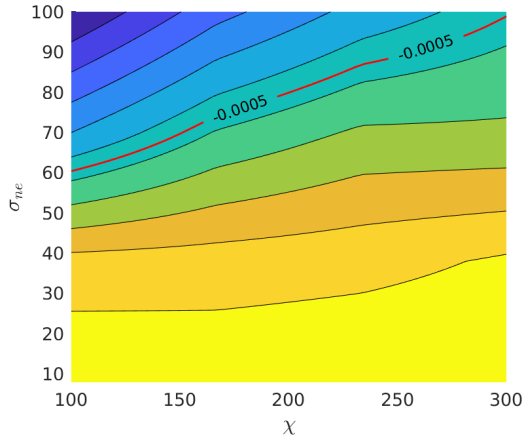
(a) HIV-Education Gradient, at the peak (1999)



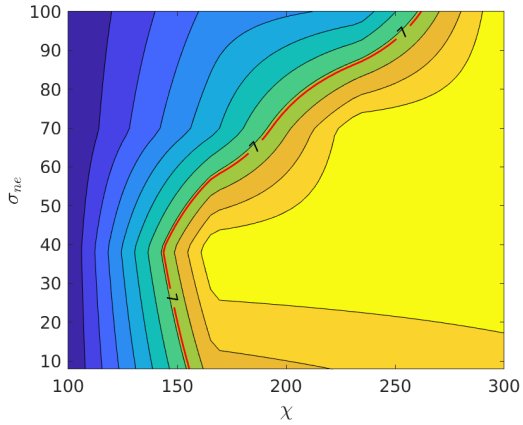
(b) Prevalence, at the peak (1999)



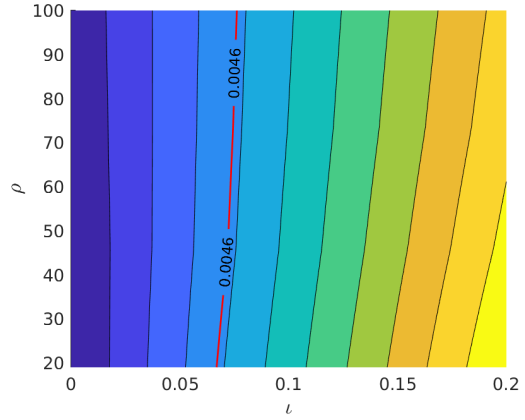
(c) HIV-Education Gradient, year before ARVs (2005)



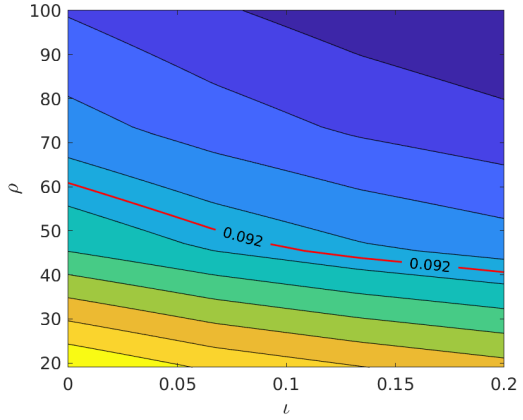
(d) Years from Peak to ARVs Arrival



(e) HIV-Education Gradient, ARV stage (2018)



(f) Prevalence, ARV stage (2018)



Notes: Panel (a) and (b) show, respectively, the sensitivity of the HIV-Education Gradient at the peak and the prevalence at the peak with respect to the infection parameter ρ and the initial proportion of sex producers $\vartheta_{-i,e=0}$. Panels (c) and (d) show, respectively, the sensitivity of the HIV-Education Gradient at the end of the learning stage (year before ARV arrivals, 2005) with respect to the preference parameter χ and the variance of the signal for less educated individuals $\sigma^2_\epsilon(e=0)$. Finally panels (e) and (f) show, respectively, the sensitivity of the HIV-education gradient and prevalence during the ARV stage (at year 2018) with respect to the coverage difference parameter ι and the new value of ρ updatd by the presence of ARVs.

5 What Explains the Evolution of the HIV-Education Gradient?

Here we assess the deterimnatns of the HIV-education gradient over the course of the epidemic. To do so, we separately remove education differences in income (Section 5.1), ability to learn about the odds of infection (Section 5.2), and access to ARV treatment (Section 5.3).

5.1 Removing income differences between education groups

For this experiment we exogenously set $y_{e=0}/y_{e=1} = 1$ by increasing $y_{e=0}$ to one. This increases the consumption of risky sex for the less educated throughout the HIV epidemic.[42] As consequence the HIV-education gradient falls below the benchmark gradient; see panel (a) in Figure 8 and column (1) in Table 5. There is a level change in the gradient throughout. Specifically, during the myopic and learning stage the gradient is approximately 65% lower than the benchmark gradient; see also column

Moreover, during the learning stage the gradient turns significantly negative reflecting the fact that the only differences left across education groups are the learning speeds. During the ARV stage the HIV gradient remains negative and lower than the benchmark, however the gap decreases over time; this occurs because during these stage there are not only learning differences across education groups but also a higher treatment coverage for the educated.

We conclude that income differences across education groups is an important driver for the level of the HIV education gradient. However, the U-shape pattern of the gradient remains largely unaltered after the removal of the income differences across education groups.

5.2 Removing learning differences between education groups

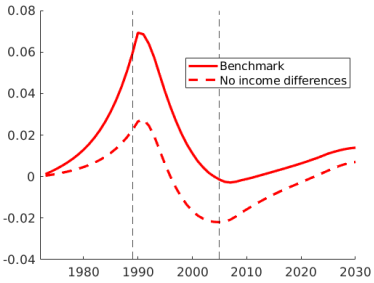
We explore two different ways of removing learning differences between the educated and the less-educated. First, we make the less-educated individuals learn at the same (fast) speed as their more-educated counterparts: we set $\sigma^2_{\epsilon,e=0} = \sigma^2_{\epsilon,e=1} = 7.9$. Second, we make the more-educated individuals learn at the same (slow) speed as their less-educated counterparts: we set $\sigma^2_{\epsilon,e=1} = \sigma^2_{\epsilon,e=0} = 84$. The results of this experiment are in panel (b) of Figure 8.

If the less-educated learn as fast as the educated, we find that 52% of the decline in HIV Education gradient and is explained by the information differences; see columns (2a) of Table 5. Moreover the gradient does not turn negative, and the effect of fewer information differences carries on to the ARV stage, where the the gradient remains high and relatively constant. On

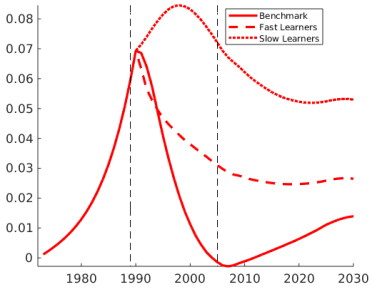
[42] We would find the opposite effects, had we chosen to decrease $y_{e=1}$ to the level of the less educated instead.

Figure 8: What Explains the Evolution of the HIV-Education Gradient?

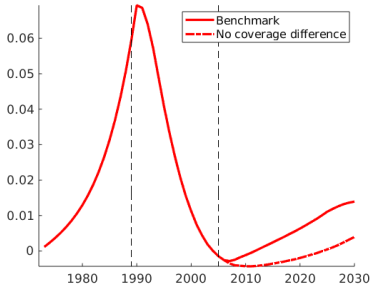
(a) Removing income differences



(b) Removing learning differences



(c) Removing ARV access differences



Notes: In all panels, the vertical axis refers to the HIV-Education gradient. We report the benchmark evolution of the HIV gradient in all panels (solid red line). Panel (a) removes income differences across education groups, raising the income of the less educated to be identical to the income of the more educated; see the discussion in Section 5.1. Panel (b) removes learning differences in two ways by forcing the less educated learn as fast as the more educated and, alternatively, by forcing the more educated learn as slow as the less educated; see the discussion in Section 5.2. Panel (c) removes the ARV access differences across education groups; see the discussion in Section 5.3.

the contrary, if the more-educated learn at the same slow rate as the less educated, the gradient increases instead of increasing; see columns (2b) of Table 5. The gradient keeps increasing, to reach a peak at 0.08, to then revert back to 0.07. In this experiment agents learn so slow that the maturity of the epidemic is delayed, the peak of the epidemic occurs two years later. Hence, it is clear that the asymmetric learning is essential for explaining the decline of the HIV-education gradient. Further, the strong effect also carries out to the ARV stage, where the gradient slowly decreases on average 25% to then remain high with a slight rebound after 2025.

5.3 Removing coverage access differences between education groups

This experiment involves setting $\iota = 0$. Specifically, The educated get lower chances of treatment whereas the less educated get higher chances of treatment compared the benchmark. By setting $\iota = 0$ both groups will have the same chance of treatment. Our results are in Column 3 of Table 5 and panel (c) of Figure 8. Lower coverage for the educated reduces the HIV-education gradient significantly, it reduces it so much that the gradient remains negative even after 20 years of rebound. This happens because the risky sex consumption of the educated decreases whereas risky sex of the less educated increases. Education differences in coverage explains on average 60% of the rebound of the HIV-education gradient, making ARV coverage crucial for explaining the HIV education gradient rebound. Notice that the slope of the gradient is positive even after eliminating coverage differences, this is because the average probability of getting treatment increases over time for all. Thus, increasing ARV coverage explains the rest of the of HIV education gradient rebound.

Table 5: Determinants of the Evolution of the HIV-Education Gradient

Moment	Benchmark	Counterfactual Experiments (1) No Income differences	(2a) Fast Learning	(2b) Slow Learning	(3) $\iota = 0$
HIV-Education gradient, end of myopic stage (1989)	0.0595	0.0221	0.0595	0.0595	0.0595
HIV-Education gradient, at the peak (1999)	0.0112	-0.0185	0.0527	0.0796	0.0112
HIV-Education gradient, end of learning stage (2005)	-0.0015	-0.0201	0.0310	0.0720	-0.0015
HIV-Education gradient, during ARV stage (2018)	0.0047	-0.0053	0.0246	0.0531	-0.0027
Prevalence, at the peak (1999)	14.1%	16.1%	10.9%	16.8%	14.1%
Prevalence, end of learning stage (2005)	13.1%	15.0%	7.1%	16.4%	13.1%
Prevalence, during ARV stage (2018)	9.4%	11.2%	5.4%	12.2%	9.8%
Time to reach the peak (in years)	28	29	21	30	28
Time from peak to ARVs arrival (in years)	6	5	13	4	6

Notes: This table shows the results of the counterfactual experiments outlined in Section 5. The first column lists the moments that were targeted in our benchmark calibration.

6 Drivers of the HIV Epidemic and Policy Experiments

In this section we explore a set counterfactual experiments designed to understand the main drivers of the HIV epidemic that can guide policy. We ask ourselves how would have the HIV epidemic evolved in the following cases: Earlier learning about the odds of infection (Section 6.1); Improving the education level of the population (Section 6.2); Earlier and universal adoption of ARVs (Section 6.3); HIV prevention through Pre-exposure prophylaxis (PrEP) (Section 6.4).

We keep tract of the evolution of the HIV epidemic looking at three endogenous outcomes generated by our quantitative model: evolution of the (1) HIV prevalence, (2) number of new HIV infections (incidence) and (3) the flow of new deaths caused by the HIV virus. We then compare the counterfactual results of the above cases with our benchmark calibration.

6.1 Earlier Learning about the Odds of Infection

In Figure 9 panels (a1)-(a3) 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. This implies that both educated and less educated start adjusting their risky sexual behaviour earlier and by a marginally larger amount with every additional year since they have more time to learn and get close to learning about their true individual probability of infection. Because of earlier learning we expect lower risky sex consumption thus an epidemic of a lower magnitude. Panels (a1)-(a3) show precisely that. Panel (a1) shows that the prevalence peak is lower the earlier the learning starts. If learning would have started 10 years earlier, the prevalence at the peak would have reduced by approximately by 60% and 45% if the learning started 5 years earlier. Whats more, earlier learning also accelerates the maturity of the epidemic, shifting the peak of the epidemic to the left. Combined, these effects translate into a significant reduction of overall number of

infections. Furthermore, the peak number of new infections and deaths due to HIV also reduce in a similar magnitude. The effect of earlier learning carries out on the ARV stage, but with slightly lower magnitudes for infections and deaths: new infections and new deaths reduce by 50% in case of the learning starting 10 years earlier; see panels (a2) and (a3), respectively. These counterfactual exercise shows that learning is a very important driver of the epidemic, having the possibility of reducing the prevalence, incidence and deaths by far more than half.

6.2 Improving the Education Level

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 were 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. In our experiment we explore the possibility of having 85% (the current level attained by South Africa) of the population with secondary education by 2030.[43] 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 $\lim_{t\to\infty}\vartheta_{e=1,i,t} = 85\%$ For this target to be reached by 2030, education growth must 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. In our model more education is associated with higher overall income, faster learning, and higher ARV coverage.

Figure 9 panels (b1)-(b3) show the results of these experiments. We see that increasing the education of the population would have translated into a higher prevalence, initially this is the result of an income effect that is partially mitigated by the fact that the educated adjust their risky sexual behaviour faster than the less educated. This is reflected in the fact that the prevalence at peak did not increase as much. However the prevalence after the introduction of ARV's is 35% higher than the benchmark. This is because educated individuals go back to increasing their risky sex consumption given the reduction of the negative effects of HIV following the introduction of ARV's. Similar effects can be seen when looking at new infections, and new deaths. Incidence increases 5 times during the ARV stage and new deaths increase by 66%.

6.3 Earlier Adoption of ARVs

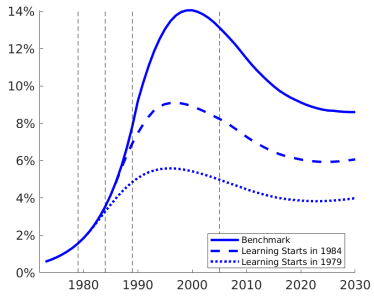
For this exercise we show how the epidemic would have evolved if ARV universal coverage would have been attained by 2019. For this to happen AR's should have been were introduced at in 1999, that is at the peak of the epidemic, (6 years earlier than in the benchmark case). The results

[43] 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.

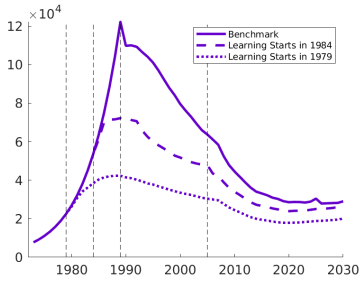
Figure 9: Drivers of the HIV Epidemic and Policy Experiments

(a) Earlier Learning about the Odds of Infection

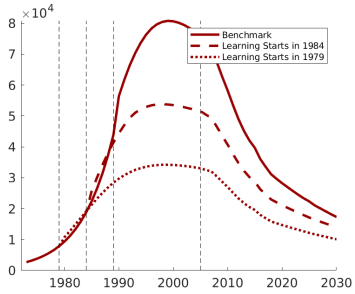
(a1) Prevalence



(a2) New Infections

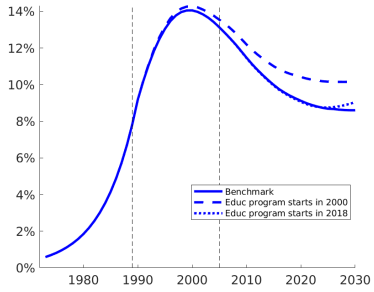


(a3) New Deaths

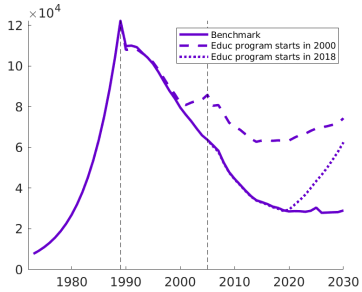


(b) Improving the Education Level

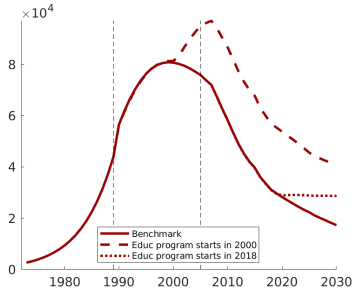
(b1) Prevalence



(b2) New Infections

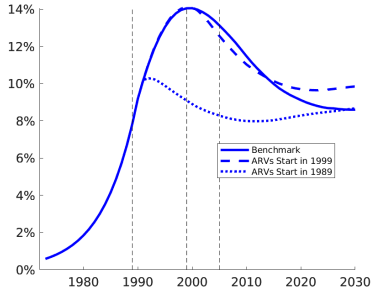


(b3) New Deaths

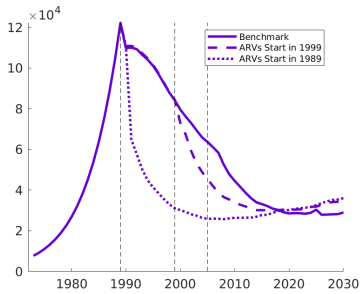


(c) Earlier Adoption of ARVs

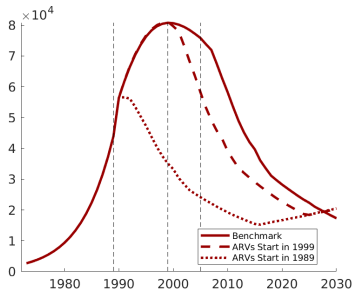
(c1) Prevalence



(c2) New Infections

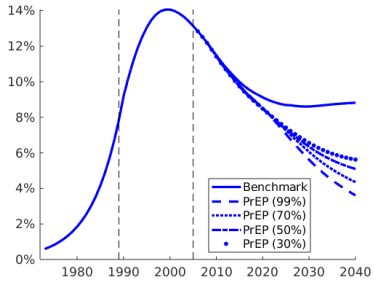


(c3) New Deaths

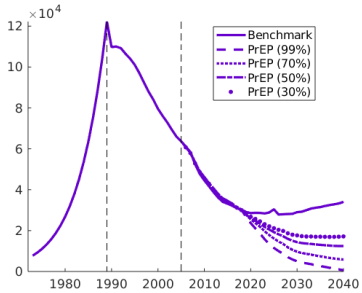


(d) Preventing HIV with PrEP

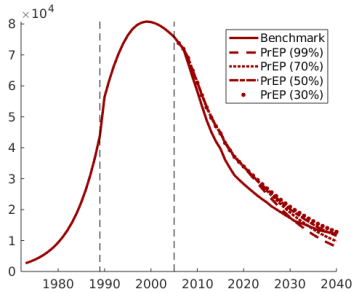
(d1) Prevalence



(d2) New Infections



(d3) New Deaths



Notes: This Figure compares our benchmark results with the effects of four counterfactual experiments: earlier learning in panel (a); improving education in panel (b); earlier adoption of ARV's in panel (c); preventing HIV with PrEP in panel (d). We show the prevalence rates (left column), new infections (center column) and new deaths (right column).

are shown in Figure 9 panels (c1) to (c3) where we see only a slight reduction in the prevalence rate. Earlier adoption of ARVs has improves the situation of the less educated but increases risky sex consumption of the educated, this as long as universal coverage is not reached. This is reflected in the the significant reduction of new infections and deaths following the early adoption of ARVs. Notice that after universal coverage is attained in 2019, infections stop decreasing but deaths keep going down, this is because at that point agents do not need to worry about getting HIV infected. If ARVs were introduced 16 years earlier, i.e. in 1989 together with the start of the learning stage, universal coverage would have been attained by 2009. In this case, the peak prevalence reduces by almost 40%, incidence and deaths by more than 70%.

6.4 Preventing HIV with 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.[44] 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. Then, we construct an experiment where we analyze the evolution of the epidemic as if PrEP was 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 ρ 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 (d1) to (d3) of Figure 9 show that in all four cases the prevalence levels reduce considerably as well as incidence and the numbers of deaths. Even with PrEP being the least effective, 30%, we see that the prevalence decreases on average 33% and incidence by 30%.

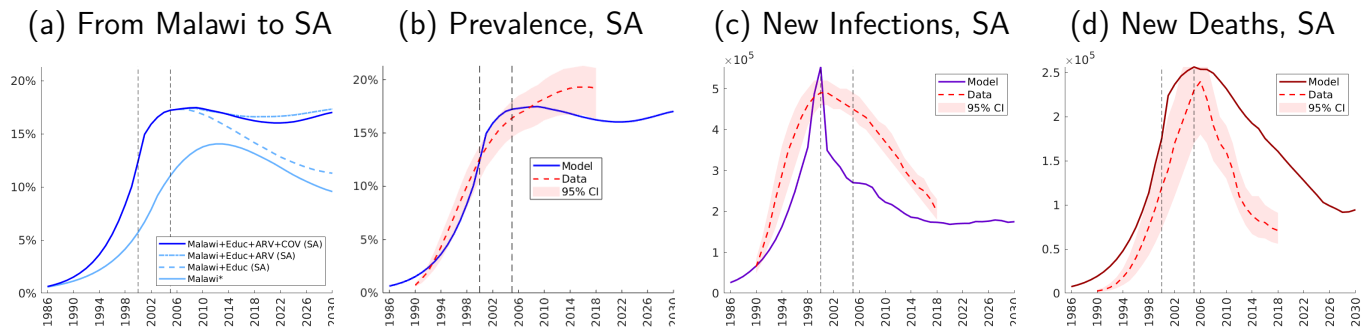
7 External Validation

Can our theory explain epidemic heterogeneity across SSA countries? To address this question, we modify our benchmark calibration set for Malawi to cumulatively include country-specific features of South Africa, as shown in panel (a) of Figure 10. First, we incorporate the specific education structure of South Africa (long-dashed light blue line) into our benchmark model calibrated to

[44] Source: Avert Info.

[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 10: Characterizing the HIV Epidemic in South Africa (SA)



Notes: The starting year of the epidemic in Malawi was normalized to that of South Africa: 1986. In panels (b), (c) and (d), the data refers to the UNAIDS model-generated data for the HIV epidemic.

Malawi (solid light blue line). In 1985, 33% of the South African population had finished primary school; by 2018, this figure increased to 85% of the population (WDI), higher than that of Malawi. Higher education implies higher prevalence rates. Second, we introduce the year of ARV introduction in South Africa (2004), leading to a substantial increase in HIV prevalence in the ARV era (short-dashed light blue line), rising from 14% in a scenario without ARVs in 2019 to 17% in a scenario with ARVs. Finally, we incorporate the country-specific rollout of ARV coverage in South Africa, reaching 59% in 2018—compared to 70% coverage in Malawi the same year—which implies a slightly lower prevalence rate in the ARV era (solid dark blue line).

After integrating education structure, ARV arrival date, and ARV coverage rates for South Africa, the model prevalence aligns with the observed path. This alignment implies, for example, an increase in the prevalence rate for 2005 from approximately 10% in the benchmark Malawi to 16% in South Africa; panel (b), Figure 10. The patterns of new infections and deaths also resemble those of South Africa; panels (c) and (d), Figure 10, respectively.

8 Conclusion

In conclusion, in order to explain the evolution of the HIV epidemic, our paper proposes a novel mechanism involving learning about odds of HIV infection due to risky sex. Our empirical analysis uncovers a U-shaped relationship between education and HIV positivity across epidemic stages, which is mirrored by a similar relationship between education and risky sex. In this context, we formalize the idea of asymmetric learning by which more educated individuals may learn faster and refine their beliefs about infection odds more accurately than their less educated counterparts, leading to earlier changes in sexual behavior among the educated. These empirical insights are integrated into a *nonstationary* quantitative theory that encompasses three stages of the HIV epidemic—myopic, learning, and ARV. Anchored in micro evidence, particularly the replication

of the HIV-education gradient throughout the epidemic for which the asymmetric learning across education groups is essential, our study reveals a large quantitative role for the learning mechanism in explaining the aggregate evolution of the HIV epidemic.

References

Beegle, K. and de Walque, D. (2009). Demographic and Socioeconomic Patterns of HIV/AIDS Prevalence in Africa. Policy Research Working Paper Series 5076, The World Bank.

Behrman, J. and Kohler, H. (2012). Sexual Transmission of HIV. In Lomborg, B., editor, *Rethink HIV*, chapter 1. Cambridge University Press.

Bethencourt, C. and Ríos-Rull, J.-V. (2009). On the Living Arrangements of Elderly Widows. *International Economic Review*, 50(3):773–801.

Bongaarts, J., Buettner, T., Heilig, G., and Pelletier, F. (2008). Has the HIV Epidemic Peaked? *Population and Development Review*, 34(2):199–224.

Brotherhood, L., Kircher, P., Santos, C., and Tertilt, M. (2020). An Economic Model of the Covid-19 Pandemic with Young and Old Agents: Behavior, Testing and Policies. Technical report.

Cameron, A. C., Gelbach, J. B., and Miller, D. L. (2008). Bootstrap-Based Improvements for Inference with Clustered Errors. *The Review of Economics and Statistics*, 90(3):414–427.

De Magalhaes, L. and Santaeulàlia-Llopis, R. (2018). The Consumption, Income, and Wealth of the Poorest: An Empirical Analysis of Economic Inequality in Rural and Urban Sub-Saharan Africa for Macroeconomists. *Journal of Development Economics*, 134(C):350–371.

de Walque, D. (2007). How Does the Impact of an HIV/AIDS Information Campaign Vary with Educational Attainment? Evidence from Rural Uganda. *Journal of Development Economics*, 84(2):686–714.

Delavande, A. and Kohler, H.-P. (2016). HIV/AIDS-related Expectations and Risky Behavior in Malawi. *Review of Economic Studies*, 83(1):118–164.

Duflo, E., Dupas, P., and Kremer, M. (2015). Education, HIV, and Early Fertility: Experimental Evidence from Kenya. *American Economic Review*, 105(9):2757–97.

Dupas, P. (2011). Do Teenagers Respond to HIV Risk Information? Evidence from a Field Experiment in Kenya. *American Economic Journal: Applied Economics*, 3(1):1–34.

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

Galor, O. and Weil, D. N. (2000). Population, Technology, and Growth: From Malthusian Stagnation to the Demographic Transition and Beyond. *American Economic Review*, 90(4):806–828.

Greenwood, J., Guner, N., and Kopecky, K. A. (2022). The Downward Spiral. Working Paper 29764, National Bureau of Economic Research.

Greenwood, J., Kircher, P., Santos, C., and Tertilt, M. (2019). An Equilibrium Model of the African HIV/AIDS Epidemic. *Econometrica*, 87(4):1081–1113.

Greenwood, J., Seshadri, A., and Vandenbroucke, G. (2005). The Baby Boom and Baby Bust. *American Economic Review*, 95(1):183–207.

Gregson, S., Waddell, H., and Chandiwana, S. (2001). School Education and HIV Control in Sub-Saharan Africa: From Discord to Armony. *Journal of International Development*, 13:467–485.

Hansen, G. D. and Prescott, E. C. (2002). Malthus to Solow. *American Economic Review*, 92(4):1205–1217.

Herrendorf, B., Rogerson, R., and Ákos Valentinyi (2014). Growth and Structural Transformation. volume 2 of *Handbook of Economic Growth*, chapter 6, pages 855 – 941. Elsevier.

Lee, R. (2003). The Demographic Transition: Three Centuries of Fundamental Change. *Journal of Economic Perspectives*, 17(4):167–190.

Lucas, R. E. (2004). The Industrial Revolution: Past and Future. *Annual Report Essay, Federal Reserve Bank of Minneapolis*, (May):5–20.

Lulseged, S., Melaku, Z. n. H., West, C., Gelibo, T., Belete, W., Tefera, F., Farahani, M., Demissie, M., Teferi, W., Abdella, S., Birhanu, S., and CE, R. (2022). Progress Towards Controlling the HIV Epidemic in Urban Ethiopia: Findings from the 2017-2018 Ethiopia Population-Based HIV Impact Assessment Survey. *PLoSOne*.

Mwale, B. (2002). HIV /AIDS in Malawi. *Malawi medical journal*, 14(2):2–3. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3346002/.

Sulaimon, M. D., Muhammad, A. A., and Shofoyeke, O. (2018). Possible Health and Growth Implications of Prostitution in Nigeria: A Theoretical Perspective. MPRA Paper 88402, University Library of Munich, Germany.