The Growth Economics of Epidemics

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Abstract

This paper examines in an endogenous growth theory perspective the mechanisms through which epidemics affect long term growth. Investment in both physical and human capital are key transmission variables in this respect. The paper distinguishes between Spanish flu like epidemics and AIDS like epidemics. Two-sector growth models are shown to better reflect the specific effects of epidemics. The effects of an AIDS like pandemic on savings and education effort are also modelled via life expectancy. The paper is closed by an extension of the celebrated Cuddington-Hancock model to account for the latter features. An application to the South African case is provided. The main finding points at a delayed effect of AIDS on economic growth due to a the recent sharp drop in life expectancy in this country.

Keywords: Epidemics, Human capital, Life expectancy, Growth theory, Spanish flu, AIDS

JEL classification: C61, C62, O41.

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1 Introduction

A few days ago, the 2005 December release of UNAIDS crudely highlighted that the devastating effects of AIDS are still not over, with an increase of infected people in 2005 at its highest level since the early eighties. The number of HIV seropositive has exceeded for the first time the symbolic threshold of 40 million, due to the registration of 5 million new cases around the world during this year. While the crisis is desperately acute in sub-Saharan Africa (with two thirds of HIV seropositive and 77% of infected females), it seriously affects all the continents and countries, notably the poorest.
More recently, the threat of an avian flu (or bird flu) worldwide pandemic has put a tremendous pressure on governments and international institution to prevent the occurrence of another major influenza crisis. Indeed, the 1918-19 influenza epidemic killed at least 40 million people worldwide in a few months, far exceeding the combat deaths experienced during the first world war.

The demographic impact of such epidemics being obviously very sharp, heavy economic consequences are unavoidable, at least via the direct (markedly declining) labor resources channel. It is also true that in certain circumstances, the reverse causality holds: a low level of development and low quality health services are likely to boost epidemics. It seems therefore clear that such health crises should interact with the economic sphere, and are likely to distort it in several relevant ways. The recent AIDS pandemic has indeed suggested an increasing number of studies aiming at identifying and quantifying these distortions.
While the operation research and mathematical demography literature has been pretty much concerned with the optimal prevention and treatment of HIV (see for example Feichtinger, Tsachev and Veliov, 2005), the economic literature has been overwhelmingly empirical. Many authors have attempted to econometrically study the relationship between epidemics (notably the Spanish flu and AIDS/HIV) and economic growth. The results are still disputed and disputable: some authors have found no evidence of such a correlation (notably Bloom and Mahal, 1997a and 1997b, for both the Spanish flu and AIDS), while others have found just the contrary (see Brainerd and Siegler, 2003, for the Spanish flu case, and McDonald and Roberts, 2005, for AIDS). It should be added that the most recent papers, a few of them based

on explicit micro-foundations (for example, Corrigan, Glomm and Mendez, 2005) point at an unquestionable negative growth impact of AIDS on sub-Saharan countries. This seems valid as far as a sufficient weight is assigned to long term growth factors like accumulation of human capital and its determinants.

In this paper, we take a much more growth theory oriented paper. What are the predictions of the traditional endogenous growth models as to the growth impact of epidemics? What would be an appropriate growth model to deal with such issues? Of course, there is no unique epidemic profile: the age profile of mortality may be U or W shaped, hitting more children and elderly or more the working ages...etc... In our theoretical analysis, we shall distinguish between Spanish flu-like epidemics, which claim victims in a matter of days, and AIDS-lie epidemics which evolve slowly in time and are typically associated with very long periods of reduced productivity and tough and expensive medication. We examine how endogenous growth models incorporating human capital accumulation respond to these epidemics. Our main objective is to identify the growth theory set-up more adapted to treat this question, and we will show that two-sector models à la Lucas-Uzawa seem to do much better the job than the typical one-sector models. To this end, we use some recent analytical results due to Boucekkine and Ruiz-Tamarit (2004a, 2004b).

Modelling AIDS-like epidemics impacts require a much deeper revisiting of existing models. In order to reflect such impacts in an acceptable way, one has to incorporate at least the mechanisms through which the subsequent dramatic fall in life expectancy affects the propensity to save and to go to school. In addition, one has also to introduce health expenditures and their impact on productivity of infected workers. We propose a comprehensive theoretical framework to deal with such issues, based on the Lucas-Uzawa model and on the Blanchard model (1985). In order to highlight in a much simpler way the impacts of the decline in life expectancy, we finally propose to extend straightforwardly the celebrated Cuddington and Hancock (1994) Solow model, and we apply it to the South African case. The results are in line with the view that AIDS is likely to have long run (or delayed) harmful effects on growth, driven by the currently dramatically dropping life expectancy.

The paper is organized as follows. Section 2 is devoted to the study of Spanish flu-like epidemics. Section 3 is a sound analysis of an AIDS-like crisis, with a

discussion of the current (mostly) empirical literature. Section 4 presents our extended Cuddington-Hancock model with the application to South Africa.

2 The case of a Spanish flu-like epidemic

In this section, we shall consider the case of the epidemics with massive and "immediate" effects, like the Spanish flu. In such a case, the whole action takes place in a very short period of time, eventually ending at period t_0. After t_0, the economy comes back to its initial natural and epidemiological environment, so that the whole story could be in principle reduced to an "initial condition shock". Suppose that the economy before the epidemics is laying in a steady state equilibrium, say $\bar{E}$. When the epidemic ends at t_0, the state of the economy is likely to be far from equilibrium, say $X(t_0) << \bar{E}$, but the economy is back to its natural environment from $t = t_0$. Therefore, the analysis of the impact of the epidemic after t_0 amounts to studying the transition dynamics from $X(t_0) << \bar{E}$ to $\bar{E}$. We shall examine this question with some details in the next section. Indeed, the answer crucially depends on the way human capital is produced in the economy.

2.1 The one-sector framework

In the one-sector model, the production of human capital is assumed to rely on the same technology as the production of physical capital: The economy produces a single final good, say Y, using a constant return to scale technology with physical and human capital as inputs, and this final good is either consumed or invested for the accumulation of either physical or human capital. The two capital assets are therefore *ex-ante* perfect substitutes. See Barro and Sala-i-Martin (1995), chapter 5 for a detailed description of the underlying economy.
Suppose now that a benevolent planner has to care about the allocation of the final good across consumption, investment in human capital, say H, and investment in physical capital, say K, so as to maximize the social welfare:

$$\int_0^\infty U\left(c(t)\right)\ N(t)\ e^{-\rho t}\ dt \tag{1}$$

subject to

$$Y(t) = A(t)K(t)^\alpha\ H(t)^{1-\alpha} = N(t)\ c(t) + I_K(t) + I_H(t),$$

$$\overset{\bullet}{K}(t) = I_K(t) - \delta_K\, K(t),$$

$$\overset{\bullet}{H}(t) = I_H(t) - \delta_H\, H(t),$$

where $H(0)$ and $K(0)$ are given, and under the positivity and irreversibility constraints: $c(t) \geq 0$, $I_H(t) \geq 0$ and $I_K(t) \geq 0$. The constraints on gross investment variables are the irreversibility restrictions, without which the optimization problem would result trivial. ρ is the strictly positive time discount rate. α is the physical capital share parameter while δ_K (Resp. δ_H) is the rate of depreciation of physical (Resp. human) capital. In such a context, we can naturally and without loss of generality assume: $\delta_K = \delta_H = \delta$. $U(.)$ is the utility function, which is assumed to be strictly concave in consumption per capita. Finally notice that the exogenous environment of the economy is defined by the pair $(N(t), A(t))$, where $N(t)$ is the size of population and $A(t)$ is the level of technological progress.
Some versions of problem (1) are treated in the growth literature. With the notable exception of Gómez (2003), the usual treatment is merely intuitive and somewhat messy (see for example, Barro and Sala-i-Martin, 1995, chapter 5). We shall provide here a different insight into the optimal control problem, and our interpretation will highlight the role of population growth in the transition dynamics, a point systematically omitted in the literature[1]. To problem (1), we shall associate the following Lagrangean:

$$\mathbf{L} = U(c)\; N\; e^{-\rho t} + \lambda\; \Big(A(t)K(t)^{\alpha}\; H(t)^{1-\alpha} - N(t)\; c(t) - I_K(t) - I_H(t)\Big) + \beta_1\; (I_K - \delta K)$$

$$+\beta_2\; (I_H - \delta H) + \mu_1\; I_K + \mu_2\; I_H,$$

where λ, β_1, β_2, μ_1 and μ_2 are trivially associated with the constraints of the optimal control problem, the multipliers μ_i, $i = 1, 2$, being the Kuhn-Tucker multipliers related to the irreversibility constraints. The first order conditions with respect to the controls c, I_K, and I_H are respectively:

$$U'(c) = \lambda, \tag{2}$$

$$-\lambda + \beta_1 + \mu_1 = 0, \tag{3}$$

$$-\lambda + \beta_2 + \mu_2, \tag{4}$$

[1]Even in Gómez (2003).

which implies that in the interior solution, that is if $I_K > 0$ and $I_H > 0$, we should have:

$$\lambda = \beta_1 = \beta_2, \tag{5}$$

because the usual slackness conditions impose $\mu_1 = \mu_2 = 0$ in such a case. The previous relationships are not surprising at all: when the irreversibility constraints do not matter, the shadow prices of both capital assets should be identical, and equal to the marginal utility of consumption. This is the unique possible outcome in a one-sector economy where the final good can be indifferently assigned to consumption or to investment in any of the two capital assets. This property has a very strong implication: **in the interior solution, the ratio physical to human capital should be constant**. Indeed, using the first-order conditions with respect to the state variables K and H, one finds:

$$\lambda \; A \; \alpha K^{\alpha-1} \; H^{1-\alpha} - \delta \; \beta_1 = - \dot{\beta}_1,$$

$$\lambda \; A \; (1-\alpha) K^{\alpha} \; H^{-\alpha} - \delta \; \beta_2 = - \dot{\beta}_2,$$

which implies by (5) that the returns to both capital assets are equal (to, say, r_i), that is:

$$A \; \alpha K^{\alpha-1} \; H^{1-\alpha} - \delta = A \; (1-\alpha) K^{\alpha} \; H^{-\alpha} - \delta = r_i,$$

ultimately fixing the ratio physical to human capital in the interior solution[2]

$$\frac{K}{H} = \left(\frac{K}{H} \right)_i = \frac{\alpha}{1-\alpha}. \tag{6}$$

The whole transition dynamics of this economy derive from this property. Naturally, if the ratio physical to human capital is not equal to $\left(\frac{K}{H}\right)_i$ at $t = 0$, the interior regime cannot set in from the initial period, and some corner regime should prevail at least temporarily. This is the story told in Barro and Sala-i-Martin, and much more rigorously in Gómez (2003). We shall consider here the case of a Spanish flu-like epidemics hitting the economy before $t = 0$. In the economic literature, the whole problem is written in per capita terms: even the objective function depends exclusively on per capita consumption,

[2]It is not hard to see that this property still holds for any constant returns production function and independently of the assumption: $\delta_K = \delta_H = \delta$.

the population size disappears from the problem and all variables are written in per capita terms[3]. Accordingly, the case of an epidemics occurring before the initial period amounts to starting from $\frac{K}{H}(0) = \frac{k}{h}(0) > \frac{\alpha}{1-\alpha}$, where k and h stand for per capita physical and human capital. In such a situation, the economy will trivially start with a corner regime $I_K = 0$ and $I_H > 0$ (or equivalently $\mu_2 = 0$. Physical capital is relatively in excess, the marginal return to this factor is lower than the return to human capital, and the desire to lower physical capital entails that the irreversibility constraint on this asset will be binding for a while. Gómez (2003) neatly shows that under standard parametric conditions, this corner regime will prevail till the returns to both capital are equalized, which happens at a finite time in this model. After this adjustment episode, the economy will stay in the interior regime, and the output of the economy will grow at the rate, g:

$$g = \frac{r_i - \rho}{\sigma} = \frac{A\,(1-\alpha)K^{\alpha}\,H^{-\alpha} - \delta - \rho}{\sigma} = \frac{A\,(1-\alpha)\,\left(\frac{\alpha}{1-\alpha}\right)^{\alpha} - \delta - \rho}{\sigma}\,, \quad (7)$$

if we assume that the utility function is a CRRA function, that is $U(c) = \frac{c(t)^{1-\sigma}-1}{1-\sigma}$, where σ represents the inverse of the intertemporal elasticity of substitution (see Barro and Sala-i-Martin (1995), chapter 5).

This is the usual story told in growth theory, and we shall examine it here regarding two important issues in the analysis of the economic effects of epidemics of the Spanish flu type.

i) First of all, it is worth pointing out that such an epidemic has no long run effect: its sole impact takes place in the short run. And in the short run, the epidemic is responded by a massive accumulation of human capital (at the expense of physical capital), in order to catch up with the interior (equilibrium) value of the ratio physical to human capital. That is the return to human capital is initially higher than the return to physical capital, and the more devastating the epidemic, the larger this return differential, and the more intense investment in human capital. What would be the growth rate of the economy along this adjustment period? Clearly, for a fixed level of technological progress, economic growth is entirely driven by the accumulation of human capital in the short run (since physical capital is decreasing at a rate δ). Such a short run growth experience could be a

[3]See Gómez (2003), page 368.

miraculous growth episode. Indeed, since

$$\frac{\dot{H}}{H} = \frac{I_H}{H} - \delta,$$

the growth rate of output can result unusually large if $\frac{I_H}{H} - \delta$, which is certainly the case if the initial level of H, in absolute value, tends to zero. According to this story, the post-epidemic period is rather a happy age with massive accumulation of human capital, and large output growth rates, especially if the experienced epidemic is truly devastating and harmful for human capital.

ii) What is the role of demography in this story? As in the standard neoclassical growth theory, it plays a fairly marginal role. Nonetheless, one can get a step further, and notice that the accumulation laws written in per capita terms, as usual in such a literature, should exhibit the growth rate of population, $n = \frac{\dot{N}}{N}$. For example, per capita human capital accumulation evolves according to:

$$\frac{\dot{h}}{h} = \frac{i_h}{h} - \delta - n, \tag{8}$$

with obvious notations. In the growth literature, we typically assume $n(t) = 0$ for every t, before and after $t = 0$: human capital accumulation is supposed to capture the whole story. While the presence of a nonzero and possibly time-dependent population growth rate will not change the basic ingredients of the transition dynamics (notably the way the interior and corner regimes do occur), since the fundamental equations (2) to (6), do not depend on such a variable, it may add some relatively interesting features.
Suppose the economy had a constant population growth rate before the epidemic, say $N(t) = e^{\bar{n}\, t}$ for $t < 0$. At $t = 0$, the economy loses a proportion ϕ of its population: $N(0) = 1 - \phi$. If the economy recovers its natural demographic growth rate after the epidemic, we get: $N(t) = (1 - \phi)\, e^{\bar{n}t}$ for $t \geq 0$, and the law of motion of human capital per capita (8) will not be altered. Now, assume that the epidemic, though not durable itself, has a demographic impact: a truly devastating epidemic is likely to significantly alter the natural demographic course. For example, it might cause the population growth rate to depart from its natural value for a while. If this growth rate exceeds the natural value during the transition, then the pace of the catching up mechanism outlined above might well be slowed down, which ultimately pushes

down the growth rate of output per capita during the transition. Otherwise, we get the opposite picture. Moreover, such a demographic ingredient will have no impact on the return to human capital either in the short or long run because the marginal productivity of this factor is given by

$$A\,(1-\alpha)K^{\alpha}\,H^{-\alpha} = A\,(1-\alpha)\,\left(\frac{K}{H}\right)^{\alpha}.$$

Finally, since the return to capital is constant and independent of n in the interior regime, the long-run growth rate of per capita variables is still equal to the value given in (7) above, which is also independent of n.

Therefore, the one-sector model leaves a tight space for demography to matter. The law of motion of population growth has no decisive role in the shape of the transition dynamics. It may mechanically reinforce or weaken the catching up mechanism characterizing the transition dynamics, but it cannot affect the return to human capital neither in the short nor in the long run. Moreover, it seems obvious that in such a one-sector framework, the effects of an epidemic are fairly the same as those of a war or a hurricane, which are supposedly more harmful for physical capital than for human capital. This symmetry between physical and human capital might be broken down within the same set-up by introducing asymmetric adjustment costs, as suggested by Barro and Sala-i-Martin (1995), chapter 5, but such a modification is, in our view, far from reproducing the specific features of human capital formation and the demographic interactions. A more serious track to follow is the two-sector growth literature with human capital. We briefly examine it hereafter.

2.2 The two-sector framework

We shall consider now the two-sector models à la Lucas (1988) two-sector endogenous growth model, with a production externality in the final good sector associated with the human capital accumulation as studied in Boucekkine and Ruiz-Tamarit (2004a, 2004b), Xie (1994) and Benhabib and Perli (1994). The main characteristic of such a framework is the introduction of a specific education sector, therefore differentiating the production of human capital from the production of the physical good. In particular, the education sector is assumed to be more human capital intensive than the final good sector. We first describe very briefly the basic model.

The economy is closed, competitive, and populated with many identical, rational agents, who have to choose the controls $c(t)$ and $u(t)$, $\forall t \geq 0$, so as to maximize the following objective function

$$\int_0^\infty U\left(c(t)\right)\ N(t)\ e^{-\rho t}\ dt \tag{9}$$

subject to

$$\dot{K}\ (t) = Y(t) - N(t)\ c(t) = AK(t)^\alpha [u(t)N(t)h(t)]^{1-\alpha} h_a(t)^\gamma - N(t)c(t)$$

$$\dot{h}\ (t) = \psi[1-u(t)]h(t)$$

where $K(0) = K_0 > 0$ and $h(0) = h_0 > 0$ are given. In order to present some analytical results, we shall assume from the beginning that the utility function is a CRRA function, that is $U(c) = \frac{c(t)^{1-\sigma}-1}{1-\sigma}$. In this model $u(t)$ is the fraction of non-leisure time devoted to goods production. The output in the consumption good sector, $Y(t)$, which may be allocated to consumption or to physical capital accumulation depends on the capital stock, $K(t)$, the effective work force, $u(t)N(t)h(t)$, and the average skill level of workers, $h_a(t)$. γ is positive and intended to capture the external effects of human capital. In our problem the representative optimizing agent takes $h_a(t)$ as given and, consequently, the competitive solution will be different from the socially optimal allocation. The efficiency parameter A represents the constant technological level in the goods sector of this economy. It is assumed that the growth of human capital does not depend on the physical capital stock, but depends on the effort devoted to the accumulation of human capital, $1-u(t)$, as well as on the achieved human capital stock. The efficiency parameter ψ represents the constant technological level in the educational sector. Technology in goods sector shows constant returns to scale over private internal factors. Technology in educational sector is linear. For the sake of simplicity, it is assumed that there is no physical nor human capital depreciation. The analytical solution technique used in this paper still applies in the presence of nonzero depreciation rates.

The current value Hamiltonian associated with the previous intertemporal optimization problem is

$$H^c(K, h, \theta_1, \theta_2, c, u; A, \sigma, \alpha, \gamma, \psi, \{N(t), h_a(t) : t \geq 0\}) =$$

$$= \frac{c^{1-\sigma}-1}{1-\sigma}N + \theta_1\left[AK^{\alpha}(uNh)^{1-\alpha}h_a^{\gamma} - Nc\right] + \theta_2\psi\,(1-u)\,h, \qquad (10)$$

where θ_1 and θ_2 are the co-state variables for K and h, respectively. The term h_a, as we have seen, is taken as given in order to calculate the competitive equilibrium. Then, the necessary first order conditions, under the equilibrium condition $h_a = h$ implying that all workers are being treated identically, are

$$c^{-\sigma} = \theta_1 \qquad (11)$$

$$\theta_1\,(1-\alpha)\,AK^{\alpha}(uNh)^{-\alpha}Nh^{1+\gamma} = \theta_2\psi h, \qquad (12)$$

$$\dot{\theta}_1 = \rho\theta_1 - \theta_1\alpha AK^{\alpha-1}(uNh)^{1-\alpha}h^{\gamma}, \qquad (13)$$

$$\dot{\theta}_2 = \rho\theta_2 - \theta_1(1-\alpha)AK^{\alpha}(uN)^{1-\alpha}h^{-\alpha+\gamma} - \theta_2\psi(1-u), \qquad (14)$$

$$\dot{K} = AK^{\alpha}(uNh)^{1-\alpha}h^{\gamma} - Nc, \qquad (15)$$

$$\dot{h} = \psi\,(1-u)\,h. \qquad (16)$$

As boundary conditions we have the initial conditions K_0 and h_0 and the transversality conditions

$$\lim_{t\to\infty}\theta_1 K\exp\{-\rho t\} = 0, \qquad (17)$$

$$\lim_{t\to\infty}\theta_2 h\exp\{-\rho t\} = 0. \qquad (18)$$

This completes the Lucas model. A quick look at equation (16) is enough to capture the following fact: in contrast to the one-sector model (see equation (8)) where population has a mechanical growth on the growth rate of human capital per capita, we don't have such a feature in the Lucas model, at least not directly. The effect of population growth on per capita human capital accumulation might operate through the variable, $1-u$, that is the fraction of human capital allocated to the education sector. Therefore, if any, the effects of demographic variables in the Lucas model are necessarily much less trivial and mechanistic than in the one-sector model. We shall examine this issue, among others, in this section.

Before, some early remarks are necessary. First of all, it is well known (see Xie, 1994, and Benhabib and Perli, 1994) that the Lucas model has multiple equilibrium paths for strong enough external effects. Given the purpose of this study, we shall abstract from this property, and to make life easier, we

will assume $\gamma = 0$ from now. In such a case, the ratio physical to human capital is uniquely determined in short and long run, as it is proved for example in Boucekkine and Ruiz-Tamarit (2004a), Proposition 5, page 12. Let us denote by $\omega = \frac{k}{h}$, and by $\bar{\omega}$ this ratio in the long run. Now let us come back to our epidemic shock: suppose that initially we have $\omega(0) > \bar{\omega}$, that is human capital is relatively scarce after the epidemic. We have performed the same exercise in the one-sector framework with $\bar{\omega}$ equal to the ratio physical to human capital in the interior regime. Will the two-sector framework supply substantially different results?

i) Having clearly differentiated the production technology of physical Vs human capital breaks down *de facto* the symmetry between the two factors, a particularly undesirable property of the one-sector seen above. To get an idea about that, have a look at the expression of the growth rate of output per capita in the case $\sigma = \alpha$ studied in Boucekkine and Ruiz-Tamarit (2004a), equation (35), page 14[4].

$$\frac{1}{y(t)}\frac{dy(t)}{dt} = \frac{\psi(1-\alpha)-\rho}{\alpha} + \psi\left[\frac{\bar{\omega}}{\omega(t)}\right]^{1-\alpha}. \tag{19}$$

In contrast to the one-sector model, there is no U-shaped relationship between the growth rate and the ratio physical to human capital. As a consequence, the impact of a Spanish flu like epidemic has nothing to do with the impact of wars or hurricanes. When $\sigma = \alpha$, the growth rate is a simple decreasing and convex function of the ratio ω. After an epidemic (Resp. a war), the ratio ω is much above (Resp. below) the long-run equilibrium value $\bar{\omega}$, which induces an adjustment period with a low (Resp. high) growth period. The lack of human capital is definitely much more penalizing than the lack of physical capital in such a framework!
The basic mechanism at work is the following: as in the one-sector model and for the same analytical reason, the epidemic is followed by an increase in the return to the relatively scarce factor, namely human capital: wages must go dramatically up. But in the two-sector framework, the economy has an additional problem to solve: how much human capital to assign to each sector? In times of human capital scarcity, the education sector is the more costly to operate because it is more intensive in human capital and wages are

[4]The analytical results below are indeed general, see Boucekkine and Ruiz-Tamarit (2004b).

high. As a consequence, the economy might find it optimal to assign more human capital to the less intensive sector in human capital, namely the final good sector, which goes against the catching up mechanism outlined in the one-sector model. Therefore, the post-epidemic period is characterized by slow growth and high wages. In contrast, the post-war (or post-hurricane) period is characterized by low wages and high growth: there is no sectoral allocation problem for physical capital, and the rising return to this factor will simply imply, as in the one-sector model, a massive accumulation of physical capital in the short run.

ii) Let us come back now to one issue raised above: if the growth rate of population has no direct effect on per capita human capital accumulation by construction, does it matter in a much less trivial, much trickier way? The answer is yes. Boucekkine and Ruiz-Tamarit (2004b) have recently solved analytically solved the model for any admissible parameterization. Among their findings, they show that variable u, the fraction of human capital resources devoted to the final good sector, is explicitly given by[5]

$$0 < u = -\frac{\psi\left(1-\sigma\right)+n-\rho}{\sigma\psi}\frac{{}_2F_1(t)}{\widetilde{{}_2F_1}\left(t\right)} < 1, \tag{20}$$

where ${}_2F_1(t)$ and $\widetilde{{}_2F_1}\left(t\right)$ are two time dependent hypergeometric functions which arguments also depend on the population growth rate n (see Propositions 1, 2 and 9 in Boucekkine and Ruiz-Tamarit, 2004b). Moreover, it can be shown that the transitional dynamics of $u(t)$ converge asymptotically to the interior constant value

$$0 < \bar{u} = -\frac{\left(\delta-\theta\right)\left(1-\sigma\right)+n-\rho}{\sigma\delta} < 1; \tag{21}$$

Finally, the population growth rate does also enter the growth rates of per capita variables in the long-run, in sharp contrast to the one-sector case (see equation (7)). For example, per capita human capital is shown to approach asymptotically the unique positive balanced growth path given by (Proposition 5, Boucekkine and Ruiz-Tamarit, 2004b):

$$\bar{h} = \frac{h_0}{\widetilde{{}_2F_1}\left(0\right)}\exp\left\{\frac{\psi+n-\rho}{\sigma}t\right\}, \tag{22}$$

[5]Provided the parameters meet some conditions for the positivity of the economic variables.

along which, given $\delta + n > \theta + \rho$, h grows permanently at a positive constant rate $\bar{g}_h = \frac{\psi + n - \rho}{\sigma}$. Henceforth, population growth is a positive determinant of the long-run growth rate. Indeed, while the short term effects of population growth are ambiguous (see the presence of the hypergeometric functions in (20) which arguments do depend on n), the long run effects are clear: from (21), one can infer that an increase in demographic growth induces a rise in the fraction of human capital devoted to education in the long run, which in turn boosts human capital accumulation, and ends up increasing long run growth. To our knowledge, this role of demographic growth in the Lucas-Uzawa model has never been mentioned before, which is not that surprising since most published papers in the field assume $n = 0$ from the beginning. We show here that the Lucas-Uzawa framework is not only better suited to track the specific outcomes of epidemics, it implicitly and explicitly assign a non-residual role to demographic growth.

2.3 Empirical evidence

The empirical evidence on the economic effects of the Spanish flu (and similar epidemics) is highly disputed, just like the theoretical predictions are, when one moves from one-sector to two-sector models. The question turns out to be whether say the Spanish flu did induce a fall or rise in per capita income growth in the short run. The answer would be yes with the one-sector model view, and just the contrary with the two-sector setting. Empirically, the question is very far from trivial. First of all, one should recall that the Spanish flu occurred after the first world war, and therefore, part of the phenomena that took place in our period of interest is also probably due to the post-war adjustment dynamics (similarly predicted by both the one-sector and two-sector models). Second, and more generally, disentangling the effects of the epidemic is markedly complicated because one has to control for many other potential explanatory factors, like urbanization, the sectoral composition of the economy, initial GDP and other numerous variables. This seems like a daunting task, there are however some very careful studies on this issue. Brainerd and Siegler (2003) and Bloom and Mahal (1997a) are among the very best[6].

[6]Another related known and even classical investigation on the growth effect of epidemics is due to Jack Hirshleifer in his celebrated book, *Economic Behaviour in Adversity*, 1987. Hirshleifer examined the case of the plague which killed one-third of the European

Bloom and Mahal studied the case of the Spanish flu in India. Precisely, they looked at the acreage sown in India across 13 Indian provinces. They found no relationship between the magnitude of population decline and the variation in acreage sown per capital across provinces. In a more detailed investigation, Brainerd and Siegler focused on the impact of the Spanish flu in the US. In their study, the dependent variable is the growth rate of per capita income from 1919-1921 to 1930, and the primary explanatory variable is the number of flu and pneumonia deaths per 1000 persons in each state of the US reported in 1918 and 1919. A nice feature of this study is the inclusion of many more variables to control for other potentially important factors (like education levels, initial income, agricultural share of personal income, ethnic composition...etc...). The main result of their econometric regressions is the significant and positive impact of the Spanish flu on the growth rate of per capita income: "...the flu coefficient ranges between 0.219 and 0.235...and is always statistically significant at the 5 percent level or lower..." (Brainerd and Siegler, 2003, page 17). And the authors conclude that along with conditional convergence and the rise of education, the Spanish flu does significantly matter in the economic growth history of US.

As usual, the available empirical evidence does not allow to settle the theoretical debate we have raised in the previous sections. Of course, it is meaningless to compare the studies of Bloom and Mahal Vs Brainerd and Siegler: they are applied to two different countries and use different regression models and different controls. Moreover, the many other contributions to this topic do not allow to extract like a perceived wisdom. This is indeed one of the main lessons drawn in the economic growth textbooks: for example, Barro and Sala-i-Martin (1995), chapter 5, take the view according to which, while the catching-up mechanism outlined in the one-sector model does work for post-war periods (see German and Japanese miracles), it is definitely unclear that it works in post-epidemic periods. As we will see in one of the next sections, this question has been also empirically unsettled in the case of the HIV/AIDS pandemic until very recently. The economic and econometric analysis of the latter pandemic is nonetheless quite different from the influenza case, and we shall devote to this issue the remaining sections of this paper.

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population between 1348 and 1351. He suggested that the plague by sharply reducing the population size led to a rapid and persistent rise in real wages for laboring classes.

3 The case of an AIDS-like epidemic

In contrast to the Spanish flu, which claimed victims all over the world in a few days, AIDS is a much more pernicious disease which can be associated with long periods of necessarily undermined activity and large and permanent health expenditures. Therefore, the economic theory relevant in this case is by no means a story of initial shortfall in human capital or any state variable else, and the subsequent short term adjustment dynamics. The needed economic theory is much more complicated, and though some very recent contributions have attempted to capture some of the crucial aspects of the AIDS epidemics (see for example, Corrigan, Glomm and Mendes, 2005), much work remains to accomplish. In order to make clear the whole set of economic implications of such an epidemic, we start with an overall view of the problem.

3.1 An overall view of the economic effects of AIDS

The direct and indirect economic effects of AIDS have been recently comprehensively identified and listed in some studies. Part of these studies have been promoted by United Nations Department of Economic and Social (Population Division). In its 2004 report, this division identifies four channels through which AIDS affects the economy (page 81):

i) The most obvious channel is through labor supply which will be cut, and its productivity reduced. Admittedly, the growth effect of such a cut can vary depending on several factors (for example, the degree to which the epidemic affects hard-to-replace skilled workers).

ii) The second channel is related to the loss of income incurred by families due to health expenditures. Such a channel can induce clearly negative long-term effects because the nutrition and education of children is likely to suffer from such an income loss.

iii) Similarly, AIDS is likely to be harmful for investment in human and physical capital by diverting public expenditures from such growth-enhancing activities. The Population Division report also points at a confidence problem that may undermine foreign investment, and reinforce the drop in the investment rate.

iv) The last important channel mentioned has to do with the probable deepening of poverty in the most affected countries as per capita income goes down.

Corrigan, Glomm and Mendes (2005) have based their overlapping generations model on the first three effects mentioned above, with some focus on the impact of the rising number of orphans (roughly captured by the second and third effects just above). A much more detailed accounting of the AIDS economic effects is performed by Freire (2002) in an excellently documented study. Freire cleverly distinguished between the effects of morbidity, and those of mortality. As to morbidity effects, she obviously put forward the fall in productivity and absenteism on one hand, and the rise in health expenditures (including the expenditures made by relatives and anticipation of future costs) on the other. Mortality induce other costs for households: cumulated cost of funerals, loss of the revenue of the sick persons, care of orphans and widows, and **the effects on education and saving due to the fall in life expectancy**. Freire devoted a substantial part of her contribution to the specific but crucial issue of AIDS impact on the savings of households, with an application to South Africa. We shall use her results in the last section of this paper. Before, we shall point at some fundamental modelling aspects of the economic AIDS problem. Concretely, we will give some insight into the modelling of saving behavior and the evolution of productivity under an AIDS-like pandemic.

3.2 Theoretical analysis

In this section, we shall focus on some crucial and subtle aspects of the theoretical economic analysis of AIDS. The aim of this section is not to present a complete closed micro-funded model of an economy subject to the AIDS pandemic, but to highlight the main sensitive specification issues. Some computable general equilibrium models have been recently built up to account for macroeconomic effects of AIDS: for example, Kambou, Devarajan and Over (1992) applied an eleven-sector computable general equilibrium model for the analysis of the impact of the epidemic in Cameroon. We don't take this approach here.

3.2.1 Saving behavior

As mentioned above, one of the main economic implications of an AIDS-like epidemic is a probable and significant distortion in the saving behavior, at the level of both the households and the governments. The modelling of such a distortion at the former level being by far trickier, we shall focus on it. Freire (2002) comprehensively summarized the determinants of such a distortion. Of course, part of the story is quite trivial: the subsequent health expenditures cut savings, and this has a clear direct negative effect on factor accumulation, thus on economic growth. We shall incorporate this aspect in our analysis in the next section. Here we focus on a much less trivial point, the way life expectancy (or equivalently, the probability of death) might affect saving. This requires a departure from the standard deterministic neoclassical model, at least formally.

The easiest tool to incorporate life expectancy into the growth model is probably the celebrated 1985 Blanchard model. Freire relied on a discrete time version of this model with constant population, we shall use the original continuous time version and allow for population growth to be consistent with the previous section of this paper. Blanchard's model entails a number of well-known drawbacks, which exclusively derive from the simplicity of the assumed analytical specifications. Nonetheless, these simplifications are absolutely necessary to get the impressively clear and meaningful aggregation formulas from relatively uneasy vintage specifications. Aggregation often yields much more complicated formulas if one depart from the basic Blanchard setting[7]. For example, if we depart from the "perpetual youth" assumption of Blanchard, that it is if we assume that the probability of death is age-dependent, then the needed algebraic steps are definitely trickier (see Boucekkine, de la Croix and Licandro, 2002, Faruqee, 2003, and Boucekkine and Diene, 2005, for some extensions with more realistic survival laws). Another drawback of Blanchard's model has been recently raised by Ascari and Rankin (2004): in effect, when labor supply is endogenized within the original Blanchard framework, labour supply might be negative, therefore requiring a major restructuring of the benchmark model[8].

For our purpose, we don't think this is a major flaw: labor resources implications of the epidemics can be treated directly on the production side of the

[7] Aisa and Pueyo (2004) is an exception.

[8] Ascari and Rankin solve the problem by introducing real money balances.

models under consideration. Moreover, in the countries most affected by the AIDS pandemic, namely sub-Saharan African countries, assuming endogenous labor supply sounds as a joke! Hereafter, we give a simple presentation of the mechanisms at work in the benchmark Blanchard model, which is largely sufficient to have an idea about its adequacy in our context.

The Blanchard model is of the Yaari type: at every instant, a new generation is born, and each member of any generation has a constant instantaneous (flow) probability to die equal to p. Therefore, an agent born at v (generation or vintage v) has a probability $e^{-p\,(t-v)}$ to survive at $t \geq v$. Accordingly, the probability to die at date t is equal to $pe^{-p\,(t-v)}$, and life expectancy is constant equal to$\frac{1}{p}$. An AIDS-like pandemic by rising the flow probability p, increases mortality and decreases life expectancy, as observed in the countries affected. How does household's saving respond to such a shock? And more generally, to which extent demographic dynamics matter in such a story? In order to complete the model a bit in this respect, let us introduce population growth in the benchmark model. Let us assume that population grows at a rate n, with $n \in \mathbf{R}$. In such a case, the total population size is given by $L_t = e^{nt}$ (with $L_0 = 1$), implying that the generation of vintage t must be of size $(p+n)\ e^{nt}$. We have now the minimal number of demographic parameters to go on, namely population growth and life expectancy.
Each agent of any generation of vintage v determines her optimal consumption stream (or equivalently, saving stream) at time $t \geq v$ by maximizing the expected lifetime utility:

$$E_t \int_t^\infty U(c_{v,s}) e^{-\rho(s-t)}\, ds = \int_t^\infty U(c_{v,s})\ e^{-p(s-t)}\ e^{-\rho(s-t)}\, ds,$$

subject to the usual budgetary constraint:

$$\overset{\bullet}{a}_{v,s} = (r(s)+p)\ a_{v,s} + w(s) - c_{v,s},$$

where $a_{v,s}$ stands for the asset holdings of the agent and $w(s)$ for the (real) wage at instant s. The presence of the flow probability p, added to r in the constraint above, reflects as usual the existence of an annuity market. All lending and borrowing contracts between generations are insured by competitive life insurance companies. When an initial condition is given, typically: no bequests, $a_{v,v} = 0$, the optimal control problem of the agent is well-posed.

The associated first-order conditions (for a CRRA utility function) are standard:

$$\frac{\overset{\bullet}{c}_{v,s}}{c_{v,s}} = \frac{r(s) - \rho}{\sigma},$$

and

$$\lim_{s \to \infty} a_{v,s}\, e^{-(p+\bar{r}_{s,t})} = 0,$$

where $\bar{r}_{s,t} = \frac{1}{s-t} \int_t^s r(u)\, du$ is the average interest rate from t. The amazingly useful virtue of the Blanchard set-up derives from the neat aggregation rules obtained. For any vintage magnitude $x_{v,t}$, aggregation across vintages gives the aggregate magnitude $X(t)$ following the formula:

$$X(t) = \int_{-\infty}^{t} x_{v,t}(p+n)\, e^{nv}\, e^{-p(t-v)}\, dv.$$

Accordingly, one could construct aggregate consumption, $C(t)$, and aggregate asset holdings, $A(t)$. In contrast, being age-independent, wages are aggregated differently:

$$W(t) = \int_{-\infty}^{t} e^{nv} \int_{t}^{+\infty} w(u)\, e^{-(p+\bar{r}_{u,t})(u-t)}\, du\, dv.$$

The following important formulas could be established by manipulating straightforwardly the aggregate magnitudes above, and using the first-order conditions in the case of a logarithmic utility function ($\sigma = 1$) [9]:

$$C(t) = (p+\rho)\ \left(A(t) + W(t)\right),$$

$$\overset{\bullet}{C}(t) = (r(t) + n - \rho)\, C(t) - (n+p)\,(p+\rho)\, A(t),$$

$$\overset{\bullet}{A}(t) = r(t)\, A(t) + w(t)\, e^{nt} - C(t).$$

Notice that if $n = 0$, we recover the results of Blanchard for the aggregate variables, notably $C(t)$ and $A(t)$. In particular, the propensity to consume aggregate income is $p + \rho$ [10]. Even if we abstract from the consumption of

[9] This is only done to allow for comparison with Blanchard's model (1985). Recall that Blanchard considers a constant population size, $n = 0$.

[10] Actually, this comes from the fact that the propensity to consume income is the same for all generations and equal to $p + \rho$.

health goods, an AIDS-like epidemic causes saving to drop: a shock increasing the death probability of death (or lowering life expectancy) have a negative impact on savings, and the more the epidemic is damaging and long-lived, the larger and more persistent is the drop in the saving rate. This makes Blanchard-like models most suitable for the analysis of the economic impact of epidemics.

3.2.2 Schooling

While the benchmark version of the Blanchard model is enough to capture the impact of a shorter life expectancy on savings, it is not sufficient to deliver the other implication of shorter horizons, namely a shorter schooling time, and therefore a slower pace of human capital accumulation. This issue has been recently the focus of some studies (see for example, de la Croix and Licandro, 1999, and Boucekkine, de la Croix and Licandro, 2002 and 2003). de la Croix and Licandro (1999) is the most elementary extension since it is closely built on the benchmark version of Blanchard model. Precisely, the authors adopt all the assumptions of Blanchard but depart from it in two respects. First of all, there is an option to go to school, and the appropriate budgetary constraint for an individual born at instant v might be written in an integral form as:

$$\int_v^{\infty} c_{v,s}\, R_{v,s}\, ds = \int_{v+S(v)}^{\infty} \omega_{v,s}\, R_{v,s}\, ds,$$

where $R(v,s) = e^{-\int_v^s (r(u)+\rho)\, du}$, and $\omega_{v,s}$ is the wage per unit of time paid to the individual of vintage v at instant s. Second, the latter wage is proportional to human capital, that is:

$$\omega_{v,s} = h(v)\, w(s), \tag{23}$$

where $w(s)$ is the wage per unit of human capital. Human capital of a generation v is a function of the time spent at school, $S(v)$, and eventually on the average human capital, $\bar{H}(v)$ at birth[11]:

$$h(v) = \bar{H}(v)\, S(v), \tag{24}$$

[11]The latter is traditionally referred to as the Lucas externality.

for example. de la Croix and Licandro found that under linear utility and linear production function (in aggregate human capital), optimal schooling time in the interior solution is:

$$S(v) = \frac{1}{\rho + p},$$

which allows to capture in the neatest way the above mentioned impact of epidemics on education: a higher probability of death, p, causes schooling time to drop, which in human capital-based growth model is a very severe limit to growth.

Another specification of the problem more connected to the Lucas-Uzawa model could be the following. At any instant, the individual of a generation v has to allocate one unit of time between education and production (or equivalently working in the final good sector). Call $u(v)$ the fraction of time devoted to production. The problem of the individual is to find the optimal production time, $u(v)$, together with the optimal lifetime consumption, in the sense of the maximization of the lifetime utility:

$$\int_t^\infty U(c_{v,s})\ e^{-p(s-t)}\ e^{-\rho(s-t)}\ ds,$$

subject to the usual budgetary constraint:

$$\dot{a}_{v,s} = (r(s) + p)\ a_{v,s} + w_{v,s}\ u(v) - c_{v,s}, \tag{25}$$

where $w_{v,s}$ is the wage per unit of time as before. One might complete the model as in the alternative specification exposed just above. The wage per unit of time is as before proportional to human capital, but the production function of human capital needs to be adapted as follows:

$$h(v) = \psi\ \bar{H}(v)\ \left(1 - u(v)\right), \tag{26}$$

where ψ is a productivity parameter. The variable $1 - u(v)$ plays exactly the same role as the schooling time $S(v)$ in de la Croix and Licandro (1999). One might specify human capital evolution as a differential equation as in the original Lucas-Uzawa model, but with the vintage index as the independent variable:

$$\frac{\partial h(v)}{\partial v} = \psi\ \left(1 - u(v)\right)\ h(v)^\beta \bar{H}(v)^{1-\beta}, \tag{27}$$

where $0 \leq \beta \leq 1$. Notice, however, that the induced optimal control problem is much more complicated with equation (27) than with equation (26) because the in the latter the human capital of a generation v only depends on its own education effort and the average human capital at birth, while integration of (26) introduces much more clearly intergenerational dependence in the production of human capital.

In any case, both specifications would deliver the same type of results as in de la Croix and Licandro (1999), and a decrease in life expectancy should drive down the schooling time again. It is worth pointing out here that we are assuming that human capital is vintage specific in both alternative models: individuals of a cohort born at date v will have the same human capital, $h(v)$, and this capital will remain constant along their whole life. That is we assume that all the members of the same generation will take the same schooling time-independent decision ($S(v)$ in the de la Croix and Licandro (1999) formulation, and $1 - u(v)$ in our Lucas-Uzawa like specifications). Of course, this need not be always the case: the individuals of the same generation, differing in abilities and other innate characteristics, may not have the same willingness to go to and stay at school, but relaxing our vintage specificity assumption would complicate dramatically the mathematical analysis of the models under consideration (see an example of vintage models with heterogenous generations in Boucekkine, de la Croix and Peeters, 2005).

3.2.3 Health expenditures

So far we have presented different modelling strategies allowing to capture the effects of a dropping life expectancy on savings and education. It remains to examine how health expenditures, which are a major characteristic of AIDS-like epidemics, may distort the individual behavior. Clearly, health expenditures might be either public or private. Its is well known, however, that such expenditures are mainly private in developing countries (including the South African case studied in the last section of this paper). For simplification, we shall consider here private health expenditures. Denote by $m_{v,t}$ the medical and epidemic-related expenditures of an individual of cohort v at time t. A relatively comprehensive individual optimization problem would consist in maximizing:

$$\int_t^\infty U(c_{v,s}, m_{v,s})\, e^{-p(\bar{m}_s)\,(s-t)}\, e^{-\rho(s-t)}\, ds,$$

subject to the usual budgetary constraint:

$$\overset{\bullet}{a}_{v,s}= (r(s) + p(\bar{m}_s))\ a_{v,s} + w_{v,s}\ u(v) - c_{v,s} - m_{v,s}, \tag{28}$$

where $\bar{m}_s = \int_{-\infty}^{s} m_{v,s}\ dv$. The wage per unit of time and the production of human capital may follow (23) and (26) as before. Note that we implicitly consider that the medical goods are produced with the same technology as the consumption good. Unless one has in mind a critical distinctive characteristic of the production technology of medical goods, this assumption seems acceptable. Private health expenditures have several effects in this mode. First of all, they tend to increase utility, just like ordinary consumption (see for example Corrigan *et al.*, 2005). Second, paying medical care out off his own pocket has a direct cost, possibly a double cost: less consumption and/or less savings. If health consumption is not included in the utility function, a lower consumption cannot be compensated by the consumption of medical goods. The second potential negative effect on saving is intrinsic and cannot be removed. Finally, medical expenditures are likely to have some effect on life expectancy. One could model this potential effect in many appealing ways. We keep in mind here that we are primarily interested in epidemic episodes. In such a case, an individual consumption of medical goods is only likely to improve marginally the life expectancy of the individual. It seems more reasonable to assume that life expectancy rather depends on the way the whole society is handling the epidemic. More generally, since every individual is supposed to live in a society and to work with colleagues...etc..., the main determinant of life expectancy of any individual is the global "medical environment", and not the individual health expenditures level. That is why we choose the specification $p\left(\bar{m}_s\right)$, with $\bar{m}_s = \int_{-\infty}^{s} m_{v,s}\ dv$. Function $p(.)$ is typically increasing, concave with appropriate boundary conditions. Notice that such a specification generates an externality in the model, implying the inefficiency of the decentralized equilibrium and promoting the role of the governments in the provision of medical goods. This is clearly a good property of the model.

Last but not least, one might argue that a more intense medication improves productivity, notably in the production of human capital, that is the parameter ψ in equation (26) could be made an increasing function of medical expenditures. Our view here is the following: a better medication by improving life expectancy will necessarily increase time devoted to education, and therefore human capital accumulation, and this is enough to capture the

impact of medication on education. We now move to the production (of the consumption good) and productivity side of the story.

3.2.4 Productivity analysis

The other crucial channel through which an AIDS-like epidemic affects economic growth is productivity. A minimal set of features to model must include: the hard loss in the labor force due to AIDS mortality, the loss in productivity due to morbidity, and the effects of (both private and public) health expenditures on productivity. We shall list hereafter some natural modelling strategies in this respect.

In a **one-sector framework**, a specification of the aggregate production function meeting the above mentioned requirements is possibly:

$$Y(t) = A(t)\ K(t)^{\alpha}\ \left([1 - \phi(t)\ \Phi\left(m(t)\right)]\ L(t)\right)^{1-\alpha}, \tag{29}$$

where $\phi(t)$ stands for the AIDS prevalence rate at date t and $m(t)$ for AIDS-related (mainly health) expenditures per capita. $\Phi(.)$ is a decreasing function, so that an increase in health expenditures lowers the productivity loss due to morbidity[12]. Such a specification allows to track the three ingredients pointed at just above: mortality must show up in total available labor force $L(t)$, morbidity plays through the prevalence rates $\phi(t)$, and we have explicitly introduced the effects of health expenditures on productivity. Nonetheless, such a formulation misses at least three fundamental points. First of all, it omits the fact that there are huge disparities across generations in terms of prevalence and mortality rates. Second, it does not account for the effects of a dropping life expectancy on productivity (via education, as repeatedly evoked in the previous sections). Last but not least, and indirectly related to the latter point, it does not model the specific effects of AIDS on human capital accumulation. While the first two ingredients can be incorporated into the one-sector model in a more less *ad-hoc* way, the last one requires to move to a two-sector structure.

In such a **two-sector setting**, the production function in the final good sector may be amended as follows:

$$Y(t) = A(t)\ K(t)^{\alpha}\ \left([1 - \phi(t)\ \Phi\left(m(t)\right)]\ h(t)\ u(t)\ L(t)\right)^{1-\alpha},$$

[12]More conditions should be imposed on function $\Phi(.)$, see Corrigan, Glomm and Mendez (2005), and our application in this paper.

where h is human capital per capita, which evolves according to:

$$\dot{h} = \psi \ (1 - u(t))\, h. \tag{30}$$

As one can see, the law of evolution of the human capital stock is a non-vintage formulation of our equation (27), with $\beta = 0$. It is closely related to the Lucas-Uzawa framework, and could be interpreted as follows. $L(t)$ measures the number of workers in the economy, assumed to be identical in our framework, with human capital $h(t)$. Every individual has a unit of time, to dispatch between production, fraction $u(t)$, and education, fraction $1-u(t)$. Therefore, $u(t)\ L(t)$ measures production work in units of time, and $u(t)\ h(t)\ L(t)$ measures labor time in efficiency units. Introducing leisure would make the story more complete but will not change it significantly.

In a partial equilibrium (or accounting exercise), (30) should be amended to account for the effect of health expenditures on the education time and human capital accumulation. In such a case, one might replace the productivity parameter ψ by a function $\Psi\left(m(t), \phi(t)\right)$ in order to capture the impact of morbidity on human capital accumulation. Notice that in such a case, $\Psi\left(m(t), \phi(t)\right)$ plays the same role as the function $1 - \phi(t)\ \Phi\left(m(t)\right)$ in the final good sector. If one believes that the impact of the epidemics and the related health expenditures are clearly not the same across sectors, he might choose function $\Psi(.,.)$ accordingly.

In a general equilibrium set-up where the consumption-saving and education behavior is described by one of the prototypes- here (30) is a re-formulation of (27), such a specification is not necessary to capture the decline of human capital induced by the epidemic, the individuals will themselves respond to the shortened life expectancy by reducing their schooling effort.

In our view, such general equilibrium growth models are by far the most appropriate to capture the salient growth effects of epidemics. And since, as we have written repeatedly before, the epidemics do not affect in the same way the young and the elderly, a vintage structure sounds as a minimal requirement. Finally, since two-sector models are more adapted to deliver the most natural predictions (see our discussion in Section 2), we do believe that the best and most comprehensive framework to deal with the growth effects of epidemics should be a combination of the Blanchard-like consumer models with private health expenditures seen in Section 3.1 and the two-sector Lucas-Uzawa type production structure seen just above. The properties of such

models have not been seriously studied so far, and we believe it is a necessary task to undertake. Hereafter, we consider a much easier framework, a one-sector accounting setting due to Cuddington and Hancock (1994), that we shall amend later to account for the growth effects of AIDS in South Africa in such a way that the impact of AIDS on savings and schooling is accounted for.

3.2.5 Limits

Our theoretical proposal has of course some limits and drawbacks. One obvious limit is the lack of modelling of epidemiological diffusion. In the Blanchard-Yaari structures described above, every agent has a probability to die (depending on his characteristics and on the epidemic-related expenditures) due to the epidemic, and that it is the way the latter primarily gets into the model. Of course, in reality things are a bit more involved: some people are more likely to be infected than others, it takes some time for the epidemic to spread over the whole society (contagion dynamics)...etc...Such important aspects of the dynamics of epidemics could be introduced in the models described above either by incorporating some realistic contagion law of motions (like those typically used for demographic projections under AIDS, see for example Wachter *et al.*, 2003) or by using some simplifying assumptions (for example, by hypothesizing simple infection probabilities depending on the stock of people already infected). Both cases seem to be largely manageable within Yaari-Blanchard structures.

Introducing endogenous population size is however a daunting task. The main interest in conducting such an extension is to account for the possible decreasing fertility of women under widespread community infection, as recently studied by Young (2005) in a stimulating empirical work. According to Young, AIDS might well lower fertility "both directly, through a reduction in the willingness to engage in unprotected sexual activity, and indirectly, by increasing the scarcity of labour and the value of a woman's time...". Whether such a fertility effect could dominate the other potential effects of AIDS (notably on education and investment) is a highly disputable issue, but one can hardly argue that the former effect is a remote aspect of the story. Unfortunately, such an endogenization would dramatically complicate the Blanchard-Yaari structure. Easier models to incorporate this extension are overlapping-generations models à la Barro-Becker (1989) with negligible life-cycle considerations (see Chakraborty and Das, 2005, for a more recent

contribution). If one believes that the life-cycle aspects are indispensable to fully reflect and accurately assess the impact of epidemics on the investment decisions (both in human and physical capital), then Blanchard-Yaari structures are the most natural. Nonetheless, putting endogenous fertility into the model in a fairly acceptable way would require a great deal of computations, and in some cases, to re-think the whole model (for example if one is willing to introduce child mortality and/or the specific role of orphans). It seems much more reasonable in such a context to introduce exogenous but realistic (ideally estimated) law of motions for the population size under epidemics, as it is actually the case in the related demographic studies.

3.3 A simple accounting set-up: Cuddington and Hancock(1994)

Cuddington and Hancock have proposed a very simple accounting exercise to measure to which extent AIDS harms (or could have harmed) GDP growth in some sub-Saharan countries. In their 1994 *Journal of Development Economics* paper, they has applied it to Malawi. As we shall see in a minute, Cuddington and Hancock's set-up is a very useful tool to get an immediate idea about the extent of damages caused by AIDS. The set-up is of the Solow type, and it is based on a simplified production function (29):

$$Y(t) = A(t)\ K(t)^{\alpha}\ \left([1-\phi(t)\ x(t)]\ \mu(t)\ L(t)\right)^{1-\alpha}.$$

With respect to (29), a new term enters the expression of the production function, namely μ_t, which is supposed to capture labor-specific productivity, and in this sense, it could be interpreted just as the human capital variable, $h(t)$ in the two-sector formulation seen in Section 3.3. Additionally, the new production function includes a term $x(t)$, which plays the same role as $\Phi(m(t))$ in (29): a larger $x(t)$ amounts to a lower effective labor effort due to morbidity. In (29), such a loss may be reduced by medical care. In Cuddington and Hancock, such a loss is assumed exogenous: the authors consider all the situations when $x(t)$ ranges from 0 to 1.
More importantly, Cuddington and Hancock introduce the age structure of the labor force in the production function. Call $E(t)$ the effective labor force, that is

$$E(t) = [1-\phi(t)\ x(t)]\ \mu(t)\ L(t),$$

such a magnitude may be rewritten in a straightforward way taking into account the age distribution:

$$E(t) = \sum_i \left[1 - \phi_i(t)\, x_i(t)\right]\ \mu_i(t)\, L_i(t), \tag{31}$$

where i stands for the age index. Cuddington and Hancock have made further simplifications. First, they assume $x_i(t) = \xi$, where ξ is a constant comprised between 0 and 1, $\forall i$ and $\forall t$. Second, they work with $\mu_i(t) = \mu_i$, $\forall t$. Third, they even postulate a simple quadratic form for age-specific productivity:

$$\mu_i = \rho_1 + \rho_2\ (i - \bar{i}) - \rho_3\ (i - \bar{i})^2, \tag{32}$$

where ρ_k, $k = 1, 2, 3$ are three positive numbers, and where $\bar{i}$ is, for example, the minimal age to enter the labor market[13]. Equation (32) merely states that productivity is a quadratic function of age, and in this sense, it exclusively captures the experience determinant of productivity.
As in any Solow growth model, the set-up is completed by a a capital accumulation equation:

$$K(t) = s\ Y(t) + (1 - \delta)\ K_{t-1}, \tag{33}$$

where s stands for the saving rate of the economy (assumed constant by Cuddington and Hancock), and with K_0 given. To be more precise, the authors retrieve a term $x\ H_t$ from the right hand side of equation (33), where H_t stands for total health expenditures, and x is the fraction of these expenditures related to AIDS. We shall consider here the saving rate s in a broader sense, it is the fraction of income not spent in consumption and health expenditures, which allows us to write capital accumulation as usual.

Using the demographic projections issued by the World Bank for Malawi, Cuddington and Hancock have studied to which extent the AIDS epidemic has affected and will affect GDP and GDP per capita in this country in the period 1985-2010. The World Bank projections entailing a comparison in terms of mortality and morbidity between AIDS and without AIDS configurations, Cuddington and Hancock have also conducted this comparison in more economic terms using such a valuable demographic information (ie. in variables L_{it}, and A_{it}). Indeed, they distinguished between an extreme AIDS scenario and a medium one: In the former scenario, average real GDP

[13] Cuddington and Hancock took $\bar{i} = 15$.

growth over the period 1985-2010 would cost 1.2 to 1.5 percentage points relative to the non-AIDS counter-factual case, while in the latter the figure drops to only 0.2 to 0.3[14]. Concerning annual growth rate of real GDP per capita, the study shows an average depression of 0.25 percentage points through the year 2010. Whatever the defects of the framework used, which does not incorporate (and is not aimed at incorporating) all the economic mechanisms induced by an AIDS-lie epidemic, the results of Cuddington and Hancock have the merit to suggest that the growth effects of AIDS could be indeed sizeable in some African countries. In a posterior paper, Bloom and Mahal (1997) questioned the treatment of Cuddington and Hancock, and the related studies, on the specific ground of the labor market in sub-Saharan countries: for Bloom and Mahal, the presence of a labor surplus could mitigate the output losses that might otherwise be associated with AIDS morbidity and mortality. Apparently, such a criticism does not seem to have a decisive scope, at least in certain countries: In a companion paper, Cuddington (1993) has shown that his results are not that sensitive to the presence of surplus labor in the Tanzanian case.

A more acceptable criticism would be the way productivity, through variable μ_i, is modelled, only relying on experience. Clearly, such a variable should also reflect the education level and other related socioeconomic determinants (like the gender). Also, the time-independence could be questioned in a more long term perspective: With life expectancy dropping from about 60 years to 40, one is tempted to suspect that human capital accumulation would be toughly affected, which would induce a downward trend in productivity *de facto*[15]. We shall amend Cuddington-Hancock's set-up in the last section of the paper to account for some of the numerous missing links one could detect in the benchmark. This is precisely what has made it so popular among the practitioners. Before, we mention briefly the state of the AIDS related empirical literature.

3.4 Empirical evidence

Unlike the Spanish flux epidemic's empirical literature, there is a growing consensus among economists that as far as one is concerned with the long term consequences of AIDS, the growth effects in sub-Saharan countries are

[14]The range is generated by letting ξ and x move from 0 to 1.

[15]Further critiques could be found in Bloom and Mahal (1997).

quite important, and should impulse the necessary international interventions to alleviate the burden of the epidemic. There are still clear quantitative differences between the studies but there are fewer and fewer papers denying the negative growth impact of AIDS. The unique recent paper arguing the contrary is the already quoted Young's (2005) paper. For this author, the impact on fertility will dominate the devastating effects on accumulation, and the AIDS epidemic will enhance the future per capita consumption possibilities (of the South African economy in his case study). One could question the conclusion of this contribution, criticize the basic (and strictly Beckerian) underlying structure and the limitations of the data used, but *in fine*and beside pointing at the fertility channel, Young's paper has the virtue of breaking down the consensus in a fundamentally fragile empirical literature (since it builds on demographics projections established under some specific epidemiological and demographic assumptions).

Ten years ago, the evidence was much more disputed: in a highly influential paper, already quoted above, Bloom and Mahal (1997) found no significant effect of AIDS on the growth rate of per capita income, and no evidence of reverse causality. They used simple cross-section regression models on a sample of 51 countries from 1982 and 1992. The estimated coefficients were found typically small and insignificant. Such result went at odds with the results obtained at the same time on more theoretically founded models à la Cuddington and Hancock, which generally predicted a relatively important growth impact of AIDS. An important reference here is the paper of Over (1992). Over used a relatively sophisticated computable general equilibrium model with three classes of workers, and the distinction between rural and urban production. This ultimately allowed him to study the impact of several AIDS scenarios, based on different assumptions about relative levels of HIV infection in educated Vs uneducated workers...etc...In the most reasonable scenarios (according to Over), the effect in the 10 most affected sub-Saharan countries would be 0.6 percentage point over the period 1990-2025 if all treatment costs were financed from savings.

More recently, some new contributions putting forward the human capital channel have found large effects of AIDS on growth. For example, Corrigan, Glomm and Mendez (2005) have used a calibrated OLG model to analyze particularly the effect of the drop in life expectancy on investment, and the large generation of orphans produced by AIDS. Their results are completely in line with Cuddington, Hancock and Over. McDonald and Roberts (2005)

use an econometric model combining growth and health capital equations. Applied on African countries, the model predicts substantial effects of the epidemic: the marginal impact on income per capita of a one percent increase in HIV prevalence rate is minus 0.59%. The authors conclude that while the human and social costs of the HIV/AIDS epidemic are major causes for concern, their results do indicate that the macroeconomic effects of the epidemic are by no way negligible.

We completely share this view. Hereafter, we show how to extend the framework of Cuddington and Hancock to account for both the dramatic drop in life expectancy, and for the fall in savings induced.

4 An application to AIDS in South Africa

Our starting point is the following: as shown by many authors, the observed fall in life expectancy in most sub-Saharan countries, one of the main consequences of the AIDS pandemic, should have induced a decline in schooling time, in addition to other dramatic effects on savings incentives. Ferreira and Pessoa (2003) find that in the face of an AIDS-like epidemic schooling time can decline by half, which is in our view quite fundamental for long run growth rate. In order to adapt the benchmark Cuddington-Hancock model to account for such crucial features, we introduce the following modifications.

(a) Concerning the production function, we have considered a specification close to (29), that is we incorporate AIDS-related health expenditures in the measurement of productivity, exactly as in Corrigan, Glomm and Mendez (2005). In order to capture the gender-specific characteristics of the epidemics, we also distinguish between males and females (index $f = 1, 2$):

$$E(t) = \sum_{i,f} \left[1 - \phi_{i,f}(t)\ \Phi\left(m_{i,f}(t)\right)\right]\ \mu_{i,f}(t)\ L_{i,f}(t). \tag{34}$$

(b) The productivity variables μ are specified in order to capture the effect of the declining life expectancy on productivity at work. We do this as follows. Notice that with the benchmark specification ({refmui1), the age at which μ_i is maximal is equal to $\bar{i} + 2\ \rho_2\rho_3$. With a declining life expectancy, such a magnitude is likely to go down, and we simply capture

this by endogenizing the coefficient ρ_2 and assuming the following ad-hoc functional relationship:

$$\rho_2(i, f, t) = \alpha_{i,f}\ \nu_{i,f,t}^{\beta},$$

where $\nu_{i,f,t}$ is the life expectancy at birth of workers of age i (thus born at $t-i$) and of gender f. $\alpha_{i,f}$ and β are calibrated[16] . With such a new specification, the age-specific productivities become time and gender dependent via life expectancy:

$$\mu_{i,f,t} = \rho_1 + \rho_2(i, f, t)\ (i - \bar{i}) - \rho_3\ (i - \bar{i})^2, \tag{35}$$

(c) Concerning the saving behavior, we rely on the previous empirical work of Freire who studied in detail in a Blanchard framework how savings are distorted by AIDS in South Africa. This seems to us better than the treatment of Cuddington and Hancock for this particular point. As pointed out by Bloom and Mahal (1997b), it seems reasonable that AIDS related health expenditures will be met by reducing both consumption and savings. As we have mentioned above, Cuddington and Hancock assume that all these expenditures will be met by an exclusive cut in savings.

Findings

The experimental setting is explained in detail in the appendix. In particular, it relies on some well-known forecasting methods developed in Hamilton (1994) and Gouriéroux and Monfort (1990). We provide here a very brief summary of the results[17]. Figures 1 and 2 depict our forecasted GDP and GDP per capita growth for the period 2010-2050. They are themselves based on forecasts per age and gender of some key variables like prevalence rates, saving rates and life expectancy at birth. The most salient ingredient is life expectancy trends: In Figures 5 and 6, one can see that the series of life expectancy reach their trough around 2009, with about 44 years for both sexes, while the starting values are about 60 years. Because we assume by (35) that life expectancy at birth is a determinant of individuals productivity

[16]In particular, the double sequence $\alpha_{i,f}$ is calibrated in such a way that $\rho_2(i, f, t_0) = \alpha_{i,f}\ \nu_{i,f,t_0}^{\beta}$, for β fixed, t_0 is the base year and $\rho_2(i, f, t_0)$ is equal to the constant coefficient ρ_2 considered by Cuddington and Hancock (1994).

[17]The figures are generated with $\beta = \frac{1}{7}$.

at their working age, we get a delayed impact of AIDS, as clearly reflected in Figure 1 and Figure 2 for the growth rates of both GDP and GDP per capita: The gap between the AIDS and no-AIDS scenarios gets larger (though not terrific) around 2020 once the less educated cohorts (due to a presumably much shorter schooling time) enter the labor market.

More precisely, the gap between the AIDS Vs without AIDS cases in terms of growth per capita is around 0.1 percentage point around 2010, and it is almost tripled 10 years later (around 2020). Our figures are lower than those put forward by Over (1992) for the ten most affected sub-Saharan countries for the period 1990-2025. In addition to some obvious experimental differences (coming from the fact that Over uses a computable general equilibrium framework), it should ne noted that we don't consider in our computations that all the treatment costs are taken from savings, so we certainly generate higher forecasts for savings in the AIDS case compared to Over, and thus higher growth in the later case. Another striking finding is that the growth differential between the two scenarios tend to be stable from 2030 to 2050 featuring a kind of long-run effect of AIDS. Of course, such a result is primarily generated by our forecasts for life expectancy, prevalence rates and savings for these horizons. Since the confidence band for our savings forecast in the AIDS case gets markedly larger after 2020 (see Figure 8), this result should be taken with caution.

Incidentally our exercise shows that there might not be any conflict between the results obtained by authors like Bloom and Mahal (1997b), and others like Corrigan, Glomm and Mendez (2005) or McDonald and Roberts (2005): A relatively short term assessment à la Bloom and Mahal might not reveal any dramatic AIDS growth effect, while a long term perspective, relying on the evolution of the determinants of human capital accumulation, might deliver the opposite message. It seems reasonable to think that the observed sharply declining pattern of life expectancy must have delayed effects, and the governments should act from now on to alleviate the expected effects of such a trend.

5 Conclusion

In this paper, we have provided with a general growth theory-oriented approach to epidemics. The use of one-sector Vs two-sector models, incorporating human capital accumulation, to deal with this issue is not neutral at

all, and we have shown that the implications in terms of transitory dynamics and the role of population growth are indeed reversed from a framework to another. We do believe that Lucas-Uzawa model are the most acceptable, but the basic specifications of such a model are clearly insufficient to measure correctly the scope of an AIDS-like crisis. We have provided some refinements of the Blanchard model to incorporate the missing channels, notably those connected with life expectancy and health expenditures. A kind of synthesis between Blanchard life cycle structures and Lucas-Uzawa technological specifications, enriched with the incorporation of health expenditures effects on savings and productivity, would be probably the best and most comprehensive set-up to tackle properly all the growth implications of AIDS. Much of the work remains to be done.

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Experimental setting

In this section, we present the forecasting and the projection of HIV prevalence. The forecasting needed first the estimation of the parameters of an assumed model, that is the assumed underlying data generating process. For this purpose, we retain a Gaussian $ARMA(p,q)$ process for with we describe below the estimation and the forecasting procedure. Further details on these statistical methods can be found in Hamilton (1994) and Gouriéroux and Monfort (1990).

Methodology

Conditional likelihood estimation

A Gaussian $ARMA(p,q)$ process is described as

$$\begin{aligned} Y_t = \alpha &+ \phi_1 Y_{t-1} + \phi_2 Y_{t-2} + \cdots + \phi_p Y_{t-p} + u_t \\ &+ \theta_1 u_{t-1} + \theta_2 u_{t-2} + \cdots + \theta_q u_{t-q} \qquad t = 1, \cdots, T \end{aligned} \tag{36}$$

where $u_t \sim$ i.i.d $N(0, \sigma^2)$, and where the vector of population parameters $\boldsymbol{\theta} = (\alpha, \phi_1, \phi_2, \cdots, \phi_p, \theta_1, \theta_2, \cdots, \theta_q, \sigma^2)'$ is to be estimated. These estimates will then be used to set the forecasting. The approximation to the likelihood function is conditioned on both initial values of the $y's$ and $u's$.

Assuming that the initial values for $\boldsymbol{y}_0 \equiv (y_0, y_{-1}, \cdots, y_{-p+1})'$ and $\boldsymbol{u}_0 \equiv (u_0, u_{-1}, \cdots, u_{-p+1})'$ are given, the sequence $\{u_1, u_2, \cdots, u_T\}$ can be computed from $\{y_1, y_2, \cdots, y_T\}$ by iterating on

$$\begin{aligned} u_t = y_t &- \alpha - \phi_1 y_{t-1} - \phi_2 y_{t-2} - \cdots - \phi_p y_{t-p} \\ &- \theta_1 u_{t-1} - \theta_2 u_{t-2} - \cdots - \theta_q u_{t-q} \qquad t = 1, \cdots, T \end{aligned} \tag{37}$$

The conditional log likelihood is given by

$$\begin{aligned} L(\boldsymbol{\theta}) &= \ln f_{Y_T, Y_{T-1}, \cdots, Y_1 | \boldsymbol{Y}_0, \boldsymbol{u}_0}(y_T, y_{T-1}, \cdots, y_1 | \boldsymbol{y}_0, \boldsymbol{u}_0; \boldsymbol{\theta}) \\ &= -\frac{T}{2}\ln(2\pi) - \frac{T}{2}\ln(\sigma^2) - \sum_{t=1}^{T} \frac{u_t^2}{2\sigma^2} \end{aligned} \tag{38}$$

In maximizing this log likelihood, we set the initial $y's$ and $u's$ to their expected values. That is $y_s = \alpha/(1-\phi_1-\phi_2-\cdots-\phi_p)$ for $s = 0, -1, \cdots, -p+1$, and $u_s = 0$ for $s = 0, -1, \cdots, -p+1$. Then, we proceed with iteration

in (37) for $t = 1, \cdots, T$. We estimate a multiple $ARMA(p, q)$ model with $p = 2$ and $q = 2$ which turns out to estimate six models, and then select the model that optimize the Schwarz Criterion. The selected model is used for forecasting purpose.

Forecasting

Now, consider forecasting the stationary and invertible $ARMA(p, q)$:

$$(1 - \phi_1 L - \phi_2 L^2 - \cdots - \phi_p L^p)(Y_t - \mu) = (1 + \theta_1 L + \theta_2 L^2 + \cdots + \theta_q L^q)u_t \quad (39)$$

where L is the lag operator and μ is the unconditional mean $E(Y_t)$. The one-period-ahead forecast ($s = 1$) is given by

$$\begin{aligned}(\hat{Y}_{t+1|t} - \mu) = \quad & \phi_1 \quad (Y_t - \mu) + \phi_2(Y_{t-1} - \mu) + \cdots \\ & + \quad \phi_p(Y_{t-p+1} - \mu) + \theta_1 \hat{u}_t + \theta_2 \hat{u}_{t-1} + \cdots + \theta_q \hat{u}_{t-q+1} \quad (40)\end{aligned}$$

with $\hat{u}$ generated recursively from $\hat{u} = Y_t - \hat{Y}_{t|t-1}$. Finally the s-period-ahead forecasts based on the *Wiener-Kolmogorov prediction formula* is

$$(\hat{Y}_{t+s|t} - \mu) = \begin{cases} \phi_1(\hat{Y}_{t+s-1|t} - \mu) + \cdots + \phi_p(\hat{Y}_{t+s-p|t} - \mu) + \theta_s \hat{u}_t + \cdots + \theta_q \hat{u}_{t+s-q} & \text{for } s = 1, \cdots, \\ \phi_1(\hat{Y}_{t+s-1|t} - \mu) + \cdots + \phi_p(\hat{Y}_{t+s-p|t} - \mu) & \text{for } s = q+1, \end{cases}$$

where $\hat{Y}_{\tau|t} = Y_\tau$ for $\tau \leq t$.

Projection of HIV prevalence

The HIV prevalence series take over 1980-2015. To compute the projected HIV prevalence from 2016-2050, one needs a guideline to chose a suitable function. The plot of the HIV prevalence is displayed on figure (3). The prevalence shows evidence of a decline.
As a result, to obtain the projected prevalence, we fit a double logistic curve of the form

$$p(t) = \left[\frac{e^{\alpha(t-\tau)}}{1 + e^{\alpha(t-\tau)}}\right]\left[\frac{ae^{-\beta(t-\tau)}}{1 + e^{\beta(t-\tau)}} + b\right] \quad (41)$$

where α is the rate of increase at the start of the epidemic, a denotes de peak value, β is the rate of convergence, b is the final prevalence level and τ shifts the whole curve backward. The value of α is chosen so that the doubling time is 1.5 years. That is value of α is chosen so that the doubling time at the

beginning of the epidemic can be $\ln(2)/\alpha$. This means that $\alpha = \ln(2)/1.5$. As a result, for a given β, we have to find (numerically) the parameters a, b and τ solution of the non linear system

$$\mathcal{S}(a,b,\tau) = \begin{cases} p(0) = 0.1916 = \frac{e^{-\alpha\tau}}{1+e^{-\alpha\tau}}\left[\frac{ae^{\beta\tau}}{1+e^{\beta\tau}} + b\right] \\ p(2015) = 0.194 = \frac{e^{\alpha(15-\tau)}}{1+e^{\alpha(15-\tau)}}\left[\frac{ae^{-\beta(15-\tau)}}{1+e^{-\beta(15-\tau)}} + b\right] \\ \dot{p}(t) = 0 \Longleftrightarrow \frac{\alpha}{e^{\alpha(t-\tau)}}\left[\frac{ae^{-\beta(t-\tau)}}{1+e^{-\beta(t-\tau)}} + b\right] = \frac{a\beta e^{-\beta(t-\tau)}}{\left[1+e^{-\beta(t-\tau)}\right]^2} \quad \text{where } t = 3. \end{cases}$$

The first equation of the system corresponds to the starting period ($t = 0$ for year 2000 where the prevalence is 0.1916), the second denotes end period ($t = 16$ for year 2015 where the prevalence is 0.194) and the third equation expresses the peak of the epidemic. The value $t = 3$ represents the difference between the peak year 2003 and the starting date 2000.

Application

Data

Economically active population Estimates and Projections, by age and sex (1980-2020): International Labor Organization.

The database contains world, regional and country estimates and projections of the total population, the activity rates and the economically active population (labour force) by sex and five-year age groups (from 10 to 64 years and 65 years and over). These estimates and projections are for international comparisons and are neither superior nor necessarily inferior to national estimates and projections, which are produced using country-specific additional information. The economically active population comprises all persons of either sex who furnish the supply of labour for the production of goods and services during a specified time-reference period.

HIV-seroprevalence (percent, 1980-2015): U.S. Census Bureau, International Programs Center.

Estimated HIV adult prevalence trends from 1980 to 2015. These estimates were derived from the Epidemic Projection Package, an epidemiologically sound computer model that allows for a "best fit" of HIV prevalence data from antenatal clinic women who come in for their first antenatal visit.

The HIV prevalence is defined as the percentage of women surveyed testing positive for HIV. Each year a national survey of HIV prevalence among women attending public antenatal clinics in South Africa is conducted by the Department of Health. The Annual HIV antenatal survey provides South Africa with annual HIV trends among pregnant women and further provides the basis for making other estimates and projections on HIV/AIDS trends.

HIV$^+$ (1990-2015): The Demographic impact of HIV/AIDS in South Africa: National Indicators for 2004. This indicator is defined as the number of people infected.

Life expectancy at birth by age and sex (1920-2060): World Development Indicators, Health Nutrition and population and League of Nations, Northwestern University. This is the number of years that a new born could live if the normal conditions of mortality at his birth should be the same ones throughout its life.

Percentage expenditure of AIDS in the total health expenditure: South African Budget Review, 2003/04 and Estimates of National Expenditure, 2003.

Total health expenditure: Health Nutrition and Population.
GDP per capita (1960-2000): Penn World Table 6.1.
Unemployed by age and sex (2000-2003): International Labor Organization.

The series on unemployment shown here relate in principle to the entire geographical area of a country. In 1982, the Thirteenth International Conference of Labour Statisticians adopted a new Resolution concerning Statistics of the Economically Active Population, Employment, Unemployment and Underemployment, in which the definition of unemployment is revised. The new definition is to a large extent similar to the earlier definition adopted by the Eighth Conference. It, however, introduces certain amplifications and modifications concerning, in particular, the criteria of seeking work and current availability for work, the statistical treatment of persons temporary laid off, persons currently available for work but not actively seeking work, etc. The changes are aimed to make it possible to measure unemployment more accurately and more meaningfully both in developed and developing countries.

Saving rates with and without AIDS: Freire (2002).

Prevalence and selected forecasting results

We now present the results of experiment. Regarding forecasting, we do not present all of them, since several series are forecasted in order to obtained series until 2050. As a result we only give some of them, others are based on the same methodology previously described and can be obtained from the authors on request. To obtain HIV prevalence projections, we first solve numerically[18] the non linear system given by $\mathcal{S}(a, b, \tau)$ for a, b and τ. We obtain $a = 0.4045$, $b = 0,0003$ and $\tau = -9.356$. These values are then plugged in relation (41) to determines HIV prevalence from 2016 to 2050. The result is provided in Figure (4). There is a clear evidence for decline in the HIV prevalence.
Now we chose to present the result of estimation and forecasting only for life expectancy at birth with AIDS for female and male, for saving without and with AIDS, and for GDP. For these series, the Schwarz Criterion is optimized by and $ARMA(2, 0)$ for life expectancy, and an $ARMA(1, 1)$ both for saving and GDP. Then, for example, the forecast of GDP at 2000 is used to compute the initial capital in the case without AIDS.

The forecasting for life expectancy with AIDS for female (Figure 5) and male (Figure 6), for saving (without and with AIDS, Figures 7 and 8 respectively) and those for GDP without AIDS (Figure 9). The figures display both median forecast and the density estimate of the forecasted values.

[18]Implemented using GAUSS 6

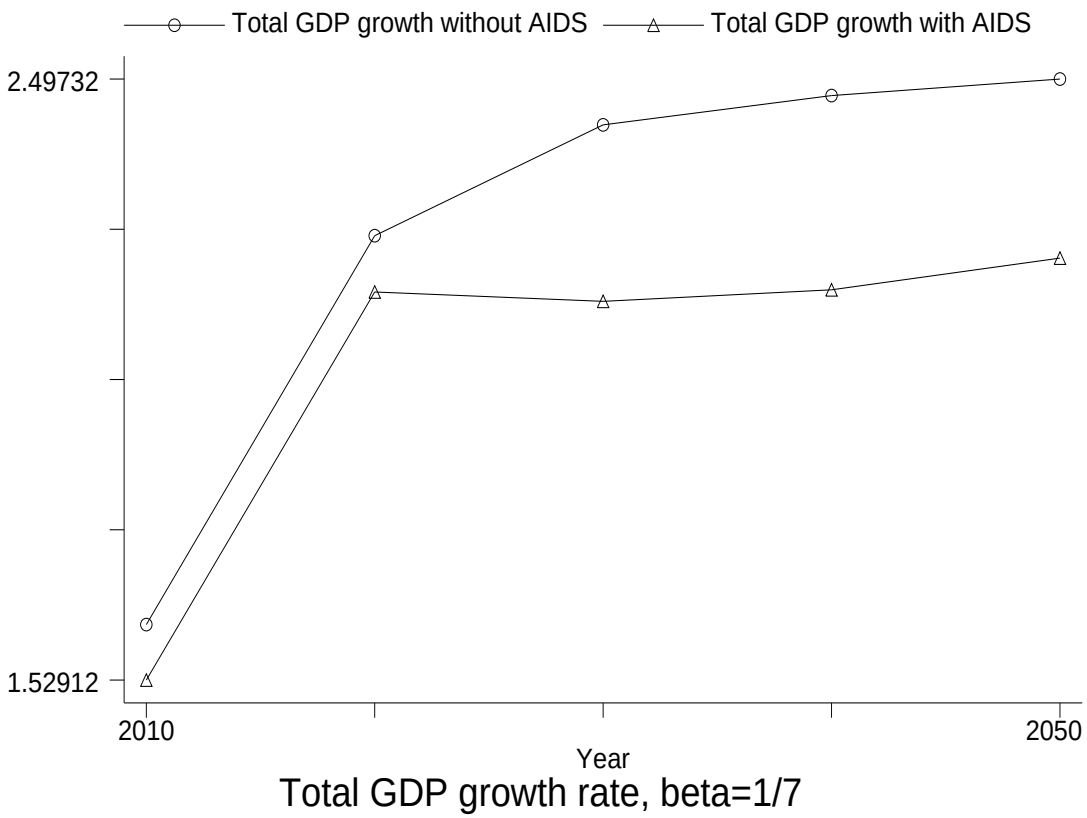

Figure 1: GDP growth rate

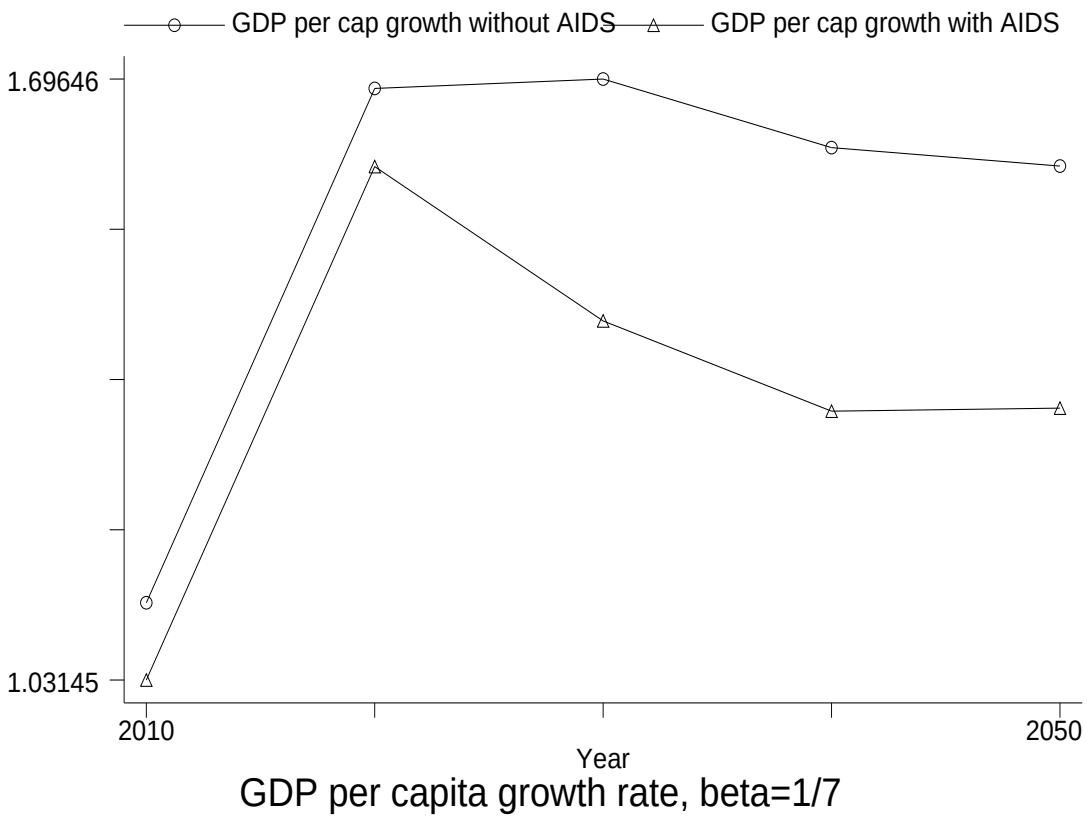

Figure 2: GDP per capita growth rate

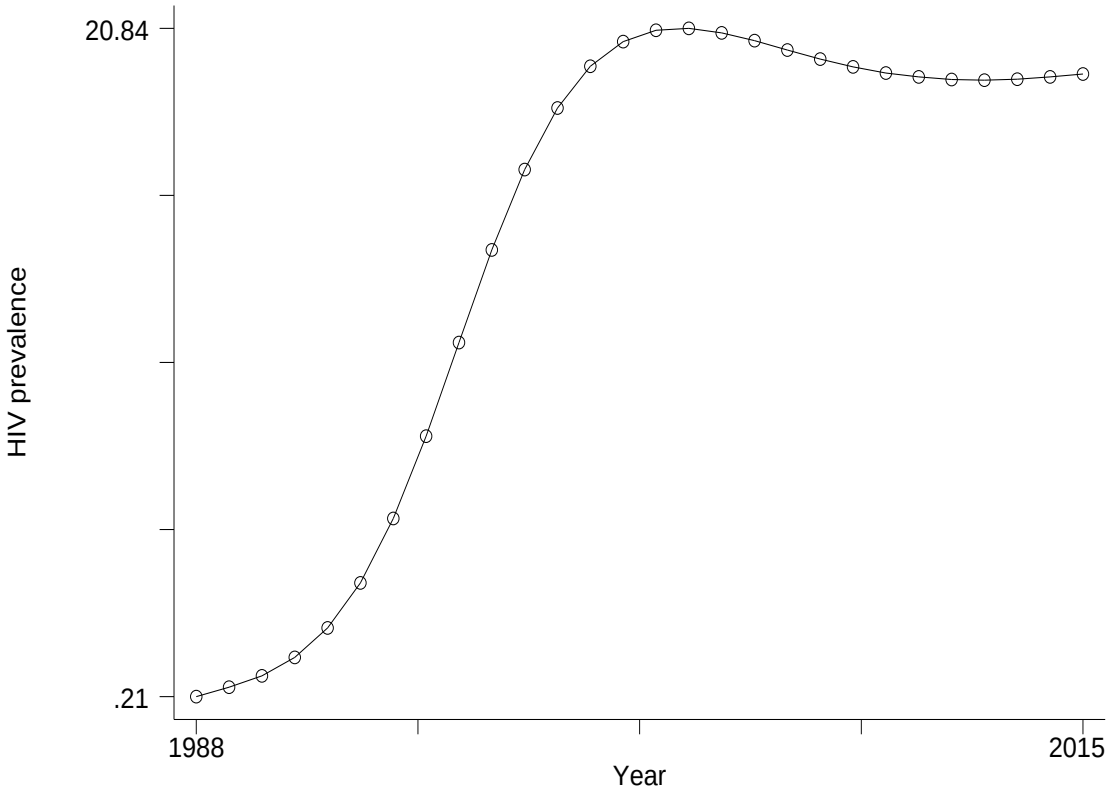

Figure 3: HIV prevalence in percent 1988-2015.

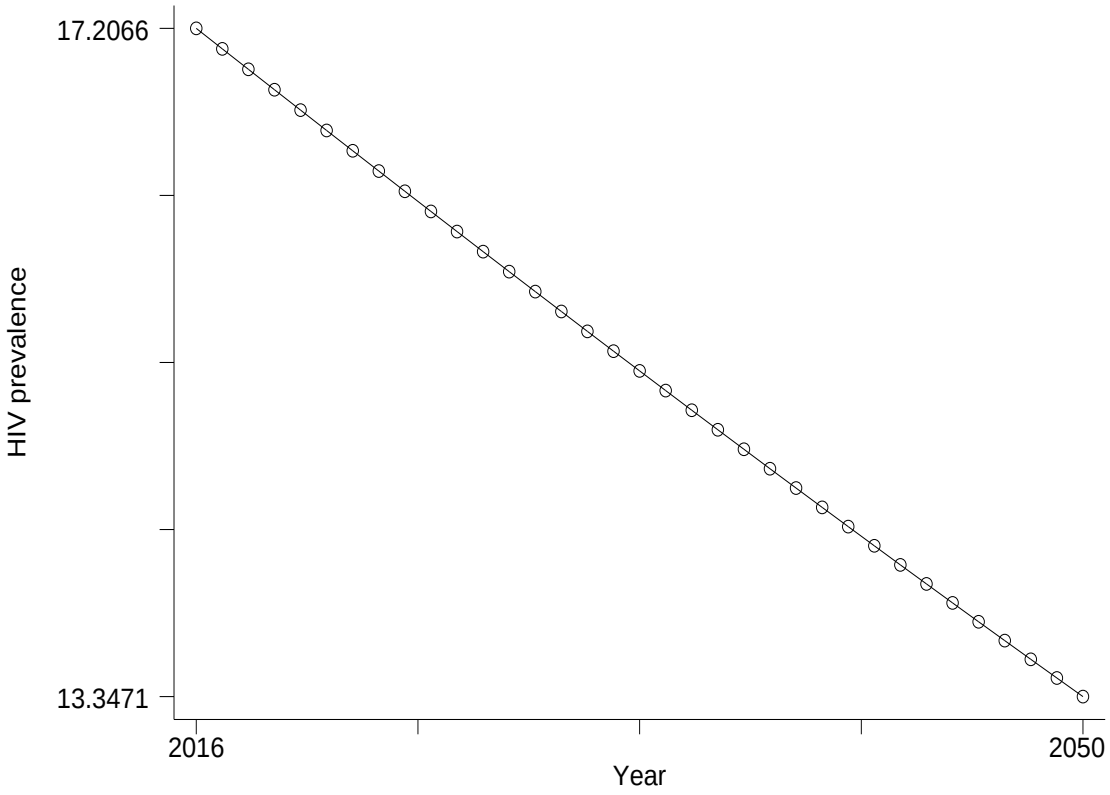

Figure 4: HIV prevalence 2016-2050.

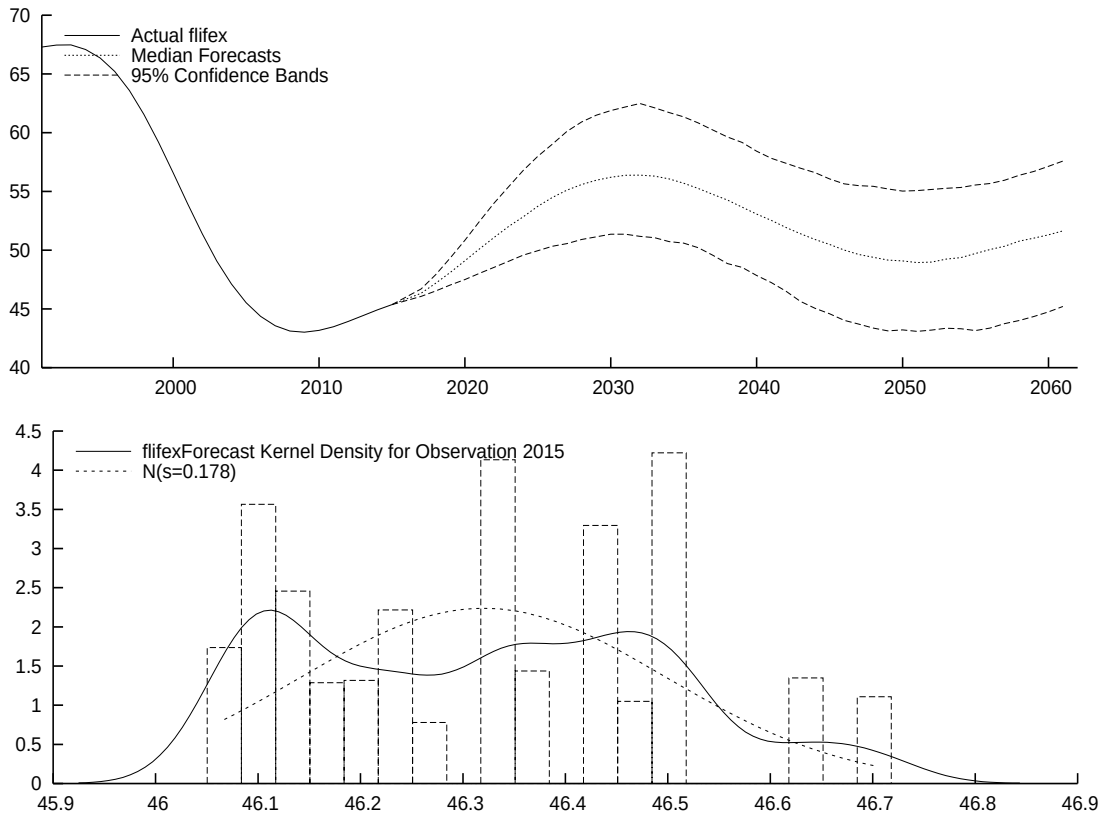

Figure 5: Forecasting of life expectancy at birth for female.

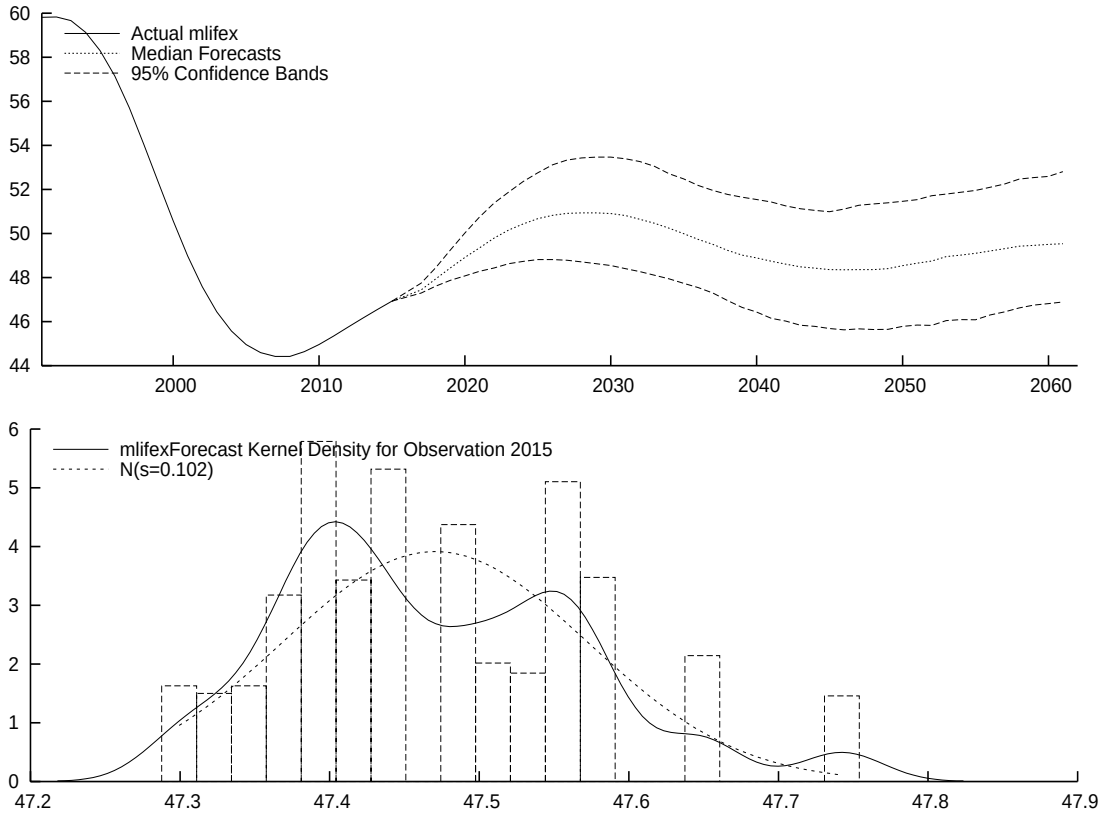

Figure 6: Forecasting of life expectancy at birth for male.

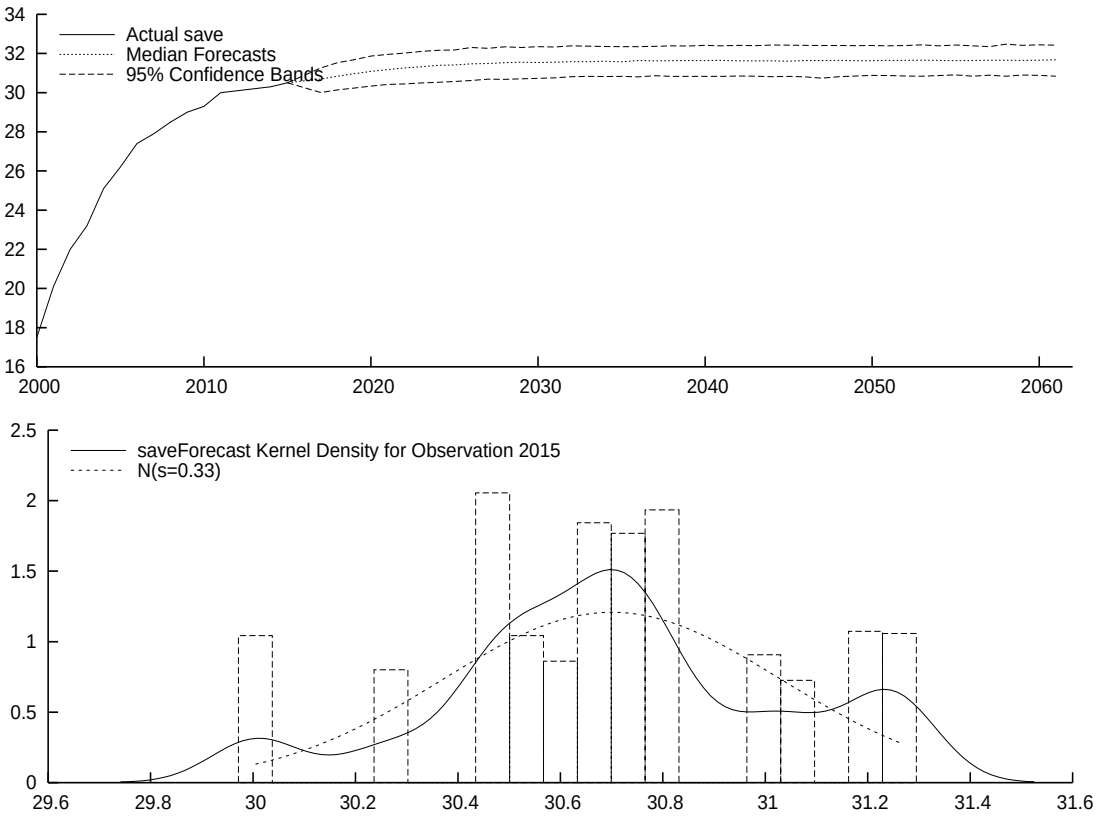

Figure 7: Forecasting of saving without AIDS based on Freire (2002).

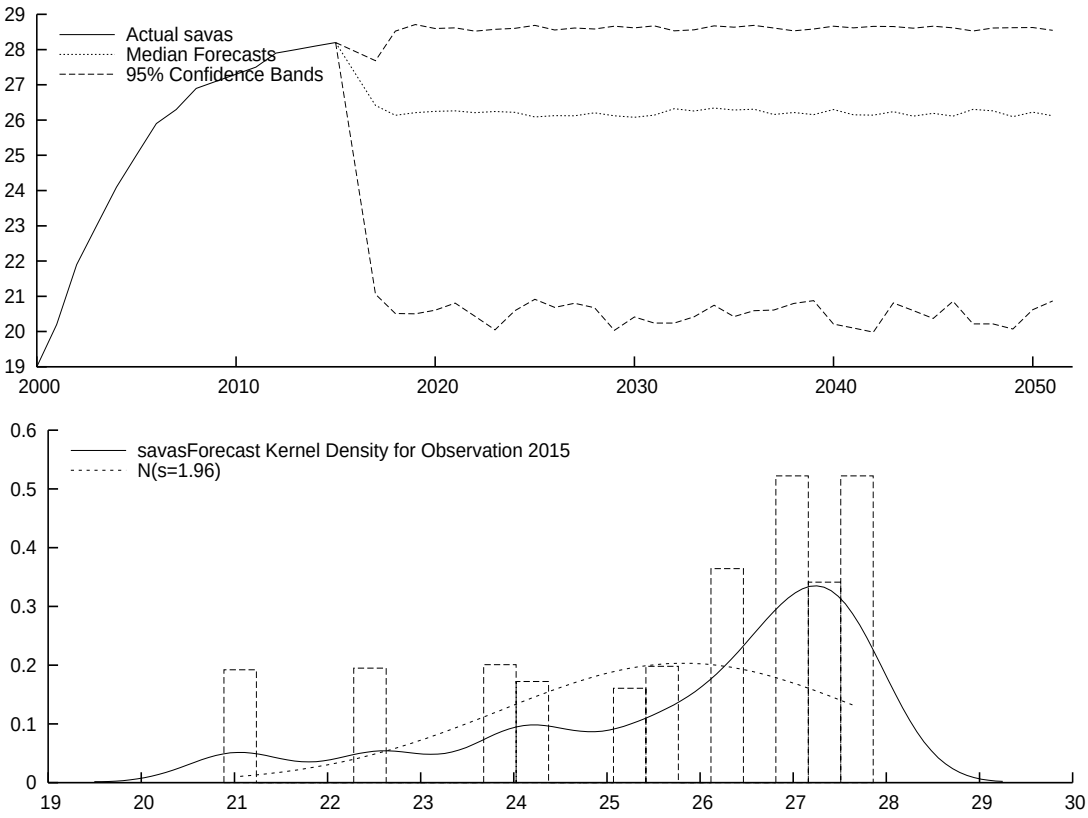

Figure 8: Forecasting of saving with AIDS based on Freire (2002).

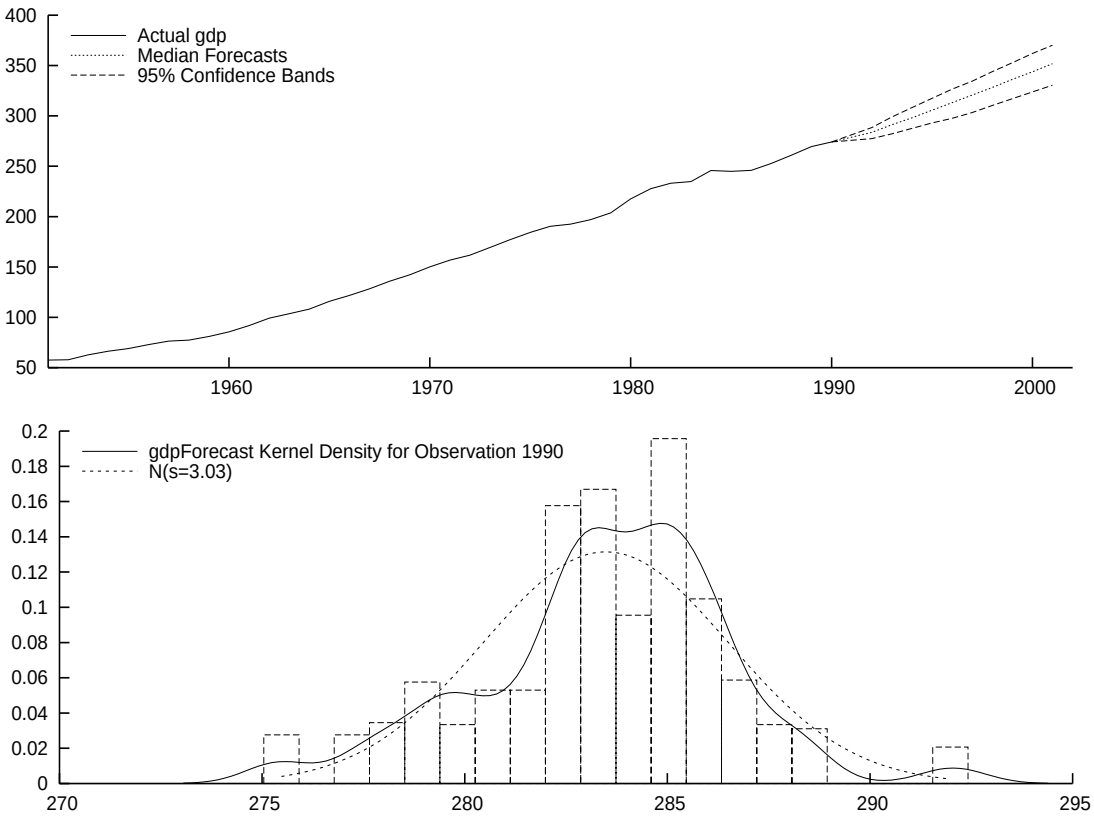

Figure 9: Forecasting of GDP without AIDS.

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