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

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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 autonomous in designing and implementing their own intervention policies against 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 mortality forced by the threat of a persistent, deadly infectious disease, such as HIV/AIDS in SSA. Second, it aims to shed light on the interplay between foreign aid and endogenous domestic public policies in SSA countries severely afflicted by HIV. Consequently, it investigates the demographic and macro-economic implications of an intervention against HIV/AIDS whose total amount is the sum of an exogenous component representing foreign aid and an endogenous public expenditure. On the assumption that these policies allow the same degree of HIV control, we show the emergence of quite different responses in terms of key demo-economic variables. These effects mainly pass 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

1 Introduction

With about 76 million people infected and 35 million people dead from related illnesses since the beginning of the epidemic [UNAIDS (2017)], HIV/AIDS represents the worst pandemic in recent history. It is however in Sub-Saharan Africa (SSA) that AIDS has reaped the highest toll, causing a tragedy of major proportions. Still in 2016, out of a total of 36.7 million people living with HIV, with an 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, 64.5% of new infections and 73% of total estimated deaths [UNAIDS (2017)]. Nonetheless, for some years now, epidemic trends have finally downturned thanks to the continued efforts aimed at increasing population awareness by prevention campaigns and expanding the proportion of seropositive and AIDS-sick people having access to effective antiretroviral therapies (ART). While in 2004-2005 (the epoch of the mortality peak in SSA), the proportion of people who had access to treatments was negligible, in 2016 54% of seropositive in SSA received ART. These efforts are allowing a substantial reduction of both HIV incidence and AIDS mortality, which has nearly halved in the last decade [UNAIDS (2017)]. However, these positive results should not obscure that AIDS remains the second most important cause of mortality in SSA [WHO (2017)].

Full success in this battle will require a further massive long-term expansion of resources devoted to HIV. This should aim to (1) making most seropositive people aware of their status, (2) providing them with effective and lifelong treatments and (3) maintaining high rates of public awareness. Given the high cost of a single therapy protocol, the overall cost of large-scale lifelong HIV treatments in SSA would be dramatic, eventually reaching levels as high as 80% of current GDP [Collier and Sterck (2018)]. This raises an issue about the affordability

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

The dramatic extent of foreign aid in the AIDS response, which in 2013 reached 90% of total expenditures on AIDS in low-income countries, can have several shortcomings. 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, concerns 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)] of developing a country-specific response entirely relying on external resources in terms of e.g., organising optimally the related public health infrastructures and keeping internal awareness high.

In prospective terms, the key emerging issue is clearly the transition towards a system where SSA countries gradually acquire autonomy in designing their HIV/AIDS policy. Public health and health economics are actively tackling this issue [Katz et al. (2014); Resch et al. (2015); Atun et al. (2016A); Remme et al. (2016)], which stands at the interplay between the epidemic and the stage of economic development, under multiple constraints arising from (a) medium-term GDP growth perspectives, (b) threats from other diseases that may absorb relevant quotas of GDP, (c) ability to develop new financial tools to face the HIV challenge [Resch et al. (2015); Atun et al. (2016B); Remme et al. (2016)].

In this article, we aim at investigating the macro-economic and fertility effects of AIDS interventions, where the overall amount of related resources is the sum of an endogenous public expenditure entirely managed by the government of the afflicted country and of an exogenous

component reflecting international donations. We assume that external donations are directly provided to households without any government intermediations to avoid, given the dramatic extent of corruption in SSA [Gossel (2018)], the inclusion of public institutions to which international donors should commit the management of resources for the private sector.

After a provocative work by Young (2005), suggesting that the AIDS tragedy will eventually improve the welfare of future SSA generations, and the ensuing response of Kalemli-Ozcan and Turan (2011), obtaining opposite conclusions by the same data, a growing macroeconomic literature has emerged on the interplay between economic development and infectious diseases [e.g., Boucekkine et al. (2009); Castro et al. (2015), Azomahou et al. (2016)]. In two influential papers, Chakraborty et al. (2010, 2016) began the investigation of the impact of deadly infections on economic growth by using a general equilibrium overlapping generations (OLG) model à la Diamond (1965). They provided a parsimonious and innovative representation of infection that was not simply modelled as a short-term exogenous shock, but was assumed to follow an explicit dynamic rule, governed by meaningful epidemiological parameters. Nonetheless, they did not explicitly consider the endogenous (general equilibrium) feedback between infections and demographic variables at the macroeconomic level. Modelling this feedback is key 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 poorest region worldwide. In addition, in their models individuals were able to internalise the effects of the epidemics by choosing disease-specific prevention investments. We believe this approach is hardly tenable for SSA, which suffers inadequate health infrastructures, severe shortages of physicians and medicines, low education and poor health causing high mortality even in the absence of HIV. This questions whether individuals living in SSA can adequately protect themselves by devot-

ing own resources to prevent infectious diseases, as this expenditure may neither substitute nor complement public interventions from domestic resources or foreign aid.

Our major task is to investigate the feasibility for AIDS-afflicted SSA countries to gradually achieve independence from international support and develop a fully internal response to the disease. As was explained above, this question cannot be set into the framework developed by Chakraborty and coauthors. Therefore, we propose a Diamond-like model where the reaction to the epidemic is based on a combination of endogenous public spending and external donations. This is done in a framework that aims at capturing some key aspects of HIV/AIDS in SSA, by including endogenous adult mortality from AIDS as well as the individual endogenous fertility response (child quantity) to AIDS-related mortality. In addition, we compare the effects of two different and opposite assumptions regarding the inter-generational transmission of inheritance under uncertain survival, namely accidental bequests versus perfect annuities. The latter hypothesis, adopted in Chakraborty et al. (2016), is clearly well suited for settings where financial markets are adequately developed, which is not the case for most current SSA countries, except for South Africa.

Our main results are as follows. Setting a prescribed outcome in terms of HIV control, namely bringing HIV incidence to about zero in a few generations, the responses of the demo-economic system largely depend on whether the interventions are funded by international donations or by an endogenous public policy. The latter scenario yields, compared to the former one, a more articulated response of income and fertility passing through an intermediate phase of fertility reversal before restoring the pathway of fertility decline. This is due to the evolving mortality during the rise and fall of the HIV epidemic but also to the general equilibrium macroeconomic response via a Malthusian effect.

The rest of the article proceeds as follows. Section 2 presents the model. Section 3 reports and discusses the main results in the light of the related economic literature. Section 4 outlines the main conclusions. The Appendix reports further details on the role of accidental bequests and perfect annuities by comparing this issue in OLG models with endogenous and exogenous fertility.

2 The model

Consider a general equilibrium OLG closed economy with identical, finite-lived and rational individuals, whose size at birth is N_t per generation ($t = 0, 1, 2, ...$). The life of the representative agent is divided into young adulthood and old age. As a child, an individual directly consumes resources in the parent's household without making any economic decisions. This is a deliberate simplification ignoring the mortality burden amongst children (including those dying by vertically transmitted AIDS) because our aim is to focus on the major source of AIDS mortality, namely adult mortality. Therefore, our key variable is the number of children surviving at the entry of adulthood [Galor and Weil (1996); Gori and Sodini (2019)]. As a young adult, an individual is economically and sexually active. He works, consumes, saves, gives birth and takes care of children. When old, he is retired and consumes based on his saving. During young adulthood, an individual may also acquire HIV infection. The HIV infection spreads along Chakraborty et al. (2016), who defined the probability $0 < p_t \leq 1$ that an HIV-susceptible young adult acquires the infection:

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

In Eq. (1), i_t represents the proportion of HIV-infective parents at time t (also termed the HIV prevalence), $0 < \pi_t \leq 1$ is the probability of acquiring the infection per sexual partnership and $\mu > 0$ represents the average number of sexual partners during young adulthood. If the population is large, the HIV prevalence at time t amongst young adults converges to the probability that a young adult is seropositive, i.e. $i_t = p_{t-1}$ for any t. As detailed in the introduction, unlike Chakraborty et al. (2016) we do not link infection spread to individual rational behaviour (i.e., private investments against HIV) because of the characteristics of the HIV epidemic and the socioeconomic context of SSA. Instead, we assume that π_t can be reduced either by appropriate interventions on sexual behaviour (first of all, by awareness of risk, e.g. by avoiding partners at risk or using condoms), or by pharmaceutical treatments aimed to reduce infectivity (e.g., antiretroviral treatments) or even by appropriate medical practices (e.g., male circumcision). These interventions can be financed in different ways. Here, we consider two main financing routes: foreign aid donations, which so far have represented the most important funding source for the response against HIV/AIDS in SSA [UNAIDS (2017)], and public expenditures managed by the government of the afflicted country. Formally, we adopt the following formulation for π_t:

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

where 1) h_t represents the amount of endogenous public expenditures against HIV/AIDS on a per worker basis, 2) D_t is the amount of foreign aids on a per worker basis (so that $h_t + D_t$ is the total expenditure per worker), 3) $0 < \pi_A \leq 1$ ($\pi_A > 1/\mu$) and $\pi_B > 0$ are exogenous constants and 4) $z > 0$ is a constant tuning the effectiveness of interventions on the transmission probability. The term $h_t + D_t$ acts as an externality that individuals take as given. We believe this captures the essence of the intervention against HIV in SSA, at odds with Chakraborty et

al. (2016), whose formulation is possibly adequate for developed countries.

Each young adult of generation t is endowed with one unit of time that can be spent working. We assume that HIV reduces the (labour) productivity of the infected of an amount $0 < \theta < 1$ of their time endowment [Chakraborty et al. (2016)]. Therefore, the remaining share $\ell_t = 1 - \theta p_t > 0$ 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, $qw_t\ell_t n_t$ is the total cost the parent incurred for caring them, where $0 < q < 1$ is the fraction of the labour income used for caring activities. 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 HIV prevalence. For simplicity, we disregard all causes of death of young adults different from AIDS so that their survival probability to old age in the absence of HIV will be 100%. In the presence of HIV, all HIV-infected young adults will prematurely die before entering old age, with probability

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

Note that including other causes of death would simply mean multiplying $1 - i_t$ by a positive constant smaller than one representing survival to causes of death different from HIV.

Unlike the recent literature on endogenous lifetime [Chakraborty (2004); Fanti and Gori (2014); Chakraborty et al. (2016)], in this work we assume the existence of accidental bequests rather than a market for annuities. We believe this assumption is more suited for capturing the working of intergenerational transfers under uncertain survival in a context, such as current SSA, where financial markets are still poorly developed. Accidental bequests imply that savings of a deceased person are equally bequeathed in full to his own descendants. Differently, perfect annuities imply that when a person dies at the onset of old age his savings are divided amongst

all the members of the generation, so that old survivors will benefit not only from their own past savings plus interest but also from savings plus interest of those who have died. Savings are then allocated to a mutual fund and invested in order to guarantee a gross return that depends on mortality rates of the surviving old agents (which are all annuitised).

Since (old) agents do not know when they will die, unintentional bequests can occur. If the typical agent of generation t prematurely dies from AIDS (with probability $1-\beta_t$) at the onset of old age, his accumulated savings (s_t) are bequeathed to his heirs. To keep the formulation tractable, we assume that the bequest-dependent wealth distribution is uniform [Hubbard and Judd (1987)] so that the level of bequests $B_{t+1} := b_{t+1}L_{t+1} = b_{t+1}\ell_{t+1}N_{t+1}$[1] should be equal to $(1-\beta_t)R^e_{t+1}s_tN_t$ [Fanti (2009)], where b_{t+1} are bequests per worker and R^e_{t+1} is the expected interest factor from time t to time $t+1$. Therefore, as $N_{t+1} = n_tN_t$ we get

$$b_{t+1} = (1-\beta_t)R^e_{t+1}\frac{s_t}{n_t\ell_{t+1}}. \tag{4}$$

Available (labour) income plus the bequest inherited from generation $t-1$ are used by each young adult of generation t to consume (c_t), save (s_t) and take care of n_t surviving children. Therefore, the budget constraint when young is:

$$c_t + s_t + qw_t\ell_tn_t = w_t\ell_t(1-\tau_t) + b_t, \tag{5}$$

where $0 \leq \tau_t < 1$ is the tax rate levied by the government on worker's income to finance HIV interventions. We assume that τ_t may vary over time as the government may alternatively have a target in terms of health expenditure or collected revenues. Old-age material consumption (d_{t+1}) is constrained by the capitalised amount of resources saved when young, that is:

$$d_{t+1} = R^e_{t+1}s_t. \tag{6}$$

[1]The equality $L_{t+1} = \ell_{t+1}N_{t+1} = (1-\theta p_{t+1})N_{t+1}$ comes from the temporary equilibrium condition in the labour market at time $t+1$, which is determined by equating labour demand and labour supply.

By using Eqs. (5) and (6), the lifetime budget constraint is

$$c_t + \frac{d_{t+1}}{R^e_{t+1}} + q w_t \ell_t n_t = w_t \ell_t (1 - \tau_t) + b_t. \tag{7}$$

The left-hand side of (7) includes the present value of lifetime material consumption plus the fraction of income used to rise children. The right-hand side represents the available (after-tax) income plus inherited bequests.

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)]:

$$U_t = \left(1 - \frac{1}{\sigma}\right)^{-1} c_t^{1-\frac{1}{\sigma}} + \beta_t \left(1 - \frac{1}{\sigma}\right)^{-1} d_{t+1}^{1-\frac{1}{\sigma}} + \gamma \left(1 - \frac{1}{\sigma}\right)^{-1} n_t^{1-\frac{1}{\sigma}}, \tag{8}$$

where $\sigma > 0$ ($\sigma \neq 1$) is the constant inter-temporal elasticity of substitution. The magnitude of σ is a debated issue in the literature. Empirical research on either micro-oriented country-level heterogeneity [Havranek et al. (2015)] or aggregate consumption [Hall (1988)] generally found values of σ smaller than one, though other analyses based either on micro data [e.g., Browning et al. (1999)] or on macro data when accounting for precautionary savings [Gomes and Ribeiro (2015)] obtained the opposite result. We pinpoint, however, that σ was not estimated in models with endogenous demographics, with the exception of Jones and Schoonbroodt (2010). In the theoretical literature employing additively separable utility functions, σ is often assumed to be larger than one to ensure that consumption in both periods and leisure are gross substitutes [Cazzavillan and Pintus (1994)].[2] Consequently, we assume gross substitutability between consumption in both periods and fertility. In addition, as this model employs the notion of expected

[2]Two commodities are called gross substitutes if an increase in the price of one of them causes an increase in the demand of the other. This is because consumption shifts towards the cheapest good.

utility, we should exclude the cases of both log-utility ($\sigma = 1$) and $\sigma < 1$ to avoid paradoxical effects of adult survival on lifetime preferences [Rosen (1988); Hall and Jones (2007)]. In fact, when $\sigma = 1$ the expression in (8) boils down to $U_t = \ln(c_t) + \beta_t \ln(d_{t+1}) + \gamma \ln(n_t)$ implying that d_{t+1} should be larger than 1 both transiently and in the long-term, otherwise an increase in the length of life would reduce utility. In order to overcome this drawback, in the numerical experiments we will take σ larger than 1 in line with Gomes and Ribeiro (2015) and Pestieau and Ponthière (2017). Some clarifications on this issue are now useful. When lifetime utility includes life expectancy [Rosen (1988)] there may exist some paradoxical effects on consumer's choices that can be avoided by adding a positive constant to the per-period utility [Hall and Jones (2007); Jones and Schoonbroodt (2010)]. However, although this assumption allowed Hall and Jones (2007) to have well-behaved preferences, in this setting it does not help overcoming the possible negative relationship between material consumption and the length (i.e., quantity) of life. This is because (8) is additively separable and has the form: per-period utility multiplied by a parameter measuring life expectancy (β_t). Indeed, at least in the class of OLG models with finite-lived individuals employing this kind of utility functions, one must postulate a positive relationship between life expectancy and the utility drawn from material consumption (this is also related to other complicate questions as those dealing with the optimal duration of life). Our approach, therefore, is more related to the value of a statistical life (VSL) [de la Croix et al. (2012)]. The VSL is defined as the ratio between the marginal utility of an extra gain in the quantity of life divided by the marginal utility of old age consumption. It measures the cost in terms of material consumption when old that would be incurred for gaining an increase in the quantity of life. At the (centralised) social planner level, the VSL can be positive, negative or zero at the social optimum. If it is positive (resp. negative), the survival rate is a corner

solution given its highest (resp. lowest) possible value, i.e. 1 (resp. 0). If it is zero, there exists an interior solution at the social optimum. At the laissez-faire (decentralised) level, one can reasonably assume that the VSL is positive so that one should expect to get the highest possible survival rate [see de la Croix et al. (2012) for details]. Therefore, assuming $\sigma > 1$ is a simple but general way to avoid both negative utility and negative VSL.

By taking as given factor prices (w_t and R^e_{t+1}), the health tax rate (τ_t), the bequests (b_t) and the HIV prevalence (i_t), the representative agent of generation t maximises the expected utility function (8) with respect to c_t, d_{t+1} and n_t subject to (7). Then, saving and fertility are given by:

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

$$n_t = \frac{\gamma^\sigma [w_t \ell_t (1-\tau_t) + b_t]}{(q w_t \ell_t)^\sigma [1 + \beta_t^\sigma (R^e_{t+1})^{\sigma-1} + \gamma^\sigma (q w_t \ell_t)^{1-\sigma}]}, \tag{10}$$

where π_t and β_t are determined by the expressions in (1) and (3), respectively, and $\ell_t = 1 - \theta p_t$. From (9) and (10), an increase in the survival probability of adults causes a positive partial equilibrium 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)], and a negative partial equilibrium effect on fertility as there are fewer resources that can be spent to take care of children. This is a standard result that mimics the unambiguous negative relationship between adult survival and fertility at the onset of the demographic transition [Blackburn and Cipriani (2002); Fanti and Gori (2014)]. Changes in the survival probability β_t negatively depend on changes in both the HIV transmission probability π_t and infection prevalence i_t. Then, a reduction in π_t and/or i_t increases β_t thus causing the direct effects on saving and fertility discussed above. However, there also exist changes in the survival probability produced by an indirect general equilibrium effect passing through prices (wage and

interest factor) due to 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) through the standard Cobb-Douglas technology with constant returns to scale:

$$Y_t = AK_t^{\alpha} L_t^{1-\alpha}, \tag{11}$$

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 $L_t = \ell_t N_t = (1 - \theta p_t) N_t$. 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:

$$w_t = w(k_t) := (1 - \alpha) A k_t^{\alpha}, \tag{12}$$

$$R_t = R(k_t) := \alpha A k_t^{\alpha - 1}, \tag{13}$$

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 devoted to fight HIV/AIDS. The ultimate amount of resources mobilised against the epidemic is given by the sum $h_t + D_t$ of external donations and internal resources. The total tax revenue $\tau_t w_t L_t$ is used to finance the HIV intervention whose amount is $H_t = h_t L_t$. Therefore, the government

constraint expressed in per worker terms is [Chakraborty (2004); Fanti and Gori (2014)]:

$$h_t = \tau_t w_t. \tag{14}$$

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

$$k_{t+1} = \frac{s_t}{n_t \ell_{t+1}} = \frac{s_t}{n_t (1 - \theta p_{t+1})}, \tag{15}$$

where s_t and n_t are respectively given by (9) and (10) and p_{t+1} is determined by the one-period forward Eq. (1). Equilibrium of the macro-economy is completely defined by two equations describing, respectively, the equilibrium in the capital market

$$k_{t+1} = \beta_t^{\sigma} (R_{t+1}^e)^{\sigma-1} \left(\frac{q}{\gamma}\right)^{\sigma} w_t^{\sigma} (1 - \theta p_t)^{\sigma} \frac{1}{1 - \theta p_{t+1}}, \tag{16}$$

and the dynamic equation of HIV prevalence

$$i_{t+1} = 1 - (1 - i_t \pi_t)^{\mu}. \tag{17}$$

From (16), capital accumulation does not depend on bequest inheritance. This is because bequests positively affect in the same qualitative way both saving and fertility by increasing the income of the young members of the working generation. Equilibrium dynamics are obtained under the assumption of perfect foresight, that is $R_{t+1}^e = R(k_{t+1})$, and are characterised by the following two-dimensional map:

$$M : \begin{cases} k_{t+1} &= (1 - i_t)^{\sigma} R(k_{t+1})^{\sigma-1} \left(\frac{q}{\gamma}\right)^{\sigma} w(k_t)^{\sigma} [1 - \theta p(k_t, i_t)] \frac{1}{1 - \theta p(k_{t+1}, i_{t+1})} \\ i_{t+1} &= 1 - [1 - i_t \pi(k_t)]^{\mu} \end{cases}, \tag{18}$$

where

$$\pi(k_t) = \frac{\pi_A}{1 + \pi_B [\tau_t w(k_t) + D_t]^z}, \tag{19}$$

and

$$p(k_t, i_t) = 1 - [1 - i_t \pi(k_t)]^{\mu}. \tag{20}$$

3 Numerical experiments

3.1 Model dynamics in the absence of interventions

The economy was initialised to reach its steady-state equilibrium in the absence of HIV with a Total Fertility Rate (TFR) varying in the range 2.4-4.0 to mimic fertility in high HIV-prevalent SSA countries prior to the onset of HIV. We recall that the most severe HIV epidemics in SSA have occurred in countries that were experiencing higher GDP compared to the average value prevailing in the region and an already initiated fertility decline [see Gori et al. (2017) for a discussion on this issue]. We assume that in the absence of HIV the TFR achieves a long-term equilibrium slightly below 2.4 (which is slightly larger than the replacement level of 2.1).

HIV was initialised starting from the steady state of the economy in the 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 largest epidemic peaks observed in SSA [UNAIDS (2017)].[3] Bequests at time $t = 0$ have been set to zero as the first existing generation did not inherit resources from anyone. Then, the amount of resources inherited over time will depend on the mortality rate, which in turn depends on HIV prevalence.

[3] 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.

3.2 The baseline control scenarios

Once the equilibrium prevalence has been achieved, intervention activities are initiated according to the following "baseline" scenarios: A) the intervention against HIV/AIDS was completely provided by foreign aid at a fixed amount D_0 per each time period until the epidemic is brought under control (*full donors* scenario); B) the intervention was completely provided by the government of the afflicted country through an HIV-specific tax rate according to (14), such that the resulting expenditure for HIV intervention is the same as in Scenario A, that is the overall expenditure against HIV/AIDS is kept unaltered by letting the tax rate adjusting over time (*full government* scenario); C) 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 replaced by the public intervention, which is set to exactly offset foreign aid by the same amount, still by keeping unaltered the ratio between HIV-specific expenditure and GDP per worker (*mixed* scenario). In all 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 to allow (perfectly foresighting) individual to be aware of the policy thus avoiding issues of time inconsistency [Calvo (1978)].

The main demo-economic and epidemiological parameters were assigned according to the values reported in Table 1, setting the key parameters proposed in this model as free simulation parameters. In particular, the HIV parameters in the absence of interventions were set ad hoc to generate a worst-case epidemic reaching an equilibrium prevalence in the range of 30-35%, while those tuning the effects of the interventions on the transmission probability were adjusted to achieve HIV elimination in the medium-long term. The initial ratio between HIV-expenditure and GDP (per worker) in the various scenarios is set to 1.12%. Finally, the initial condition on

HIV prevalence was set to 10%.

[**TABLE 1 ABOUT HERE**]

In the absence of HIV, economic and demographic variables are predicted to achieve their neoclassical long-term steady-state values, as is shown by the flat portions of the curves for $t < 6$ (Fig. 1, Panels B and C, black solid line). The HIV epidemic is initialised at time $t = 6$ (red dotted line in the figures) in the absence of any interventions and shows its S-shaped free temporal course achieving its equilibrium prevalence of $\bar{\imath} = 0.35$ in a span of about 25 periods (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 woman, and a reduction in the willingness to save during young adulthood, due to the individual expected shorter life span [Chakraborty (2004)], as is clear from (10) and (9). The phenomenon of HIV-induced fertility reversal was first predicted by Kalemli-Ozcan (2012) based on empirical analysis of DHS data from SSA and then investigated in a Unified Growth Theory model by Gori et al. (2017). Therefore, capital accumulation and GDP per worker (Fig. 1, Panel C, black dash-dotted line) dramatically decline following increasing HIV prevalence by approaching substantially lower steady-state values.

The intervention against HIV starts at $t = 31$ in the three Scenarios A (dotted lines), B (dashed lines) and C (red lines). Consistently with the hypothesis that the same amount of resources is allocated for all scenarios, the outcome in terms of HIV/AIDS control is the same, yielding an identical temporal trend of HIV prevalence. The rapid (convex shaped) decline of HIV prevalence arises from setting the intervention to its maximal level from start rather than

considering a gradual increasing intervention (as considered in Section 3.3).

Under Scenario A (full donors), fertility responds to the reduced mortality allowed by the decline in HIV prevalence by re-approaching its pre-AIDS steady-state (Fig. 1, Panel B, black dotted line). At the same time, saving recovers due to the reduced adult mortality thus causing, together with the reduction in fertility, an increase in capital accumulation and GDP per worker that eventually restores the pre-AIDS steady-state.

The more interesting result occurs when an endogenous public policy is considered (Scenarios B and C). Under Scenario B (full government), the fertility response to the declining epidemic is dramatically different compared to the full donors' scenario. Fertility in this case remains permanently lower than its pre-AIDS level (Fig. 1, Panel B, black dashed line) because the continuation of the policy for the whole relevant horizon of the epidemic will continuously display its depressing effects on fertility. Clearly, one could argue that once the emergency of AIDS will disappear, this will also remove the need for a dedicated policy. At that stage, by re-setting to zero the tax rate the model will restore its natural equilibrium in the absence of the epidemic along the model time scale. The results of this scenario are the consequence of its greater complexity compared to the full donors' case. In fact, the endogenous public policy generates a direct effect by relapsing adult survival following the reduced HIV prevalence (as in Scenario A, this has a depressing effect on fertility). However, Scenario B also predicts another, indirect, depressing effect on fertility passing through the reduced (after tax) disposable income, which also contributes to reduce saving. This second effect can be interpreted as a standard Malthusian response of individual fertility to the success of the policy in controlling HIV. 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 rates. This is because of the different response of individuals to saving and fertility. Surprisingly, the net balance on GDP is essentially neutral: individuals save less but they also have less children than in the full donors' case.

[**FIGURE 1 ABOUT HERE**]

3.3 Alternative control scenarios

In this section we report results about a few alternative (possibly more realistic) circumstances.

Epidemic onset at still high levels of fertility Here we investigate (Figure 2) the three scenarios above considered by letting the HIV epidemic to begin at when the process of fertility decline is still ongoing with levels of the Total Fertility Rate far distant ($n = 2.9$) from its long-term equilibrium. The resulting dynamics are in line with those reported in Figure 1.

[**FIGURE 2 ABOUT HERE**]

Increasing levels of AIDS expenditure This scenario investigates the situation where the intervention against HIV initiates before the epidemic reached its equilibrium level by an increasing rate of expenditure (as empirically documented). The latter aspect is motivated by the fact that awareness of AIDS in SSA took a long time also at the level of international institutions. In this experiment, the temporal profile of donors' intervention is: (i) initiated when the epidemic prevalence has achieved the level of almost 10%, (ii) implemented by a linearly increasing external expenditure curve, reaching in five periods the level D_0 set in Scenario A.

As is shown in Figure 3, the low initial intensity of intervention is not sufficient to halt the epidemic increase calling for additional resources. However, as increases in D_t mitigate the epidemics by preventing prevalence to reach its equilibrium level, thus causing the downturn when prevalence is almost 20%, and eventually achieving disease elimination (Figure 3, left). This is consistent with what has been observed e.g., in South Africa. The resulting effects show that the HIV-induced fertility reversal brings the TFR uprising from the achieved level of 2.9 in the pre-AIDS era (still made broadly consistent with the case of South Africa) to a level of almost 3.1 before returning to a decreasing path only when prevalence starts declining (Figure 3, centre). The pattern of income per worker is consistent with the trends of the disease and fertility, going down during the major phase of the epidemics and subsequently relapsing on its growth path up to its pre-AIDS long-term equilibrium (Figure 3, right).

[**FIGURE 3 ABOUT HERE**]

4 Conclusions

AIDS has been a major tragedy capable to halt and reverse the slow but continued growth in life expectancy in Sub-Saharan Africa. WHO and UNAIDS have launched a global plan aiming to bring AIDS under full control by 2030. A critical debated issue about this target is how to finance the huge amount of resources needed to treating and taking care of sick people as well as maintaining a high degree of societal awareness [Haacker (2009); Resch et al. (2015); Remme et al. (2016)]. In this study, we have combined general equilibrium macroeconomic dynamics with a temporal trend of HIV epidemic under a range of possible options to finance the policy aimed to control the disease, ranging from external foreign aid (full donors) to internal public policy

(full government). Given the same target in terms of HIV control and the same expenditure in the two opposite scenarios, the ultimate consequences for demo-economic outcomes are different. The latter scenario implies a more articulated response than the former one passing from the rise-and-fall of fertility to the evolving mortality during the rise and fall of the epidemic but also capturing the general equilibrium macroeconomic response via a Malthusian effect. The fertility response under full government is larger than under full donors. Though the predictions of the model on the magnitude of the response of fertility to AIDS mortality are surely unreliable on a quantitative standpoint, as the model was not calibrated on actual data, they should be considered carefully for their qualitative content. These predictions add theoretical support on the possibility of a paralysis in the fertility decline in SSA following the mortality upturn due to HIV/AIDS. This issue, which is potentially critical for the perspectives of economic development in SSA, is widely debated in the economic literature receiving empirical support by Kalemli-Ozcan and Turan (2011), Kalemli-Ozcan (2012), Akbulut-Yuksel and Turan (2013), Juhn et al. (2013) and Chin and Wilson (2018) as well as theoretical support by Gori et al. (2017).

This work intended to start a debate on a critical issue by using a simple theoretical model. Future contributions should primarily account for the public finance issue on the side of both the expenditure and financing components, which is key in the cited public health economics literature. Beyond this, there are some issues to be considered. Much related to the core of this article, there exists a debate about the possibility to develop financial tools specifically targeted at supporting public interventions against HIV/AIDS in SSA [Atun et al. (2016A, 2016B)] without compromising development perspectives. In this framework, the length of the horizon in the battle against HIV suggests that in the meanwhile financial markets could

develop substantially, which makes it of interest to deepen the issues of private insurance (i.e., markets for annuities) and inheritance transmission. Although we assumed accidental bequests, given current perspectives it might become relevant to consider also the alternative assumption of purposeful (or voluntary) bequests. More in general, the issue of inheritance transmission in countries (such as SSA) where high-mortality coexists with complicate sociocultural settings deserves more careful investigation and empirical support (think, e.g., to the evidence suggesting that people at higher risk of acquiring the infection were, at least in the first phase of the HIV epidemic, the richer, the more educated, and the more mobile individuals).

Further open questions exist. For example, in this work we allocated the amount of (foreign and domestic) interventions against HIV/AIDS on a per worker basis, which is a convenient simplification. However, empirical analyses have suggested that the criteria adopted to allocate external financing to fight HIV in SSA have been variable but mostly related to GDP per capita in afflicted countries and somewhat unrelated to 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 its initiation adopted in the numerical simulations have 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. These 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 epidemic and its impact on society.

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

Appendix

This appendix shows that the OLG model with HIV/AIDS, *endogenous fertility* and perfect annuities leads to the same qualitative dynamic outcomes than the corresponding version with accidental bequests presented in the main text. This is because the amount of bequests unintentionally inherited from the previous generation does not affect the dynamics of capital accumulation. Differently, accidental bequests and perfect annuities imply different outcomes in the counterpart version with *exogenous fertility* [Chakraborty et al. (2016)].

Let us consider a version with perfect annuities of the model reported on the main text. The budget constraints when young and when old modify to become the following:

$$c_t + s_t + qw_t\ell_t n_t = w_t\ell_t(1-\tau_t), \tag{21}$$

and

$$d_{t+1} = \frac{R^e_{t+1}}{\beta_t} s_t. \tag{22}$$

Therefore, the lifetime budget constraint can be written as follows:

$$c_t + \frac{\beta_t d_{t+1}}{R^e_{t+1}} + qw_t\ell_t n_t = w_t\ell_t(1-\tau_t). \tag{23}$$

Therefore, the maximisation of the utility function (8) subject to (23) yields:

$$s_t = \frac{\beta_t (R^e_{t+1})^{\sigma-1} w_t\ell_t(1-\tau_t)}{1+\beta_t(R^e_{t+1})^{\sigma-1} + \gamma^\sigma (qw_t\ell_t)^{1-\sigma}}, \tag{24}$$

$$n_t = \frac{\gamma^\sigma w_t \ell_t (1-\tau_t)}{(q w_t \ell_t)^\sigma [1 + \beta_t (R^e_{t+1})^{\sigma-1} + \gamma^\sigma (q w_t \ell_t)^{1-\sigma}]}. \tag{25}$$

By using the market clearing equation (15) together with (24) and (25) one gets the equilibrium condition in the capital market in the case of perfect annuities:

$$k_{t+1} = \beta_t (R^e_{t+1})^{\sigma-1} \left(\frac{q}{\gamma}\right)^\sigma w_t^\sigma (1-\theta p_t)^\sigma \frac{1}{1-\theta p_{t+1}}. \tag{26}$$

Eqs. (16) in the main text and (26) above are identical except for the term β_t, which is raised to the power of σ in the former equation while entering linearly in the latter one. This is the outcome if the different effect played by the rate of longevity on both the lifetime budget constraint and the Euler equation describing the individual substitution rule between consumption when young and consumption when old at the optimum [see Fanti et al. (2014) for a discussion of this issue]. As the dynamics of the prevalence rate of HIV is still given by (17), assuming accidental bequests or perfect annuities in a model with endogenous fertility (child quantity) gives the same qualitative dynamic outcomes. Of course, in the case of log-utility ($\sigma = 1$) the two versions exactly coincide.

Under exogenous fertility ($\gamma = 0$, i.e. no individual preferences for and marginal benefits of having children, and $q = 0$, i.e. no marginal costs of children), accidental bequests and perfect annuities imply substantial differences. We now briefly sketch these two models to clarify these differences in a Chakraborty-like economy with public (rather than private) health prevention. Let us assume $\gamma = q = 0$. The individual of generation t maximises the utility function (8) with respect to the unique control variable s_t, subject to the exogenous fertility version of the lifetime budget constraint (7), under the hypothesis of accidental bequests, and the exogenous fertility version of (23), under the hypothesis of perfect annuities. Results are the following:

$$s_t = \frac{\beta_t^\sigma (R^e_{t+1})^{\sigma-1} [w_t \ell_t (1-\tau_t) + b_t]}{1 + \beta_t^\sigma (R^e_{t+1})^{\sigma-1}}, \tag{27}$$

in the case of accidental bequest, where $b_t = (1-\beta_{t-1})R_t^e \frac{s_{t-1}}{n\ell_t}$ (n is a constant) and

$$s_t = \frac{\beta_t (R_{t+1}^e)^{\sigma-1} w_t \ell_t (1-\tau_t)}{1+\beta_t (R_{t+1}^e)^{\sigma-1}}, \tag{28}$$

in the case of perfect annuities. Knowing that the market clearing condition in the capital market is $k_{t+1} = \frac{s_t}{n\ell_{t+1}}$, it is clear that the mechanics and the outcomes of the model are different when one replaces the assumption of perfect annuities with the one of accidental bequests in the framework developed by Chakraborty et al. (2016). This is because, unlike the model with endogenous fertility, in the latter case bequests affect the dynamics of capital accumulation.

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Figures and tables

Parameter	Value	Source
A	200	Free simulation parameter
α	0.33	Gollin (2002)
q	0.000308	Free simulation parameter
γ	0.00438	Free simulation parameter
σ	1.25	Pestieau and Ponthière (2017)
z	1	Chakraborty et al. (2016)
π_A	0.01	Chakraborty et al. (2016)
π_B	0.03	Free simulation parameter
μ	123	Free simulation parameter
θ	0.05	Free simulation parameter

Table 1. Parameter values for the numerical simulations distinguishing between (a) parameters borrowed from the cited economic literature, and (b) "free simulation parameter" assigned to obtain reasonably realistic values of output.

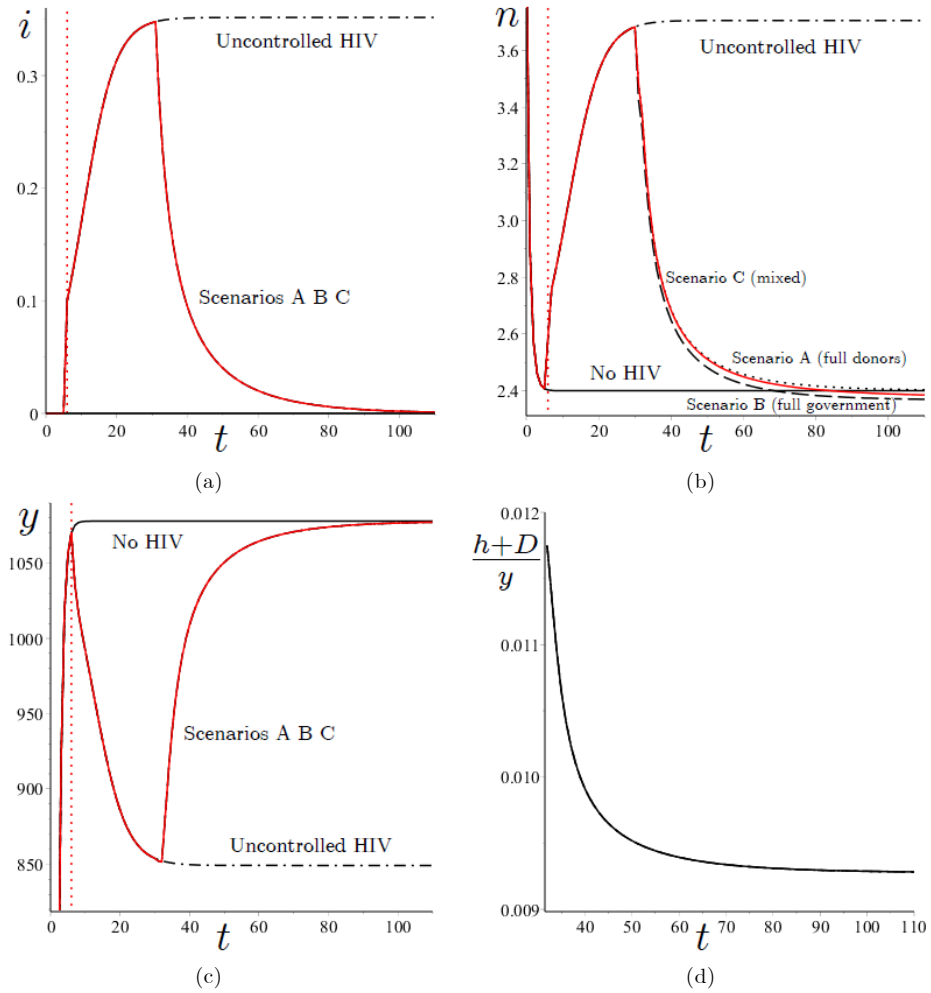

Figure 1. Temporal trends of HIV prevalence (Panel A), Fertility (Panel B), GDP per worker (Panel C) and the ratio between HIV-related expenditure and GDP per worker (Panel D). The vertical red dotted lines indicate the timing at which HIV initiates.

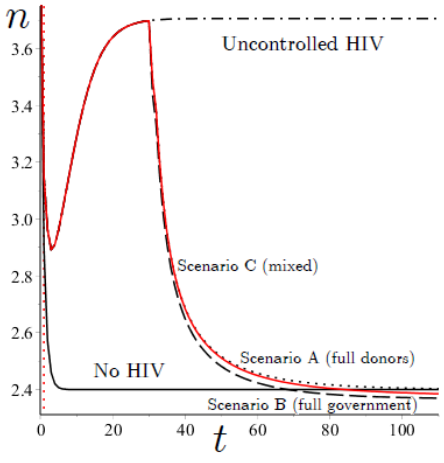

Figure 2. Temporal trend of fertility when HIV initiates before the completion of fertility decline.

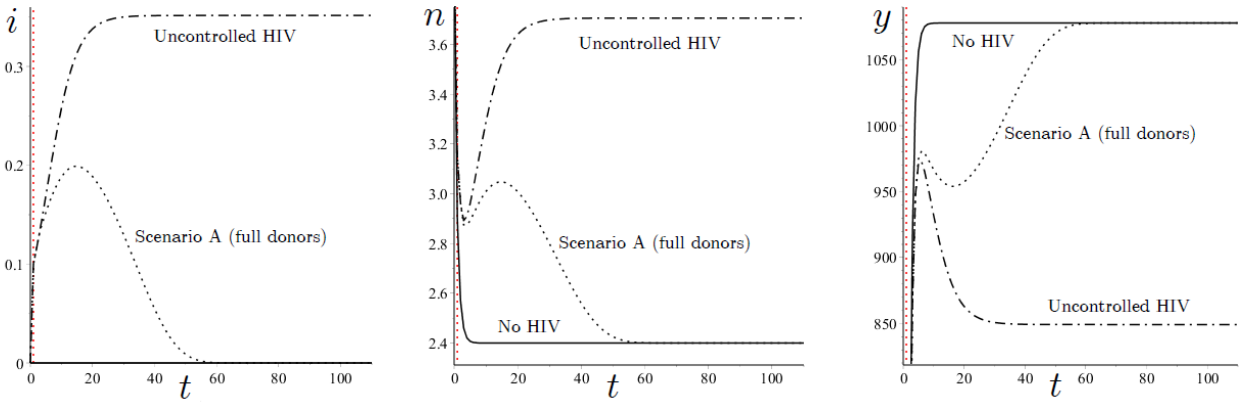

Figure 3. Temporal trends of HIV prevalence (left), fertility (centre) and GDP per worker (right) on the hypothesis of a time-increasing external expenditure.