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HIV/AIDS IN PREGNANCY PROCESS AND ENDOGENOUS LIFE FLUCTUATION IN LEAST DEVELOPED ECONOMICS

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ABSTRACT: *this article studies transmission risk from mother to child during the pregnancy process where the immune system acts like health defense in an endogenous growth model with epidemiological literature. When the transmission risk becomes positive, the system defense also increases in order to stop the pandemic propagation. Since a given level is crossed, fixed points are ruled out, then cycles and chaos arise, thus makes population growth tends to zero. The social planner's intervention in order to ensure population growth reaches the steady state defines a unique optimal path where per-capital capital must be monotonic and increasing in development economics. The application of the theory shows the existence of a stable locus where the gap between lives fluctuations and the steady state tends to zero. Whereas diseases or epidemiological studies are mostly related to economic variable in growth studies, this article explicitly models the medical system action in presence of infection and acts such as a disease fight power mechanics for health recovery in order to contribute to the debate on population growth.*

KEYWORDS: Epidemiology, Infectious diseases, Growth, Spectrum Convergence, Chaos, Cycles, optimal steady state

JEL Classification: E32, I10, E63, E13, D90, O11, O44

INTRODUCTION

This article is based on the mathematical tools used by Goenka-Liu (2012) and also introduces the epidemiological literature in a one sector growth model but through the modeling of HIV/AIDS disease transmission from mother to child during the pregnancy process in order to capture the existence of the stable steady state in contrast to the basic model where the infectious disease is more general. The logistic map conjugates topologically to the disease dynamics in order to determinate the link between the mother and the baby as well as the pregnancies dynamics which are able to ensure a healthy new born. The disease gravity depends on the parameter of risk transmission, α which is associated to the immune system fight capability against the disease expressed by the parameter γ. Since $\alpha<0$ and $\gamma<1$ and / or $\gamma<\alpha\leq\gamma+2$, the gap of life dynamics and the steady state is almost zero. In contrast since $\alpha>0$, $\gamma>1$ and $\alpha>\gamma+2$, cycles and chaos arise from the structure of lives dynamics. The social planner's program unifies growth analysis to the epidemiology literature and determines a policy which reduces the fluctuations in lives dynamics around the steady state through a monotonic increase of per-capita capital or medical care support and

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yields fixed points existence. Finally, the application of the theory highlights the existence of a unique stable solution.

While disease in economic models are mainly focused on its impact on GDP (Kambou, Devarajan and Over, 1992) or on population growth (Theodore, 2001, Young, 2005) as well as on Human capital (Oster et al, 2013) and mostly found an opposite relation between economic growth and epidemiologic literatures (Goenka –Liu, 2012) and Malthus (1798) theory mostly forms the basis of discussions even extended after while economic variables still to explain disease, the feature of this article holds on the explicit modeling of medical mechanics matters in growth absence explanation i.e in population fluctuations. We highlight the existence of immune system mechanics on which medical care must act in life maintenance target and play a great role on health recovery faculty. Thus the model closely related medical tools to financial support where diseases are not only caused by economic limitations and its efficiency to explain the stability of the sustainability system. Moreover, since disease are not only the outcomes of economic shortages but comes mostly from information lack on medical interactions, the model provides tools to study the convergence of life dynamics to its steady state in contrast to all the models quoted because economic tools alone are unable to create life, thus disease meets natural resistance of the body and creates fluctuations despite of the economic system ex-ante and related to it ex-post with medical care function of production which can capture endogenously the situation in order to make population growth tends towards its optimal locus. Indeed, the model is not limited to growth and economic variables faculties only it is deeper than that and shows the existence of hide mechanics able to explain population growth fluctuations.

The human immunodeficiency virus (HIV) pandemic is one of the most serious health crisis the world poor is facing today. AIDS has killed more than 25 million people since 1981 and an estimated 38.6 million people are now living with HIV, about 2.3 million of whom are children *(2006 Report on the global AIDS epidemic.* Geneva, UNAIDS, 2006*)*. Since 1999, primarily as a result of HIV, average life expectancy has declined in 38 countries. In the most severely affected countries, average life expectancy is now 49 years – 13 years less than in the absence of AIDS *(Questions & Answers (November 2005)*, Geneva, UNAIDS, 2005*)*. A disproportionate burden has been placed on women and children, who in many settings continue to experience high rates of new HIV infections and of HIV-related illness and death. In 2005 alone, an estimated 540 000 children were newly infected with HIV, with about 90% of these infections occurring in sub-Saharan Africa. Epidemics of infectious diseases have led to the study of their impact on the economy first and by modeling the disease transmission explicitly like in the epidemiology literature, this paper studies the effect of HIV/AIDS transmission risk on life dynamics of the babies during the pregnancy process of their mothers, by modeling the dynamics of disease transmission, new insights on their effects emerge. We show that varying parameters and looking at steady states can be misleading as the disease dynamics are a source of non-linearity, then the infectivity of the disease increases the nature of steady states change and endogenous fluctuations can emerge.

There is a growing empirical literature on the effects of infectious diseases on economic variables. This literature tries to measure the effect of diseases on economic growth (Bell et al. (2003, 2004), Cuddington (1993a and 1993b), Cuddington and Hancock (1994)). Some papers find the effect of control of diseases to be large (Bloom et al. 2009), while others find the effect is modest (Ashraf et al. 2009) or there might even be an adverse effect due to the

dilution effect of a larger population and increase in dependency ratio (Acemoglu and Robinson 2007; Young 2005). The underlying theoretical models in these papers largely look at steady state behavior with a fixed savings rate and exogenous labor supply (Thirumurthy et al. 2007) to change in response to changes in disease incidence. Those papers do not simultaneously model both capital accumulation and the epidemiological structure of the diseases. One of the key insights of the epidemiology literature is that variations in infectivity change the dynamic properties of diseases. Thus, because it is not sufficient to know how steady states change in the economic model as the dynamic properties of the economy may not be invariant to changes in the disease incidence, Goenka and Liu (2012) show that the incidence of disease has level effects and also cause economic fluctuations and model the disease transmission explicitly through the epidemiology literature (see Anderson and May 1991; Hethcote, 2000) into dynamic economic analysis to examine the effect of the canonical epidemiological structure for recurring diseases—*SIS* dynamics—in a discrete time growth model. *SIS* dynamics characterize diseases where upon recovery from the disease there is no subsequent immunity to the disease. This covers many major infectious diseases such as tuberculosis, malaria, dengue, schistosomiasis, trypanosomiasis (human sleeping sickness), typhoid, meningitis, pneumonia, diarrhoea, acute haemorrhagic conjunctivitis, strep throat and sexually transmitted diseases (STD) such as gonorrhea, syphilis (see Anderson and May 1991) and HIV/AIDS (Loubaki, 2012a,b,c; Young, 2005) etc.. Infectious diseases affect the economy mainly through three channels: labor productivity (Thirumurthy et al. 2007; Weil, 2007), human capital accumulation (Bell et al. 2003; Bleakley, 2007) and population size (Kalemli et al. 2000; Young, 2005). A decrease in the first two will have adverse effects on economic outcomes, but a decrease in the population size may have a positive effect contingent on the dependency ratio through increases in capital per capita. For the diseases mentioned above the major impact is making infected individuals ill and reducing labor productivity. For several of these, disease related mortality is low for adults.

The article is organized like follow section2 presents the epidemiological literature and some results highlighted by this structure in terms of lives dynamics, section3 presents the economic environment, section4 introduces the epidemiological literature in the one sector growth model and studies the equilibrium dynamics, section5 applies the theory to the concrete case and section6 concludes on the analysis.

DISEASE TRANSMISSION DYNAMICS

We abstract away from all the demographics in the model and assume the population consists of a continuum of individuals of mass *N*. The total size of the population is categorized into two classes: the healthy pregnant women and the infective HIV/AIDS pregnant women. Let H_t and I_t denote the number of the healthy and the infective pregnant women respectively, and $h_t=H_t/N$, $i_t=I_t/N$ denote the per-capita healthy and infective pregnant women where $h_t + i_t = 1$. Let α be the transmission risk of HIV/AIDS to the future baby and γ be the percentage of chance for the baby to remain healthy. Since the healthy future mother becomes infected, then $\gamma I_t(\alpha b_t^i)$ is the number of new cases for which the baby b_t^i may be infected at the probability rate α and/ or remains healthy at the rate γ whereas, $(1-\alpha)$ is the fraction of healthy babies , thus b_t^h is per-capita stock of healthy baby even from the already infected mothers which yields $\gamma I_t(1-\alpha)b_t^h$ cases for which the baby is not infected recalling that b_t^i is per-capita

infected babies' stock and b_t^h is per-capita healthy babies' stock. Therefore, the dynamics of the mothers in connection with their babies are given by the following difference equations:

$$H_{t+1} = H_t - \gamma I_t(\alpha b_t^i) + \gamma I_t(1-\alpha) b_t^h \quad (1)$$

$$I_{t+1} = I_t - \gamma I_t((1-\alpha) b_t^h) + \gamma I_t \alpha b_t^i \quad (2)$$

Where I_t , $H_t \geq 0$ given.
Since $H_t + I_t = N$, one of the above equations is redundant. Therefore, we can express the HIV/AIDS prevalence and transmission risk in terms of healthy pregnant women stock evolution only which simplifies the dynamics to:

$$H_{t+1} = H_t - \gamma I_t(\alpha(1-b_t^h)) + \gamma I_t(1-\alpha) b_t^h \quad (3)$$

Therefore per-capita pregnant woman law of motion of health evolution can be expressed by equation (4) i.e

$$h_{t+1} = h_t + \gamma(b_t^h - \alpha)(1-h_t) \quad (4)$$

Where $h_{t+1} = g(h_t, b_t^h)$
If $b_t^h = \alpha$, all the babies are infected, indeed $b_t^h = 0$ and $h_{t+1} = h_t$, population in growth and level terms converges to 0 in the long run as the dynamic of population depends on pregnant women positively related to the babies' stock and the increase of the parameter α decreases the stock of babies and lead women in pregnancy to death.

Lemma 1 $\forall$ h_0 , $b_0^h \in [0, 1]$ *it yields* $0 \leq h_t \leq 1$ *and* $0 \leq b_t^h \leq 1$

Proof: showing $0 < h_t \leq 1$ *and* $0 < b_t^h \leq 1$ *where* $0 \leq \alpha \leq \gamma$ *and* $0 < \gamma < 1$ $\forall t$, is equivalent to prove - $\gamma\alpha \leq g(h_t, b_t^h) \leq 1$, $\forall$ h_t , b_t^h $[0,\alpha] \cup [\alpha,1]$. We notice that $g(0, b_t^h) = \gamma(b_t^h - \alpha)$ and $g(h_t, 0) = (1+\gamma\alpha)h_t - \gamma\alpha$, $\frac{\partial g}{\partial h_t} = 0 \Rightarrow b_t^{h*} = \alpha + 1/\gamma > 0$ and $\frac{\partial g}{\partial b_t^h} = 0 \Rightarrow h_t^* = 1$ therefore $g(1,0) > 0$ and $g(1, b^{h*}) \leq 1$, thus $g^{max} = 1$. In parallel, $g(0, \alpha + 1/\gamma) > 0$ and $g(1, \alpha + 1/\gamma) \leq 1$ thus $g^{max} = 1$. Consequently, $0 \leq g(,) \leq 1$, thus $0 \leq h_t \leq 1$ *and* $0 \leq b_t^h \leq 1$.

Assumption1*:* $b^h = \alpha + \frac{(2\gamma - \alpha)}{\gamma} - h = b^h(h)$

Lemma 2 *For* $b^h = b^h(h)$*, the mapping g is topologically conjugate to the logistic map* $L(h_t) = \mu h_t(1-h_t)$ *with* $0 \leq \mu \leq 4$

PROOF: define $\mu = 1 + \alpha - \gamma$ and the map $\psi(h) = (\gamma/\mu)(1-h)$, ψ is a homeomorphism[3]. and we can verify that $L \circ \psi = \psi \circ g$

$$L \circ \Psi = (1+\alpha-\gamma)\frac{\gamma}{1+\alpha-\gamma}(1-h)\left[1 - \frac{\gamma}{1+\alpha-\gamma}h\right] = \gamma\left(1 - \frac{\gamma(1-h)}{1+\alpha-\gamma}\right)(1-h)$$

[3] Given topological spaces X and Y and a bijective application on them, f , then there is a double continuity of f: X→Y and f^{-1} Y→X

$\Psi \circ g = \frac{\gamma}{1+\alpha-\gamma}(1-h-\gamma(b^h-\alpha)(1-h)) = \gamma\left[1-\frac{\gamma(1-h)}{1+\alpha-\gamma}\right](1-h)$ if the mother and the baby joint lives dynamics is expressed such that $b^h-\alpha=\frac{(2\gamma-\alpha)}{\gamma}-h$, thus, $L\circ\psi=\psi\circ g$ *indeed* g and L are topologically conjugate with $\mu=1+\alpha-\gamma$. In addition, for $0\leq\mu\leq4$, the mapping L lies entirely in the interval [0,1], which is consistent with h_t *and* b_t^h [0, 1]. As we know, mappings that are topologically conjugate are completely equivalent in terms of their dynamics. Hence, we can deduce the joint dynamics of h_t and b_t^h i.e g in terms of the parameters α, γ from the well-examined logistic map, as shown in Table 1, with $\mu=1+\alpha-\gamma$, the map has both stable and unstable orbits, and in Table 1 the stable orbits are reported.

Table 1 The dynamics of the logistic map

μ	Attractors
$0 \leq \mu \leq 1$	$x^* = 0$
$1 < \mu \leq 3$	$x^* = \frac{\mu-1}{\mu}$
$3 < \mu \leq 1+\sqrt{6}$	$x_t^* = \frac{\mu+1+\sqrt{(\mu-3)(\mu+1)}}{2\mu}, x_{t+1}^* = \frac{\mu+1-\sqrt{(\mu-3)(\mu+1)}}{2\mu}$
$1+\sqrt{6} < \mu \leq \mu_\infty$	Cycles of period $2^r, r = 2, 3, 4, \ldots$
$\mu_\infty < \mu \leq 4$	Chaotic attractor

μ_∞ is the accumulation point of cycles of period 2^r $(r = 2, 3, 4, \ldots)$ and $\mu_\infty = 3.57\ldots$. Source: Weisstein (2009)

Proposition1 *per-capita mother dynamics can be expressed by equation (6) i.e:*

$$h_{t+1} = h_t + [(2\gamma-\alpha)-\gamma h_t](1-h_t) \tag{6}$$

Proof: replacing $b_t^h + h_t = \alpha + \frac{(2\gamma-\alpha)}{\gamma}$ inside equation (4) determined fully the law of motion of the mothers dynamics expressed by equation (6).

Corollary 1*: according to proposition 1, the baby's disease dynamics is defined by*

$$b_{t+1}^h = \alpha + \frac{(2\gamma-\alpha)}{\gamma} - h_{t+1}$$

Proof: introducing equation (6) in the joint dynamics expression, $b_t^h + h_t = \alpha + \frac{(2\gamma-\alpha)}{\gamma}$, we get the expression of the babies' dynamics in function of their mother law of motion of life dynamics. The babies' dynamics can thus, be located in reference to their mothers' dynamics through the parameters α and γ.

Assumption 2: according to assumption1, in order for the transmission risk to be low, we must have $\gamma<1$ for $\alpha<0$ to hold, otherwise since $\gamma\geq1$ it yields $\alpha\geq0$ and may become too high making the sum of the mothers and the babies stock becoming negative

Definition1: *f* is a stages function if there exist a subdivision $\Lambda=\{z_0 , z_1 ,\ldots, z_n \}$ such that for each $x \in]z_i , z_{i+1}[_{j\geq 0}$, *f* is a positive and increasing function

Lemma 3: *when $\gamma<1$ and $\alpha<0$, the system converges to the stable steady state, otherwise when $\gamma\geq 1$ the system becomes unstable in order to resist to the attacks of the infection highlights by the positivity of HIV/AIDS in the immune system expressed by the risk parameter, $\alpha>0$. Then the system describes several stages i.e cycles and chaos arise expressing the fight of the body for both health maintenance and human reproduction worsened by medical care shortages in Sub-Saharan Africa.*

Proposition 2: *a given disease transmission risk, $\alpha(\gamma)$ defines a gap G between the lives dynamics of the mothers and the babies, $G=,b_{t+1}^{h} - h_{t+1}$ such that (h_{t+1} ,b_{t+1}^{h}) converge to some locus around the steady state, indeed:*
If $\alpha\leq\gamma<1$, there exits $G>0$,such that (h_{t+1} ,b_{t+1}^{h}) converges to $(1, \gamma)$, then because $\gamma<1$, $\alpha<0$, the path is optimal otherwise if $\gamma>1$, α is too high and the sum of the mothers and the babies stock becomes negative.
If $\gamma<\alpha\leq\gamma+2$, the steady state where $G=0$ is reached at some point in the area, (h_{t+1} ,b_{t+1}^{h}) converges to $(1-2/\gamma, 1+\gamma)$ just before the mother reaches the HIV state i.e when $\gamma<1$
Since $\alpha>\gamma+2$, and $\gamma>1$, the gap between the lives dynamics and the steady state G is a stages function such that: if $\gamma+2<\alpha\leq(6)^{1/2}+\gamma$; (h_{t+1} ,b_{t+1}^{h}) converges to $(1-(6)^{1/2}/\gamma, \sqrt{6}+\gamma)$, there are cycles of period 2; if $\gamma+(6)^{1/2}<\alpha\leq\gamma+\mu_\infty -1$, there are cycles of periods $(2^r)_{r=2,3,4}$ and (h_{t+1} ,b_{t+1}^{h}) converges to $((\gamma+1-\mu_\infty)/\gamma, \gamma+\mu_\infty+1)$; if $\gamma+\mu_\infty-1<\alpha\leq\gamma+3$, the dynamic process of pregnancies is chaotic and (h_{t+1} ,b_{t+1}^{h}) converges to $(1-3/\gamma , \gamma+3)$, the mother has crossed the AIDS threshold, the baby is unable to survive.

Proof: because the map *g* is topologically conjugate to the logistic map $L(h_t)$ and the dynamics expressed in function of the transmission risk, α and the risk neutral transmission parameter, γ then for a given value of $\mu=\mu(\alpha,\gamma)$, we can find the corresponding value of α in function of γ which will give the corresponding deaths optimal dynamics of the mothers and determinate the lives dynamics of the babies. For a given value of $\alpha(\gamma)$ the stable orbit of new forthcoming healthy babies can thus be determined. The steady states are calculated in the usual way of letting $h_t= g(\alpha, \gamma)$ and $b_t^h= b^h(h,\alpha,\gamma)$ for the bifurcation points of the logistic map, define $G= b_{t+1}^{h} - h_{t+1}$ as the basic reproductive stock which guarantees the species continuity, in reference to the epidemiology literature, it is the key parameter which determines whether the disease spectrum doesn't exist in the long run (when G=0 because $\gamma<1$ and $\alpha<0$) or becomes endemic (when $G>0$ because $\gamma\geq 1$ and $\alpha\geq 0$). When $\alpha\leq\gamma<1$, the lives dynamics converge to its highest locus as proved by the following corollary 2. *In contrast, when* $\gamma+\mu_\infty-1<\alpha\leq\gamma+3$, there is a chaotic behavior of the dynamics of the mothers and the babies' because the spectrum which expresses the gap in lives term around the steady state reaches its highest level where $\gamma>1$ and $\alpha>0$. In the area $\gamma+(6)^{1/2}<\alpha\leq\gamma+\mu_\infty -1$, the dynamics of the mothers defines a 2r periods steady state ; in the area $\gamma+2<\alpha\leq (6)^{1/2}+\gamma$, there is a stable cycle of period 2 pregnancy process; in the area $\gamma<\alpha\leq\gamma+2$, the dynamic process of pregnancies is divided in two parts where at the left of G=0 because $\gamma<1$ and $\alpha<0$ the fluctuation of the dynamics is small and almost stable but at the right of G=0, the increasing and decreasing curves of the dynamics accelerates its progression toward a kind of non stability, the stable orbit is ruled out when $\gamma\geq 1$ and $\alpha\geq 0$. Indeed, $b^h > \gamma$ means that the

infection impact on babies' life is positive since $\gamma \geq 1$, thus, disease is no more free and G>0. In contrast, the steady state arises from the structure of the lives dynamics when G=0 leading to the highest steady state since $b_{t+1}^{h} = h_{t+1}$. For $\gamma < \alpha \leq \gamma + 2$, *the infection risk transmission exists and determines a stable endemic case. But when* $\gamma + 2 < \alpha \leq (6)^{1/2} + \gamma$ *, there are* cycles of period 2. After the cross of the AIDS state or close to death, the system is chaotic and there are no lives possible since the dynamics of reproduction is too far from the steady state (see figure 1).

Corollary 2: *denote the mapping H where H={(m(γ),n(γ)) such that there exists α(γ), γ for which,* (h_{t+1}, b_{t+1}^{h}) *converges to (m(γ),n(γ))}, H is a convex function meaning that in presence of decreasing HIV/AIDS, the sustainable path converges to the steady state.*

Proof: the limits of the joint dynamics found in proposition 2 for $\gamma < 1$ and $\alpha \leq 0$ when drawn in a figure lead to a decreasing curve or figure 2 which represents the relationship of the mothers and the babies limits of the dynamics and highlights the fact that the optimal solution is reached at $h_t = 1$ and $b_t^{h} = \gamma < 1$, *thus* $\alpha < 0$. Therefore h_t and b_t^{h} inside the range 0 and 1 *tend to the* optimal solution where only one solution reaching the steady state exists (see figure 2).

Proposition 3: *the conditions for the spectrum convergence to 0 i.e the existence of the couple of scalars* (γ, α) *for which* $\lim_{\alpha(\gamma)} G = 0$ *yield: if* $\alpha \leq \gamma$*, then G converges to 0 if and only if* $(\gamma, \alpha) \to Min\{(1,1),(1,0)\} = (1,0)$*; if* $\gamma < \alpha \leq \gamma + 2$*, then G converges around 0 if and only if* $(\gamma, \alpha) \to Min\{(\sqrt{2}; 3.41),(-\sqrt{2}; 0.586)\} = (-\sqrt{2}; 0.586)$*; if* $\gamma + 2 < \alpha \leq \gamma + (6)^{1/2}$ *, then G converges around 0 if and only if* $(\gamma, \alpha) \to Min\{(1; 3.45),(2.75; 5)\} = (1; 3.45)$*; if* $\gamma + (6)^{1/2} < \alpha \leq \gamma - 1 + \mu_\infty$ *, then G converges around 0 if and only if* $(\gamma, \alpha) \to Min\{(1; 3.5),(-1; 1.5)\} = (-1; 1.5)$*; if* $\gamma - 1 + \mu_\infty < \alpha \leq \gamma + 3$*, then G converges around 0 if and only if* $(\gamma, \alpha) \to Min\{(-3; 0),(1; 4)\} = (-3; 0)$*;*

Proof: for the first case, setting $\alpha = \gamma$ in the expression of G and making it equal to zero, we find *G converge to* $\gamma - 1 = 0$ yields γ *converge to 1 or to* α then replacing that value in the linking equation with α yields, α *converge to* 0^{+} *or to* 1^{-}. Doing the same thing for the following cases, we obtain the limits announced in the proposition 3 where $\mu_\infty \approx 3.5$.

Proposition 4: *if the transmission risk doesn't exist or* $\alpha = 0$ *then* $\gamma = 1$ *or* $\gamma = -3$ *meaning that* $\alpha \leq \gamma$ *or* $\gamma - 1 + \mu_\infty < \alpha \leq \gamma + 3$*. Otherwise, if* $0 < \alpha < 1$ *then* $\gamma < 0$ *or* $\gamma < \alpha \leq \gamma + 2$*. Finally, if* $\alpha > 1$ *then* $\gamma = 1$ *or* $\gamma = -1$ *respectively when* $\gamma + 2 < \alpha \leq \gamma + (6)^{1/2}$ and $\gamma + (6)^{1/2} < \alpha \leq \gamma - 1 + \mu_\infty$

Proof: in the chaotic behavior of the dynamics, transmission risk doesn't exist because birth not exist either, the chaotic behavior leaves nothing possible when $\gamma = -3$ *for* $\gamma - 1 + \mu_\infty < \alpha \leq \gamma + 3$ or when the future mother is not infected i.e $\gamma = 1$ *for* $\alpha \leq \gamma$. When the transmission risk is low, the system still able to defend itself in the second case where $\gamma < 0$ *for* $\gamma < \alpha \leq \gamma + 2$. Finally when the transmission risk begins to widen, cycles appear which can be of period 2 or of period 2r.

Next section presents a one sector growth model in which the epidemiological model is introduced.

THE ECONOMY

There is a continuum of individuals of mass N. We assume the medical production function is composed of sick and healthy pregnant women expressed by $f(k_t, b_t^h, h_t)$ where k_t is per-capita capital stock or medical-care stock and the depreciation rate of capital is n $(0,1]$.

Assumption3 The production function $f(k_t, b_t^h, h_t)$: $R_+^3 \rightarrow R_+$ is a C^2 function and satisfies:
1. f is strictly increasing and concave in k, b^h, and h;
2. f satisfies Inada conditions, i.e $lim_{k\rightarrow 0} f_1=\infty$, $lim_{k\rightarrow\infty} f_1=0$ and $lim_{b^h\rightarrow 0} f_2=\infty$, $lim_{b^h\rightarrow\infty} f_2=0$; $lim_{h\rightarrow 0} f_3=\infty$, $lim_{h\rightarrow\infty} f_3=0$;
3. $f(0, b_t^h, h)=f(k, 0, h)=f(k, b^h, 0)=0$.
We assume that for each individual, the instantaneous utility function is time additively separable and depends on consumption c, and health state, $d=(d^b, d^h)$ i.e respectively the baby and his mother's health state.

Assumption 4 The individual utility function $u(c, d)$ is a C^2 function and satisfies:
1. u is strictly increasing and concave in c , d;
2. u satisfies Inada conditions, i.e. $\lim_{c\rightarrow 0} u_1 =\infty$, and $\lim_{d\rightarrow 0} u_2 =\infty$;
3. u is additively separable in consumption as well as in health and $u_{12}=u_{21}=0$.
Let $\theta(c, d)$ denote the marginal rate of substitution between health and consumption i.e. $\theta(c, d)=u_2/u_1$ .Additive separability implies that $\theta(c,d)$ admits an optimal solution, thus doesn't oscillate.

We want to study the social planner's problem. The objective of the social planner is to maximize the average individual utilities which is discounted at the rate β with $0<\beta<1$. As the utility function of each individual is additively separable, the optimal allocation will have full insurance of consumption i.e $c_t^h=c_t^i=c_t$ (i.e healthy and infective pregnant women per-capita consumption are equal) and the same for per-capita health state i.e $d_t^h=d_t^i=d_t$ *for* simplicity. Thus, we do not have to keep track of individual health histories and we can show the results analytically.
Denote the welfare function by: $U(c_t, d_t, b_t^h, h_t)=h_t u(c_t, d_t)+b_t^h u(c_t, d_t)$

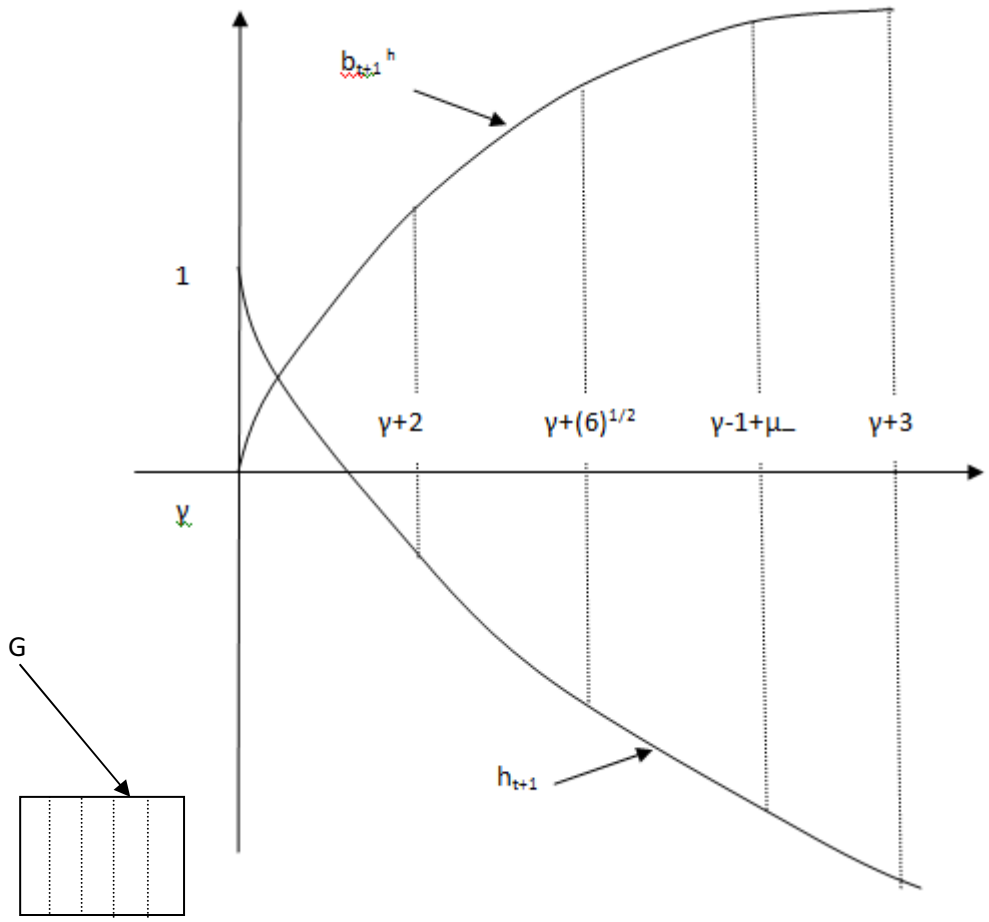

Figure1 (proposition 2 representation)

Lemma 4 *The social planner's objective function U(c, d, b_t^h, h): $R_+^4 \rightarrow R$ is a C^2 function which satisfies:*
1. *U is strictly increasing in both c and d, and strictly concave in (c,* d*);*
2. *U satisfies Inada conditions, i.e.* $\lim_{c\rightarrow 0} U_1 = \infty$ *and* $\lim_{d\rightarrow 0} U_2 = \infty$*;*
3. *U is additively separable in consumption and health i.e.* $U_{12} = U_{21} = 0$.

Proof From the assumption on *u*, we have
$U_1 = hu_1(c, d) + b^h u_1(c, d) > 0$
$U_2 = hu_2(c, d) + b^h u_2(c, d) > 0$
Assuming pregnant individuals work to finance their stays at the hospital, indeed we have:
$U_{11} = hu_{11}(c, d) + b^h u_{11}(c, d) < 0$
$U_{22} = hu_{22}(c, d) + b^h u_{22}(c, d) < 0$
$U_{12} = U_{21} = hu_{12}(c, d) + b^h u_{12}(c, d) = 0$
$U_{11}U_{22} - U_{12}U_{21} > 0$.

Hence, for the social planner's objective function, the marginal rate of substitution $\theta(c,d) = U_2/U_1$ is also an increasing function of *c*, then it is possible to show the monotonicity of the optimal capital stock.

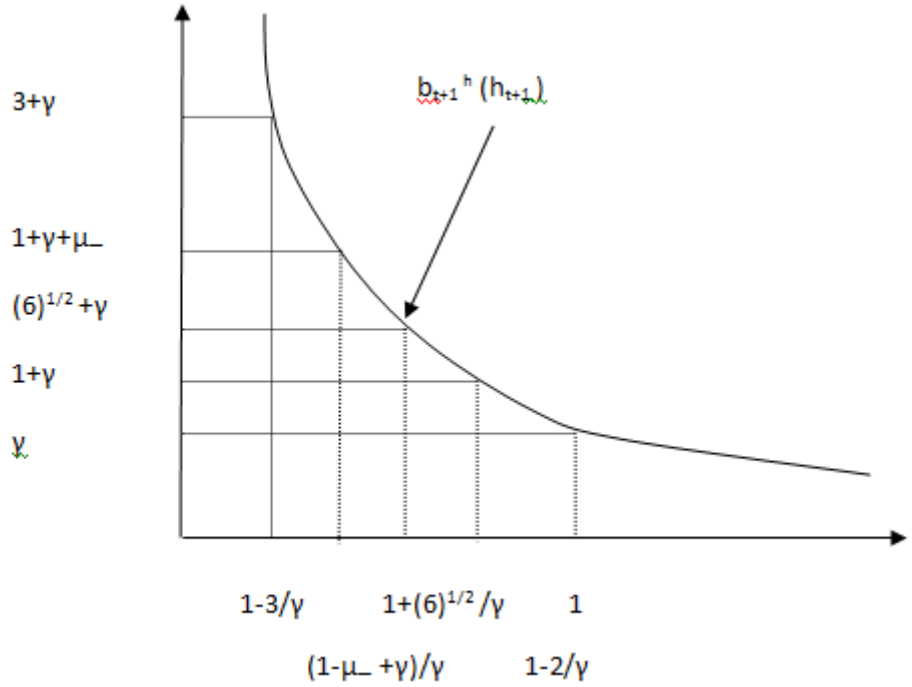

Figure 2 relative to corollary 2

The social planner's maximization problem is given by:

$$\underset{(c_t,d_t,k_{t+1})}{Max}\sum_{t=0}^{\infty}\beta^t U\left(c_t,d_t,b_t^h,h_t\right)$$

sc

$$k_{t+1}-(1-\delta)k_t+c_t+d_t \le f\left(k_t,b_t^h,h_t\right)\ \ \forall t$$
$$h_{t+1}=h_t+\left[(2\gamma-\alpha)-\gamma h_t\right](1-h_t)$$
$$b_{t+1}^{\ h}=\alpha+\frac{(2\gamma-\alpha)}{\gamma}-h_{t+1}=b_{t+1}^{\ h}(h_t)$$
$$k_t \ge 0,\ c_t \ge 0,\ d_t \ge 0\ \ \forall t$$
$$k_0>0,\ 0\le b_0^{\ h},h_0 \le 1 \text{ given}$$

The state variables are k_t , b_t^h , h_t and the control variables are c_t , d_t and k_{t+1}

THE EQUILIBRIUM DYNAMICS

Define $F(k_t, b_t^h, h_t)=f(k_t, b_t^h, h_t)+(1-n)k_t$. Since $lim_{k\to 0}F_1=\infty$, we have $k_t>0\ \ \forall t$ if $k_0>0$. Since $lim_{k\to\infty}F_1<1$, there exists a maximum sustainable stock $k^h>0$ for each fixed (b^h, h) and k^h is increasing in (b^h, h) by the implicit function theorem[4]. Hence k^{max} with $F(k^{max}, \alpha+1/\gamma, 1)=k^{max}$ is the maximum sustainable stock for all h, $b^h \in [0, 1]$. Therefore we have $(k_t, b_t^h, h_t) \in X=[0,k^{max}]x[0,1]x[0,1] \subset R^3$ and the set X is closed and bounded.

[4] If $f(x_0, y_0)=0$ and $f_2'(x_0, y_0)\neq 0$, then the equation $f(x,y)=0$ defines y as an "implicit function" of x i.e $y=\rho(x)$ of x near x_0 with $y_0=\rho(x_0)$ and with its derivative given by $y'=-f'(x,y)/ f_2'(x,y)$

According to dynamic programming methods and the Bellman equation, the social planner problem can be written such that:

$$V(k_t, b_t^h, h_t) = \underset{(c_t, d_t, k_{t+1})}{Max} \left[u(c_t, d_t, b_t^h, h_t) + \beta V(k_{t+1}, b_{t+1}^h, h_{t+1}) \right]$$

St

$$k_{t+1} - (1-m)k_t + c_t + d_t \leq f(k_t, b_t^h, h_t) \quad \forall t$$

$$h_{t+1} = h_t + [(2\gamma - \alpha) - \gamma h_t](1 - h_t)$$

$$b_{t+1}{}^h + h_{t+1} = \alpha + \frac{(2\gamma - \alpha)}{\gamma}$$

$$k_t \geq 0,\ c_t \geq 0,\ d_t \geq 0 \quad \forall t$$

$$k_0 > 0,\ 0 \leq b_0^h,\ h_0 \leq 1 \text{ given}$$

Isolating the choice of consumption and of labor supply from the investment choice in decomposing the planning problem into a static problem and a dynamic problem[5], the static problem is given by:

$$W(k, k', h) = \underset{(c_t, d_t) \in B(k, k', b^h, h)}{Max} U(c, d, b^h, h)$$

Where

$$B(k, k', b^h, h) = \left\{ (c, d) \in R^2 \ / \ 0 \leq d \leq 1,\ c + k' \leq F(k, b^h, h) \right\}$$

Lemma 5: *W is continuous and bounded, and the maximizers c(k, k', b^h, h) and d(k, k', b_t^h, h) are continuous functions; W is strictly concave in (k, k') for each fixed (b^h, h); W is continuously differentiable and $W_1>0$, $W_2<0$, $W_{12}>0$, $W_{22}<0$ for each fixed h and b^h*

Proof: since *U* is continuous and *B(k, k', b^h, h)* is continuous and compact, by the theorem of the maximum, the function *W* is continuous and the maximizers are non empty, compact valued and upper hemi-continuous. Since the objective function is strictly concave, the maximizers are unique and continuous functions. Since *(k, k', b_t^h, h)* $\epsilon X=[0,k^{max}]x[0,1]x[0,1]$ which is compact, *W* is bounded. Fixing (*h*, b^h) and choosing any $\lambda \epsilon [0,1]$ and letting *(c_1 , d_1)* being the maximizers of *(k_1 ,k_1', b_t^h ,h)* and *(c_2 , d_2)* being the maximizers of *(k_2 , k_2', b_t^h , h)* then *($\lambda c_1 + (1-\lambda)c_2$, $\lambda(1-d_1) + (1-\lambda)(1-d_2)$)* is feasible for *($\lambda k_1 + (1-\lambda)k_2$, $\lambda k'_1 + (1-\lambda)k'_2$, b^h ,h)* and

$W[\lambda k_1+(1-\lambda)k_2$,$\lambda k'_1+(1-\lambda)k'_2$, b_t^h ,h]$\geq U[\lambda c_1+(1-\lambda)c_2$,$\lambda(1-d_1)+(1-\lambda)(1-d_2)$, b_t^h ,h]

$>\lambda U(c_1$,1-d_1 , b_t^h ,h)+(1-λ)U(c_2 ,1-d_2 , b_t^h ,h)=$\lambda W(k_1$,k_1' ,b_t^h ,h)+(1-λ)W(k_2 ,k_2' ,b_t^h , h) by strict concavity of *U*, thus, *W* is strictly concave for each fixed (b^h , *h*). Let *(c*, d*)* be the optimal consumption and health care supply, by the envelope theorem, *W* is continuously differentiable, $W_1=U_1F_{1/(c^*,d^*)} >0$ and $W_2=-U_{1/(c^*,d^*)} <0$. Given the Inada conditions, the corner solution can be ruled out, thus the optimal solution given by *c*=c(k, k', b^h ,h)* and *d*=d(k, k' b_t^h, h)* is determined by

F_2(k, hd+b^hd)-θ(c,1-d, b^h , h)=0 and *F(k, dh+db^h)-c - k'=0*. By the implicit function theorem *c(k, k', b^h ,h)* and *d(k, k', b^h ,h)* are differentiable in the neighborhood of (*k*, b^h , *h*) and *(k', b^h , h*).

[5] Aiyagari et al. 1992

Based on the properties of the function *W* above, the dynamic problem is given by:

$$V\left(k_t, b_t^h, h_t\right) = \underset{\left(b_{t+1}^h, ht+1_t, k_{t+1}\right) \in A\left(b_t^h, k_t, h_t\right)}{Max} \left[W\left(k_t, k_{t+1}, b_t^h, h_t\right) + \beta V\left(k_{t+1}, b_{t+1}^h, h_{t+1}\right)\right]$$

Where

$$A\left(k_t, b_t^h, h_t\right) = \left\{ \begin{array}{l} \left(k_{t+1}, b_{t+1}^h, h_{t+1}\right) \in R^3 \;/\; k_{t+1} \le F\left(k_t, b_t^h, h_t\right) \text{ and } h_{t+1} = h_t + \left[(2\gamma - \alpha) - \gamma h_t\right](1 - h_t), \\ \text{with } \; b_{t+1}^h + h_{t+1} = \alpha + \dfrac{(2\gamma - \alpha)}{\gamma} \end{array} \right\}$$

and $0<\beta<1$

Let the optimal capital investment be $k_{t+1}=g^1(k_t, b_t^h, h_t)$ where h_{t+1} and $b_{t+1}{}^h$ are governed by the mapping $g'(h_t)$ and $b(h_t)$

Lemma 6: *V is continuously differentiable, strictly concave in k_t for each fixed (b^h , h) and g' is a continuous function*

Proof: since X is a convex subset of R^3_+ and the correspondence *E: $X \to X$* is non empty, compact-valued and continuous, the function *W* is continuous and bounded, from Lucas and Stockey (1989) theorem, there exists a unique value function which solves the dynamic programming problem and the policy correspondence is compact valued. Since *W* is strictly increasing in *k* in the way that $k_1 \le k_2$ implies $A(k_1, b_t^h, h) \subset A(k_2, b_t^h, h)$, *V* is strictly increasing in k in the way that $k_1, k_2 \in [0, k^{max}]$•

Given $\left(k_0, b_0^h, h_0\right)$, the sequence $\left(k_t, b_t^h, h_t\right)_{t=0}^{\infty}$ defined by $\left(k_{t+1}, b_{t+1}^h, h_{t+1}\right) = \left(g^1\left(k_t, b_t^h, h_t\right), g'(h_t), b^h\left(h_{t+1}\right)\right)$ is the unique solution which satisfies: (S) i.e

$$W_2\left(k_t, k_{t+1}, b_t^h, h_t\right) + \beta V_1\left(k_{t+1}, b_{t+1}^h, h_{t+1}\right) = 0$$
$$W_1\left(k_t, k_{t+1}, b_t^h, h_t\right) = V_1\left(k_t, b_t^h, h_t\right)$$
$$h_{t+1} = h_t + \left[(2\gamma - \alpha) - \gamma h_t\right](1 - h_t) = g(h_t)$$
$$b_{t+1}{}^h(h_t) = \alpha + \frac{(2\gamma - \alpha)}{\gamma} - h_{t+1}$$
$$\lim_{t \to \infty} \beta^t W_1\left(k_t, k_{t+1}, b_t^h, h_t\right) k_t = 0$$

The dynamical system has three state variables k_t and (b_t^h , h_t). Therefore, the dynamical system is given by the vector *(X, g, b^h)*, where $X=\{[0, k^{max}]\times[0,1]x]0,1]\}$ is the state space.

Lemma 7: *the sequence $\left(k_t\right)_{t=0}^{\infty}$ is strictly monotonic for every fixed h_t , b_t^h*

Proof: from the system of equations (S), we have $W_2\left(k_t, g^1, b_t^h, h_t\right) + \beta V_1\left(g^1\left(k_t, b_t^h, h_t\right), g'(h_t), b^h\left(h_{t+1}\right)\right) = 0$ by substitution into the policy

function. By the implicit theorem, g^1 is differentiable in k for a fixed (b^h, h) and the chain rule $W_{21}+W_{22}g^1{}_{11}+\beta v_{11}g^1{}_{11}=0$, thus $g_1{}^1=-W_{21}/W_{22}+\beta v_{11}$. Since $W_{21}>0$, $W_{22}<0$ and $V_{11}<0$, we have $g_1{}^1(k, b^h, h)>0$ i.e g^1 is strictly increasing in the first argument. Hence for every fixed (b^h, h), if $k_1>k_0$, $k_2=g^1(k_1, b^h, h)>g^1(k_0, b^h, h)=k_1$ and by induction $k_{t+1}>k_t$ for every t. Similarly if $k_1<k_0$, $k_{t+1}=k_t$ for every t. Thus the sequence $(k_t)_{t=0}{}^{\infty}$ is strictly monotonic for every fixed (b^h, h).

Fixed Points

Definition2: let the set Ω be such that $\Omega \subset R^n$, the map $l: \Omega \rightarrow \Omega$ is said to have a fixed point if there exists $x^* \in \Omega$ such that $l(x^*)=x^*$

Proposition 5: *if $\alpha \leq \gamma$ and $\gamma<\alpha \leq \gamma+2$ where, $\alpha<0$ and $\gamma<1$ as well as $\alpha \geq 0$ and $\gamma \geq 1$, the sequence $(b^h, h_t)_{t=0}{}^{\infty}$ converge to some locus around the steady state. where for fixed (b^{h*}, h^*) the sequence $(k_t)_{t=0}{}^{\infty}$ is strictly monotonic (lemma 6) and bounded i.e $k_t \in [0, k^{max}]$. By the monotone convergence theorem, $(k_t)_{t=0}{}^{\infty}$ converge to some fixed point k^*. Because of the uniqueness of the limit and the continuity of the mapping, $h^*=g^1(0,0, h^*)$, $b^{h*}=g^1(0, b^h, (0)$ and $k^*=g^1(k^*, b^{h*}, h^*)$ as the set X is closed, it yields $(k^*, b^{h*}, h^*) \in X$*

Cycles

Definition3: let the set Ω be such that $\Omega \subset R^n$, the map $l: \Omega \rightarrow \Omega$ is said to have cycles of period $r>1$ *if we have* $l^r(x)=x$

Proposition6: if $\gamma+(6)^{1/2}<\alpha \leq \gamma+\mu_{\infty}-1$, the system has cycles of period 2^r , $r=1,2,3,\ldots$ and for each α there is a unique periodic orbit which is attracting

Proof: consider the case of $r=1$ first, if $\gamma+2<\alpha \leq \gamma+(6)^{1/2}$ from lemma 6, the sequence $(k_t)_{t=0}{}^{\infty}$ has a unique attracting cycles of period 2. So that for any $t \geq T$, $(k_t)_{t=0}{}^{\infty}$ takes value from the set $O(g)=((b_1{}^h, b_2{}^h),(h_1, h_2))$, and we substitute the value of $b_t{}^h$, h_t into the mapping g^1 for $t>T$:

$$k_{T+1} = g^1\left(k_T, b_1^h, h_1\right)$$

$$k_{T+2} = g^1\left(k_{T+1}, b_2^h, h_2\right) = g^1\left(g^1\left(k_T, b_1^h, h_1\right), b_2^h, h_2\right)$$

$$k_{T+3} = g^1\left(k_{T+2}, b_1^h, h_1\right) = g^1\left(g^1\left(k_{T+1}, b_2^h, h_2\right), b_1^h, h_1\right)$$

$$k_{T+4} = g^1\left(k_{T+3}, b_2^h, h_2\right) = g^1\left(g^1\left(k_{T+2}, b_1^h, h_1\right), b_2^h, h_2\right)$$

$$k_{T+5} = g^1\left(k_{T+4}, b_1^h, h_1\right) = g^1\left(g^1\left(k_{T+3}, b_2^h, h_2\right), b_1^h, h_1\right)$$

$$\ldots\ldots$$

We define a new sequence $\{(k_{T+2j})_{j=0}{}^{\infty} = \{k_T, , k_{T+2}, k_{T+4}, \ldots\}$ with $k_{T+2(j+1)}=g^1(g^1(k_{T+2j}, b_1{}^h, h_1), b_2{}^h, h_2)$. If $k_T<k_{T+2}$, $g^1(k_T, b_1{}^h, h_1)<g^1(k_{T+2}, b_1{}^h, h_1)$ since $g^1(k, b_1{}^h, h_1)$ is increasing in k for fixed $b_1{}^h$, h_1 , and thus, by the same reasoning $k_{T+2}=g^1(g^1(k_T, b_1{}^h, h_1), b_2{}^h, h_2)<g^1(g^1(k_{T+2}, b_1{}^h, h_1), b_2{}^h, h_2)=k_{T+1}$. Hence, by induction $k_{T+2j}<k_{T+2(j+1)}$ j . Similarly, if $k_T>k_{T+2}$ we have $k_{T+2j}>k_{T+2(j+1)}$ j , and if $k_T=k_{T+2}$ we have $k_{T+2j}=k_{T+2(j+1)}$ j. Hence, the sequence $\{k_{T+2j}\}_{j=0}{}^{\infty}$ is strictly monotonic. Moreover, since $\{k_{T+2j}\}_{j=0}{}^{\infty}$ is a subsequence of $\{k_t\}_{t=0}{}^{\infty}$ *then* $\{k_{T+2j}\}_{j=0}{}^{\infty}$ is bounded. By the monotone convergence theorem, $\{k_{T+2j}\}_{j=0}{}^{\infty}$ converge to some fixed point $k_1{}^*$. By the uniqueness of the limit and continuity of the mapping $g^1(g^1(k, b_1{}^h, h_1), b_2{}^h, h_2)$, $k_1{}^*$ is determined *by* $k_1{}^*=g^1(g^1(k_1{}^*, b_1{}^h, h_1), b_2{}^h, h_2)$ and $k_1{}^* \in [0, k^{max}]$. Similarly, we can define the sequence $\{k_{T+1+2j}\}_{j=0}{}^{\infty}=\{k_{T+1}, k_{T+3}, k_{T+5}\ldots$

.} with $k_{T+1+2(j+1)} = g^1(g^1(k_{T+1+2j}, h_2), h_1)$. This sequence, $\{k_{T+1+2j}\}_{j=0}^{\infty}$ converge to some fixed point, k_2^* determined by $k_2^* = g^1(g^1(k_2^*, b_2^h, h_2), b_1^h, h_1)$ and k_2^* $[0, k^{max}]$. Therefore, by construction we decompose the sequence $\{k_{T+j}\}_{j=0}^{\infty}$ into two subsequences, $\{k_{T+2j}\}_{j=0}^{\infty}$ consisting all the even terms and converging to k_1^* and $\{k_{T+1+2j}\}_{j=0}^{\infty}$ consisting all the odd terms and converging to k_2^*. Thus, $\{k_{T+j}\}_{j=0}^{\infty}$ will fluctuate between k_1^* and k_2^* for sufficient large j. Moreover $\{k_{T+j}\}_{j=0}^{\infty}$ is the tail of the sequence $\{k_t\}_{t=0}^{\infty}$, so $\{k_t\}_{t=0}^{\infty}$ has a cycle of period 2. Hence, the system has a cycle of period 2, which is attracting.

Second, if $\gamma - 2 < \alpha \leq \gamma + u_{\infty} - 1$, from Lemma 6 the sequence $\{h_t\}_{t=0}^{\infty}$ has a cycle of period $2r$, $r = 2, 3, 4, \ldots$. Moreover for each α, there is a unique attracting periodic orbit. Following the same argument above we can show the sequence $\{k_t\}_{t=0}^{\infty}$ has a cycle of the same period as $\{b_t^h, h_t\}_{t=0}^{\infty}$,. Hence, the system has a cycle of period $2r$, $r = 1, 2, 3, 4, \ldots$.

Chaos

For a one dimensional system, if the Lyapunov[6] exponent is positive, then the orbits from two infinitesimally close initial conditions diverge exponentially over time and there is sensitive dependence on initial conditions. For higher dimensional systems[7], the local behavior of the system depends on the direction and the nearby points may be moving apart in one direction but the other move together in another[8] direction. To prove chaos existence, it is necessary to study the behavior of the entire system, i.e. $g = (g^1, g)$[9].

Definition4 Let l be a smooth map on $\Omega \subset R^n$ and $J^T = Dh^T(x_0)$, $J^{T'} = D(b^h)^T(y_0)$ be the Jacobian of $h^T(x)$ and $(b^h)^T(y)$ respectively evaluated at an orbit starting point from the initial condition x_0 and y_0. Let λ_q^T, $q = (1, \ldots, n)$ and λ_p^T, $p = (1, \ldots, n)$ be the eigenvalues of J^T and $J^{T'}$ respectively, then the qth and pth Lyapunov number of x_0 and y_0 is defined by $L_q = \lim_{T\to\infty} (\lambda_q^T)^{1/T}$ and $L_p = \lim_{T\to\infty} (\lambda_p^T)^{1/T}$ if this limit exists, then the qth and pth Lyapunov exponent of x_0 and of y_0 is respectively such that:

$$\Gamma_q = \underset{T\to\infty}{Lim} \frac{1}{T} Ln\left|\lambda_q^T\right| \text{ and } \Gamma_p = \underset{T\to\infty}{Lim} \frac{1}{T} Ln\left|\lambda_p^T\right|$$

The set of all the Lyapunov exponents i.e $\Gamma = \{\Gamma_1, \Gamma_2, .. \Gamma_q, ... \Gamma_n\}$ and $\Gamma' = \{\Gamma_1, \Gamma_2, .. \Gamma_p, ... \Gamma_m\}$ are called the Lyapunov spectrum of the dynamical systems h_{t+1} and b_{t+1}^h. Using the concept of Lyapunov exponents, we can give the definition of chaotic orbits of higher dimensional maps[10]. The conditions 1 and 3 below are standard and hold for the conventional definitions of one-dimensional chaos. Condition 2 is to rule out quasi-periodic orbits which are predictable.

Definition5 Let l, m be a map on Ω, $\Omega' \subset R^n$ and let $\{x_0, x_1, \ldots\}$ and $\{y_0, y_1, \ldots\}$ be a bounded orbit of l, m. The orbit exhibits multi-dimensional chaos if:

1. It is not asymptotically periodic,
2. No Lyapunov exponent is exactly zero, and
3. At least one Lyapunov exponent is positive.

[6] The Lyapunov exponents are away to study the divergence of orbits on an attractor and other invariant sets.

[7] The definition of higher dimensional chaos is provided by Alligood at al, 1997, Chap.5

[8] The definition of higher dimensional chaos that we use (see Alligood et al. 1997, Ch. 5) is equipped to deal with such issues. This definition also relies on the Lyapunov exponent. Note that while it is well known that $g2$ on its own exhibits chaos (Ott 2002, Section 2.2) as it is topologically conjugate to the logistic map and has a positive Lyapunov exponent,

[9] Note that g exhibits chaos, see Ott 2002, Section 2.2

[10] Alligood et al. 1997, p. 196

Proposition 7: if $\gamma+\mu_{\infty}-1<\alpha\leq\gamma<\alpha\leq\gamma+3$, *the dynamical system exhibits multidimensional chaos.*

Proof: the orbit $O(g^1) = \{(k_0 , b_t^h\ h_0), (k_1 , b_1^h\ h_1), \ldots\}$ is bounded since (k_t , b_t^h , h_t) X. Beyond the accumulation point of cycles of period $2r$, that is $\mu\infty<\mu\leq4$ where $\mu\infty = 3.570\ldots$, the logistic map L has a chaotic attractor with an infinite number of embedded unstable periodic orbits. Since the mapping g and L are topologically conjugate and the Lyapunov exponent is invariant with respect to topological conjugacy, g has the same Lyapunov exponent as the logistic map, L which is positive when $\gamma+\mu\infty-1<\alpha<\gamma+3$ (from Lemma 4). It turns out that $\Gamma^g = (\Gamma_q , \Gamma_p)$ i.e the Lyapunov exponent of g, is one of the Lyapunov exponent of mapping g as well, that is $\Gamma_2^g = \underset{T\to\infty}{Lim}\frac{1}{T}\left|Ln\left(\lambda_{2,,q}^T\right)+Ln\left(\lambda_{2,p}^T\right)\right| \succ 0$ because the Jacobian of the system is given by $J^T+J^{T'} = J=\begin{pmatrix} g_1^1 & g_2^1 \\ 0 & g_2^2 \end{pmatrix}$ and the second Lyapunov exponent is $\Gamma_1^g = \Gamma_{1,p}+\Gamma_{1,q} = \underset{T\to\infty}{Lim}\frac{1}{T}\left|Ln\left(\lambda_{1,p}^T\right)+Ln\left(\lambda_{1,q}^T\right)\right|$ where $\lambda_1^T = \Pi_{t=1}^T\left|g_1^1\left(k_t, b_t^{\ h}, h_t\right)\right|$ and $\lambda_2^T = \Pi_{t=1}^T\left|g_2^1\left(k_t, b_t^{\ h}, h_t\right)\right|$. For this Lyapunov exponent to be exactly equal to zero it must be that $g^1(k_t , b_t^h , h_t)=1$, t. As $g^1{}_1(k_t , b_t^h, h_t)=-W_{21}/W_{22}+\beta v_{11}$, we can see that this will not be true in general. The orbit $O(g^1)$ is not asymptotically periodic as the dynamics are governed by the well-known logistic map. Thus, the orbit $O(g^1)$ is not asymptotically periodic as well. Therefore, the system exhibits multi-dimensional chaos. Note that, $\alpha=\gamma+3$ is equivalent to $\mu=4$ in the logistic map, when $\mu=4$ the orbit is dense in [0, 1]. Hence, b_t^h , h_t may be equal to zero and the population cease.

In summary, the monotonicity of capital accumulation (for fixed b^h , h) implies that the dynamics of the system are fully determined by the disease transmission dynamics. Non-linearity in the disease transmission dynamics can induce fluctuations in the population growth and dynamics. We find that sufficiently large HIV/AIDS transmission risks and unhealthy babies' stock can generate complex dynamics in population growth. Intuitively, when the transmission rate is low (relative to the babies' healthy rate), that is $\alpha\leq\gamma$, $\alpha<0$ and $\gamma<1$ the inflow into the infective class is small while the outflow is relatively larger. Thus, the number of infective people is decreasing which leads to an even smaller inflow as less pregnant women are infected. Therefore, the number of infective pregnant women will keep declining and eventually becomes zero which leads to upper babies stock. This is the disease-free steady state, and is the same as the one sector neo-classical growth model where the disease is absent. When the HIV/AIDS transmission rate is such that $\gamma<\alpha\leq\gamma+2$, the inflow into the infective class is larger than the outflow and the system reaches the stable steady state with a fixed proportion of infective people. This is called the endemic or disease-persistent steady state and has a higher level of physical capital or medical care stock and lower economic performance compared with the disease-free case. This can easily be shown by the comparative statics of the steady states. The larger the proportion of healthy people is, the lower needs of the capital per capita for medical care use is. The disease-free and endemic steady states have been the focus in the economic literature. As we abstract away from change in population size, the disease prevalence has an adverse effect on the economy through its impact on labor productivity. When both the transmission rate and the stability of health rate are high non optimality establishes because the mechanics of the pandemic

HIV/AIDS are stronger than those of the immune system, cycles and chaos emerge in the population growth and the economy do not have a locally stable steady state. If the transmission rate is high, a large number of healthy pregnant women are getting infected and babies too, thus the both move into the infective class. Indeed, the number of healthy future mothers will decline and the number of infective future mother will rise. In contrast to the epidemiologic literature, here many pregnant women are unable to move from the infective class to the healthy class as a result of recovery from the disease only if the system didn't reach cycles of period 2r and chaos. If the condition of $\alpha \leq \gamma+2$ is satisfied, then the number of healthy future mothers increases while the number of infective future mothers drops. Therefore, the number of healthy pregnant women in each class fluctuate sharply, and eventually they may not reach two cycle of period $2r$, r = 1, 2, 3, 4, . . . or exhibit chaotic behavior. Which situation is realized depends on the relative rate of flows in and out of the classes, that is on the magnitudes of α and γ. Moreover, the oscillation in the number of healthy pregnant women causes the fluctuations of the capital stock, and consequently, causes population growth fluctuates endogenously..

Application of the theory

The utility function of the agents is $u(c_t, d_t) = \ln(c_t) + \varepsilon \ln(d_t)$ because of the hypothesis of full insurance of consumption and health care. Therefore the social planner objective function is $U(c_t, d_t, b_t^h, h_t) = \ln(c_t) + h_t \ln(d_t^{\,h}) + b_t^h \ln(d_t^{\,b})$ [11]

The medical production function is of Cobb-Douglas i.e $y_t = Ak_t^{\sigma}(b_t^h)^{\delta}(h_t)^{1-\delta-\sigma}$ where $0 < \sigma, \delta < 1$ and capital fully depreciate on use.

The social planner's problem (S') is:

$$V(k_t, h_t) = \underset{(c_t, d_t, k_{t+1})}{Max} \left[\ln(c_t) + h_t \ln(d_t^{\,h}) + b_t^h \ln(d_t^{\,b}) + \beta V(k_{t+1}, b_{t+1}^h, h_{t+1})\right]$$

St

$$k_{t+1} + c_t + d_t \leq F(k_t, b_t^h, h_t) \quad \forall t$$

$$h_{t+1} = h_t + [(2\gamma - \alpha) - \gamma h_t](1 - h_t)$$

$$b_{t+1}^{\,h} + h_{t+1} = \alpha + \frac{(2\gamma - \alpha)}{\gamma}$$

$$k_t \geq 0,\ c_t \geq 0,\ d_t \geq 0 \quad \forall t$$

$$k_0 > 0,\ 0 \leq b_0^{\,h}, h_0 \leq 1 \text{ given}$$

The first order conditions are:

$1/c_t - \beta V_1(k_{t+1}, b_{t+1}^h, h_{t+1}) = 0$ (i)

$h_t / d_t^{\,h} - \beta V_1(k_{t+1}, b_{t+1}^h, h_{t+1}) = 0$ (j)

$b_t^{\,h} / d_t^{\,b} - \beta V_1(k_{t+1}, b_{t+1}^h, h_{t+1}) = 0$ (k)

$V_1(k_t, b_t^h, h_t) = \beta V_1(k_{t+1}, b_{t+1}^h, h_{t+1}) F_1(k_t, b_t^h, h_t)$ (l)

[11] See Goenka and Liu, 2012, p.137

Combining (1) and (3) as well as (2) and (3) it yields

$$1/c_t = \beta\frac{1}{c_{t+1}}F_1\left(k_{t+1}, b_{t+1}^h, h_{t+1}\right) = A\sigma\beta\frac{1}{c_{t+1}}\frac{y_{t+1}}{k_{t+1}} = A\sigma\beta\frac{1}{c_{t+1}}\frac{y_{t+1}}{y_t - c_t - d_t}$$

$$h_t / {d_t}^h = \beta\frac{h_{t+1}}{d_{t+1}}F_2\left(k_{t+1}, b_{t+1}^h, h_{t+1}\right) = A(1-\delta-\sigma)\beta\frac{h_{t+1}}{{d_{t+1}}^h}\frac{y_{t+1}}{h_{t+1}} = A(1-\delta-\sigma)\beta\frac{y_{t+1}}{{d_{t+1}}^h}$$

$${b_t}^h / {d_t}^b = \beta\frac{{b_{t+1}}^h}{{d_{t+1}}^b}F_2\left(k_{t+1}, b_{t+1}^h, h_{t+1}\right) = A\delta\beta\frac{{b_{t+1}}^h}{{d_{t+1}}^b}\frac{y_{t+1}}{{b_{t+1}}^h} = A\delta\beta\frac{y_{t+1}}{{d_{t+1}}^b}$$

In the long run we have: $c_t = c_{t+1}$; $y_t = y_{t+1}$; ${d_t}^j = {d_{t+1}}^j$ added with the assumption ${d_t}^b = {d_t}^h \quad \forall t$, therefore re-writing the equations in dynamical terms we get:

$$b_t^h = A\delta\beta y_t \tag{7}$$

$$h_t = A\beta(1-\delta-\sigma)y_t \tag{8}$$

$$c_t = (1-A\beta\sigma)y_t - d_t \tag{9}$$

Indeed,

$$k_{t+1} = y_t - (c_t + d_t) = A\sigma\beta y_t \tag{10}$$

Proposition8: *both the mother and the baby dynamics are expressed in function of per-capita capital expressed by* $h_t = \Delta_1 {k_t}^M$, $b_t^h = \Delta_2 {k_t}^N$ *define per-capita capital law of motion given by equation (11) i.e*

$$k_{t+1} = \left(\sigma\beta A^4\Delta_1^\delta\Delta_2^{1-\sigma-\delta}\right){k_t}^{\sigma+N\delta+M(1-\sigma-\delta)} \tag{11}$$

Where $\Delta_2 \succ \Delta_1$ and $N \succ M$

Proof: introducing the expression of the production function inside equations (7)-(9), then we determinate the dynamics given where each constant variable depends on parameters and thus express parameters inside the range 0 and 1.

Proposition9: *the economy admits a unique optimal solution* $(k_t, h_t, b^h)_{t=0}^{\infty}$ *for* $\alpha\leq\gamma$ *and the system achieves the disease free steady state satisfying:*

$$k^* = \left(\sigma\beta A^4\Delta_1^\delta\Delta_2^{1-\sigma-\delta}\right)^{-[\sigma+N\delta+M(1-\sigma-\delta)]};\ b^{h*} = \Delta_2\left(\sigma\beta A^4\Delta_1^\delta\Delta_2^{1-\sigma-\delta}\right)^{-N[\sigma+N\delta+M(1-\sigma-\delta)]};$$

$$h^* = \Delta_1\left(\sigma\beta A^4\Delta_1^\delta\Delta_2^{1-\sigma-\delta}\right)^{-M[\sigma+N\delta+M(1-\sigma-\delta)]}$$

If transmission risk is located such that $\gamma<\alpha\leq\gamma+2$*, then the system achieves the endemic steady state and* ${b_t}^h + h_t = 100\%$ *leads to one feasible solution for health maintenance i.e* $\gamma = -1/2 + \sqrt{3}/2$ *; if transmission risk is located such that* $\gamma+2<\alpha\leq\gamma+\mu_\infty-1$*, then the system has cycles of period* 2^r *and* ${b_t}^h + h_t$ =100% lead to $\mu_\infty = \left(1-\gamma-\gamma^{-1}\right)/1-\gamma^{-1}$ *where r=1, 2, 3, 4,… because the dynamics presents cycles. If* $\gamma+\mu_\infty-1<\alpha\leq\gamma+3$*, the system is chaotic and* ${b_t}^h + h_t$ =100% yields multiple solutions where the evident are $\gamma = (\sqrt{21}+3)/2$ and $\gamma' = (-\sqrt{21}+3)/2$ and summarize a chaotic behavior.

Proof: solving the equations of proposition 8 in the long run terms i.e fixing the time for the variables, determinate the steady state locus ensuring population growth equilibrium i.e the stable optimal path existence. The following calculus aim is to look for the stable equilibrium in fixing the joint dynamics to hundred percent.

CONCLUSION

The model began with the disease transmission dynamics in conformity with the epidemiological literature and established that since transmission risk is positive, the immune system rise in order to fight against HIV/AIDS and protect the baby whom life dynamics depends on his mother's health state. Therefore at a given levels of infection and protection, the pandemic force is higher than the natural health state power which thus arise cycles and chaos in opposite to the stable steady state. The spectrum is a stages function after the cross of a given infection gravity. The introduction of that literature inside the one sector growth model highlights the medical production function to use for health care, then the social planner aim is to maximize the utility function of babies and the mothers to ensure life continuity and make population growth reached its stable steady state. Unfortunately, in contrast with the SIS model, the recovery mechanics doesn't work and life fluctuates endogenously. That is the state of evolution in Africa where still prevailing lake of information on that illness evolution and limitations on medical care. The model is useful to understand the way disease such that HIV/AIDS in infant appear in Africa as well as gives tools to understand how to deal with it.

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