A contribution to the theory of economic development and the demographic transition: fertility reversal under the HIV epidemic

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Abstract   According to the conventional theory of the demographic transition, mortality decline has represented the major trigger for fertility decline and eventually sustained economic development. In Sub-Saharan Africa (SSA), the HIV/AIDS epidemic has had a devastating impact on mortality, by dramatically reversing (in high HIV-prevalence countries) the long-term positive trend in life expectancies. Despite SSA is suffering a delayed and slowed fertility transition compared to other world's regions and despite the existence of a robust empirical evidence showing the potential for a fertility reversal in countries with severe HIV epidemics, there seems to be a little concern amongst international organisations about the ultimate impact that HIV might have on SSA fertility. This work builds on a Unified Growth Theory model supporting the existing evidence of a HIV-triggered fertility reversal in SSA countries. The model predicts the reversal because of the fall in education and human capital investments due to the drop in life expectancy for young adults. This mechanism eventually breaks down the virtuous circle promoting the switch quantity-to-quality of children. Results suggest that the current evidence on the stall in fertility decline and the declining education in high HIV-prevalent SSA countries should be seriously taken into consideration to prioritise international interventions.

Keywords   Sub-Saharan Africa, fertility transition, quantity-quality switch, HIV/AIDS epidemics, human capital accumulation, fertility reversal

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1. Introduction

The current magnitude and shape of human populations worldwide have been the outcome of that major process of mortality and fertility decline known as the *demographic transition* (DT). The DT debuted in Europe after 1750 [Chesnais (1987); Livi-Bacci (2017)] by a phase of mortality decline which interrupted the long-term stagnation of western populations around their *Malthusian* regime, characterised by high levels of mortality and fertility. Mortality decline, which was responsible for a rapid population growth, was later followed (after 1850) by fertility decline, landing (around 1930) on a *modern* stationary regime at low levels of fertility and mortality [Chesnais (1987); Bulatao and Casterline (2001); Livi-Bacci (2017)]. The other world's regions followed later along their own paths [Bulatao and Casterline (2001); Bongaarts and Casterline (2012)]. Modern demo-economic theories of the DT emphasise its interplay with the industrial revolution and the related endogenous nature of mortality decline. The latter triggered fertility decline by promoting investments in education, thus favouring the switch in children's demand from "quantity" (typical of Malthusian regime) to "quality" (typical of the modern regime) [Galor (2011) and references therein] and eventually resulting a main engine of sustained economic growth and economic development.

In Sub-Saharan Africa (SSA), which is the less-developed world region also as a consequence of the highest burden from infectious diseases [Bloom and Canning (2004); IHME (2013)], mortality decline was halted in 1980s by the HIV/AIDS epidemic (Fig. 1a). In high-prevalence settings, HIV was able to reverse [UN (2015)] the long-term positive trend in life expectancies (Fig. 1b). The memory of HIV on mortality might persist for long time (Fig. 1b). For example, in Lesotho, the life expectancy at age 15 (e_{15}), which was estimated to fall from 53 to 35 yr during 1990-2010, is projected by the UN medium variant to return to its pre-HIV level only by 2060 (Fig. 1b).

In addition, the fertility transition in SSA, which was markedly delayed and slower compared to other world regions started from similar initial conditions, such as Asia and the Latin America [Bongaarts and Casterline (2012)], with several countries still showing total fertility rates (TFR) about six [UN (2015)], is now experiencing symptoms of stalling or even relapsing [Bongaarts and Casterline (2012); UN (2015)].

Worryingly, most of the countries showing the clearest symptoms of a stall in their fertility decline are amongst those with the higher prevalence of HIV (Fig. 1c). Notably, many such countries departed at the onset of the epidemic (1980) from higher income and lower fertility conditions. On the one hand, evidence of stalling or relapsing fertility at still high TFR levels is clear for Botswana, Zimbabwe, Lesotho and Namibia. In addition, in South Africa the TFR – that was fast declining long before 1990 – sharply slowed down thereafter when HIV prevalence became substantial. Currently, Swaziland is the only country with high prevalence of HIV not showing evidence of stalling fertility. On the other hand, in countries suffering intermediate HIV epidemics – most of which were characterised by higher fertility at the onset ofHIV– fertility decline slowed down during 1995-2005 (e.g., Tanzania, Malawi, Zambia, Mozambique and Kenya) but accelerated thereafter.

Obviously, the reported association between HIV and fertility does not necessarily imply a causal relationship. Nonetheless, the possibility that the injury caused by massive HIV epidemics might compromise major societal processes, such as the fertility decline, should be considered so scaring for the development of Africa to deserve careful consideration. Instead, this possibility is acknowledged neither by observers who pointed out the anomalous pattern of fertility decline in SSA [Bongaarts and Casterline (2012)], nor by the last UN population projections [UN (2015)]. Notably, while UN projections included AIDS mortality, possible feedbacks on fertility were deliberately ruled out: "The fertility projections for sub-Saharan Africa follow the general path from high to low fertility observed in other regions." [UN (2015), p. 19]. However, the resulting fertility scenarios appear inconsistent both in relation with previously observed trends and also on conceptual grounds, due to the lack of dynamic plausibility. This is well shown by the case of

Zimbabwe (Fig. 1d), which seems to postulate a Deus ex Machina suddenly restoring the right fertility pathway at 2020 while the HIV epidemic is still fully on.

This work stems from the recent empirical evidence on the potential for a HIV-triggered fertility reversal in SSA countries [Kalemli-Ozcan (2012); Kalemli-Ozcan and Turan (2011); Juhn et al. (2013); Chin and Wilson (2017)] to develop a theoretical framework bringing macroeconomic dynamic results supporting the "reversal hypothesis". In our framework, the reversal appears as a robust phenomenon arising from the fall in education and human capital investments following the drop in life expectancy *for young adults*. The underlying mechanism is at odds with the one universally acknowledged to be responsible for the (forward) fertility transition, namely the drop in *child mortality*. Moreover, although the reversal can eventually be mitigated by effective intervention programmes against HIV, such interventions cannot prevent fertility to remain high for at least several decades by compromising development perspectives in the region.

The rest of the article proceeds as follows. Section 2 reviews the macro-economic literature highlighting the interplay between HIV/AIDS and fertility. Section 3 builds on the macro-dynamic model and discusses the parametrisation used in the numerical simulations. Section 4 reports the main results. Section 5 outlines the conclusions.
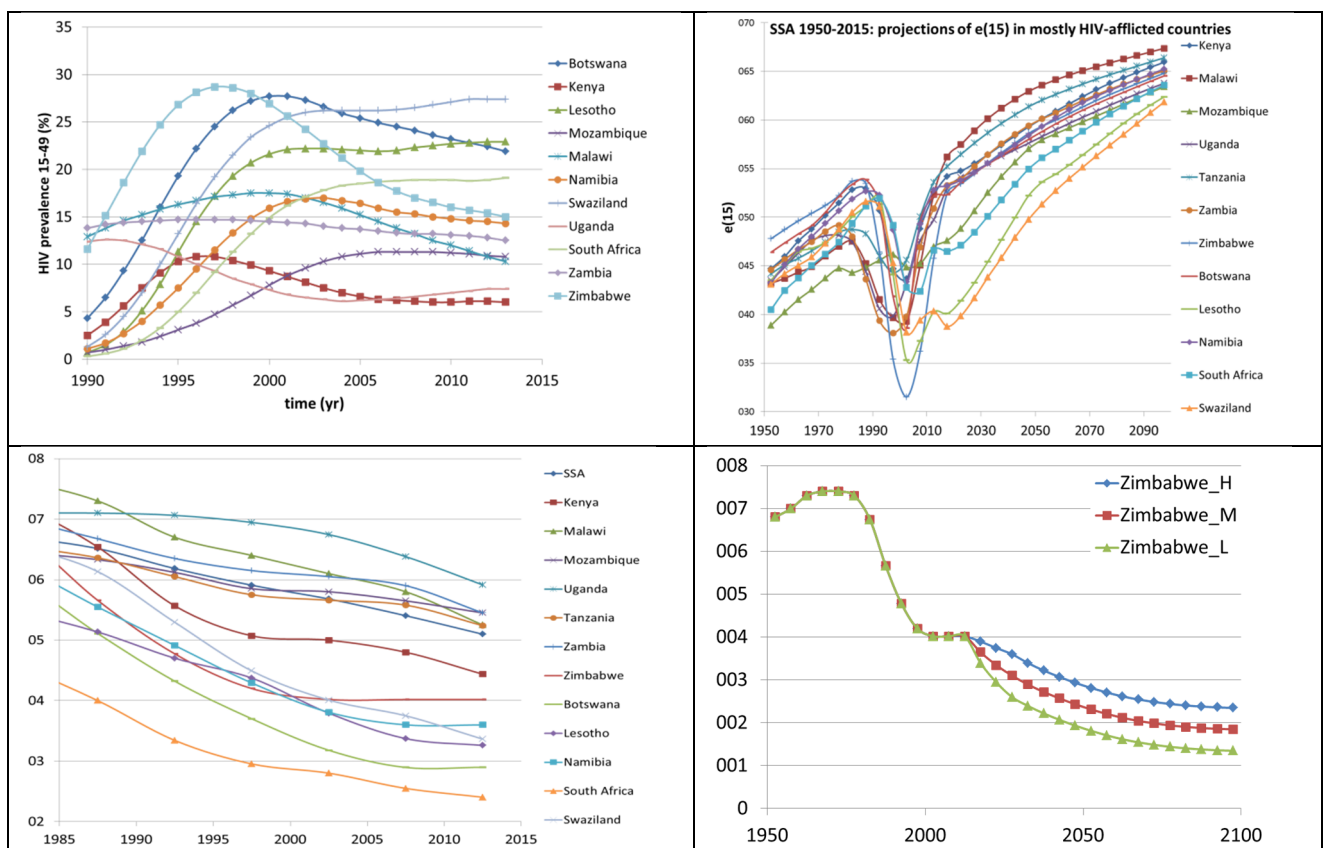

Fig. 1. Demographic trends in SSA countries with higher HIV prevalence: (a) Estimated trend of HIV prevalence in 15-49 yr old individuals [source: (UNAIDS) 2017]; (b) estimated and projected future evolution of mortality of young adults, as measured by e_{15}, the life expectancy at 15 years [source: UN (2015)]; (c) estimated trend 1980-2015 in the total fertility rate (TFR), [source: UN (2015)]; (d) the case of Zimbabwe: projected future evolution of TFR according to UN projection variants (M="medium", L="low", and H="high") [source: UN (2015)].

2. The economic literature on the impact of HIV/AIDS on fertility and development in SSA

Given the critical role (pointed out in the introduction) of fertility decline as an engine of sustained economic development, a major conundrum regards the ultimate impact of HIV on the pace and extent of the fertility transition. Two major opposite positions have emerged on this issue in the

economic literature. At the one extreme, by focusing on South Africa [Young (2005)] reaches the conclusion that AIDS, once brought under control, will allow future generations to enjoy higher welfare than current ones. This scenario, remindful of the "world of opportunities after the Black death" allowing an epoch of sustained growth in 15th century Europe, would eventually prevail because of the increase in the capital- and output-labour ratios due to the direct and indirect effects of AIDS mortality on the labour supply. This conclusion, based on a model à la Becker with quantity-quality trade-off and fixed saving rate as in Solow (1956), holds both in the medium term, due to the mortality of young individuals, and in the longer term, due to the fertility decline allowed by the increased female labour market participation [Young (2005, 2007)]. These effects will dominate the main negative one of HIV i.e., the disruption of human capital [Young (2005)]. In particular, [Young (2005), p. 460] concluded that: "The AIDS epidemic is a humanitarian disaster of millennial proportions, one that cries for assistance. It is not, however, an economic disaster."

At the other extreme, based on World Bank and Demographic Health Surveys (DHS) data, [Kalemli-Ozcan (2012)] showed a positive (resp. negative) correlation between HIV prevalence and fertility (resp. school enrolment) and highlighted the risk that HIV might reverse the fertility transition in SSA. This stems from the upward pressure that the upturn in mortality due to AIDS has on the precautionary demand for children (i.e., the preference for "quantity"), and to the downward pressure on the demand for their education. Further results on South Africa showing no effects of HIV on fertility behaviour were used by [Kalemli-Ozcan and Turan (2011)] in [Young's (2005)] framework, finding that the future generations of South Africa are actually worse off. This scenario was further supported by the empirical analysis (still based on DHS data) on all over SSA carried out by [Juhn et al. (2013)] as well as by the new estimates reported in [Chin and Wilson (2017)] providing further evidence that HIV has increased the TFR and the number of surviving children in SSA countries. This led to the conclusion that: "Together with the results from other papers that document substantial declines in human capital accumulation, the results here suggest that HIV/AIDS is likely to decrease rather than increase future per capita incomes in Africa." [Juhn et al. (2013), p. 851]. This view was also supported by [Bell and Gersbach (2013)], who emphasised the possibility of an "economic collapse" (p. 2083) following the HIV-induced disruption of human capital. All these results are in sharp contrast to [Young (2005, 2007)].

In between these two extreme positions, there is a number of further demo-economic contributions [Corrigan et al. (2005); Boucekkine et al. (2009); Boucekkine and Laffargue (2010); Chakraborty et al. (2010, 2016); Boucekkine (2012); Azomahou et al. (2016)]. In particular, Corrigan et al. (2005) studied an OLG growth model where the large number of orphans caused by the HIV/AIDS epidemics negatively affects the accumulation of both human and physical capital. Boucekkine et al. (2009), using an OLG economy where HIV/AIDS acts as an exogenous shock, provided empirical evidence that the epidemic ambiguously affects net fertility in SSA because adult HIV negatively affects fertility, whereas the opposite holds for child HIV. Nevertheless, the only effort explicitly accounting for a dynamics of HIV/AIDS infection in a macroeconomic set up are [Chakraborty et al. (2010, 2016)]. They innovated on the side of the interplay between infectious disease and economic variables but did not model the endogenous feedback between HIV/AIDS and demographic variables. More generally, none of the above mentioned works considered an economy where an infectious disease with high long-term mortality, such as HIV/AIDS, affects individual decisions on fertility (directly) and mortality (indirectly through education investments).

This work aims at contributing to this debate by a novel model integrating the temporal spread of HIV following the mechanism proposed by [Chakraborty et al. (2010, 2016)] into a general equilibrium model relying on the Unified Growth Theory (UGT) [Galor and Weil (1996, 2000); Galor (2011)], which represents the economic theory of the Demographic Transition. The model aims to represent a conceptual framework for the empirical evidence about the fertility reversal hypothesis cited above.

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3. The macroeconomic dynamic framework

3.1. The model: key ideas

The building block of our model is represented by the assumption of endogenous fertility and endogenous child and adult survival probabilities in the absence of HIV. These probabilities are taken as appropriately parametrised increasing functions of human capital in order to reproduce, in the absence of HIV, a regular DT pattern triggered by the interplay amongst increasing survival, investments in education and the level of income, aiming to reflect the pre-AIDS setting in SSA countries, where the DT was ongoing (although delayed) prior to the HIV onset. In the presence of HIV, these survival probabilities are assumed to scale with infection prevalence to mirror the disruption of human capital caused by AIDS mortality on the assumption that all HIV-infected die by AIDS. The major news of the present work is that while in the absence of HIV the fertility transition is triggered especially by the decline in *child mortality* (exactly as in standard UGT models), the fertility reversal occurs mainly because of the HIV-induced upsurge in *adult mortality*, which contributes to reduce resources for education [Kalemli-Ozcan (2012)] eventually eroding the growth of human capital.

3.2. The model: equations

We consider an OLG closed economy accounting for fertility, mortality, education and human capital accumulation along the UGT lines. The economy is populated by a continuum of rational and identical individuals of size N_t (at birth) per generation, where time is discrete and indexed by $t = 0,1,2,....$ The length of each period is conventionally set at 20 years. An individual lives for three periods: *childhood/adolescence*, *young adulthood* (or adulthood, which represents the working period) and *old age*. A new born of generation t may either die early (before parents spend time for his education), with probability $1-\Gamma_{t+1}$, or he may survive, with probability $\Gamma_{t+1} \in [0,1]$, thus receiving education (e_{t+1}) according to parents' decisions. As an adolescent, he does not make economic decisions and does not work, but he can become sexually active and acquire HIV infection. If he survives at the onset of childhood/adolescence, he becomes economically active at the beginning of adulthood (time $t+1$). In particular, Γ_{t+1} is assumed to depend positively on the level of the human capital of parents h_{t+1} and negatively on the proportion i_{t+1} of parents who are HIV infective (which represents the rate of *HIV prevalence*), i.e. $\Gamma_{t+1} = \Gamma(h_{t+1}, i_{t+1})$.

An adult maximises his expected lifetime utility and may acquire HIV infection. He has a probability $\Pi_{t+1} = \Pi(h_{t+1}, i_{t+1})$ to survive up to the onset of old age. As for the case of child survival, adult survival positively depends on the endowment of human capital (h_{t+1}) and negatively on HIV prevalence. This endowment is inelastically supplied to firms in exchange for wage income w_{t+1} per unit of labour. The (expected) lifetime utility function captures the individual preferences towards consumption (c_{t+1}) and the number of surviving children (n_{t+1}) during young adulthood as well as the number and quality of children during old age.

3.3. Disease transmission

We represent HIV spread by following [Chakraborty et al. (2010, 2016)], who first proposed a parsimonious approach to represent the diffusion of fatal infectious diseases as persistent phenomena characterised by a specific temporal profile induced by the patterns of transmission between individuals rather than a mere mortality shock.

Let p_t be the probability that an HIV-susceptible young adult acquires HIV infection. By following [Chakraborty et al. (2016)], this can be represented as a function of HIV prevalence amongst individuals of generation t, that is

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

where $0 < \lambda \le 1$ is the constant probability of being infected 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 $t+1$ amongst young adults converges to the probability of a young adult to be HIV-infected, i.e. $i_{t+1} = p_t$. Therefore,

$$i_{t+1} = 1 - (1 - \lambda i_t)^\mu. \tag{2}$$

Unlike [Chakraborty et al. (2016)], we did not link HIV spread to agents' rational behaviour for two main reasons related to the characteristics of HIV and to the socio-economic context prevailing in SSA during a greatest part of the HIV epidemic. The first one is the fact that essentially the entire HIV incubation period is asymptomatic i.e., individuals remain in a good health state during the entire period in which they are able to retransmit the infection to others. The second one has been the almost total lack of effectiveness of past public policies aimed to contrasting HIV diffusion by raising awareness of the epidemic in lowest resource settings. As for SSA there are a very few exceptions, such as the Uganda "success story" [Green et al (2006)], which is however contradicted by some more recent works such as [Shafer et al (2008)], or the recent decline in HIV prevalence in Zimbabwe attributed to a success of awareness and prevention [Halperin et al. (2013)]. As a consequence, from a modelling point of view it appears unreasonable assuming that agents from SSA countries can rationally choose their own private health investments for HIV prevention, given their utility function and budget constraint. The ability to affecting such a mechanism is the main aspect to be considered for public policies (i.e., health investments provided by international organizations or private foundations, as the well-known Bill and Melinda Gates foundation) aiming at reducing the impact of the epidemics on the economic system. This is actually considered later in Section 3.5, where we will account for the effects of public policies still fully *exogenous* in view of the reasons discussed above and other motivated later.

3.4. Preferences and solutions
By normalizing the utility from death to zero, preferences of the representative individual that is economically active at time $t+1$ are captured by the following inter-temporal (expected) utility function whose formulation accords with the UGT tradition, that is:

$$U_{t+1} = \ln(c_{t+1}) + \rho \ln(n_{t+1}) + z \Pi_{t+1} \ln(n_{t+1} h_{t+2}), \tag{3}$$

where $\rho > 0$ captures the parent's relative taste for children and $z > 0$ is a scaling parameter tuning the relative degree of altruism. This allows receiving utility (with certainty) from material consumption and the quantity of children when young, and from both quantity and quality of children when old, which holds with probability Π_{t+1}. The quality of children is represented by their own level of human capital (education). Weighting the flow of utility when old with the adult survival probability is an essential ingredient of the model, which allows to capture the effects of premature death agents due to HIV/AIDS preventing any utility flow from their educated children.

The relationship between the total number of born children and the number of surviving ones at every time $t \ge 0$ is given by:

$$n_t = \Gamma_t n_t^g. \tag{4}$$

A t-generation individual choice is made at time $t+1$ subject to the budget constraint:

$$c_{t+1} = w_{t+1} \left(m - \psi \frac{n_{t+1}}{\Gamma_{t+1}} - \phi_{t+1} n_{t+1} - e_{t+1} n_{t+1} \right), \tag{5}$$

where m is his time endowment. Eq. (5) implies that consumption is constrained by the amount of resources available after accounting for the portions of the time endowment for giving birth to n_{t+1}^g children ($\psi \in (0, m)$), and raising ($\phi_{t+1} \in (0, m)$) and educating (e_{t+1}) those who survive (n_{t+1}).

The human capital of each child (h_{t+2}) depends on the human capital of parents (h_{t+1}) and their time expenditure in education (e_{t+1}), according to the following evolution equation:

$$h_{t+2} = q(x + e_{t+1}) h_{t+1}^\alpha, \tag{6}$$

where $q > 0$ and $\alpha > 0$. Given that $x + e_{t+1} > 0$, it follows $h_{t+1} \in (0, +\infty)$ for every $t \geq 0$ as $h_1 > 0$ (the initial condition) must hold. The term $x > 0$ guarantees that children's human capital is positive even if parents do not invest in education.

The maximisation of the utility function with respect to material consumption, the number of children and the time spent in education, subject to the budget constraint, the rule for the accumulation of human capital and the condition $c_{t+1} > 0$, gives the solution to the optimisation programme. This solution may be either interior ($e_{t+1} > 0$) or on the corner ($e_{t+1} = 0$). The interior solution is:

$$e_{t+1} = \frac{z \Pi_{t+1}[\Gamma_{t+1}(\phi_{t+1} - x) + \psi]}{\rho \Gamma_{t+1}} - x, \tag{7}$$

$$n_{t+1} = \frac{m \rho \Gamma_{t+1}}{[\Gamma_{t+1}(\phi_{t+1} - x) + \psi](1 + \rho + z \Pi_{t+1})}, \tag{8}$$

$$c_{t+1} = \frac{m w_{t+1}}{1 + \rho + z \Pi_{t+1}}. \tag{9}$$

The condition $\Gamma_{t+1}(\phi_{t+1} - x) + \psi > 0$ must hold. In addition, the condition that guarantees the existence of an interior solution is $\dfrac{z \Pi_{t+1}[\Gamma_{t+1}(\phi_{t+1} - x) + \psi]}{\rho \Gamma_{t+1}} > x$. Then, if $\dfrac{z \Pi_{t+1}[\Gamma_{t+1}(\phi_{t+1} - x) + \psi]}{\rho \Gamma_{t+1}} \leq x$, we get the corner solution, which is given by:

$$e_{t+1} = 0, \tag{10}$$

$$n_{t+1} = \frac{m \Gamma_{t+1}(\rho + z \Pi_{t+1})}{(\phi_{t+1} \Gamma_{t+1} + \psi)(1 + \rho + z \Pi_{t+1})}, \tag{11}$$

$$c_{t+1} = \frac{m w_{t+1}}{1 + \rho + z \Pi_{t+1}}. \tag{12}$$

The interior and corner solutions are in line with [Galor and Weil (2000)] and [de la Croix and Doepke (2003, 2004)]. It is important to emphasise the dependence of the optimal values of education and fertility on the adult survival probability. An increase in this probability reduces fertility and raises the time investment in education per child, as is shown by previous equations. The reason for the switch in the allocation of resources from quantity to quality of children depends on the higher probability to receiving a utility inflow in old age. The main effect for the next period output is a higher level of human capital due to a larger investment in education when individuals live longer. Beside the classical dependence of education and fertility on child survival, which is the cornerstone of the UGT allowing to capture the main stylised facts of the DT, our equations also show a dependency of education and fertility on adult survival. This actually reflects the main

mechanism of our model, that is the feedback of AIDS-related mortality of adults on education and eventually on the dynamics of the system.

The production of final output (Y_t) occurs under perfect competition according to the technology $Y_{t+1} = H_{t+1} + \theta L_{t+1}$, where $H_{t+1} = h_{t+1}L_{t+1}$ is the aggregate stock of human capital, L_{t+1} is the labour input and $\theta \geq 0$ is a parameter capturing the worker's productivity. By normalising to 1 the price of Y_{t+1}, profit maximisation implies that labour is paid according to its marginal product, that is $w_{t+1} = \theta + h_{t+1}$. Compared to [Chakraborty et al. (2010, 2016)], who considered a model with physical capital, we deliberately did not include any negative feedback of HIV on labour productivity not only because HIV-infected individual are asymptomatic and in good health until they develop full-blown AIDS (as stated before), but especially because in this model the wage only affects material consumption and it has no effects on education and fertility as well as on the dynamics of the system.

By using the interior or corner solution together with the equation describing human capital accumulation and the expression of the wage rate, we obtain the following two-dimensional map describing the evolution of human capital and HIV prevalence over two subsequent periods, which completely characterise the dynamics of the economy:

$$h_{t+1} = \begin{cases} qxh_t^\alpha & if \quad e_t = 0 \\ \dfrac{qz\Pi_t[(\phi_t - x)\Gamma_t + \psi]}{\rho\Gamma_t} h_t^\alpha & if \quad e_t > 0 \end{cases}. \tag{13}$$

$$i_{t+1} = 1 - (1 - \lambda i_t)^\mu$$

3.5. Intervention programmes

Intervention programmes against HIV are modelled in the simplest way, i.e. by a fully exogenous reduction in the transmission probability per partnership λ (it would be equivalent to intervene on the number of sexual partners μ). This is motivated by the known fact that the large majority (currently, more than 80%) of HIV therapies in SSA are paid for through external financial donations [UNAIDS 2017]), mostly from international organisations and charities, rather than endogenously managed by the government of the afflicted country. The chosen approach can capture a range of different medical, e.g. anti-retroviral treatments having the potential to reduce individual infectivity as well as non-medical interventions, e.g. aimed to increase awareness of HIV risk and favour spontaneous behaviour changes, such as reducing the number of sexual partners and increasing the use of condoms. The intervention is introduced at time $t = T$ as follows:

$$\lambda_t = \begin{cases} \lambda & if \quad t < T \\ \lambda + (\lambda_t - \lambda)\dfrac{f(t-T)}{1 + f(t-T)} & if \quad t \geq T \end{cases}. \tag{14}$$

Where $f > 0$ is a parameter capturing the effectiveness of the policy over time, and λ_t summarises the strength of the policy in terms of reduction in the transmission probability.

3.6. Model parametrisation

3.6.1. Functional forms for child and adult survival

Consistently with Section 3.2, we model the child survival probability Γ_{t+1} as follows:

$$\Gamma_{t+1} = \xi_{t+1}(1 - \delta i_{t+1}), \tag{15}$$

where ξ_{t+1} represents the child survival probability in the absence of HIV and $0 < \delta \leq 1$ is the conditional probability that a child of HIV-infected parents is vertically transmitted at birth, so that δi_{t+1} is the probability for a child to be born infected (and of dying soon after). We adopt the following functional form for ξ_{t+1}:

$$\xi_{t+1} = \Gamma_{pre} + (\Gamma_{post} - \Gamma_{pre})\frac{b\ln(1+h_{t+1})}{1+b\ln(1+h_{t+1})}, \tag{16}$$

where $\Gamma_{pre}, \Gamma_{post} \in [0,1]$ are, respectively, the child survival at birth in the Malthusian regime prevailing before mortality decline and the child survival at birth in the modern regime at the end of the mortality transition [Galor (2005, 2011)], and $b > 0$ is a parameter which governs the shape of the mortality transition pattern along the growth of human capital. The functional form (16) depicts a pattern of increasing child survival during the mortality transition, departing from its Malthusian level Γ_{pre} and eventually ending at its modern level Γ_{post} by the end of the mortality transition. The temporal pattern (16) of the endogenous mortality transition is modelled through a logarithmic function of human capital, but many other functions could play the same role.

Similarly, the probability Π_{t+1} to enter old age conditioned on being entered the adult state is modelled as follows:

$$\Pi_{t+1} = \left[\Pi_{pre} + (\Pi_{post} - \Pi_{pre})\frac{B\ln(1+h_{t+1})}{1+B\ln(1+h_{t+1})}\right](1 - i_{t+1}), \tag{17}$$

where $\Pi_{pre}, \Pi_{post} \in [0,1]$ and $B > 0$ have the corresponding meaning of Γ_{pre}, Γ_{post} and b in Eq. (16). Finally, the cost of raising children is represented as an increasing function of human capital accumulation as in (16) and (17):

$$\phi_{t+1} = \phi_{pre} + (\phi_{post} - \phi_{pre})\frac{\ln(1+h_{t+1})}{1+\ln(1+h_{t+1})}. \tag{18}$$

3.6.2. Parameter assignment

Summary information about model parameters is reported in Tab. 1. Model parametrisation was carried out in order to achieve the following empirically relevant targets:

(A) reproducing the scale of fertility and mortality transition in SSA. Demographic parameters were assigned from UN data for SSA [UN (2016)] in order to coarsely (i) reproduce the range of decline of TFR during the fertility transition in SSA, departing from a pre-transitional level of about 6 and ending to about 2 (i.e., the replacement level) in line with UN projections medium variant [UN (2016)], and (ii) the pre- and post transitional transitional levels of the survival probabilities Π_{pre}, Π_{post} and $\Gamma_{pre}, \Gamma_{post}$ in (16)-(17);

(B) matching HIV prevalence data. The parameters (μ, λ) were assigned to allow the HIV equation (2) to bound *at equilibrium* (in the absence of any intervention) the range of prevalence levels observed in the adult population in medium-high prevalence SSA countries, according to the last UNAIDS estimates [UNAIDS (2017)]. This was motivated by the fact that in most cases UNAIDS estimates of prevalence showed a constant trend for several years before 2015 (Fig. 1a). Inparticular, we considered the following idealised scenarios in terms of the HIV equilibrium prevalence (EP): "low": EP=10%, "medium": EP=15%, "high": EP=20%, "very high": EP=30%.

(C) keeping in a correct empirical balance the relative time scales of the epidemic and of the fertility transition.

Finally, economic parameters were either borrowed from the literature or assigned for simulative purposes.

Parameter	Description	Value or range	Source
μ	Average number of sexual partnerships of a young adult individual during his entire young adulthood phase	450-500	Assigned to match predicted equilibrium prevalence and UNAIDS estimates.
λ	Transmission probability of HIV per sexual partnership	[0.0022,0.0028]	Assigned to match predicted equilibrium prevalence and UNAIDS estimates.
δ	Probability that a newborn is vertically transmitted from HIV-positive parents.	0-0.25	[Chakraborty et al. (2010, 2016); WHO (2016)]
z	Relative degree of individual's altruism	70	Assigned to match pre-transition level of TFR [UN (2015)]
ρ	Parent's taste for children	2.04	Assigned to match pre-transition level of TFR [UN (2015)]
m	Time endowment of households	5	Simulation
θ	Coefficient capturing natural endowment of rural economy	8	Simulation
ψ	Time cost of bearing a child	0.002	Simulation
ϕ_{pre}	Parameter regulating pre-transition time cost of rearing a child	0.394	[30,31]
ϕ_{post}	Post-transition time cost of rearing a child	0.475	[Haveman and Wolfe (1995); de la Croix and Doepke (2004)]
v	Speed of change of the time cost of rearing	1/8	Simulation
Γ_{pre}	Parameter tuning pre-transition child survival probability	0.1381	[UN (2015)]
Γ_{post}	Post-transition child survival probability	0.9	[UN (2015)]
b	Speed of child mortality transition	1	Simulation
Π_{pre}	Parameter tuning pre-transition adult survival probability	0.5767	[UN (2015)]
Π_{post}	Post-transition adult survival probability	1	[UN (2015)]
B	Speed of adult mortality transition	1	Simulation
α	Relative weight of parental human capital in the production of children's human capital	1	[de la Croix and Doepke (2004)]
q	Coefficient of the human capital technology	0.6	[de la Croix and Doepke (2004)]
x	Level of automatic transmission of human capital	0.34	Simulation
f	Effectiveness of the policy over time	0.01	Simulation
λ_t	Strength of the policy in reducing the transmission probability	0.0001	Simulation

Tab. 1. Summary information about model parameters; t denotes time in OLG units i.e., it represents the length of an OLG time period (set to 20 years in the model). Consequently values of Π_{pre}, Π_{post} and $\Gamma_{pre}, \Gamma_{post}$ were assigned to match the corresponding observed probabilities to survive from age 20 to age 40 (for a person alive at age 20), and from birth to age 20, respectively. The large value assigned to μ was based on data from South Africa by multiplying the average number of prostitutes partner of an average sexually active male for the duration of the sexually active period, here set to 20 years.

4. Results

4.1. The case of an uncontrolled HIV epidemics

In the absence of HIV, the model predicts the onset and gradual completion of the fertility transition as an endogenous response to mortality reduction through the increase in education investments, with the TFR declining from about six to replacement level (Fig. 2, left axes).

Following the scenarios hypothesised in Point B, Section 3.6.2, the HIV epidemics (Eq. (2)) takes off and gradually increases up to its equilibrium prevalence where it persists thereafter. Fig. 2 mirrors the observed variability in the stage of the fertility transition of SSA countries at the onset of large HIV epidemics, by contrasting the case where HIV took-off at an advanced stage of the fertility transition (left panel), as has been the case of South Africa, with the case where HIV onset occurred at an earlier stage (right panel).
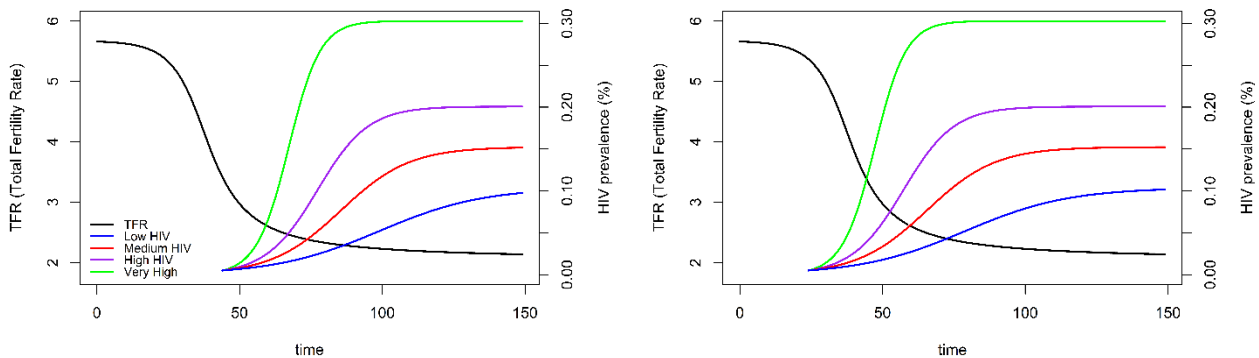
Fig. 2. The hypothesised trajectories of SSA fertility in the absence of HIV (left axis) and of HIV prevalence (right axis) under uncontrolled HIV epidemics of different intensity. Left panel: the onset of HIV occurs at an advanced stage of the fertility transition. Right panel: the onset of HIV occurs at an earlier stage of the fertility transition.

The impact predicted by map (13) of a persistent uncontrolled HIV epidemic on the fertility transition in SSA would be dramatic (Fig. 3), with fertility landing on levels well above the replacement threshold. For high levels of the EP, the TFR eventually stalls on levels in excess of three. Moreover, medium-high HIV epidemics have the potential to cause a sharp reversal in the fertility transition pathway. Two main qualitative scenarios emerge depending on whether the onset of HIV occurred either at an advanced or at an earlier stage of the fertility transition as in Fig. 2. In the former case, at the onset of HIV the TFR has already experienced most of its decline. In this case, the reversal occurs by a marked increase in TFR that remains essentially constant thereafter. Instead, in the latter case the TFR shows a temporary relapse, after which it resets on a declining path. This is because the fertility decline triggered by mortality progress from causes of deaths different from HIV was just initiated, so that there still remains large room for further fertility decline.

Notably, the HIV-induced fertility relapse is not a full reversal in the clock of history with fertility returning to its pre-transitional level. The range of the fertility relapse is indeed strictly bounded by the relapse in the mortality of adults, which is only one of the two components of the fertility transition, the other one being the decline in child mortality, which is only minorly affected (at the aggregate level) by HIV.

The causation chain (illustrated in Fig. 4 for the scenario of late onset of HIV) goes as follows: (a) HIV breaks-down the long-term declining trend in adult mortality, which suddenly upturns (Fig. 4, left panel); the upturn in adult mortality reverses the positive trend on education investments (Fig. 4, central panel); (c) the collapse in education investment sets the growth rate of human capital at lower levels, which are insufficient to further fuel the quantity-quality switch, and therefore to further promote fertility decline (Fig. 4, right panel).
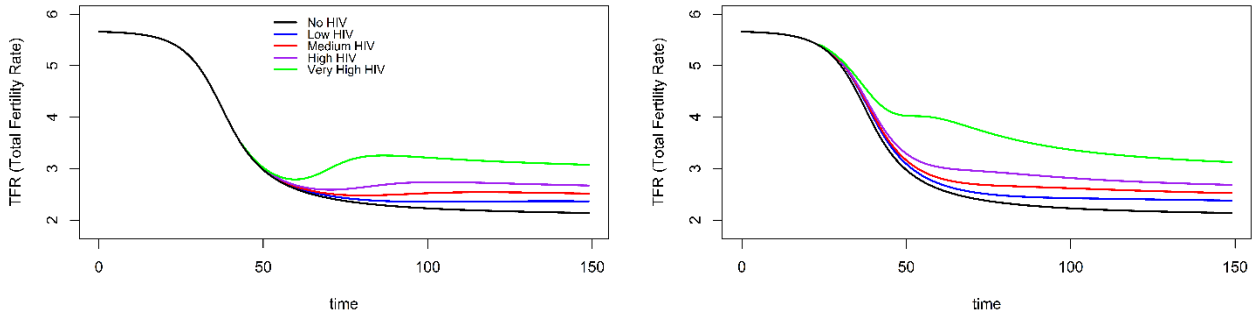
Fig. 3. Potential impact of uncontrolled HIV epidemics on the fertility transition in SSA for different levels of HIV endemic equilibrium prevalence (EP). Left panel: the HIV onset occurs at an advanced stage of the transition. Right panel: the HIV onset occurs at an earlier stage of the transition.
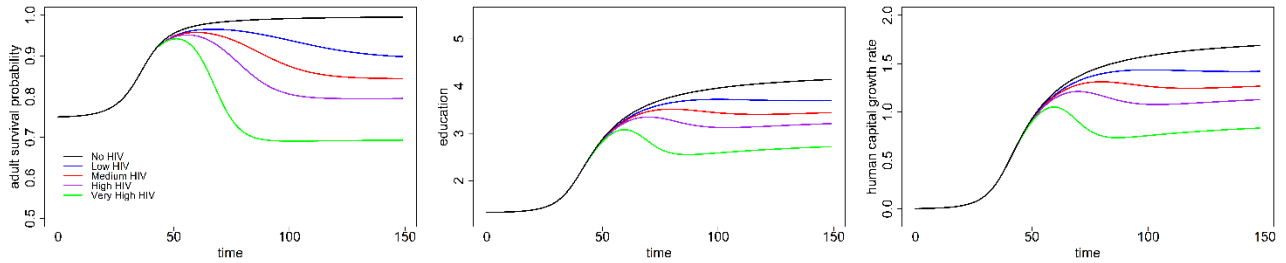

Fig. 4. Fertility reversal in SSA under an uncontrolled HIV epidemics. Temporal trends of: adult survival probability Π_{t+1} (left panel), education e_{t+1} (central panel), growth rate of human capital h_{t+1} (right panel).

4.2. The effects of interventions

The previous analysis described a useful theoretical benchmark against which grounding the possible effects on fertility as predicted by our model of the current realistic scenarios of HIV control [WHO (2013)]. We now report the implications of exogenous interventions as is explained in Section 3.5. For the sake of simplicity, we only consider interventions able to eventually eliminate HIV, and compare the effects of different timings, constrasting an *early* intervention, initiating shortly before the epidemics has reached its equilibrium prevalence, with a number of delayed interventions (Fig. 5). In particular, we hypothesise that the time span needed to bringing HIV to elimination from the intervention onset is coarsely comparable with the time span that the HIV epidemics took to reach its peak (Fig. 5, left panel), which in SSA lasted approximately 2-3 decades [UNAIDS (2017)]. This is motivated by the fact that in SSA a large proportion of currently seropositive individuals is still unaware of his health status and might therefore remain infective for decades in absence of large scale screening, while, on the other hand, effective anti-retroviral therapies will extend the duration of the sero-positive period for treated individuals [WHO (2016)].

For brevity we report predictions of controlled HIV scenarios (Fig. 5) just focusing on the case of HIV onset occurring in an advanced stage of the fertility transition. Bringing HIV under full control (left panel) will remove obstacles to investments in education, so that the TFR will eventually land to its replacement level (right panel). Nonetheless, even under the early interventions scenario, the time span needed for bringing HIV to elimination, has the potential to delay the completion of the fertility transition. Obviously, this scenario can substantially worsen in the presence of delays in the effectiveness of interventions (right panel).
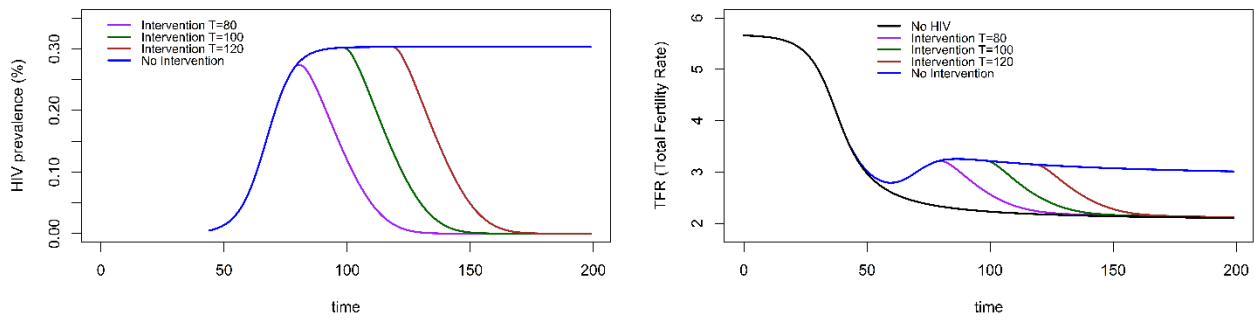
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Fig. 5. The case of HIV onset at an advanced stage of the fertility transition. Temporal trend of HIV prevalence (left panel) and total fertility (right panel) under different effective intervention programmes able to eventually achieve HIV elimination. The intervention programmes differ in the timing of initiation (t=80,100,120).

4.3. Implications of the results and further discussions

Despite its poorest health conditions, with the highest impact of communicable diseases worldwide ([Bloom and Canning (2004); WHO (2016)]), SSA experienced continued – though slow – mortality decline until the onset of HIV/AIDS. In high-prevalence countries, HIV has become the leading cause of mortality [IHME (2013)], able to reverting the increasing trend in life expectancy. However, so far the ultimate effects of HIV on fertility and economic development in SSA are more controversial, as discussed in Section 2. This work has built on a parsimonious novel macroeconomic framework including HIV spread, endogenous child and adult mortality (the latter factor not previously considered), endogenous fertility and private education (i) to reassess the ultimate impact of HIV on fertility in SSA countries in its natural modelling context, namely Galor's UGT of economic determinants of fertility transitions [Galor (2011)]; (ii) to serve as a theoretical framework for the empirical evidence on the HIV-induced fertility reversal in SSA ([Kalemli-Ozcan (2012)]; [Kalemli-Ozcan and Turan (2011)], [Juhn et al. (2013)]; [Chin and Wilson (2017)]), (iii) to prioritise future economic intervention programmes against HIV based on a more general societal perspective.

The adopted parametrisation is robust on the timing of the interplay between HIV and the fertility transition as it keeps in a correct empirical balance the relative time scales of the epidemic and of the fertility transition. In the absence of HIV, the model predicts the completion of the fertility transition as an endogenous response to the decline in both young and adult mortality, which in turn promotes investments in education and eventually fertility decline via the quantity-quality switch, as is in the standard UGT models.

In the theoretical case of a large uncontrolled HIV epidemic (that is, yielding an endemic prevalence above 15-20%), HIV has the potential to fully prevent the completion of the fertility transition. In particular, reversals in the fertility trajectory are more likely to occur in countries where the onset of HIV occurred at a later stage of the fertility transition. However, the ultimate level of fertility i.e., how far the TFR will eventually land from the replacement level, only depends on the intensity of the HIV epidemic and not on the stage of the transition where the epidemics actually debuted. The critical obstacle to the completion of the fertility transition is the AIDS-related increase in adult mortality, which reduces the parents' educational investment on children and therefore prevents the quantity-quality switch.

Hopefully, the increasing adoption and effectiveness of interventions against HIV/AIDS worldwide [WHO (2016); UNAIDS (2017)] should make an uncontrolled HIV epidemic only a theoretical worse case. Although the achievement of HIV full control would remove the obstacles to the ultimate completion of the fertility transition, our results however show that the timing with which HIV control measures will be enacted becomes critical. Indeed, any delay in controlling HIV would not only allow the direct negative effects of HIV (namely, its large mortality burden) to

persist, but it might also cause fertility to remain persistently high due to the decline in private investments in education. In addition, the persistence of high fertility would promote continued population growth, therefore loosing epochs potentially favourable to economic development.

We believe that the relationships between HIV, education and fertility theoretically inferred here should be carefully considered on a policy standpoint. This is because – on the one hand – the recent empirical evidence han shown a robust negative relationship between HIV prevalence and education [Fortson (2011); Akbulut-Yuksel and Turan (2013)], and – on the other hand – the time required for full HIV control depends on the effectiveness and diffusion of interventions, which in turn depend on the resources allocated for HIV control. Given the large number of seropositive individuals in SSA [UNAIDS (2017); WHO (2016)], most of which unaware of their sero-status, timing and effectiveness of interventions are currently hard to predict for SSA. From this standpoint, there seems to be only one possible policy recommendation for international organisations and policy makers, that is the maximal control effort on HIV should be done right now, without delays, and with priority to those high-prevalence countries where the long-lasting mortality crisis due to HIV, which is projected to require several decades before being reabsorbed, could threaten the fertility transition. Therefore, the first candidate country would le Lesotho, where life expectancy is predicted to return to the pre-HIV level by 2060 and UN fertility data [UN (2016)] show clear symptoms of fertility stalling. This also makes it important emphasising the danger of conservative approaches, as in the last round of the UN projections, where the possible feedbacks of HIV on fertility in SSA were deliberately ruled out [UN (2015)]. This optimistic view does not seem to be acceptable.

The present model must be considered as a departure point whose primary goal was to develop a framework for the impact of HIV of fertility in SSA in its "natural" setup, namely the UGT for endogenous fertility transitions. Consequently, we disregarded other possible channels of influence of HIV on fertility that have been considered in various literatures (epidemiologic, demographic, economic, etc) ranging from e.g., the lower fecundity observed in sero-positive women, up to the long-term general equilibrium effect of higher mortality that increases women's labour participation. These effects are clearly important to consider for detailed quantitative explanations of the phenomenon but they are secondary here.

Further extensions of the present model should include, for instance, saving and the accumulation of physical capital. This was deliberately avoided here to mirror SSA economies, which show the lowest aggregate saving [World Bank (2016)], the highest income inequality [IMF (2015)], and the most under-developed political and financial systems worldwide [Easterly and Levine (1997)]. This would allow to consider also the impact of the HIV/AIDS on labour productivity, as in [Chakraborty (2010, 2016)]. This hypothesis might become relevant under widespread diffusion of highly effective anti-retroviral therapies allowing good health survival of individuals with full-blown AIDS. Another limitation lies in the use of a model with homogeneous agents. Extensions to heterogenous agents settings may actually allow capturing the issue of *HIV orphanhood*, as considered in [Bell and Gersbach (2013)]. Finally, it would be desirable building on continuous-time models to realistically capture the time scales of the interplay between HIV and fertility timescales on the one hand and the economic timescale on the other hand.

5. Conclusions

This article tackled a major open question about economic development, namely the impact of HIV/AIDS on the fertility transition in Sub-Saharan Africa, as is also pointed out by [Lorentzen et al. (2008)], and consistently built on a theoretical framework based on existing empirical evidence about HIV-induced fertility reversal by using a UGT model.

All successful stories of economic development initiated by mortality decline, which in turn triggered a drop in fertility, thus making resources available to be used for investments in education and fuelling a virtuous circle of sustained economic growth and development [Becker et al. (1990);

Fogel (2004); Galor (2005, 2011); Soares (2005); Livi-Bacci (2017)]. In some SSA countries suffering large HIV/AIDS epidemics, life expectancies are projected to return to pre-HIV levels only by 2060 [UN (2015)], fertility decline is stalling [UN (2015)], and there is evidence of negative effects of such a disease on educational investments [Fortson (2011); Kalemli-Ozcan (2012); Akbulut-Yuksel and Turan (2013)]. The present research stemmed from these facts by providing model-based results that even under successful disease control in the long term, HIV might substantially delay the fertility transition in SSA by halting economic development for decades as it compromises the children quantity-quality switch, which has represented the major trigger of economic development all over the world. The results challenged the optimistic view, which seems to be adopted amongst international institutions due to the trust that the increasing degree of epidemic control will rapidly take place. This contribution aimed to shed light in the economic theory of fertility by giving unambiguous predictions on demo-economic outcomes that the HIV epidemic may generate, especially on the welfare of future generations. Unlike [Young (2005)], we believe that the HIV/AIDS epidemic is a humanitarian and economic disaster that should receive the highest priority amongst international institutions and policy makers.

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

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