HIV/AIDS, demography and development: individual choices versus public policies in SSA

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Abstract Despite the increasing rate of diffusion of effective therapies, the battle against HIV/AIDS in Sub-Saharan Africa (SSA) is far from being over. Three main challenges are that the epidemics might paralyse or reverse the fertility transition, the expansion of the resources needed to finance the fight against HIV, and the emerging resistance to anti-retroviral treatments. This research proposes a UGT-like model showing the complexity of the interplay amongst the (macro)economy, the epidemics, their endogenous feedback on mortality and fertility and the central role of policy actions aimed to fight HIV. The disease-induced increase in adult mortality can hamper economic development by its upward pressure on the precautionary demand for children and downward pressure on education. This can dramatically reduce physical and human capital accumulation.

Keywords Sub-Saharan Africa, HIV/AIDS epidemics, fertility, public versus international financing of AIDS interventions, physical and human capital accumulation

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1. Introduction

For more than three decades since its debut in 1980, the HIV/AIDS pandemic has ravaged Sub-Saharan Africa (SSA) with a devastating mortality burden especially frightening young adults of both sexes. However, in the last few years the epidemic seems to have finally entered a declining phase (UNAIDS, 2018) thanks to the massive efforts aiming to (i) expand HIV testing to make seropositive people (i.e., people in the HIV stage) aware of their serostatus, (ii) increase the number of seropositive and sick people (i.e., people with full blown AIDS stage) accessing effective antiretroviral therapies (ART), and (iii) maintain high rates of population awareness on AIDS risks, by continuing and extending ongoing prevention and screening campaigns. For example, in 2016 the proportion of people accessing ART amongst those living with HIV in the overall SSA region reached the level of 54% (though with large inter- and intra-country variation) compared to essentially none 10 years before. This is a great success that, compared to 2004-2005, representing the epoch of the AIDS-related mortality peak in SSA, has contributed to nearly halve AIDS mortality over a span of one decade (UNAIDS, 2017). Indeed, this has radically changed overall perspectives of AIDS control in SSA, as summarised in the so-called 90-90-90 plan launched by the UN in 2014 (UNAIDS, 2017), upon which the slogan "ending AIDS by 2030" was coined (UNAIDS, 2017) – referring to a major reduction in incidence and obviously not to disease eradication.

Clearly, going beyond the current – already burning – debate about the feasibility of the 90-90-90 plan (Levi et al., 2016), the success achieved so far should not let us forget that AIDS currently remains the second main cause of mortality in SSA (WHO, 2017), with a huge number of seropositive individuals unaware of their serostatus (UNAIDS, 2017) and therefore able to continue to transmit the infection to others for a long time, suggesting that the battle is only initiated. In relation to this, several challenges have arisen, whose outcomes are surrounded by large uncertainties. In our opinion, these include (amongst others) three strictly inter-related areas at the interplay of epidemiology and public health on one hand, and demo-economics on the other hand,

deserving maximal attention by scholars and policy makers. The first one deals with the ultimate impact of HIV on the fertility transition in SSA and its consequences for economic development. The second regards the dramatic amount of resources that will have to be mobilised at both the national and international levels for successfully winning the AIDS battle, and its ambiguous effect on economic development (WHO, 2018). The third one – perhaps the true major challenge – deals with the emergence of resistance to HIV treatment (Hamers et al., 2018), whose effects are at present largely uncertain. These effects, which might be reasonably kept under control in industrialised countries, where HIV epidemics are mild, might reveal devastating in SSA by dramatically worsening the first two threats and eventually questioning the current optimism in the fight against AIDS (Hamers et al., 2018, Phillips et al 2017).

Amongst the three challenges, the first one on the eventual impact of HIV on the fertility transition in SSA and economic development has already been the object of a sustained debate amongst economists, with two dramatically opposite positions. On one extreme, Young (2005, 2007) concluded that AIDS is contributing to lowering fertility in sub-Saharan Africa, and although representing a humanitarian disaster, does not cause an economic disaster. Rather, AIDS will allow future SSA generations to enjoy higher welfare than current generations thanks to the increase in the capital- and output-labour ratios because of AIDS mortality's impact on labour supply that will dominate the main negative effect of HIV namely, the disruption of human capital. At the other extreme, (Kalemli-Ozcan and Turan, 2011) and (Kalemli-Ozcan, 2012) were first in corroborating an earlier intuition by Kalemli-Ozcan (2002) that HIV has the potential to reverse the fertility transition in SSA. In particular (Kalemli-Ozcan and Turan, 2011) revisited Young's (2005, 2007) analyses using the same data and concluded that "the effect of HIV prevalence on fertility turns out to be positive". Moreover, Kalemli-Ozcan (2012, p. 891) empirically showed the potential for HIV to reverse the fertility transition in SSA via both a positive direct effect on the quantity of children and a negative effect on the quality of children because of the existence of a negative correlation between HIV prevalence and school enrolment, and explained this phenomenon with the upward

pressure that the mortality upturn due to AIDS causes on the precautionary demand for children (quantity) and to the downward pressure on the demand for their education (quality). Within these two extreme positions there exists an entire spectrum of intermediate ones, which are reviewed in Gori et al. (2017).

On the previous topic, we have investigated (Gori et al., 2017) the possible consequences of the HIV epidemics on the fertility transition in SSA by using a novel model integrating an explicit dynamics of a fatal epidemics along the formulation firstly proposed in Chakraborty et al. (2010, 2016) into a basic Unified Growth Theory (UGT) general equilibrium model (Galor and Weil, 2000; Galor, 2011) including only human capital accumulation, to capture the main feature of the HIV context in SSA, and endogenous fertility and child and adult survival. Child and adult mortality schedules were made dependent on both human capital accumulation, to mirror the pre-AIDS mortality transition, and the HIV prevalence to capture the additional effects of HIV/AIDS-related mortality. The aim was to track the ultimate consequences – at the general equilibrium level – of the fall in education and human capital accumulation following the HIV-related blow-up of mortality amongst young adults on the quantity-quality switch, and therefore the fertility transition, in SSA. By choosing a parametrisation of SSA mortality allowing an appropriate balance between the time scales of the demographic transition and AIDS spread, we indeed concluded that an HIV-induced stall (or even a reversal) of the fertility transition is a potentially robust phenomenon that, even under reasonably optimistic hypotheses on the timing and effects of AIDS control, might delay the trajectory of fertility in SSA of several decades thus compromising economic development in the region. These results bring theoretical evidence to the findings supporting the fertility "reversal" hypothesis, formulated first by Kalemli-Ozcan (2012) and Kalemli-Ozcan and Turan (2011) (see Gori et al., 2017, and references therein).

The possibility that the fertility transition in SSA might be paralysed by HIV makes it imperative to rapidly bring the epidemics under full control, thereby yielding to the second challenge above. Indeed, successfully fighting this battle will in turn call for unprecedented further mobilisation of

resources specifically devoted to fight HIV/AIDS in the short as well as in the medium and long terms. Recent estimates (Collier and Sterck, 2018) indicate that, given current high costs of a single therapy, the overall economic cost of generalised lifelong HIV treatment in the poorest SSA countries would be in the range of 80% of current GDP! The magnitude of this figure can hardly be covered by a massive expansion of international donations, representing the main funding source of the intervention against HIV carried out in SSA so far (UNAIDS, 2017). As was noted by Resch et al. (2015) and Remme et al. (2016) – based on current perspectives –, international aids might not be able to expand significantly beyond current levels. On the other hand, in the light of the dramatic extent of foreign financing of the AIDS response, which has reached levels above 90% of total AIDS funding in low income countries in 2013, there have been concerns about the fact that an HIV response completely dependent on external resources might have potentially destabilising effects on the afflicted countries (Mohiddin and Johnston, 2006; UNAIDS, 2013).

The previous considerations therefore highlight not only the need for massive resources deployment but also the delicate balance between foreign aid and the ability, for HIV afflicted SSA countries, to develop an autonomous endogenous response to AIDS. This is a major challenge whose response depend from an endless list of factors including primarily: (i) the magnitude of the HIV epidemic, (ii) the stage of economic development in the afflicted country and its perspectives for GDP growth in the medium term, (iii) the threats from other diseases, first of all malaria and TB, that in turn require allocation of large significant quotas of GDP, (iv) the ability to develop new financial tools to face the HIV challenge (Resch et al., 2015; Atun et al., 2016A; Remme et al., 2016). These complicate questions have already raised a tough debate in the areas of public health and health economics (Katz et al., 2014; Resch et al., 2015; Atun et al., 2016B; Remme et al., 2016).

On this topic, in a different work (Gori et al., 2018) we have initiated an investigation of the macro-economic dynamic implications of the affordability (by the government of HIV-afflicted countries) of control programmes mainly relying on public domestic expenditure. In that article,

also motivated by the need to provide a well-funded formulation of intervention against HIV (taken as fully exogenous in Gori et al. (2017) we used a simplified OLG macroeconomic framework à la Diamond (1965) with endogenous population and endogenous HIV transmission. About the latter, we represented the intervention against HIV by modelling the probability of transmission of HIV infection per single sexual contact as the sum of an endogenous component, representing public expenditure (financed at a balanced budget) specifically targeted to fight against HIV, and of an exogenous component reflecting international donations.

The main aim of the present work is to go one step further compared to both Gori et al. (2017) and Gori et al. (2018) by superimposing to the standard Diamond setup also private education and human capital accumulation, *thereby enabling us to tackle the issue of HIV spread and control in a model with the quantity-quality trade off and consequently capable to also offer predictions on the fertility issue as well*. For the macroeconomic component of the model we adopt a UGT-like structure along the lines of the contribution of Yakita (2010) with both physical and human capital accumulation. This macroeconomic setup is combined with an endogenous representation of the transmission and control of HIV/AIDS following the pioneering works of Chakraborty et al. (2010, 2016), who were first in proposing an explicit representation of the transmission process into an OLG model. However, unlike Chakraborty and co-authors, whose agents optimally chose their private health preventive expenditure against HIV, in our formulation the transmission probability is determined by (i) the public interventions managed by the government of the afflicted country, (ii) foreign aid donations, which historically have represented the main channel of interventions carried out so far in SSA, (iii) a component tuning the positive impact of private education on the awareness of being at risk and ultimately capable to enhance the ongoing policies. Foreign aids are assumed to be scaled by the two main factors that should drove international programmes in SSA, namely the rate of HIV prevalence and per capita income of the afflicted country, although it is documented that GDP has been the main driver of the intervention while prevalence seemingly played a secondary role (Haacker, 2009; UNAIDS, 2017). Compared to the formulation adopted in

Chakraborty et al. (2016), which is clearly well suited for settings where financial markets are adequately developed, we believe that ours can better fit the current context of HIV in SSA, which is – with the sole exception of South Africa – the poorest region worldwide with inadequate health infrastructures, severe shortages of physicians and medicines, low education and poor health (UNDP, 2018) causing the highest mortality setting worldwide even in the absence of HIV.

Our formulation inherits the growth structure from Yakita (2010) showing the two development regimes characterising UGT growth models (e.g., Galor and Weil, 2000; Kalemli-Ozcan, 2002; de la Croix and Doepke, 2003, 2004; Galor, 2011), that is an initial exogenous growth phase (representing the paradigm of the poverty trap), where private education does not play any role and there exists only a precautionary demand for children, and a subsequent endogenous growth phase where education promotes the quantity-quality switch, which in turn plays a critical role in both human capital accumulation and economic development. However, the inclusion of the endogenous dynamics of HIV generates a rich interplay amongst the macroeconomic set up, the diseases and its possible intervention strategies, which have the potential to affect in a complicate manner the possibility to switch between the two development regimes as well as to promote significant quantitative differences within each of them. Additionally, thanks to the flexibility of our formulation of control strategies (public policy versus donors versus private education) the model allows to pinpoint a rich number of endogenous policy options that can be used by the afflicted country in the battle against the HIV disease, which however can have profoundly different impacts on the development regimes depending on the specific mix of the interventions adopted.

On one hand, high levels of HIV prevalence have the potential to relegate the economy in its poverty trap, where the lack of ability to sustain education makes unavoidable the need for international foreign aid donations. Obviously, in low-resource settings, that is countries already entrapped in poverty, AIDS can dramatically worsen the conditions for escaping underdevelopment. On the other hand, lower levels of HIV prevalence and/or the ability to effectively control the epidemics can promote the escape from the poverty trap, entering a regime where the development

of education and the accumulation of human capital make available further tools for successfully fighting the disease, eventually entering a virtuous circle where HIV-afflicted countries can afford to successfully fight the battle exclusively with internal resources. Unlike Young (2005), this model shows that the increase in adult mortality due to HIV/AIDS can represent a serious threat to economic development by its upward pressure on the precautionary demand for children and downward pressure on education, as first stressed by Kalemli-Ozcan (2002), which in turn can dramatically reduce physical and human capital accumulation.

The rest of the article proceeds as follows. Section 2 reviews the main facts about the demographic and economic impact of the HIVAIDS epidemic in SSA, with a special emphasis on the critical issue of funding HIV control programmes. Section 3 presents the macro-economic dynamic framework. Section 4 studies the dynamics of the model. Section 5 reports and discusses the main results of the work. Section 6 outlines the main conclusions.

2. HIV/AIDS in SSA: natural history, mortality, fertility, and the demographic transition

2.1. Global facts about HIV/AIDS

Several scholars have identified the still dramatic levels of mortality, with the highest toll from communicable diseases worldwide, and the underlying poor health conditions, as the major issue responsible for the underdevelopment in SSA (e.g., Bloom and Canning, 2004; Lorentzen et al., 2008). Notwithstanding these tragic circumstances, SSA countries were able to achieve continued (though slow) progresses in life expectancy until the early 1980 i.e., prior the HIV/AIDS initiated to ravage the region yielding a tragedy of major proportions.

While in several other regions of the world the HIV epidemics remained confined for years in special and therefore relatively small risk groups such as men having sex with men, intravenous drug users, and blood transfused, since the very beginning of the pandemics the spread of HIV in SSA was mainly driven by heterosexual transmission in the young adult population thereby favouring high rates of disease spread as well as large rates of vertical transmission to new-born

children from infected mothers. Fast diffusion was further enhanced by a number of factors highly specific to the context of SSA including high rates of sexual partner change, high rates of prostitute attendance, almost no use of protections during sexual intercourse, large human mobility due to seasonal works, sporadic and largely ineffective awareness campaigns – with a few notable exceptions – such as the Ugandan case (Green et al 2006) – up to the phenomenon of AIDS denialism, which in some cases, as that of South-Africa, was unbelievably promoted at the highest policy level, namely by the country prime minister (Fassin and Schneider, 2003, Simelela and Venter, 2014). These circumstances allowed HIV to reach large prevalence levels in several SSA countries, thereby causing AIDS to rapidly emerge – and to remain for about 25 years – the leading cause of mortality (UNAIDS, 2017).

The updated UNAIDS central estimates (UNAIDS, 2017) document that HIV/AIDS - by far the major pandemic of the current epoch, with 76 million people infected by HIV and 35 million died from AIDS related illnesses (primarily Tuberculosis) since the onset of the epidemic in the late seventies up to 2016 – has devastated SSA. Still in 2016, out of a total of 36.7 million people living with HIV worldwide, with an incidence of about 1.8 million new HIV infections and 1 million deaths from AIDS-related illnesses, SSA – which in the same period was homing 13% of the world population – accounted for 25.5 million people living with HIV, i.e. 70% of total HIV prevalence worldwide, for 64.5% of new infections, for 73% of total estimated deaths from AIDS, and for 85,5% new infections among children (UNAIDS, 2017). However, these figures represent a dramatic decline (-42%) compared to the disease peak phase occurred between 2001 and 2005, when the number of AIDS-related deaths in SSA was in the region of 1.7 million per year. The factors underlining this decline are complicate and include awareness campaigns – that essentially represented the only available weapon against the infection until 2005 – aimed to reduce risky behaviour, possible saturation effects in the at-risk population – and the growing access of SSA population to antiretroviral treatments. Considering that essentially no treatments were available in SSA still in 2005, progresses have been dramatic: in 2016 about 50% of those living with HIV in

SSA were accessing to therapies though with large regional disparities (about 60% in southern and eastern Africa vs only 35% in western and central Africa).

2.2. Country-level epidemics

A major distinctive trait of HIV/AIDS in SSA, as by the way all over the world, is the marked heterogeneity in prevalence amongst different communities as clear from the temporal trends of country-level prevalence of HIV infection in the most afflicted countries (Fig. 1). For example, in Zimbabwe, that possibly suffered the most aggressive HIV epidemics worldwide with a peak in the region of 29% in the 1997-1998, the epidemics already entered in a declining phase since the 2000, thus well before the introduction of therapies. This has been primarily attributed to the success in the awareness campaigns conducted therein, which were subsequently reinforced by the spread of therapies (Halperin, 2013). However, in the recent years the prevalence curve seems stalling at levels still exceptionally high (more than 15%) suggesting that the battle is far from being won. A similar trend was observed in Botswana, and at lower prevalence regimes in Malawi and Namibia. Somewhat differently, in Swaziland (27,5% prevalence at 2014), Lesotho (23% prevalence at 2014) and South-Africa (about 19% prevalence at 2014), HIV prevalence has reached a plateau between 2000 and 2005, remaining essentially constant or even slightly increasing thereafter. These slightly increasing trends also reflect the positive effects of therapies, which extend the duration of sojourn in the seropositive (i.e., HIV) phase, thereby increasing the size of the HIV population. Nonetheless, this effect is also illustrative of the complexities of intervention against HIV: any success of therapies in delaying the transition to full-blown AIDS disease has the effect of increasing the size of the HIV population. As the latter includes individuals in good health state and infective, that is capable to retransmit the infection to others, efforts to keep awareness high and ensure safe sexual behaviour will require a lot of effort in the very long-term.

[Figure 1 about here]

2.3. HIV/AIDS and life expectancy

The growth in HIV prevalence curves, followed with a delay of some years, by the corresponding growth in full-blown AIDS prevalence curves, and ensuing rapid death (in the absence of therapies) was mirrored by a dramatic impact on life expectancy all over SSA but especially in countries with large HIV epidemics. Figure 2 reports UN (2017) medium variant estimates and projections for the post-1950 trend in the life expectancy at age 15 (e_{15}) in the SSA countries suffering the most severe HIV epidemics (as reported in Figure 1). Life expectancy at age 15 was chosen as representative of the expected lifespan for individuals entering young adulthood in SSA. As previously stated, HIV/AIDS was able to dramatically reverse the long-lasting – though slow – increasing trend in life expectancy showed everywhere in SSA. In the worst epidemics, in Zimbabwe, the life expectancy at age 15 that was estimated in the range of 54 years between 1985-1990 i.e., before the onset of HIV/AIDS, fell between 2000 and 2005 (that is, as expected, a few years after the predicted peak in prevalence reported in Figure 1) to a level only slightly in excess of 30 years. Though the decline in HIV prevalence observed in Zimbabwe in subsequent years is predicted to allow life expectancy to return to the pre-AIDS levels already by 2015-2020, things are dramatically different in other contexts. For example, in Lesotho and Swaziland e_{15} is projected by the UN medium variant to remain substantially below its pre-AIDS level for decades, and to return to it only between 2060 and 2070 (Fig. 1b). Which will be the ultimate consequences of the persistent fall in life expectancy for young adults on economic decisions (e.g., investment in children and their education) is presently unclear. Surely, mortality decline has represented the major trigger of the fertility transition in both conventional demographic explanations (Bongaarts and Casterline, 2012; Livi-Bacci, 2017), emphasising fertility decline as a direct homeostatic response to increasing survival, and modern demo-economic theories (Galor, 2011), emphasising the endogenous nature of mortality decline which promoted investments in education, thus favouring the switch in children's demand from "quantity" (Malthusian growth regime) to "quality" (modern growth regime).

Focusing on the role played by the exceptionally high mortality of adults in SSA, Lorentzen et al. (2008) showed that it was the main cause of the collapsed economic growth and development in the region via shortening time horizons, increasing risky behaviours and eventually raising fertility.

[Figure 2 about here]

2.4. HIV/AIDS and the fertility transition

The issue of the possible effects of HIV/AIDS on fertility decisions in SSA raised in the previous section, is currently the object of a tough debate in the recent empirical economic literature.[i] The resulting conclusions are however dramatically variable, with some studies suggesting that AIDS mortality is already having a substantial positive effect on fertility in SSA and others predicting that AIDS is not having any effects or even lowering fertility in SSA (an extensive literature review on the subject is reported in Gori et al., 2017).

As remarked by influential scholars (Bongaarts and Casterline, 2012), the fertility transition in SSA has been dramatically delayed (the most dramatic evidence is represented by the case of the Democratic Republic of the Congo, Romaniuk, 2011) compared to other world regions started from similar initial conditions, such as Asia and the Latin America (Bongaarts and Casterline, 2012). In this general context, a worrying news has been represented by the generalised slowing down of fertility decline in the early 2000's (Schoumaker, 2009) and especially by the symptoms of stalling or even relapsing fertility in the group of countries suffering the most severe HIV/AIDS epidemics, which notably were SSA leaders in fertility decline at the onset of the HIV epidemics, according to UN data (UN, 2017). Additionally, in South Africa, where fertility was decreasing since already 1950, the decline was dramatically slowed down after HIV prevalence became substantial in the 1980's. This is a remarkable fact since, on the contrary, in SSA countries suffering intermediate HIV epidemics (e.g., Malawi, Zambia, Mozambique and Kenya), fertility decline slowed down

during 1995-2005 but accelerated thereafter (UN, 2017), while no slowing down was observed in countries suffering mild epidemics.

Though these are just temporal associations based on aggregate data, the possibility that the persistent mortality blow-up due to AIDS might cause a paralysis or even a reversal in the fertility transition in SSA might have such dramatic implications for the development of the region so to represent, in our opinion, one of the major conundra for current economic development studies.

3. The model

This section builds on a modified version with HIV spread of the growth model of Yakita (2010) with physical and human capital accumulation. The OLG (general equilibrium) closed economy is populated by a continuum of finite-lived (perfectly) rational and identical individuals of size N_t per generation ($t = 0,1,2,...$ is the time index). The length of each generation is conventionally set at 25 years. The life of the typical agent belonging to generation t is divided into childhood, young adulthood and old age. As a child, an individual does not make any economic decisions and directly consumes resources in the parent's household, while also receiving education from his parents. We consequently avoid including AIDS-related child mortality in the model as justified by the fact that the AIDS-mortality burden amongst children (following by vertically transmitted AIDS) is steadily decreasing thanks to pre-natal treatments, and anyhow second-order compared to AIDS-related adult mortality in SSA (UNAIDS, 2018). Therefore, we only consider the children surviving at the entry of adulthood (n_t) as the key variable of the model (Galor and Weil, 1996; Gori and Sodini, 2019). As a young adult, an individual is economically and sexually active. He works, saves, gives birth and takes care of children, and may also acquire HIV infection. The HIV spread follows the rule developed by Chakraborty et al. (2016), which will be clarified below. In addition, he invests the amount e_t per child for educational purposes of their children. When young, an individual is endowed with 1 unit of time. We assume that raising children is a purely time-consuming activity

(see Guryan et al., 2008 for empirical evidence). The child rearing technology requires a positive exogenous fraction $z > 0$ of the parent's time endowment to raise a child (representing parent's foregone earnings), i.e. the time required to care for children cannot be spent working. As n_t represents the number of surviving children at time t, zn_t is the time needed to care for n_t descendants of a parent that belongs to generation t. This implies that the opportunity cost of children is proportional to the wage rate per effective labour (w_t), which is provided to firms for production purposes. Therefore, the budget constraint when young reads as follows:

$$s_t + e_t n_t = (1-\tau)(1-zn_t)w_t h_t, \tag{1}$$

where s_t is saving, $e_t n_t$ is the total private expenditure that parents devote to educate their own n_t children, $0 \le \tau < 1$ is the constant labour income tax rate levied by the government to finance a public expenditure specifically devoted to fight HIV, and $w_t h_t$ is the total labour income of an individual of the working-age generation who has h_t efficient units of human capital. Of course, the condition $n_t < 1/z$ should always hold to guarantee economic feasibility.

By following Yakita (2010), the labour productivity of a young adult individual is given by the stock of human capital endowed at the beginning of the working period. The stock of human capital of an individual belonging to the next (working-age) generation (h_{t+1}) depends on the educational expenditure per child provided by the current working-age generation to their children (e_t) and the existing stock of human capital (h_t). The human capital accumulation rule therefore is the following:

$$h_{t+1} = \varepsilon(e_t + \theta h_t)^{\delta} \bar{h}_t^{1-\delta}, \quad \varepsilon, \theta > 0, \quad 0 < \delta \le 1, \tag{2}$$

where $\bar{h}_t$ is the average or economy-wide stock of human capital reflecting the spillovers from the society at time t, ε is a scaling parameter, δ is the constant elasticity of the overall private component $e_t + \theta h_t$ in human capital accumulation. This term $e_t + \theta h_t$ implies perfect substitutability in the process of human capital accumulation between the expenditure in education

provided by parents to children and the ability of children to absorb knowledge by looking at their own parents at work (parental background). This ability is tuned by the positive parameter $\theta > 0$ (see Yakita, 2010 for details).

The survival probability of a young adult agent belonging to generation t, $0 < \beta_t \leq 1$, is endogenous and determined by his individual state of health. The existence of HIV/AIDS makes the individual health status worse off so that adult survival negatively depends on the prevalence rate of HIV infection i_t, representing the proportion of agents infected at time t. We assume that this probability has the following simplified form:

$$\beta_t = \beta_A(1-i_t), \tag{3}$$

where $\beta_A < 1$ represents (exogenous) survival from causes of death different from AIDS. This formulation is obviously over-simplified, but it aims to reflect the well documented facts that prior to the HIV/AIDS crisis: 1) life expectancies at all ages were increasing everywhere in SSA (UN, 2017), despite the dramatic burden of mortality claimed by other communicable diseases such as malaria and tuberculosis; 2) HIV/AIDS has represented for three decades the most important cause of death in SSA. The purpose of the formulation of endogenous survival implicit in Eq. (3) is to reflect the role of premature death of adults due to HIV/AIDS as the dominant effect of the epidemic at the macroeconomic level. We are aware that there are several other important effects of HIV, first the burden of child mortality through vertical transmission. Although dramatically relevant from a humanitarian point of view, this factor does not affect the structure of the model as it deals with non-economically active agents (children).

Young adults may acquire HIV infection by sexual transmission only. The probability p_t that an HIV-susceptible agent will become infected is defined as follows (Chakraborty et al., 2016):

$$p_t = 1-(1-i_t\pi_t)^{\mu}. \tag{4}$$

where $0 < \pi_t \leq 1$ is the probability of acquiring the infection per sexual partnership and $\mu > 0$ is the average number of sexual encounters that a young agent has during his entire 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. In this model, the probability π_t of acquiring HIV per sexual partnership negatively is assumed to depend on three main factors, which are: (i) private education (e_t) that contributes to raise individual awareness against the risk of HIV infection and therefore promotes subsequent actions such as the use of protections during sexual intercourse, (ii) public interventions, which are endogenously managed by the government of the afflicted country, that are provided in the model on a per worker basis (g_t),[ii] and (iii) foreign aid donations that are instead provided on a per young basis (d_t). The last component is assumed to depend on two main factors that should drive international programmes in SSA, namely the rate of HIV prevalence and the GDP of the afflicted country (Haacker, 2009), which is measured on a per young basis for analytical convenience. We will turn to the specifics of all these three components affecting π_t later in this chapter. Then,

$$\pi_t = \frac{\pi_A}{1+\pi_B(qe_t^* + g_t + d_t)^{\omega}}. \tag{5}$$

where $\omega > 0$ is a parameter that controls the degree of effectiveness of the overall amount of resources aimed at fighting the HIV disease, $0 < \pi_A \leq 1$ ($\pi_A > 1/\mu$), $\pi_B > 0$ and $e_t^* := e_t / w_t h_t$ is the expenditure per child over the individual total income and $q > 0$ represents the relative weight of the educational component in reducing the probability of acquiring HIV per sexual partnership.

If an individual survives at the onset of old age, his material consumption is based on accumulated saving. The existence of a perfect market for annuities allows an agent to make his own saving being intermediated through mutual funds. This assumption follows a recent established literature on endogenous lifetime in growth models dealing with economic development issues (Chakraborty, 2004; Fanti and Gori, 2014; Chakraborty et al., 2016), and implies that the savings of those who prematurely die (due to AIDS in this context) are allocated amongst those who are still alive.[iii] Therefore, old-age material consumption (c_{t+1}) is constrained by the capitalised amount of

resources saved when young divided by the survival probability of a young person of generation t, that is:

$$c_{t+1} = \frac{R^e_{t+1}}{\beta_t} s_t, \tag{6}$$

where R^e_{t+1} is the interest factor that individuals of generation t expect will prevail at time $t+1$.

The individual representative of generation t has preferences toward the number of children when young and material consumption and the quality of children (represented by the human capital accumulated through education) when he will be old. Therefore, the expected lifetime utility function has the following form:

$$U_t = \ln(n_t) + \beta_t[\ln(c_{t+1}) + \gamma \ln(h_{t+1})], \tag{7}$$

where $\gamma > 0$ measures the relative degree of altruism towards (the quality of) children and β_t, which resembles the subjective discount rate, is given by the expression in (3) and then it is negatively affected by the prevalence rate of the HIV disease, i_t. In other words, an increase in i_t causes an increase in adult mortality (i.e., a reduction in the intertemporal discount factor) and this in turn implies that individuals of generation t reduce the relative importance (in the utility function) of consuming material goods and enjoying having educated children when old because they live shorter. By taking as given factor prices, the tax rate and the prevalence rate of HIV, the representative agent maximises the expected lifetime utility function in (7) with respect to material consumption (c_{t+1}), the number of children (n_t) and the amount of educational expenditure per child (e_t) subject to the lifetime budget constraint

$$\frac{c_{t+1}\beta_t}{R^e_{t+1}} + e_t n_t = (1-\tau)(1-z n_t) w_t h_t, \tag{8}$$

obtained by combining (1) and (6). Then, he gets the following optimal values for the saving rate s_t, the number (precautionary demand) of children n_t and the quality (education) of children e_t:[iv]

$$s_t = \frac{\beta_t(1-\tau)w_t h_t}{1+\beta_t}, \tag{9}$$

$$n_t = \begin{cases} \dfrac{1}{z(1+\beta_t)} & w_t \le \bar{w}_t \\ \dfrac{(1-\beta_t\delta\gamma)(1-\tau)w_t}{(1+\beta_t)[z(1-\tau)w_t-\theta]} & w_t > \bar{w}_t \end{cases}, \tag{10}$$

and

$$e_t = \begin{cases} 0 \quad if \ w_t \le \bar{w}_t \\ \dfrac{h_t[z(1-\tau)\beta_t\delta\gamma w_t-\theta]}{1-\beta_t\delta\gamma} \quad if \ w > \bar{w}_t \end{cases}, \tag{11}$$

where $\bar{w}_t := \dfrac{\theta}{z(1-\tau)\beta_t\delta\gamma}$ is the threshold value of the wage rate per effective labour above which the representative individual has an incentive to invest in child quality. We note that the condition $w_t > \bar{w}_t$ also guarantees a positive value of the precautionary demand for children. For details regarding the discussion of marginal benefits and costs of investing in child quality see Yakita (2010). A specific feature of our model in comparison with the one developed by Yakita (2010) is the role played by the survival probability β_t on the critical value $\bar{w}_t$ that discriminates between the corner solution (a state in which parents do not want to invest in child quality) and the interior solution (a state in which parents are likely to spend on education of their children rather than having more children). As β_t negatively depends on i_t, it is straightforward to see that an increase in the rate of HIV prevalence reduces the survival probability of adults by increasing $\bar{w}_t$ thus making less convenient investing in education, as the marginal cost of educating children increases. This model therefore includes the quantity-quality trade-off as well as the substitutability between education provided by parents and intergenerational human capital transmission within the family in the process of human capital accumulation in presence of an endogenous mechanism of HIV transmission. These are reasonable ingredients to characterise SSA economies.

At time t, the government provides the amount G_t of public expenditure to fight HIV. The term G_t therefore includes any type of relevant actions such as building health infrastructures, providing effective antiretroviral therapies (ART) for seropositive and AIDS-sick individuals, physicians and medicines, promoting awareness on the risk of infections and therefore the adoption of safe sexual practices such as e.g. the use of condoms and so on, with the purpose to reduce the probability of acquiring HIV per sexual partnership.[v] These expenditures are devoted to young-adult people, which are the ones who are HIV-susceptible and may then acquire HIV through sexual transmission. The HIV spending is financed by the government of the afflicted country at a balanced budget through labour income taxation. As the tax rate is $0 < \tau \leq 1$, the total amount of tax revenues is $\tau w_t L_t$, where L_t is the quantity of effective labour employed. Defining now $g_t := G_t / L_t$ as the amount of public expenditure per worker, the government accounting rule at a balanced budget is the following:

$$g_t = \tau w_t . \tag{12}$$

In addition to the public (internal) HIV-related expenditure, there exist external resources provided by international organisations (e.g., UN and Bill & Melinda Gates Foundation) to high HIV-prevalent SSA countries. These resources (defined on a per young basis) depend on the GDP of the afflicted country (UNAIDS, 2018) and the rate of HIV prevalence according to the following rule:

$$d_t = \begin{cases} ai_t - by_t & if\ i_t > (b/a)y_t \\ 0 & if\ i_t \leq (b/a)y_t \end{cases}, \tag{13}$$

where $a, b > 0$ are the share of external resources devoted to each single HIV-afflicted individual at time t (based on the rate of HIV prevalence) and the share of external resources per head devoted to each young living at time t (based on income), respectively. The rule in (13) implies that international organisations increase foreign aid in SSA as HIV prevalence increases and reduce it as the HIV-afflicted country gets richer. When the HIV prevalence in one country is large (resp. small)

enough, external donations are based on a mix between the two quotas discussed above (resp. become null). In the last case, the fight against the HIV/AIDS disease is entirely based on internal resources of the afflicted country, which then becomes fully autonomous in the battle against HIV, according to the auspices raised by the deputy director of UNAIDS (UNAIDS, 2013).

As individuals are identical, the condition $h_t = \bar{h}_t$ holds in a symmetric equilibrium. Therefore, depending on the level of education coming from the interior or corner solution in (11) of the individual optimisation problem, human capital accumulation (2) becomes the following:

$$h_{t+1} = \begin{cases} \varepsilon\theta^{\delta} h_t & \text{if } w_t \leq \bar{w}_t \\ \varepsilon(\beta_t \delta\gamma)^{\delta} \left[\dfrac{z(1-\tau)w_t - \theta}{1-\beta_t\delta\gamma} \right]^{\delta} h_t & \text{if } w > \bar{w}_t \end{cases}. \tag{14}$$

When the wage is small enough ($w_t \leq \bar{w}_t$) parents choose to do not provide education to their children. In this case, the human capital growth rate is constant and depends only on parental background. It may grow indefinitely, shrink or follow a stationary pathway depending on whether the term $\varepsilon\theta^{\delta}$ is larger than, smaller than or equal to one, respectively. In contrast, when the wage is large enough ($w_t > \bar{w}_t$) there exists a child quantity-quality trade-off as parents want to provide education to their children and the growth rate of human capital depends on the wage rate per effective labour.

Regarding the production side of the economy, firms are identical and act competitively on the market. The time-t representative firm produces a homogeneous good Y_t by combining physical capital K_t and the amount L_t of effective labour. These inputs are combined by using a Cobb-Douglas technology with constant returns to scale, that is:

$$Y_t = K_t^{\alpha} L_t^{1-\alpha}, \quad 0 < \alpha < 1. \tag{15}$$

The temporary equilibrium condition in the labour market at time t is determined by equating labour demand and labour supply, that is $L_t = (1 - zn_t) h_t N_t$. 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 rate and the interest factor are equal to the marginal product of efficient labour and the marginal product of capital, respectively, that is:

$$w_t = (1-\alpha)v_t^{\alpha}(1-zn_t)^{-\alpha}, \tag{16}$$

and

$$R_t = \alpha v_t^{\alpha-1}(1-zn_t)^{1-\alpha}, \tag{17}$$

where $v_t := k_t / h_t$ is the physical to human capital ratio, $k_t := K_t / N_t$ is the stock of physical capital per young person and GDP per young person is defined as $y_t := Y_t / N_t = v_t^{\alpha}(1-zn_t)^{1-\alpha}$.

Equilibrium in the capital market is determined by equating aggregate saving by the representative individual and aggregate investment by the representative firm, that is $S_t = I_t$. The inter-temporal equilibrium condition in the capital market requires that the aggregate capital stock installed at time $t+1$ is equal to the gross aggregate investment at time t. As physical capital fully depreciates at the end of each period, we get $K_{t+1} = I_t = S_t = s_t N_t$. Knowing that $K_{t+1} := k_{t+1}N_{t+1}$ and $N_{t+1} = n_t N_t$, equilibrium is characterised as follows:

$$k_{t+1} = \frac{s_t}{n_t}. \tag{18}$$

For the sake of simplicity, from the next section we set the constant β_A (representing survival from causes of death different from AIDS) to one, as in this context it merely represents a scaling parameter without relevant qualitative effects.

4. Main theoretical predictions

Depending on the size of the wage rate w_t compared to the threshold $\bar{w}_t$, there exist two different development regimes, as in Yakita (2010). If individuals are relatively poor ($w_t \le \bar{w}_t$), the economy is entrapped in a phase of underdevelopment. If they become richer ($w_t > \bar{w}_t$), the economy may follow a sustained development trajectory.

(1) [Underdevelopment, $w_t \leq \bar{w}_t$]. The individual optimal choices are described by the saving function defined in (9) as well as by the values of the first expression of n_t and e_t in (10) and (11), respectively. In this regime, individuals do not invest in education and there exists only a precautionary demand for children. By using the same strategy of Yakita (2010), it is possible to study the evolution of the physical to human capital ratio and the HIV prevalence through the following map M_1 defined in the variables v_t and i_t, that is

$$M_1 : \begin{cases} v_{t+1} = \dfrac{(1-\alpha)(1-\tau)z}{\varepsilon\theta^{\delta}} v_t^{\alpha}(1-i_t)^{1-\alpha}(2-i_t)^{-\alpha} \\ i_{t+1} = 1-\left[1-i_t\dfrac{\pi_A}{1+\pi_B(g_t+d_t)^{\omega}}\right]^{\mu} \end{cases}, \tag{19}$$

where $g_t = \tau w_t = \tau(1-\alpha)v_t^{\alpha}\left(\dfrac{2-i_t}{1-i_t}\right)^{\alpha}$ is the amount of internal public spending against HIV on a per worker basis and foreign aid d_t is defined in (13). If foreign aids and/or the internal public health are strong enough to eliminate HIV in the phase of underdevelopment, the model essentially falls within the one-dimensional system studied by Yakita (2010), in which the evolution of all the variables can be characterised by studying the dynamics of v_t.

Unfortunately, studying system (19) is not an easy task as it cannot be dealt with in a neat analytical form. Indeed, it can exhibit a plethora of stationary states with positive values of the HIV prevalence ($i_t > 0$) in addition to the one characterised by the absence of HIV/AIDS ($i_t = 0$). In this regard, we must distinguish between the case in which international organisations devote resources in the fight against HIV, $v_t < \hat{v}_t$, to the case in which foreign aids are absent, $v_t \geq \hat{v}_t$, where $\hat{v}_t := \left(\dfrac{ai_t}{b}\right)^{\frac{1}{\alpha}}\left(\dfrac{1-i_t}{2-i_t}\right)^{\frac{\alpha-1}{\alpha}}$ represents the threshold value of the physical to human capital ratio that discriminates between international intervention and no international intervention, according to the

rule specified in (13). We recall that the existence of (one or more) stationary states in the underdevelopment regime is possible only whether the condition $v_t \leq \bar{v}_t := \left[\frac{\theta}{z(1-\tau)\delta\gamma(1-\alpha)(1-i_t)}\right]^{\frac{1}{\alpha}}\left(\frac{1-i_t}{2-i_t}\right)$ is satisfied, where $\bar{v}_t$ is evaluated at its stationary state value. This threshold is obtained by replacing n_t with its optimal value, through the first expression of Eq. (10), in the equation of the marginal product of efficient labour in (16) and using $\bar{w}_t$. In this regard, we recall that, apart from the infectious dynamics, a stationary state in this model can be related to unbounded growth of physical and human capital if $\varepsilon\theta^{\delta} > 1$ or to a situation where the human capital continuously reduces and the physical capital tends towards its stationary state level if $\varepsilon\theta^{\delta} < 1$ (see Yakita, 2010, for details). If the physical to human capital ratio is larger than the critical value $\bar{v}_t$, there exist no stationary states with a positive value of v in the underdevelopment regime. The stability properties of the stationary states of the dynamic system (19) are studied by resorting to numerical analyses given the complexity of the expressions of the map.

(2) [Sustained development, $w_t > \bar{w}_t$]. In this case, the individual optimal choices are described by the saving function defined in (9) as well as by the values of the second expression of n_t and e_t in (10) and (11), respectively. In this regime, individuals are rich enough to invest in education and therefore they decide to trade off child quantity against child quality. Then, equilibrium dynamics of v_t and i_t are described by the following map M_2:

$$M_2 : \begin{cases} v_{t+1} = \dfrac{\dfrac{1-i_t}{2-i_t}\dfrac{1-\alpha}{(1-zn_t)^{\alpha}}}{\varepsilon\left[\dfrac{(1-i_t)\gamma\delta}{1-(1-i_t)\gamma\delta}\right]^{\delta}\left[z(1-\tau)(1-\alpha)\left(\dfrac{v_t}{1-zn_t}\right)^{\alpha}-\theta\right]^{\delta} n_t} v_t^{\alpha} \\ i_{t+1} = 1-\left[1-i_t\dfrac{\pi_A}{1+\pi_B(e_t^{*}+g_t+d_t)^{\omega}}\right]^{\mu} \end{cases}, \qquad (20)$$

where $e_t = \frac{h_t[z(1-\tau)\beta_t\delta\gamma w_t - \theta]}{1-\beta_t\delta\gamma}$, $g_t = \tau w_t = \tau(1-\alpha)v_t^{\alpha}(1-zn_t)^{-\alpha}$, d_t is defined in (13) and n_t is determined as the *unique* solution of $n_t = \frac{[1-(1-i_t)\delta\gamma](1-\tau)(1-\alpha)v_t^{\alpha}(1-zn_t)^{-\alpha}}{(2-i_t)[z(1-\tau)(1-\alpha)v_t^{\alpha}(1-zn_t)^{-\alpha} - \theta]}$. Given the complexity of the dynamic system (20) and the problems related to the analytical tractability of characterising the possible switches between the two distinct regimes governed by maps M_1 and M_2, in what follows we will resort to numerical simulations to concentrate on some demo-economic results of the model. In doing this, we will follow the methodology introduced by Chakraborty et al. (2010, 2016) by fixing the main parameters of the problem at economically meaningful values for SSA countries and then studying the effects of both internal and external health policies on the HIV prevalence, the economic dynamics and their (macroeconomic) interplay.

5. Dynamics of the model and discussion of the main results

This section builds on a simulative exercise aiming at studying the economic and epidemiological dynamics characterised by Eqs. (19) and (20). The parameters of the model are chosen as follows: $\alpha = 0.37$ (Gollin, 2002), $\delta = 0.535$, $\theta = 0.011$, $\varepsilon = 22.3$ (these values are similar to those used by Yakita, 2010), $\gamma = 1.52$, $z = 0.12$ (assigned to obtain a fertility rate in line with SSA), $\pi_A = 0.0123$, $\pi_B = 35$, $\mu = 100$, $q = 0.1$ and $\omega = 1$ (assigned to get an equilibrium prevalence rate of 35% in the case of an uncontrolled HIV in the phase of underdevelopment).

First, we considered an economy without the HIV disease that moves away (at time $t = 3$) – thanks to human capital accumulation – from the underdevelopment regime, where parents did not invest in education and there existed only a precautionary demand for children, to a sustained development regime, where there is a child quantity-quality trade off. We have considered a parameterisation excluding the existence of poverty traps due to the initial conditions (history). This is because the main aim of the work is to stress the role of HIV in determining a fall back in

poverty. The economy begins with a situation where fertility is constant and fixed at 4.16 children per woman in a phase of underdevelopment, ending up in context of sustained development where each parent has 2.85 children on average. In this last regime, education helps reducing the precautionary demand for children by changing the marginal conditions for having an extra child. At $t = 6$, the HIV disease begins ($i_6 = 0.01$) and the prevalence rate would reach 35% (in line with the values of some high-prevalent SSA countries) in the case of an uncontrolled spread. HIV is responsible for the falling back of the economy into a phase of underdevelopment due to a lowering life expectancy leading to a lower saving and higher fertility due to increased mortality by also reducing education to zero (child quality-quantity trade off). It is important to note that education has partially slowed down the development of HIV (clearly, education becomes zero when one falls back into the early stage of economic development).

An important role in addressing the humanitarian emergency due to HIV infection has been played by international aids from both private and public institutions (donors). In our simulative exercise, we calibrated these interventions by linking them positively to the prevalence rate and negatively to the income produced by the afflicted country (measured on a per young basis) according to the rule: the higher income, the lower international donations. Foreign aids start at $t = 48$ when the HIV prevalence is almost $i_{48} = 0.23$ succeeding in bringing the amount of infected people in the afflicted country below the threshold of 1% and letting the economy move on a sustained development path with positive levels of education and lower fertility. Once donors are reset to zero because the prevalence rate has gone below the critical threshold ($i_t = (b/a)y_t$, which was set at 1% with $a = 4$ and $b = 0.17$), we explored the possibility of an endogenous intervention by the government of the afflicted country aiming at replacing external aids with an internal policy financed by levying taxes on labour income ($\tau = 0.1$). We note that by setting the tax rate τ at too high a level would greatly reduce individual disposable income and saving, which in turn reduce physical capital accumulation by also stimulating the precautionary demand for children.

Differently, setting the tax rate at too low a level would be ineffective and would make the disease spreading again. Therefore, the public expenditure against HIV initially causes a reduction in the demand for education (child quality) and increases fertility (child quantity) but at lower birth rates than those observed when the donors were active. This is because the direct negative effect on income due to labour income taxation is high and dominates the indirect positive effect of a lower HIV on mortality reduction. However, this taxation can be decreased over time, thus making the endogenous policy dynamically reactive and linked to the prevalence rates observed. Also, in this phase education plays a positive role and contributes to reducing both the number of children and HIV, but with a much lower intensity than in a pre-AIDS setting. We note, however, that long-term fertility is higher than the values observed in the (pre-AIDS) early stages of demographic transition. AIDS therefore represents a major cause depressing the development pathway of SSA countries (Figures 3 and 4).

[Figures 3 and 4 about here]

6. Conclusions

For more than three decades, HIV/AIDS has ravaged SSA and still now is claiming dramatic toll of deaths. Nonetheless, the increasing diffusion of anti-retroviral therapies and awareness campaigns have finally downturned the epidemic curve and brought optimistic perspectives on the control of the disease, as summarised in the global 90-90-90 plan (UNAIDS, 2018) aiming to bringing AIDS under full control by 2030. However, the battle against HIV in SSA is far from its end and several challenges are still open. Amongst these, we believe that there are at least three major threats that are critical for their potential effects to economic development and therefore should deserve major attention at the highest policy level, that is (i) the potential for an HIV-induced paralysis of the fertility transition, (ii) the need for a dramatic expansion of the amount of resources devoted to funding the interventions against HIV, and (iii) the unclear magnitude of the

emerging phenomenon of resistance to treatments. This work developed a macroeconomic dynamic framework capable to account for both the first two previously mentioned factors within a UGT-like growth set up. The model includes an explicit HIV transmission mechanism – as first proposed by Chakraborty et al. (2010, 2016) – and different funding rules of the interventions against HIV, covering almost all the kinds of interventions used in actual SSA countries. We believe that the explicit inclusion of the epidemic dynamics goes far enough beyond the possibility offered by models simply treating HIV as an exogenous mortality shock, as it allows to pinpoint the dynamic interplay between the time scales of the epidemics with those of fertility decline and, ultimately, of economic development.

Our main conclusions dramatically contrast Young's (2005) results, according to which AIDS is a humanitarian disaster but not an economic disaster. Indeed, we found that HIV has the potential to constrain an economy in an underdevelopment regime with high HIV-prevalence rates and high fertility, or to worsen the conditions for being entrapped in poverty by reversing the long-term trend of fertility decline. The issue of the AIDS-induced fertility reversal is currently a controversial one. In Gori et al. (2017), we have surveyed several empirical contributions on the subject, showing a wide degree of disagreement in the responses, with a number of papers suggesting that HIV has a potential not only for stalling but also reversing the fertility transition in SSA, as first suggested in the seminal works of Kalemli-Ozcan and Turan (2011) and Kalemli-Ozcan (2012), and a similar number of papers reaching opposite conclusions. However, we found that countries suffering a combination of the following ingredients: 1) a stall in the fertility transition, 2) a large HIV epidemic, 3) negative perspectives on life expectancies requiring long intervals of time to restore the pre-AIDS levels, as is the case for example of Lesotho, should represent the highest priority for international interventions in SSA. We feel that this message is not so far at the top of the debate on the topic. On top of this, the present work has added the issue of the financing of the battle against HIV/AIDS. The model clearly enlightens the conditions under which an international intervention is necessary versus the circumstances that may allow afflicted countries to support the AIDS battle by

using internal resources without compromising the escape form the poverty trap. In this last case, private education might play a key role in the battle against HIV by enhancing the internal public policy, therefore setting in motion a virtuous circle capable to simultaneously control epidemics and fertility.

We did not explicitly include in the model the third challenge, namely the resistance issue. This was due to the lack of clear evidence on the magnitude of the phenomenon in SSA which prevented a meaningful parametrisation of it. Nonetheless, the widespread diffusion of resistance would simply worsen the control conditions of HIV/AIDS predicted by our model and ultimately the perspectives for economic development.

The list of possible future developments is clearly endless. A point of major interest in our research agenda regards the implications (in terms of both survival and education) for the children of adults prematurely dying of AIDS. In this regard, a naturally raising question is whether orphans suffer higher mortality and lower education opportunities than non-orphans (particularly in high-prevalence HIV settings) and then how to build a proper model for this, for instance along the lines of the approach proposed by Bell and Gersbach (2013). In general, this is a complicate problem for the model considered in our chapter that cannot be solved simply by adding (endogenous) child mortality, as this is a general shortcoming of OLG settings regardless of whether fertility is endogenous (for instance, the UGT set up) or exogenous (Diamond, 1965). In fact, both frameworks did not account for an economically active childhood generation. In our opinion, a solution to the problem would require to fully account for the heterogeneity between HIV/AIDS-sick versus non-sick parents and then accounting the possible different options available to, or behaviours of, children. This would imply at least the building of a heterogenous-agents OLG settings, with heterogenous utility functions in turn implying to also consider the effects of the peculiar socio-cultural SSA settings, such as for example the presence or not of extended households, which can offer care to orphans.

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

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Endnotes

[i] On the demographic side, it should be recalled the work of Schoumaker (2009) pointing out that part of the fertility stall observed in SSA since the 2000 might partly be spurious due to data quality issues. Though clearly this is an important problem to be tackled in the future, we believe that the policy prescriptions of the present work are at all unaffected.

[ii] We chose to define the public expenditure against HIV on a per worker basis rather than in per young terms (in line with the definitions of the other main variables used later in this work) for reasons of analytical tractability.

[iii] We are aware that the assumption of perfect annuities does not perfectly describe the behaviour of intergenerational transmission of inheritance under uncertain survival in contexts such as SSA countries, as it is clearly well suited for settings where financial markets are adequately developed. However, the use of the alternative hypothesis of accidental bequests does not allow to characterise the behaviour of individual fertility and thus the dynamics of the physical to human capital ratio and the rate of HIV prevalence.

[iv] The first order conditions define the optimal solution of the problem if and only if $\beta_t \delta\gamma < 1$ (this condition is assumed to hold throughout the work). As β_t beta depends on the prevalence rate of HIV, which in turn may vary depending on internal and external resources aimed at fighting the disease, in our analysis we will use the most restricting assumption $\delta\gamma < 1$.

[v] The issue of contraception, which has been the object of a recent interest in development studies about the fertility transition in industrialised countries (Bhattacharya and Chakraborty, 2017; Prettner and Strulik, 2017) is of interest in relation to the interplay between HIV/AIDS and the fertility transition in SSA. Indeed, SSA is also characterised as the only region in the world where low levels of contraception are persisting (Greenwood et al., 2013) thus representing a main factor of the permanence of high fertility (Cleland, 2009). Therefore, the awareness of the risk of HIV might have an important by product in terms of fertility control.