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 mortality forced by the threat of a deadly enduring 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 those SSA countries severely afflicted by HIV. It investigates the demographic and macro-economic implications 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. On the assumption that these policies allow the same degree of HIV control, we show 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 – 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,

*L. Gori (corresponding author), Department of Law, University of Pisa, Via Collegio Ricci, 10, I–56126 Pisa (PI), Italy, e-mail: luca.gori@unipi.it or dr.luca.gori@gmail.com., tel.: +39 050 22 12 847.

†P. Manfredi, Department of Economics and Management, University of Pisa, Via Cosimo Ridolfi, 10, I–56124 Pisa (PI), Italy, e-mail: piero.manfredi@unipi.it, tel.: +39 050 22 16 363, fax: +39 050 22 10 603.

‡M. Sodini, Department of Economics and Management, University of Pisa, Via Cosimo Ridolfi, 10, I–56124 Pisa (PI), Italy, e-mail: mauro.sodini@unipi.it, tel.: +39 050 22 16 234, fax: +39 050 22 10 603.

HIV/AIDS 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 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, 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 the 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 and are allowing a dramatic reduction of AIDS mortality. 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 most important 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 the HIV/AIDS disease. This should be done with the aim of making most seropositive people aware of their status and providing them with a fully effective and lifelong treatment and to maintain high rates of public awareness of HIV [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, which in 2013 reached the level of 90% of total expenditures devoted to AIDS in low income countries, can have several 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 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. (2016A); Remme et al. (2016)]. The problem is a complicate one, stemming from the interplay between HIV and AIDS prevalences and on the afflicted country stage of economic development, but extending to several 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. (2016B); Remme et al. (2016)].

Based on this line of reasoning, we aim at contributing to this debate from the perspective of macro-economic dynamic modelling. As a first step, we aim at investigating the macro-economic and fertility 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. We pinpoint that in this article we disregard the problem of HIV on the side of public finance by avoiding to concentrate on the components of both the financing side and expenditure side, which instead is the focus of the above cited medical literature. In other words, the type of external donations considered in this article is directly provided to households (without the intermediation of the government) in order to enact prevention actions against HIV. This avoids the need to postulate the presence of transparent and efficient public institutions in the afflicted country, to which international donors should commit the management and transfer of resources to the private sector.

After a provocative work of Young (2005), suggesting that the tragedy of HIV/AIDS will eventually improve the welfare of future (post epidemic) SSA generations, and the ensuing response of Kalemli-Ozcan and Turan (2011), showing – by using the same data – that Young's conclusions were wrong, there has been a growing macroeconomic literature on the interplay between economic growth, development and infectious diseases at both theoretical and empirical levels [see also Boucekkine et al. (2009); Castro et al. (2015), Azomahou et al. (2016)]. In two influential works that focused on Africa as the world's region still suffering the highest burden from infectious disease, Chakraborty et al. (2010, 2016) began the investigation of the impact of a range of deadly infections within the general equilibrium overlapping generations (OLG) framework à la Diamond (1965) with exogenous fertility. They provided a parsimonious and innovative representation of the infection dynamics which is not simply modelled as a short-term exogenous shock, but it was assumed to follow an explicit dynamic 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 a long time prevented effective interventions at the population level. In addition, Chakraborty et al. (2010, 2016) considered a model where individuals were 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 whether individuals living in SSA can 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.

Our major task in this article 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. As was explained above, this main research question, 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 and co-authors. 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 in 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. 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 and perfect annuities. The latter hypothesis, which was adopted in the exogenous fertility model of Chakraborty et al. (2016), is clearly well suited for settings where financial markets are adequately developed, which is not the case for most of the current SSA countries with high prevalence of HIV. Indeed, with the sole exception of South Africa, these are low-resource settings for which the former assumption is surely an appropriate simplification of reality. For the sake of simplicity, 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).

Our main results are as follows. Given a certain outcome in terms of success in controlling the HIV epidemics, namely bringing the incidence of HIV to about zero in a number of generations, the responses of the demo-economic system largely depend on how the interventions are financed, that is whether they are entirely funded by international donations or by

an endogenous public policy of the afflicted country. The endogenous policy scenario yields a much richer 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 epidemics but also to the general equilibrium macroeconomic response via a Malthusian effect.

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. 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 (macro)economy closed to international trade and comprised of finite-lived 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 assuming that the mortality burden amongst children (including those dying by vertically transmitted AIDS) is a constant background steadily affecting the population in SSA countries. Therefore, we only consider the number of 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, 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 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 the 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) because of the characteristics of the HIV epidemic and the socioeconomic context prevailing in SSA. Instead, we assume that the transmission probability per single sexual intercourse π_t can be reduced either by appropriate interventions on sexual behaviour (e.g., first of all, by 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 a number of different ways. Here, we consider two main financing routes: foreign aid donations, which so far represented the most important way of financing the public health response against HIV/AIDS in SSA, and public expenditures against HIV endogenously managed by the government of the afflicted country. This leads to the following formulation of the transmission probability:

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

where h_t represents the amount of endogenous public expenditures against HIV/AIDS on a per worker basis, D_t represents the amount of foreign aids still on a per worker basis (so that the sum $h_t + D_t$ is the total amount of internal and external resources devoted to fight HIV on a per worker basis), $0 < \pi_A \leq 1$ ($\pi_A > 1/\mu$) and $\pi_B > 0$ are exogenous constants and $z > 0$ is a parameter that controls the degree of effectiveness of the overall amount of resources devoted to the intervention against AIDS in affecting the transmission probability. 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 $0 < \theta < 1$ 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 the absence of HIV will be 100%. In the presence of HIV, all HIV-infected young adults will prematurely 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 but 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 that 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 might be an adequate representation of disease prevention in developed countries.

Different from 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)], in this work we assume the existence of accidental bequests rather than a market for annuities. Indeed, we believe this assumption allows capturing in a better way the working of intergenerational transfers under uncertain survival in a context, such as current SSA, where financial markets are not adequately developed. The existence of accidental bequests implies that savings of a deceased person are equally bequeathed in full to his own descendants. Differently, the assumption of the availability of perfect annuities implies 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).

Let us now turn to the working of the model. Since (old) agents do not know when they will die (given the HIV epidemic and health prevention), unintentional bequests can occur. If the typical agent of generation t prematurely dies (with probability $1-\beta_t$) at the onset of old age due to HIV, his accumulated savings (s_t) are in full bequeathed to his heirs. To keep the representative agent 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 expressed in per worker terms and 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 $t+1$). Therefore, knowing that $N_{t+1} = n_tN_t$ determines the evolution of population, 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 accidentally 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 (whose cost per child is $qw_t\ell_t$). Therefore, the budget constraint when young reads as

[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.

follows:

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

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. Therefore, old-age material consumption (d_{t+1}) is constrained by the capitalised amount of resources saved when young, that is:

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

By using Eqs. (5) and (6), the lifetime budget constraint can be written as follows:

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

The left-hand side of (7) includes the present value of 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 plus inherited unintentional 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); Fanti and Gori (2013); Spataro and Fanti (2011)]:

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

where $\sigma > 0$ ($\sigma \neq 1$) is the constant inter-temporal elasticity of substitution. Empirical research focused on explaining country-level heterogeneity at the micro level [Havranek et al. (2015)] or the one analysing the behaviour of aggregate consumption data [Hall (1988); Guvenen (2006); Jones and Schoonbroodt (2010)] generally found values of the elasticity of substitution in consumption smaller than one, though other analyses based on micro data [Blundell et al. (1994); Attanasio and Browning (1995); Browning et al. (1999)] or macro data by taking into account the precautionary savings motive [Gomes and Ribeiro (2015)] obtained the opposite result. We pinpoint, however, that this index was not estimated in models including endogenous mortality and, more in general, under (endogenous) demographic variables, with the sole exception of the work of Jones and Schoonbroodt (2010) that considers endogenous fertility. In the theoretical literature employing additively separable utility functions, the elasticity of substitution is often assumed to be larger than one to guarantee that consumption in both periods and leisure are gross substitutes as, e.g., in the works of Cazzavillan and Pintus (1994) and de Vilder (1996).[2] As this model employs the notion of expected utility due to the existence

[2] Two commodities are called gross substitutes if an increase in the price of one of them implies that an individual wants to increase the quantity consumed of the other commodity. This is because consumption shifts towards the cheapest good.

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 preferences [Rosen (1988); Hall and Jones (2007)]. This is because when $\sigma = 1$ the expression in (8) boils down to $U_t = \ln(c_t) + \beta_t \ln(d_{t+1}) + \gamma \ln(n_t)$ and we should always be able to guarantee values of d_{t+1} larger than 1 both in transition and long-term. Otherwise, an increase in the length of life would reduce utility. In order to overcome this problem, in the numerical experiments we will use values of σ larger than 1 in line with those used by Pestieau and Ponthière (2017), which are in turn consistent with the empirical result of Gomes and Ribeiro (2015). This also allows us to avoid problems related to normalising the utility from death at zero. Some clarifications on this issue are now useful. On theoretical grounds, similar to Cazzavillan and Pintus (1994) and de Vilder (1996), who assumed that consumption in both periods and leisure are gross substitutes, we may assume that consumption in both periods and fertility are gross substitutes. In addition, as was pointed out by Rosen (1988) when lifetime utility includes life expectancy 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, as they considered a model where spending in health directly affected the quality of life of an individual, 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 the lifetime utility 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 and this class of utility functions, one must postulate a positive relationship between life expectancy and the utility drawn from material consumption, otherwise the utility would reduce by shortening the lifespan. This would also open complicate questions including the possibility of the existence of an optimal duration of life. Our problem, therefore, is much more related to the value of a statistical life (VSL), as was pointed out in the OLG model of 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 should be borne for gaining of 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]. Definitely, we pinpoint that assuming $\sigma > 1$ is a simple but general way to avoid having both a negative utility and negative VSL.

By taking as given factor prices, the health tax rate, the bequests inherited from the past and the economic effects of the epidemic, the individual representative of generation t maximises

the expected utility function (8) with respect to c_t, d_{t+1} and n_t subject to the lifetime budget constraint (7). Then, saving and fertility are given by:

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

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

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 (that is, in the quantity of life) 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, other things being equal. 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 depend 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) through the following 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 $L_t = \ell_t N_t = (1-\theta p_t)N_t$. The previous equation includes the possibility that HIV/AIDS reduces labour productivity in line with Chakraborty et al. (2016). However, the issue of HIV-reduced productivity is not relevant for the main results of this work and is included only for generality. We better discuss its implications later in this article. 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:

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

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

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 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:

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

Market-clearing in the capital market is determined by equating aggregate investment and aggregate saving and it is given by $K_{t+1} = S_t = s_t N_t$. 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:

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

where s_t and n_t are respectively given by the expressions in (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), it is clear that capital accumulation does not depend on accidental bequest inheritance. This is because (in this model with endogenous fertility) bequests positively affect in the same qualitative way both saving and fertility by increasing the income of the young members of the working generation. Definitely, 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. Indeed, 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 SSA and an already initiated fertility decline [see Gori et al. (2017) for a discussion on this issue]. In particular, we assume that in the absence of HIV the TFR achieve a long-term equilibrium slightly below 2.4, which is slightly in excess of the ideal replacement level of 2.1, to cope with the possibility that some mortality in child-bearing ages from causes different from AIDS (this component of mortality is not included in the model) remains even after the complete control of the epidemics.

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 peaks in epidemic incidence observed in the most severe epidemics in SSA countries [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 generation per generation will depend on the mortality rate, which in turn depends on the prevalence rate of HIV.

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 epidemics 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 revenue - to be used to finance HIV intervention - is exactly identical to the one set in Scenario A, that is the overall expenditure against HIV/AIDS is kept unaltered by letting the

[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.

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 they are replaced by the endogenous 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 awareness of the policy and avoid issues of time inconsistency [Calvo (1978)].

The main demo-economic and epidemiological parameters were assigned according to the values reported in Table 1, where some economic parameters were borrowed from the literature, whereas the remaining ones were used as free simulation parameters and were set in order to illustrate some of the main features of the issue of financing the interventions against HIV/AIDS. In particular, the HIV parameters in the absence of interventions were tuned ad hoc in order to generate a worst case 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 taken as free simulation parameters and adjusted to essentially achieving the target of HIV elimination in the medium-long term. The initial ratio between HIV-specific expenditure and GDP (per worker) in the various scenarios is set to 1.12%. Finally, the initial condition of the prevalence rate in the free HIV epidemics was set to 10%.

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 assignment used in the numerical simulations. Whenever possible, parameter assignment were drawn from available estimates from the cited economic literature. By "free simulation parameter" we mean a parameter whose value has been freely assigned with the purpose to obtain reasonably realistic values of the output trajectories. In particular, the parameters A, q and γ are those ultimately tuning the fertility function, while parameters π_B and μ tune the HIV prevalence equations (1) and (2).

In the absence of HIV, economic and demographic variables are predicted to achieve 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 $t < 6$ (Fig. 1, Panels B and C, black solid line). The HIV epidemic is initialised at $t = 6$ (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 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 woman, and a reduction in the willingness to save during young adulthood due the individual expected shorter life span [Chakraborty (2004)], as is clear from (10) and (9). This phenomenon of HIV-induced fertility reversal - though still controversial [see the discussion in Gori et al. (2017)] - was first predicted by Kalemli-Ozcan (2012) based on empirical analysis of DHS data from SSA and investigated from a Unified Growth Theory theoretical perspective in Gori et al. (2017). As a consequence, capital accumulation and GDP per worker (Fig. 1, Panel C, black dash-dotted line) dramatically fall as a consequence of increasing HIV prevalence, and will approach 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) 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 identical temporal trend of the HIV prevalence curve. 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.

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 restore the steady-state level that was prevailing in the pre-AIDS era.

The more interesting result occurs 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 epidemic 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 is because the continued presence of the policy for the whole relevant horizon of the epidemics 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 scenario. In fact, the endogenous public policy has first of all a direct effect by relapsing 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, 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 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 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.

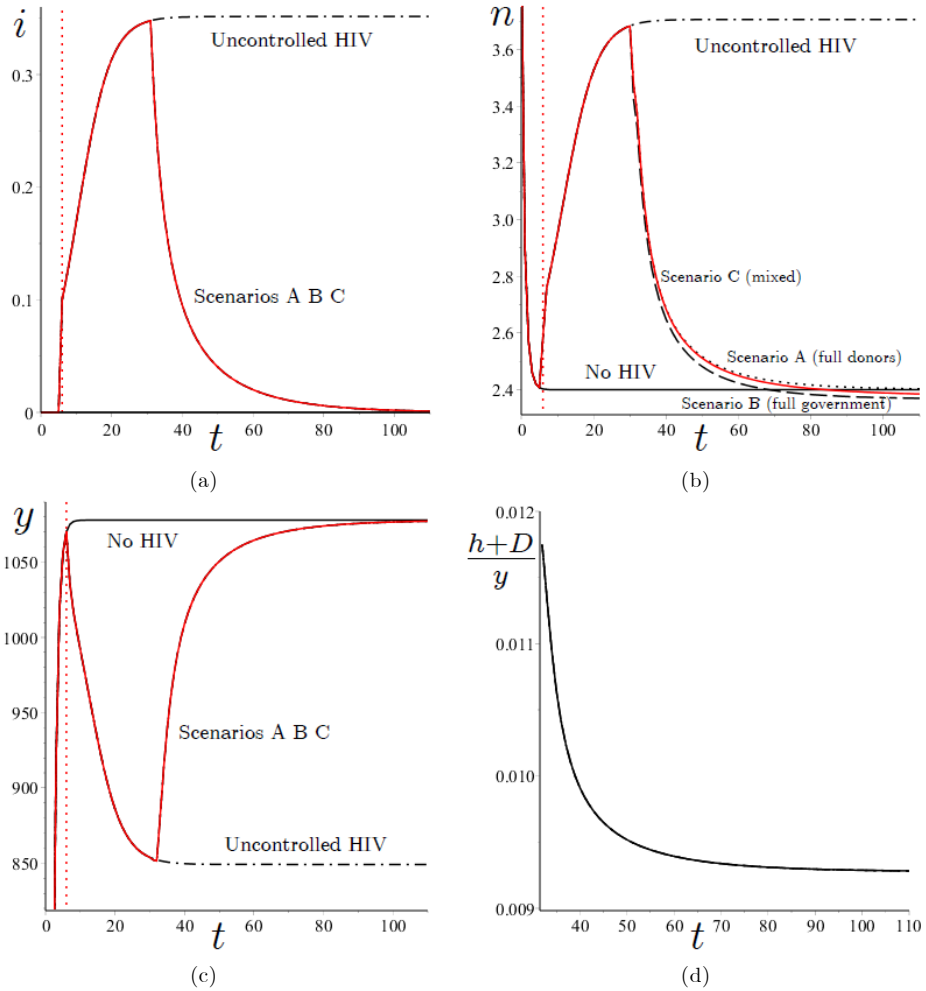

Figure 1. Temporal trends of HIV prevalence (Panel A), Fertility (Panel B), GDP per worker (Panel C) and the ratio between HIV-related expenditure and GDP per worker (Panel D). The vertical red dotted lines in the figures indicate the timing of initiation of HIV.

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 the three scenarios above considered by letting the HIV epidemics to begin at when the process of fertility decline is still ongoing with levels of the Total Fertility Rate still far distant ($n = 2.9$) from its long-term equilibrium. Subsequent dynamics are much in line with those reported in Figure 1.

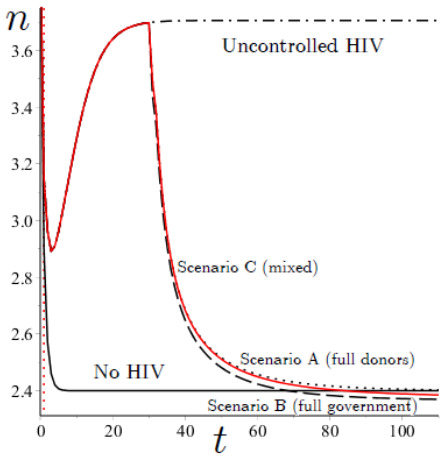

Figure 2. Temporal trend of fertility in the case HIV initiates before the completion of fertility decline.

Increasing levels of AIDS expenditure This scenario is designed to investigate the situation where the intervention against HIV initiates before the epidemics has reached its endemic equilibrium level by, as empirically documented, an increasing rate of expenditure. The latter aspect is motivated by the fact that awareness of the AIDS tragedy 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 time-varying external expenditure curve D_t, which is taken as a linearly increasing function of time, reaching in a few periods the level D_0 set in Scenario A.

As shown in Figure 3, in this case the initial intensity of intervention is not sufficient to stop the epidemic increase calling for additional resources. However, as D_t increases over time it has the potential to mitigate the impact of the HIV/AIDS epidemics by preventing prevalence to reach its equilibrium level, by achieving the downturn when prevalence is almost 20%, consistently with what has been observed e.g., in South Africa, and eventually achieving disease elimination (Figure 3, left panel). The resulting effects on fertility 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, 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 long-term equilibrium in the absence of AIDS (Figure 3, right).

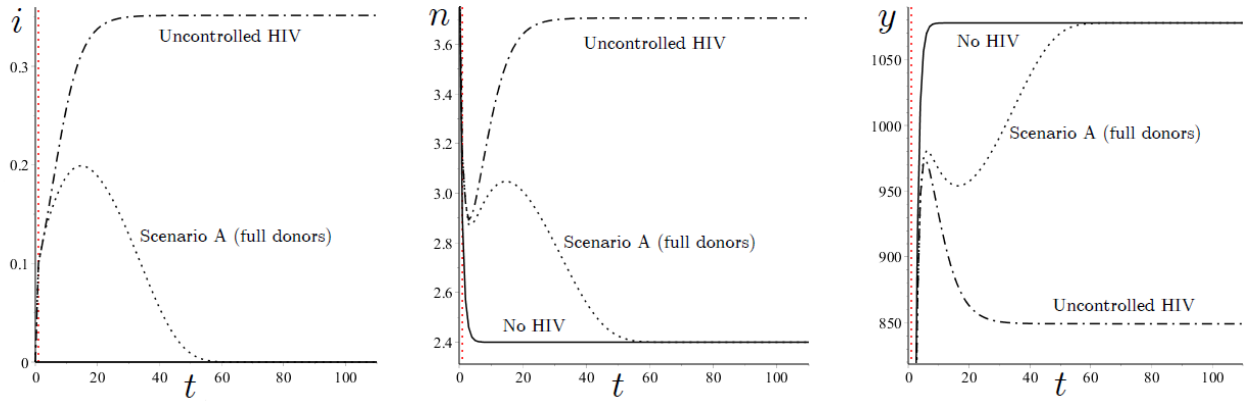

Figure 3. Temporal trends of HIV prevalence (left), fertility (centre) and GDP per worker (right) on the hypothesis of a time-increasing external expenditure.

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. Briefly, our results show that given a target in terms of HIV control and by keeping the same expenditures for HIV control in the two scenarios (full donors and full government), the direct effect of the policy on HIV/AIDS is obviously the same, but its ultimate consequences for demo-economic variables are different. Indeed, 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. In particular, the full government scenario emphasises the fertility response which is dramatically larger than that in the full donors scenario. 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 simple theoretical model. The primary task of future works should be that of the formally including the public finance issue on the side of both the expenditure components and financing components, which has been the key topic of the cited recent public health economics literature. Beyond this, there is a number of issues to be considered. Much related to the core of this article, in the current moment there is a debate about the possibility to develop innovative financial tools specifically targeted at supporting public interventions against HIV/AIDS in SSA [Atun et al. (2016A, 2016B)] in the forthcoming decades without compromising development perspectives in the region. 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 issue of private insurance and financial markets (i.e., markets for annuities) and related problems as those of inheritance transmission. Although in the present work we used the assumption of accidental bequests to incorporate inheritance transmission, given current perspectives it might become relevant to also consider the alternative assumption of purposeful (or voluntary) bequests. More in general, the issue of inheritance in countries (such SSA) where high-mortality coexists with complicate socio-cultural settings - 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 epidemics, the richer, the more educated, and the more mobile individuals, for which the issue of inheritance is surely more involved than the simplistic assumption of accidental bequests used here - and possibly rapid economic structural changes is one which should deserve a more careful support based on empirical findings.

However, the list of open questions is endless. For example, we allocated the amount of (foreign and domestic) interventions against HIV/AIDS on a per worker basis, which is a convenient modelling simplification. However, 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 on 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.

Appendix

The aim of this appendix is to show that the OLG model with HIV/AIDS, *endogenous fertility* and perfect annuities will qualitatively lead to the same 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 (and represented by the addendum b_t in the budget constraint (5), which contributes to increase the individual income but it is not subject to any optimisation) does not affect the dynamics of capital accumulation. Differently, in the case of *exogenous fertility* [Chakraborty et al. (2016)] accidental bequests and perfect annuities will imply different dynamic outcomes.

Let us consider now a version with perfect annuities rather than accidental bequests of the model built 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

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

By using Eqs. (21) and (22), 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 the lifetime budget constraint (23) gives the individual optimal amount of saving and fertility, that is:

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

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

By using the expression of the market clearing condition in (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) and (26) are identical except for the term β_t, which enters linearly in (26) and it is raised to the power of σ in (16). This difference comes from 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 qualitatively 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. Here we briefly sketch these two models to clarify these differences in a Chakraborty-like economy with public health investments to fight HIV. Therefore, let us assume $\gamma = q = 0$. In this case, 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 (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 $b_t = (1-\beta_{t-1}) R^e_t \frac{s_{t-1}}{n\ell_t}$ (n is a constant) and

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

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) with exogenous fertility. This is because, unlike the model with endogenous fertility, in the latter case bequests affect the dynamics of capital accumulation.

References

[1] Akbulut-Yuksel, M. and B. Turan (2013) Left behind: intergenerational transmission of human capital in the midst of HIV/AIDS. *Journal of Population Economics* 26, 1523–1547.

[2] Attanasio, O.P., and M. Browning (1995) Consumption over the life cycle and over the business cycle. *American Economic Review* 85, 1118–1137.

[3] Atun, R., S. Silva, M. Ncube and A. Vassall (2016A) Innovative financing for HIV response in sub–Saharan Africa. *Journal of Global Health* 6, e010407.

[4] Atun, R., A.Y. Chang, O. Ogbuoji, S. Silva, S. Resch, J. Hontelez and T. Bärnighausen (2016B) Long-term financing needs for HIV control in sub-Saharan Africa in 2015–2050: a modelling study. *British Medical Journal Open* 6, e009656.

[5] Azomahou, T.T., R. Boucekkine and B. Diene (2016) HIV/AIDS and development: a reappraisal of the productivity and factor accumulation effects. *American Economic Review* 106, 472–477.

[6] Blackburn, K. and G.P. Cipriani (2002) A model of longevity, fertility and growth. *Journal of Economic Dynamics & Control* 26, 187–204.

[7] Blundell, R., M. Browning and C. Meghir (1994) Consumer demand and the life-cycle allocation of household expenditures. *Review of Economic Studies* 61, 57–80.

[8] Boucekkine, R., R. Desbordes and H. Latzer (2009) How do epidemics induce behavioral changes? *Journal of Economic Growth* 14, 233–264.

[9] Browning, M., L. Hansen and J.J. Heckman (1999) Micro data and general equilibrium models. In: Taylor, J. and M. Woodford (eds.) Handbook of Macroeconomics, Vol. 1A. Elsevier Science.

[10] Calvo, G.A. (1978) On the time consistency of optimal policy in a monetary economy. *Econometrica* 46, 1411–1428.

[11] Castro, R., J.R. Behrman and H.P. Kohler (2015) Perception of HIV risk and the quantity and quality of children: the case of rural Malawi. *Journal of Population Economics* 28, 113–132.

[12] Cazzavillan, G., and P.A. Pintus (2004) Robustness of multiple equilibria in OLG economies. *Review of Economic Dynamics* 7, 456–475.

[13] Chakraborty, S. (2004) Endogenous lifetime and economic growth. *Journal of Economic Theory* 116, 119–137.

[14] Chakraborty, S., C. Papageorgiou and F. Pérez Sebastián (2010) Diseases, infection dynamics and development. *Journal of Monetary Economics* 57, 859–872.

[15] Chakraborty, S., C. Papageorgiou and F. Pérez Sebastián (2016) Health cycles and health transitions. *Macroeconomic Dynamics* 20, 189–213.

[16] Chin, Y.-M. and N. Wilson (2018) Disease risk and fertility: evidence from the HIV/AIDS pandemic. *Journal of Population Economics* 31, 429–451.

[17] Collier, P. and O. Sterck (2018) The moral and fiscal implications of antiretroviral therapies for HIV in Africa. *Oxford Economic Papers* 70, 353–374.

[18] de la Croix, D. and P. Michel (2002) A theory of economic growth. Dynamics and policy in overlapping generations. Cambridge University Press, Cambridge.

[19] de la Croix, D., P. Pestieau and G. Ponthière (2012) How powerful is demography? The Serendipity Theorem revisited. *Journal of Population Economics* 25, 899–922.

[20] de Vilder R. (1996) Complicated endogenous business cycles under gross substitutability. *Journal of Economic Theory* 71, 416–442.

[21] Diamond, P.A. (1965) National debt in a neoclassical growth model. *American Economic Review* 55, 1126–1150.

[22] Fanti, L. (2009) Longevity, fertility and Demographic Transition in an OLG model. *Economics Bulletin* 29, 348–357.

[23] Fanti, L. and L. Gori (2013) Fertility-related pensions and cyclical instability. *Journal of Population Economics* 26, 1209–1232.

[24] Fanti, L. and L. Gori (2014) Endogenous fertility, endogenous lifetime and economic growth: the role of child policies. *Journal of Population Economics* 27, 529–564.

[25] Fanti, L., L. Gori and F. Tramontana (2014) Endogenous lifetime, accidental bequests and economic growth. *Decisions in Economics and Finance* 37, 81–98.

[26] Ferreira, P.C., S. Pessôa and M.R. Dos Santos (2011) The impact of AIDS on income and human capital. *Economic Inquiry* 49, 1104–1116.

[27] Galor, O. and D.N. Weil (1996) The gender gap, fertility, and growth. *American Economic Review* 86, 374–387.

[28] Gollin, D. (2002) Getting income shares right. *Journal of Political Economy* 110, 458—474.

[29] Gomes, F.A.R. and P.F. Ribeiro (2015) Estimating the elasticity of intertemporal substitution taking into account the precautionary savings motive. *Journal of Macroeconomics* 45, 108–123.

[30] Gori, L., and M. Sodini (2018) A contribution to the theory of fertility and economic development. GLO Discussion Paper No. 170.

[31] Gori, L., E. Lupi, P. Manfredi and M. Sodini (2017) Can HIV alter the quantity-quality switch and delay the fertility transition in Sub-Saharan Africa? GLO Discussion Paper No. 75.

[32] Guvenen, F. (2006) Reconciling conflicting evidence on the elasticity of intertemporal substitution: a macroeconomic perspective. *Journal of Monetary Economics* 53, 1451–1472.

[33] Haacker, M. (2009) Financing HIV/AIDS programs in sub-Saharan Africa. *Health Aff (Millwood)*. 28, 1606–1616.

[34] Hall, R.E. (1988) Intertemporal substitution in consumption. *Journal of Political Economy* 96, 339–357.

[35] Hall, R.E. and C.I. Jones (2007) The value of life and the rise in health spending. *Quarterly Journal of Economics* 122, 39–72.

[36] Havranek, T., R. Horvath, Z. Irsova and M. Rusnak (2015) Cross-country heterogeneity in intertemporal substitution. *Journal of International Economics* 96, 100–118.

[37] Hubbard, R.G. and K.L. Judd (1987). Social security and individual welfare: precautionary saving, borrowing constraints, and the payroll tax. *American Economic Review* 77, 630–646.

[38] Jones, L.E. and A. Schoonbroodt (2010) Complements versus substitutes and trends in fertility choice in dynastic models. *International Economic Review* 51, 671–699.

[39] Juhn, C., S. Kalemli-Ozcan and B. Turan (2013) HIV and fertility in Africa: first evidence from population-based surveys. *Journal of Population Economics* 26, 835–853.

[40] Kalemli-Ozcan, S. (2012) AIDS, "reversal" of the demographic transition and economic development: evidence from Africa. *Journal of Population Economics* 25, 871–897.

[41] Kalemli-Ozcan, S. and B. Turan (2011) HIV and fertility revisited. *Journal of Development Economics* 96, 61–65.

[42] Katz, I., S. Routh, R. Bitran, A. Hulme and C. Avila (2014) Where will the money come from? Alternative mechanisms to HIV donor funding. *BMC Public Health* 14, e956.

[43] Mohiddin, A., and D. Johnston (2006) HIV/AIDS mitigation strategies and the State in sub-Saharan Africa – the missing link? *Globalization and Health* 2, 1–5.

[44] Pestieau, P. and G. Ponthière (2017) Optimal fertility under age-dependent labour productivity. *Journal of Population Economics* 30, 621–646.

[45] Remme, M., M. Siapka, O. Sterck, M. Ncube, C. Watts and A. Vassall (2016) Financing the HIV response in sub-Saharan Africa from domestic sources: moving beyond a normative approach. *Social Science & Medicine* 169, 66–76.

[46] Resch, S., T. Ryckman and R. Hecht (2015) Funding AIDS programmes in the era of shared responsibility: an analysis of domestic spending in 12 low-income and middle-income countries. *Lancet Global Health* 3, 52–61.

[47] Rosen, S. (1988). The value of changes in life expectancy. *Journal of Risk and Uncertainty* 1, 285–304.

[48] Spataro, L. and L. Fanti (2011) The optimal level of debt in an OLG model with endogenous fertility. *German Economic Review* 12, 351–369.

[49] UNAIDS (2012) http://www.unaids.org/en/resources/presscentre/pressreleaseandstatementarchive/2

[50] UNAIDS (2017) Global HIV Statistics. www.unaids.org.

[51] WHO (2017). Global Health Estimates 2015: Estimated deaths by age, sex, and cause.

[52] Young, A. (2005) The gift of the dying: the tragedy of AIDS and the welfare of future African generations. *Quarterly Journal of Economics* 120, 423–466.