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A parsimonious model of longevity, fertility, HIV transmission and development

Luca Gori*• Piero Manfredi†• Mauro Sodini‡

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Abstract

A central policy issue in the battle against HIV in Sub-Saharan Africa (SSA) is whether and when high-prevalence countries might become fully autonomous in designing and implementing their own intervention policies aimed to control the disease. The aim of this research is twofold. First, it develops a framework for explaining economic development in a general equilibrium growth model with endogenous fertility and endogenous longevity under the threat of a deadly enduring infectious disease, as is the case of HIV/AIDS in SSA. Second, it aims to shed light on the interplay between foreign aid and endogenous domestic public policies in those SSA countries severely afflicted by HIV. In particular, it investigates the macro-economic dynamic feasibility and related effects of an intervention policy where the overall amount of resources devoted to HIV/AIDS is the sum of an exogenous component representing foreign aid and an endogenous public expenditure. Both these policies allow to bring HIV under control, but show quite different responses in terms demo-economic variables, mainly passing through the fertility response to the evolving epidemic conditions.

Keywords HIV transmission; Economic development; Endogenous fertility; Endogenous longevity;

JEL Classification C61; C62; J1, J22; O41; O47

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1 Introduction

With about - according to the last UNAIDS estimates [UNAIDS (2017)] - 76 million people infected and 35 million people died from related illnesses since the start of the epidemic, HIV/AIDS definitely represents the major dramatic pandemic of the current era. It is however in Sub-Saharan Africa (SSA) that AIDS has reaped the most dramatic toll, yielding a tragedy of major proportions. Still in 2016, out of a total of 36.7 million people living with HIV, with and incidence of 1.8 million new HIV infections and 1 million AIDS-related deaths per year worldwide, SSA - which hosted 13% of the world population - accounted for 69.5% of those living with HIV, for 64.5% of new infections and for 73% of total estimated deaths, respectively [UNAIDS (2017)]. Nonetheless, since several years epidemic trends have finally downturned thanks to the continued efforts aiming both at developing prevention campaigns - to rise and keep population awareness - and expanding the proportion of seropositive and AIDS-seek people to access effective antiretroviral therapies (ART). Indeed, while in 2004-2005, the epoch of the mortality peak in SSA, the proportions accessing treatment were negligible, in 2016 54% of those living with HIV received ART in SSA, though with large difference between Eastern and Southern Africa compared to Western and Central Africa.

These efforts are reducing the incidence of new HIV infections on one hand, and are allowing a dramatic reduction of AIDS mortality on the other hand. The latter indeed has nearly halved in a span of one decade [UNAIDS (2017)]. However, these positive results should not obscure the fact that AIDS still represents the second cause of mortality in SSA [WHO (2017)] and that many complicate issues remain to be solved.

Indeed, eventual full success against HIV will require a further massive long-term expansion of the resources specifically devoted to the fight of HIV/AIDS, in order to make most seropositive people aware of their status and provide them with a fully effective and lifelong treatment on one hand, and to maintain high rates of public awareness of HIV on the other hand [UNAIDS (2017)]. Given the high cost of a single therapy protocol, the overall economic cost of generalised lifelong HIV treatment in the poorest SSA countries would be dramatic. In these settings, this cost has been estimated to reach levels as high as 80% of current GDP [Collier and Sterck (2018)].

This raises a serious issue of the affordability of an endogenous AIDS response by the afflicted SSA countries. Until recently, most of the intervention carried out in SSA has relied on international financing [UNAIDS (2017)] either by international institutions or by donors, such as partnerships as e.g., the Global Fund against AIDS, Tuberculosis and Malaria.

The dramatic extent of foreign financing of the AIDS response, with 90% of AIDS funding in low income countries in 2013, can have a number of drawbacks. First, the magnitude of this source might not be able to expand significantly beyond current levels [Resch et al. (2015); Remme et al. (2016)]. Second, as expressed by the UNAIDS executive director, concern should be raised about the potentially destabilising effects of an HIV response completely dependent

on external resources [UNAIDS (2012)]. We believe that the latter is an important concern underlining the possible difficulties [e.g.,Mohiddin and Johnston (2006)] to develop a country-specific response to HIV by entirely relying on external resources in terms of e.g., optimally organising the related public health infrastructures and keeping high the degree of internal awareness.

In prospective terms, the key emerging issue is clearly that of the transition towards a system where SSA countries gradually acquire degrees of autonomy in the management of domestic HIV/AIDS epidemic. Public health and health economic research are currently confronting with this issue that is stimulating an increasing number of contributions [Katz et al. (2014); Resch et al. (2015); Atun et al. (2016); Remme et al. (2016)]. The problem is a complicate one, stemming from the interplay between HIV and AIDS prevalences on one hand, and on the afflicted country stage of economic development on the other hand, but extending to a number of further aspects including (a) perspectives for GDP growth in the medium term, (b) threats from other diseases that require to distract significant quotas of GDP, (c) ability to develop new financial tools for front the HIV challenge, etc. [Resch et al. (2015); Atun et al. (2016); Remme et al. (2016)].

Based on this line of reasoning, we aim at contributing to this debate from the perspective of macro-economic dynamic modeling. As a first step, we aim at investigating the macro-economic dynamic feasibility and related effects of an intervention policy where the overall amount of resources devoted to fight against HIV/AIDS is the sum of two components namely (a) a fully endogenous public expenditure (financed at a balanced budget), which is assumed to be entirely managed by the government of the afflicted country, and (b) a completely exogenous component reflecting international donations.

The macroeconomic literature on the interplay between economic growth and development and infectious diseases is still in its infancy. In two influential works, [Chakraborty et al. (2010, 2016)], focusing on Africa as the world's region still suffering the highest burden from infectious disease, started the investigation of the impact of a range of deadly infections within the general equilibrium overlapping generations (OLG) framework à la [Diamond (1965)]. In particular, they provided a parsimonious and innovative representation of the infection dynamics which is not simply modelled as a short-term exogenous shock, but it is assumed to follow an explicit dynamics governed by meaningful epidemiological parameters. Nonetheless, they did not explicitly model out the endogenous (general equilibrium) feedback between HIV/AIDS and demographic variables at the macroeconomic level. Modelling this feedback is the key ingredient to understand the effects of an infection such as HIV/AIDS in SSA, which combines long-term persistence at very high levels of prevalence and high mortality in the lowest resource setting worldwide. This combination of factors has for long time prevented effective interventions at the population level. In addition, [Chakraborty et al. (2010, 2016)] considered a model where individuals are able to internalise the effects of the epidemics by choosing optimally disease-specific prevention investments. We believe this approach is hardly tenable

from an empirical point of view if one concentrates on SSA, which is the poorest region worldwide with inadequate health infrastructures, severe shortages of physicians and medicines, low education and poor health causing high mortality even in the absence of HIV. Then, it is at least questionable to think about individuals living in SSA are able to adequately protect themselves (by devoting their own resources) against (different kind of) infectious diseases, as this expenditure may neither substitute nor complement public interventions coming from domestic resources or foreign aid. The onset of HIV has represented a major tragedy producing highly detrimental effects on the life expectancy after many decades of continued growth despite the presence of many other deadly pathologies. For these reasons, the main research question of this paper, namely the feasibility of an HIV response entirely sustained by resources coming from the afflicted country, could not be set into the framework developed by Chakraborty et al. Therefore, we propose a model where the endogenous response to the HIV/AIDS epidemic is based on a combination of endogenous public spending and exogenous donations. This is set up within a framework aiming at capturing some key aspects of the current context of HIV in SSA, by including endogenous adult mortality from HIV/AIDS and the agents' endogenous fertility response to AIDS mortality. Our major task is to investigate the feasibility for HIV-afflicted SSA countries to gradually achieve independence from international support and develop a fully internal response to the disease. For the sake of clarity, in the present model we did include neither education, human capital accumulation and their HIV-related effects nor the mortality and fertility transition, which have been already considered in Gori et al. (2017).

The rest of the article proceeds as follows. Section 2 discusses the main assumptions of the model and its key ingredients. Section 3 reports and discusses the main results in the light of the related economic literature. Section 4 outlines the main conclusions.

2 The model

Consider a general equilibrium OLG (macro)economy closed to international trade and comprised of rational and identical individuals of size N_t (at birth) per generation, where $t = 0, 1, 2, ...$ is the time index. The life of the representative agent is divided into young adulthood and old age. We follow [Chakraborty et al. (2010, 2016)] and did not model out an explicit childhood cohort. As a child, an individual directly consumes resources in the parent's household without making any economic decisions. This is a deliberate simplification because our aim is to focus on the major source of AIDS mortality, namely adult mortality. This amounts to assume that the mortality burden amongst children (including those dying by vertically transmitted AIDS) is a constant background steadily affecting the population of SSA countries and, therefore, we only consider the children surviving at the entry of adulthood as the key variable of the model [Galor and Weil (1996); Gori and Sodini (2018)]. As a young adult, an individual is economically and sexually active. He works, saves, gives birth and takes care of

children. As an old-aged, he is retired and consumes on the basis of his saving. *During young adulthood*, an individual may also acquire HIV infection. The HIV spread follows the parsimonious approach developed by [Chakraborty et al. (2016)]. Let $0 < p_t \leq 1$ be the probability that an HIV-susceptible young adult acquires HIV, defined as:

$$p_t = 1 - (1 - i_t \pi_t)^{\mu}, \tag{1}$$

where i_t is the proportion of parents who are HIV infective at time t (which represents the rate of HIV prevalence), $0 < \pi_t \leq 1$ is the probability of acquiring infection per sexual partnership with an infected individual and $\mu > 0$ represents the average number of sexual partnerships of a young adult individual during his entire young adulthood. If the population is large, the prevalence rate at time t amongst young adults converges to the probability that a young adult agent can be HIV-infected, i.e. $i_t = p_{t-1}$ for any t. As detailed in the introduction, unlike [Chakraborty et al. (2016)] we did not link HIV spread to agents' rational behaviour (i.e., private investment activities against HIV) for reasons related to the characteristics of HIV and the socioeconomic context prevailing in SSA. Instead, we assume that the probability π_t negatively depends on public treatments per worker against HIV (h_t), which are endogenously managed by the government of the afflicted country, and the amount of foreign aid donations on a per worker basis (D_t). Then,

$$\pi_t = \frac{\pi_A}{1 + \pi_B (h_t + D_t)^z}, \tag{2}$$

where $z > 0$ is a parameter that controls the degree of effectiveness of the overall amount of resources devoted to the intervention aimed at fighting the disease, $0 < \pi_A \leq 1$ ($\pi_A > 1/\mu$) and $\pi_B > 0$. We will turn to the discussion of both the expenditure and revenue sides of the government health policy later in this article.

Each young member of generation t is endowed with one unit of time that can be spent working. We assume that HIV reduces the (labour) productivity of an individual who is infected with probability p_t of an amount equal to $0 < \theta < 1$ of his time endowment [Ferreira et al. (2011); Chakraborty et al. (2016)]. Therefore, the remaining share $\ell_t = 1 - \theta p_t > 0$ of time is supplied to firms in exchange for wage w_t per unit of labour. Let $n_t > 0$ be the number of (surviving) children at time t. Then, $q w_t \ell_t n_t$ is total cost for caring n_t surviving descendants of a parent that belongs to generation t, which represents a proxy for the time cost of children, where $0 < q < 1$ is the fraction of the labour income used for upbringing purposes. The probability of surviving from youth to old age ($0 < \beta_t \leq 1$) is endogenous and determined by the individual's state of health when young, which in turn negatively depends on the prevalence rate of HIV infection i_t. For simplicity, we disregard all causes of death of young adults different from AIDS so that their survival probability to old age in absence of HIV will be 100%. In the presence of HIV, HIV-infected young adults are all assumed to die before entering old age. Therefore,

$$\beta_t = 1 - i_t. \tag{3}$$

Note that the inclusion of other causes of death would simply amount to multiply $1 - i_t$ by a positive constant smaller than one representing survival to everything nut HIV, as above hypothesised.

The formulation of endogenous lifetime in (3) allows to account for the most important effects of premature death of adults due to HIV/AIDS at the macroeconomic level (through the individual reaction of saving and the number of children). The overall amount of expenditure comprised of external donations and internal public investments acts exactly as a positive externality which individuals take as given. We believe this captures the essence of the intervention against HIV in SSA, quite unlikely compared to the formulation used by [Chakraborty et al. (2016)], which instead may be an adequate representation of disease prevention in developed countries.

Available (labour) income is used by each young adult to consume (c_t), save (s_t) and take care of n_t surviving children (whose cost per child is $qw_t\ell_t$). Therefore, the budget constraint when young reads as follows:

$$c_t + s_t + qw_t\ell_t n_t = w_t\ell_t(1 - \tau_t), \tag{4}$$

where is $0 \leq \tau_t < 1$ the HIV-specific tax rate levied by the government on worker's income. We assume that the tax rate may vary over time as the government may alternatively have a target in terms of health expenditure or collected revenues. The existence of a perfect market for annuities allows an agent to make his own saving being intermediated through mutual funds. This assumption follows a recent established literature on endogenous lifetime in growth models dealing with economic development issues [Chakraborty (2004); Fanti and Gori (2014); Chakraborty et al. (2016)]. Therefore, old-age material consumption (d_{t+1}) is constrained by the capitalised amount of resources saved when young divided by the survival probability of a young person of generation t (given the HIV epidemic and health prevention). Under this hypothesis, the savings of those who prematurely die due to HIV are allocated amongst those who are still alive (see [Chakraborty (2004)] for details). Therefore,

$$d_{t+1} = \frac{R^e_{t+1}}{\beta_t} s_t, \tag{5}$$

where R^e_{t+1} is the interest factor that an individual of generation t expects will prevail from time t to time $t+1$ (it will become the realised interest factor at the beginning of period $t+1$). By using Eqs. (4) and (5), the lifetime budget constraint can be written as follows:

$$c_t + \frac{\beta_t d_{t+1}}{R^e_{t+1}} + qw_t\ell_t n_t = w_t\ell_t(1 - \tau_t). \tag{6}$$

The left-hand side of (6) includes the present value of material consumption of an individual during the course of his lifetime plus the fraction of income that should be used to rise children. The right-hand side represents the available (after-tax) income.

By normalising the utility flow from death to zero, the expected lifetime utility function captures individual preferences towards material consumption (c_t) and the number of children (n_t) when young and material consumption when old (d_{t+1}). We use the following additively separable formulation, which is usual in the OLG context [e.g., de la Croix and Michel (2002); Fanti and Gori (2013); Spataro and Fanti (2011)], resembling the standard Constant Inter-temporal Elasticity of Substitution (CIES) function, that is

$$U_t = \left(1-\frac{1}{\sigma}\right)^{-1} c_t^{1-\frac{1}{\sigma}} + \beta_t \left(1-\frac{1}{\sigma}\right)^{-1} d_{t+1}^{1-\frac{1}{\sigma}} + \gamma \left(1-\frac{1}{\sigma}\right)^{-1} n_t^{1-\frac{1}{\sigma}}, \tag{7}$$

where $\sigma > 0$ ($\sigma \neq 1$) is the constant inter-temporal elasticity of substitution. Empirical evidence [Hall (1988); Guvenen (2006); Jones and Schoonbroodt (2010); Havranek et al. (2015)] finds that the elasticity of substitution in consumption is consistently smaller than one (although this index was not estimated under endogenous mortality). As this model employs the notion of expected utility due to the existence of a surviving probability of adults, we should exclude the cases of both log-utility ($\sigma = 1$) and $\sigma < 1$ to avoid paradoxical effects of adult mortality on lifetime utility [Rosen (1988); Hall and Jones (2007)]. This is because when $\sigma = 1$ the expression in (7) boils down to $U_t = \ln(c_t) + \beta_t \ln(d_{t+1}) + \gamma \ln(n_t)$. As a consequence, we should always be able to guarantee values of d_{t+1} larger than 1. Otherwise, an increase in the length of life would reduce utility. This argument also holds for the case $\sigma > 1$ irrespective of the value of d_{t+1}. In order to overcome this problem, in the numerical experiments we will use values of σ larger than 1 but close enough to the case of log-utility.

By taking as given factor prices, the health tax rate and the economic effects of the epidemic, the individual representative of generation t maximises the expected utility function (7) with respect to c_t, d_{t+1} and n_t subject to the lifetime budget constraint (6). Then, saving and fertility are given by:

$$s_t = \frac{\beta_t (R^e_{t+1})^{\sigma-1} w_t \ell_t (1-\tau_t)}{1+\beta_t (R^e_{t+1})^{\sigma-1} + \gamma^\sigma (q w_t \ell_t)^{1-\sigma}}, \tag{8}$$

$$n_t = \frac{\gamma^\sigma w_t \ell_t (1-\tau_t)}{(q w_t \ell_t)^\sigma [1+\beta_t (R^e_{t+1})^{\sigma-1} + \gamma^\sigma (q w_t \ell_t)^{1-\sigma}]}, \tag{9}$$

where π_t and β_t are given by (1) and (3), respectively, and $\ell_t = 1-\theta p_t$. As expected, an increase in the survival probability of adults causes a positive direct short-run effect on saving because individuals live longer (i.e., high mortality leads individuals to significantly underestimate their future lifetime by reducing savings today) [Chakraborty (2004)]. It also causes a negative direct short-run effect on fertility because individuals become healthier and decide to reduce the number of children as they will assistance to a lesser extent when they will be old. This is a standard result that mimics the unambiguous negative relationship between adult survival and fertility at the onset of the demographic transition pattern [Blackburn and Cipriani (2002); Fanti and Gori (2014)]. Changes in the survival probability β_t negatively depends on changes in both the probability of being infected per sexual partnership with an infected individual, π_t,

and the rate of HIV prevalence, i_t. Then, a reduction in π_t and/or i_t increases β_t and this in turn produces the direct effects discussed above on saving and fertility. However, there also exist changes in the survival probability produced by an indirect general equilibrium effect passing through prices (wage and interest factor) due the dynamics of capital and HIV prevalence. This will be analysed later together with the incidence of foreign aid and internal HIV-related expenditure on GDP and fertility.

Firms are identical and act competitively on the market. At time t the representative firm produces a homogeneous good (Y_t) by combining capital (K_t) and labour (L_t) by means of the following Cobb-Douglas technology with constant returns to scale:

$$Y_t = AK_t^{\alpha} L_t^{1-\alpha}, \tag{10}$$

where $0 < \alpha < 1$ is the output elasticity of capital and $A > 0$ is a constant production scaling parameter that weights technological progress (Total Factor Productivity). The temporary equilibrium condition in the labour market at time t is determined by equating labour demand and labour supply, that is $L_t = \ell_t N_t = (1 - \theta p_t)N_t$. The previous equation includes the possibility of HIV-reduced labour productivity in line with [Chakraborty et al. (2016)]. The issue of reduced productivity is not relevant for the conclusions of this paper and is included only for generality. We better discuss its implications in the discussion. By assuming full depreciation of capital, a unit price of output and taking factor prices as given, profit maximisation by the representative firm implies that the wage and the interest factor are equal to the marginal product of labour and the marginal product of capital, respectively, that is:

$$w_t = w(k_t) := (1 - \alpha)Ak_t^{\alpha}, \tag{11}$$

$$R_t = R(k_t) := \alpha Ak_t^{\alpha-1}, \tag{12}$$

where $k_t := K_t/L_t$ is the stock of capital per worker.

The government of the afflicted country collects resources in every period specifically dictated to fight HIV/AIDS. The ultimate amount of resources mobilised against the epidemic is given by the sum of external (exogenous) donations coming from foreign aid and internal (endogenous) resources. The former is simply a fully exogenous time dependent variable $D_t \geq 0$ reflecting any exogenous expenditure impacting on the probability of transmission, which is supplied in any form to the population of the afflicted country. The latter takes the usual form of public expenditure against HIV financed at a balanced budget through labour income taxation [Chakraborty (2004); Fanti and Gori (2014)]. The total government expenditure is $H_t = h_t L_t$, whereas the revenue aimed at covering health prevention and caring against HIV for young adults is given by the amount $\tau_t w_t L_t$. Therefore, the governement constraint expressed in per worker terms reads as follows:

$$h_t = \tau_t w_t. \tag{13}$$

Market-clearing in the capital market is determined by equating aggregate investment and aggregate saving and it is given by $K_{t+1} = S_t = s_t N_t$. Knowing that $N_{t+1} = n_t N_t$ determines the evolution of population, $k_{t+1} := K_{t+1}/L_{t+1}$ is the stock of capital per worker and $L_{t+1} = \ell_{t+1} N_{t+1} = (1 - \theta p_{t+1}) N_{t+1}$, equilibrium implies:

$$k_{t+1} = \frac{s_t}{n_t(1 - \theta p_{t+1})}, \tag{14}$$

where s_t and n_t are respectively given by the expressions in (8) and (9) and p_{t+1} is determined by the one-period forward Eq. (1). Equilibrium of the macro-economy is completely defined by a two equations describing the equilibrium in the capital market

$$k_{t+1} = \beta_t (R^e_{t+1})^{\sigma-1} \left(\frac{q}{\gamma}\right)^{\sigma} w_t^{\sigma} (1 - \theta p_t)^{\sigma} \frac{1}{1 - \theta p_{t+1}}, \tag{15}$$

and the equilibrium of the prevalence rate of HIV

$$i_{t+1} = 1 - (1 - i_t \pi_t)^{\mu}. \tag{16}$$

Definitely, equilibrium dynamics are obtained under the assumption of perfect foresight, that is $R^e_{t+1} = R(k_{t+1})$, and are characterised by the following two-dimensional map:

$$M : \begin{cases} k_{t+1} &= (1 - i_t) R(k_{t+1})^{\sigma-1} \left(\frac{q}{\gamma}\right)^{\sigma} w(k_t)^{\sigma} [1 - \theta p(k_t, i_t)] \frac{1}{1 - \theta p(k_{t+1}, i_{t+1})} \\ i_{t+1} &= 1 - [1 - i_t \pi(k_t)]^{\mu} \end{cases}, \tag{17}$$

where

$$\pi(k_t) = \frac{\pi_A}{1 + \pi_B [\tau w(k_t) + D_t]^z}, \tag{18}$$

$$p(k_t, i_t) = 1 - [1 - i_t \pi(k_t)]^{\mu}. \tag{19}$$

3 Numerical experiments

Consistently with the major goal of the work, this section compares three main scenarios: A) the case where the intervention against HIV/AIDS was completely provided by foreign aid at a fixed amount D_0 per worker each time period until the epidemics is brought under full control (we also call this the *full donors* scenario); B) the case where the intervention was completely provided by the government of the afflicted country through an HIV-specific tax rate on workers' income according to (13), such that the resulting revenue - to be used to finance HIV intervention - is exactly identical to that set in Scenario A (*full government*); C) the case where foreign aid donations are reduced over time according to the rule $D_t = D_{t-1}(1 - a)$, where $0 < a < 1$ is a decay rate, and they are replaced by the endogenous public intervention, which is set in order to exactly offset foreign aid by the same amount to keep unaltered the ratio between HIV-specific expenditure and GDP (per worker) (*mixed*). In all intervention

scenarios, the policy is announced by the policy maker who is managing the intervention (that is, either the donors or the government of the afflicted country or both) one period before the official start of the programme.

Simulations were designed as follows. The economy was initialised and allowed to reach its steady-state equilibrium in the absence of HIV with a Total Fertility Rate varying in the range 3.0-5.0 to mimic fertility in high HIV-prevalent SSA countries prior to the onset of HIV. Indeed, we recall that the most severe HIV epidemics in SSA have occurred in countries that were experiencing higher GDP and an already initiated fertility decline [see Gori et al. (2017) for a discussion on this issue]. Then, HIV was initialised starting from the steady state of the economy in the complete absence of any control interventions ($D_t = \tau_t = 0$) and parametrised in order to achieve an equilibrium prevalence of 30-35%, consistently with the peaks observed in the most severe epidemics in SSA countries [UNAIDS (2017)].[1] Once the equilibrium prevalence has been achieved, intervention activities are initiated according to Scenarios A, B and C described above. The assignment of the main demo-economic and epidemiological parameters was carried out according to the values reported in Table 1. Briefly, the main economic parameters were borrowed from the literature; the HIV parameters in the absence of interventions were tuned ad hoc in order to replicate an epidemic reaching an equilibrium prevalence in the range of 30-35%; the parameters tuning the effects of the interventions on the HIV-transmission probability were set as simulation parameters, by setting a desired target in terms of epidemic mitigation; policy parameters were adjusted in order to achieve this target, with an initial ratio between HIV-specific expenditure and GDP (per worker) in the various scenarios of about 2.5%; finally, the initial condition of the prevalence in the free HIV epidemics was set to 1%.

Parameter	Value	Source
A	200	Simulation
α	0.33	[Gollin (2002)]
q	0.2	Simulation
γ	6	Simulation
σ	1.01	Simulation
z	1	[Chakraborty et al. (2016)]
π_A	0.01	[Chakraborty et al. (2016)]
π_B	0.028	Simulation
μ	123	Simulation
θ	0.15	[Chakraborty et al. (2016)]

Table 1. Parameter assignment used in the numerical illustrations.

In the absence of HIV, the model predicts the achievement of economic and demographic

[1] Sensitivity analyses on the target prevalence and a number of simulation parameters have been carried out and are available on request, but they do not modify the key findings of this work.

variables to their long-term steady-state values, according to the standard prediction of the neoclassical model, as is shown by the flat portions of the curves for $t < 8$ (Fig. 1, Panels B and C, black solid line). The HIV epidemic is initialised at $t = 8$ in the absence of any interventions, and shows its S-shaped free temporal course achieving its equilibrium prevalence of about $\bar{\imath} = 0.35$ in a span of about 50 periods of time (Fig. 1, Panel A, black dash-dotted line). The main demo-economic consequences of the uncontrolled epidemic is via the direct effect following from the increase in adult mortality, which in turn causes both a fertility relapse, with an increase in the number of children per women, and a reduction in the willingness to save during young adulthood due the individual expected shorter life span [Chakraborty (2004)], as is clear from (9) and (8). As a consequence, capital accumulation and GDP per worker (Fig. 1, Panel C, black dash-dotted line) will approach lower steady state values.

The initiation of the intervention against HIV starts at the same time $t = 50$ in all the three intervention Scenarios A, B and C described above. Consistently with the hypothesis that the same amount of resources is allocated for all scenarios, the outcome in terms of HIV/AIDS control results to be the same, yielding an (almost) identical temporal trend of the HIV prevalence curve in all control scenarios. The rapid convex shaped decline of the HIV prevalence is the consequence of the hypothesis stated above of setting the intervention to its maximal level since its very beginning. More realistic patterns of epidemic control, as observed in the data [UNAIDS (2017)], can be easily obtained by simply assuming an increasing profile of the amount of resources devoted to interventions over time.

Under Scenario A (full donors), the fertility response to the reduced HIV prevalence, which allows adult survival to gradually relapse to its level prevailing before the AIDS crisis, is to straightforwardly react to the resulting mortality decline, by re-approaching the pre-AIDS steady-state level (Fig. 1, Panel B, black dotted line). At the same time, also saving recovers, due to the reduced adult mortality, which in turn causes, together with the reduction in fertility, an increase in capital accumulation and GDP per worker that eventually approach the higher steady state level that was prevailing in the pre-AIDS era.

The more interesting result occur when an endogenous public policy is considered as in Scenarios B and C. Under Scenario B (full government), the fertility response to the more benign epidemics conditions is much more dramatic than in the full donors scenario. It is to be remarked that in this case the ultimate fertility remains permanently lower than its pre-AIDS crisis level (Fig. 1, Panel B, black dashed line). This follows from the fact that the present result postulates the continued presence of the policy for the whole relevant horizon of the epidemics and this will continuously display its depressing effects on fertility (clearly, one could argue that once the emergency of AIDS will fully disappear, this will also remove the need for a dedicated policy, but this is outside of the interest of this article). The results of this scenario are the consequence of its greater complexity of compared to the full donors case. In fact, the endogenous public policy implies as a direct effect the relapse in adult survival following the reduced HIV prevalence (as in Scenario A, this has a direct depressing effect on fertility).

However, Scenario B also predicts another depressing effect on fertility that passes through the reduction in the disposable income of individuals following taxation, which also contributes to reduce saving. This second effect can be interpreted as a standard Malthusian response of individual fertility to the success in epidemic control allowed by the policy. The results of the mixed scenario (C) are intermediate between the two previous cases and can be fully explained by previous arguments (Fig. 1, red lines). Notably, the long-term capital accumulation and GDP per worker are the same as in the pre-AIDS era despite the different saving because of the different response of individuals to saving and fertility. Surprisingly, the net balance on GDP is essentially neutral: individuals save less but also they have less children than in the full donors case.

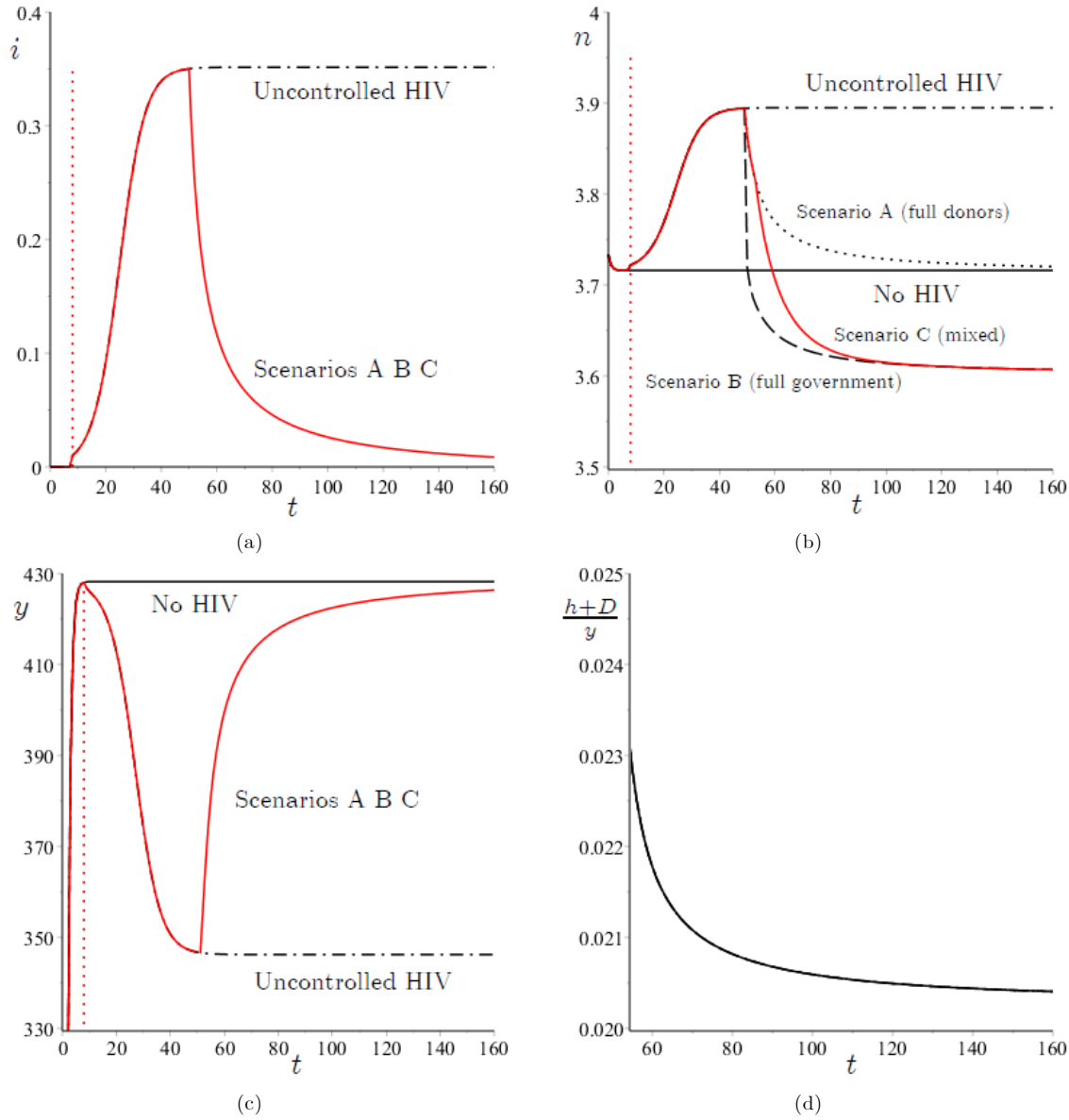

Figure 1. Time series of HIV prevalence (Panel A), Fertility (Panel B), GDP per worker

(Panel C) and the ratio between HIV-related expenditure and GDP per worker (Panel D). The vertical red dotted lines in the figures indicates the timing of initiation of HIV.

4 Conclusions

WHO and UNAIDS have launched a global plan aiming to bring AIDS under full control by 2030. A critical debated issue in relation to this target is how to finance the huge amount of resources needed to fight the epidemics, by treating and taking care of the seek people and by maintaining a high degree of societal awareness [Haacker (2009); Resch et al. (2015); Remme et al. (2016)]. In this study, we have provided the first (to the best of our knowledge) attempt to combine general equilibrium macroeconomic dynamics together with an explicit temporal trend of the epidemic under a range of possible options with regard to the funding of the policy aimed to control HIV, ranging from a fully external policy based on foreign aid up to a fully endogenous policy managed by the government of the afflicted country. The endogenous policy scenario yields a much richer response of the economic and demographic variables, which passes through the rise-and-fall of fertility to the evolving mortality during the rise and fall of the epidemics but also to the general equilibrium macroeconomic response via a Malthusian effect. Though the predictions of our model on the magnitude of the fertility response to AIDS mortality are surely unreliable on a quantitative standpoint, given that the model was not calibrated on real data, they should be considered carefully for their qualitative content. Indeed, these predictions add theoretical evidence of the possibility of a paralysis in the fertility decline in SSA following the mortality upturn due to the HIV/AIDS epidemic. This issue, which is a potentially critical one for the perspectives of development of SSA, is widely debated in the current empirical economic literature, where it has received a number of supports [Kalemli-Ozcan and Turan (2011); Kalemli-Ozcan (2012); Akbulut-Yuksel and Turan (2013); Juhn et al. (2013); Chin and Wilson (2018)], and has been predicted in a theoretical article dealing with the impact of HIV/AIDS on the fertility transition by [Gori et al. (2017)].

We are aware of the many limitations of this work, which only intended to start a debate on a critical issue by using a toy model. For example, in our framework, we allocated the amount of (foreign and domestic) interventions against HIV/AIDS on a per worker basis, which is a convenient modelling simplification. Empirical analyses have suggested that the criteria adopted for allocating external financing to fight HIV in SSA have been somewhat variable but mostly related with gross domestic product per capita in afflicted countries and, instead, somewhat unrelated with the severity of the epidemic [Haacker (2009)]. Both these criteria could be accommodated in a refined model. Also, the amount of interventions and the timing of initiation adopted in the numerical simulations has been set crudely with respect to the complexity of the interventions against HIV in SSA but consistently with the simplistic timing of the OLG model. Moreover, the model does not include the fact that in the presence of

effective therapies, also AIDS-seek individuals can be cured and would then benefit of an extended life expectancy. However, the previous ones are only examples amongst the endless list of important missing details that should be included in a more realistic description of Sub-Saharan Africa economies, with their amazing socioeconomic and cultural heterogeneities, as well as a more faithful representation of the HIV epidemics and its impact of the society as a whole. These aspects represent non-trivial complications that should be integrated in a much more extended research programme than was the objective of this work.

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Conflict of Interest The authors declare that they have no conflict of interest.

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