How do epidemics induce behavioral changes? *

Raouf Boucekkine[†] Rodolphe Desbordes[‡] Hélène Latzer[§]

July 6, 2007

Abstract

This paper is concerned with the impact of epidemics on economic behavior, and in particular on fertility and schooling. Special attention is paid to the fertility effect, which has been at the heart of a recent controversy around the AIDS crisis. An illustrative model is proposed where agents choose labor supply, life-cycle consumption and the number of children. We show that the optimal response in terms of fertility and labor supply to an epidemic shock depends on the relative strength of two forces at work, deriving from: (i) the induced decrease in the survival probability, and (ii) the impact of epidemics on wages. A comprehensive empirical study is then proposed to disentangle the latter effects in the HIV/AIDS and malaria cases. Using data from 69 developing countries over the period 1980-2004, we find that HIV/AIDS has a robust negative effect on fertility and a robust positive effect on education, while opposite results are found in the case of malaria. We argue that this discrepancy can be attributed to a sizeable wage effect in the AIDS case while such an effect is rather negligible under malaria at least in the short term, as higher malaria prevalence depresses wages in the long term.

*We acknowledge the financial support of the Belgian research programmes PAI P5/10 and ARC 03/08-302. The usual disclaimer applies.

[†]CORE-IRES, Université Catholique de Louvain; University of Glasgow. E-mail: boucekkine@ires.ucl.ac.be.

[‡]Corresponding author. IRES, Université Catholique de Louvain; University of Strathclyde. Address: Place Montesquieu 3, 1348 Louvain La Neuve, Belgium. Telephone number: (0032) (0)10 473 508. E-mail: rodolphe.desbordes@univ-paris1.fr.

[§]IRES, Université Catholique de Louvain. E-mail: latzer@ires.ucl.ac.be.

1 Introduction

Recently, a controversy has been taking place around the fertility impact of HIV/AIDS. While the study of Young (2005a) concerning the South-African case has concluded that the epidemic is decreasing fertility, Kalemli-Ozcan (2006) has identified the opposite effect on a panel of African countries over the 1985-2000 period.[1] An argument often brought forward is that the sexual transmission of such an epidemic would naturally induce a sort of sexual behavior adjustment (more protected relationships, more contraception) which should drive fertility down. However, Kalemli-Ozcan (2006) reviews the medical literature and concludes that uninfected people, or people believing not to be infected, do not modify their fertility-related behaviors: indeed, in the African case, numerous social and political circumstances are particulary unfavorable to awareness of HIV/AIDS (World Bank, 1997). On the other hand, Young (2005b) argues that fertility can drop under HIV/AIDS because this epidemic may induce changes in both sexual *and* contraceptive behaviors. He uses microdata of Sub-Saharan African countries and econometrically finds that higher HIV prevalence leads to an increased use of *all* forms of contraception, suggesting that the behavioral changes induced by the epidemics may not only reflect a willingness to reduce unprotected sexual activity but also to purposely control fertility. The main economic mechanism put forward to explain this change in individual behaviors is highly interesting: the desire of women to have less children may be driven by a rise in the opportunity cost of rearing them. Thus, at the heart of Young's argument lies the idea that similarly to what occurred during the Black Death in the fourteenth century, the heavy labor shortage induced by epidemics like HIV/AIDS should have a significant positive effect on wages, which might well raise female participation in the labor market and ultimately lead to a lower fertility. Set in more theoretical terms in line with Kremer and Chen (2002), it might be the case that the substitution effect induced by higher wages (lowering fertility through the rising opportunity cost of rearing children) ends up dominating the income effect (increasing fertility through a higher amount of resources available to bring up children).

On the other hand, Kalemli-Ozcan's argument is tightly linked to the so-called insurance effect deriving from an increasing uncertainty about children's survival to the adult age (see a related theory in Kalemli-Ozcan (2002)). The perception of an increasing infant mortality may generate a rationale for increasing the number of offsprings. It is already clear that such an argument is

[1] Kalemli-Ozcan (2006) has nevertheless stressed that South-Africa may be a specific case in her empirical study, evoking a "weak negative relation between HIV/AIDS and the total fertility rate" (p.19).

likely to be relevant when the epidemic kills or is supposed to kill a large number of children, which is indisputably the case of malaria. However, epidemics like HIV/AIDS or the Spanish flu (see Brainerd and Siegler (2003) for example) tend to kill many more adults than children.[2] Such a massive adult mortality implies a sizeable labor supply shortage, which in turn gives rise to the wage effect discussed above. While this effect is at the heart of Young's appraisal, it is not at all disentangled in Kalemli-Ozcan's empirical assessment. We argue in this paper that the behavioral impact of epidemics cannot be finely apprehended by only considering decreasing survival rates, whether it be for adults or children. As far as one is concerned with fertility and educational choices under epidemics, the wage effect should be accurately isolated. We will show that such a distinction between mortality and wage effects could be most useful and decisive to understand the impact of epidemics on economic behavior in general, and to get through the current fertility debate around the HIV/AIDS crisis in particular.

We shall proceed in two steps. We first present a simple overlapping generations model depicting the individual behavior modifications in response to an epidemic shock, taking into account the two distinct effects outlined just above: on one hand, the sharp decrease in survival probabilities, and on the other hand a dramatic shortage in labor supply, leading to an increase in real wages. We show that while a decrease in the survival probability always leads to higher fertility and lower labor supply, a positive shock on wages can generate the exact opposite consequences, reducing fertility and raising labor supply. The main mechanism lying behind the "wage effect" at work in our model is exactly the one pointed out by Young (2005a): higher wages increase the opportunity cost of rearing children and can therefore decrease fertility. Actually our model can be seen as a reasonable combination between his static model and the overlapping generations setting proposed by Zhang and Zhang (2005). The "mortality effect" inherent to our theory stems from the diminishing need for life-cycle consumption with respect to fertility. A lower survival probability to the senior age decreases the need for life-cycle consumption and increases the desired number of offsprings. Clearly this is neither the infant mortality effect nor the insurance mechanism outlined by Kalemli-Ozcan (2002) but it yields exactly the same outcome, opposite to the wage effect under certain conditions that will be specified along the way. Hence, our simple model perfectly illustrates that, in the presence of the two competing effects described above, the overall impact of an epidemic shock on fertility (and on labor supply) is not straightforward,

[2] Recent demographic projections (see for example, the 2004 UNAIDS annual report) are nevertheless showing quite an alarming trend for HIV-related infant mortality in some Sub-Saharan African countries for the next two decades. However, it is undisputable that the vast majority of AIDS-related deaths are active adults.

and primarily depends on the relative strength of the two forces at work. It should be already noted that for simplicity we do not model explicitly schooling decisions. Of course, one can straightforwardly figure out the same competing mortality vs wage effects on this decision: lower life expectancy is associated to lower education through the Ben Porath mechanism (Boucekkine et al., 2002), while higher wages would induce opposite effects.

In the second step, we conduct a comprehensive empirical analysis to disentangle the impacts of the two forces in the case of two distinct epidemics: HIV/AIDS and malaria, another infectious disease plaguing the African world. By checking the behavioral effects of HIV/AIDS on a sample of 69 developing countries over the 1980-2004 period, and after taking into account outliers, reverse causality, interdependencies and the potential existence of an African specificity, we find that a higher HIV prevalence has a negative effect on both life expectancy and fertility, while it has a positive and robust effect on education. The conjecture that the "wage channel" seems to dominate is then further strengthened by testing for the actual impact of the epidemics on the evolution of wages: we find that HIV prevalence indeed exerts a positive direct impact on wages. On the other hand, we find that malaria, although having as well a negative impact on life expectancy, has a negative impact on education and a positive effect on fertility. Such results rather suggest that the wage effect is tenuous under malaria or that malaria has a negative impact on wages. We find that malaria has no significant effect on wages in the short run and a negative effect in the long run. The lack of short-run impact should not be regarded as a surprising result since malaria mostly kills children in contrast to AIDS, which massively hits active adults at first, and thus affects much more directly labor supply. However, in the long term, chronic malaria prevalence reduces both economic activity and labor demand, causing a drop in wages. In addition to the cognitive and learning effects of malaria, this may explain why educational attainment remains persistently low in countries chronically exposed to malaria.

To summarize, the original contributions of this paper are twofold. On the theoretical ground, and though our model is illustrative to a large extent, it provides a useful benchmark for the study of epidemics. As we have already mentioned, the model can be closely linked to the literature on life expectancy and growth. While a comprehensive theoretical analysis of the relationship between life expectancy, education and fertility has already been provided by Hazan and Zoabi (2006), our model contributes to the literature by tackling the problem of behavioral answer to an epidemic shock and studying in a transparent and tractable way the *combined* impact of a shorter survival probability *and* a positive effect on wages. On the empirical side, the contributions are

markedly more significant. In the particular case of HIV/AIDS, we contribute to the fertility debate. By closely examining the behavioral impacts of shorter lives and higher wages within the same framework, we are able to integrate the opposite effects on fertility put forward by Young and Kalemli-Ozcan. Our results confirm the intuitions of Young (2005a). Incidentally, we develop a more global approach to the assessment of behavioral impacts of epidemics by testing for behavioral consequences of malaria, which broadens the scope of analysis to another type of epidemics, and exemplifies possible divergences in the impact on individual behavior of life-threatening diseases.

The paper is organized as follows. Section 2 presents our simple theoretical model and its predictions concerning rational behavior under an epidemic shock. Section 3 describes the empirical tests conducted and reports the results obtained. Section 4 concludes.

2 Rational behavior under epidemics: a simple theory

We start presenting a benchmark model illustrating how individual behaviors could be altered by epidemic shocks. In order to gather enough ingredients to comprehensively tackle the HIV/AIDS overall impact on fertility, we shall consider as explained in the introduction that epidemics have two distinct direct effects on the individuals. First, the "mortality effect" consists in a decrease in survival probability of individuals, consistently with decreasing life expectancy patterns associated with AIDS-like epidemics.[3] Second, the "wage effect" lies in the increase in real wages induced by the heavy shortage in labor supply resulting from massive epidemics like the Black Death or AIDS. This positive effect on wages is not only emphasized by Young (2005a) in his inspection of the South African AIDS tragedy, but is also commonly admitted by sociologists and historians (Herlihy, 1997) concerning epidemics such as the Black Death.[4]

In this section, we shall in particular characterize optimal fertility and labor supply behavior under epidemic shocks triggering the two direct effects mentioned above. The illustrative model presented here can be seen as a reasonable combination of the Young (2005a) static model and Zhang and Zhang (2005) overlapping generations model. It is however important to signal right away that in order to keep this analytical part as algebraically transparent as possible, we don't explicitly allow for the traditional trade-off "quality vs quantity of children" that is present in both

[3] It goes without saying that such a feature does not fit into episodes of short-lived epidemics such as the Spanish flu. See a modeling of the latter in Boucekkine et al. (2007).

[4] This positive effect on wages is actually even acknowledged by economy textbooks (Mankiw, 2005).

mentioned models through the educational choices of the parents. For the sake of computational clarity, we here choose to focus on the fertility decision (quantity of children), not including an education variable. One should however notice that adding such a trade-off to our theoretical set-up would simply deliver the typical outcome that in response to the epidemic shocks, parents move the quality and quantity of children in opposite directions. We hence keep things as simple as possible, since our aim in this theoretical part is merely illustrative: we wish to isolate as clearly as possible the respective impacts of the two competing direct effects we identified on fertility. In other words, how could fertility and labor supply respond to a simultaneous rise in wages ("wage effect") and decline in life expectancy ("mortality effect")? Implications for optimal education decisions will then result quite naturally, as we will see in the concluding sub-section.

2.1 The model

The model is a 3 periods, one good overlapping generations model. An individual born in period t spends all his time endowment (say one unit of time) of his first life period (childhood), that is period t, having leisure. In period $t+1$, he becomes for sure a young adult (no child mortality). A young adult consumes c_{t+1}, has n_{t+1} children, works a proportion l_{t+1} of his unit time endowment, paid at an exogenous wage per unit of time w_{t+1}, and saves s_{t+1} for consumption in his old age (if he survives). As usual, rearing children costs a proportion θn_{t+1} of time endowed, with $\theta > 0$. The young adult becomes a senior adult with probability p. A senior adult consumes c_{t+2} out of the savings made in $t+1$. A classic feature in this kind of framework is then to assume the existence of an annuity market which guarantees that survivors get the savings plus interests of the young adults who die before reaching the seniority. Accordingly, the return rate to savings is given by $\frac{R_{t+2}}{p}$.

As already signaled and justified, with respect to Zhang and Zhang (2005), one can notice that we omit schooling and human capital accumulation. Indeed, introducing them would result in a system of 7 equations as opposed to 5 below: the economic gains from such an extension are completely overshadowed by the algebraic cost. Instead we choose to stick to a benchmark model, more adapted to the illustrative purpose of this section. A more fundamental difference that we will comment now comes from preferences, whose specification is closer to the spirit of Beckerian behavioral models such as the one developed by Young (2005a). Indeed, we assume that the preferences of an individual of generation t are given by:

$$U\left(c_{t+1}, n_{t+1}, l_{t+1}, c_{t+2}\right) = \frac{c_{t+1}^{1-\sigma_c}}{1-\sigma_c} + \alpha_1 \frac{n_{t+1}^{1-\sigma_n}}{1-\sigma_n} + \alpha_2 \frac{\left(1-\theta n_{t+1} - l_{t+1}\right)^{1-\sigma_l}}{1-\sigma_l} + p\,\alpha_3 \frac{c_{t+2}^{1-\sigma_c}}{1-\sigma_c}$$

where α_i, $i = 1, 2, 3$, are strictly positive constants, and σ_c, σ_n and σ_l, are the usual positive elasticity parameters respectively related to consumption (either "young" or "old" consumption), number of children and leisure.[5]

Hence, consistently with Young's static preferences, and since labor supply response to epidemic shocks is a fundamental aspect of our paper, we introduce disutility of working (and rearing children) in the second period of life: young adults enjoy leisure, and working and rearing children reduces their utility. In contrast, Zhang and Zhang (2005) introduce such a leisure term for children, necessary to their purpose of featuring educational choices.

Finally, the general iso-elastic specification for utility terms is aimed for generality. As we shall see later, some important behavioral implications of epidemics do depend on the elasticity parameters.

The budgetary constraints for periods $t+1$ and $t+2$ are as follow:

$$c_{t+1} + s_{t+1} = w_{t+1}\, l_{t+1}, \tag{1}$$

$$c_{t+2} = \frac{R_{t+2}}{p}\, s_{t+1}, \tag{2}$$

inducing the intertemporal budgetary constraint

$$c_{t+1} + \frac{p}{R_{t+2}}\, c_{t+2} = w_{t+1}\, l_{t+1}. \tag{3}$$

Optimal behavior is obtained by maximization of the utility function with respect to the four decision variables $(c_{t+1}, n_{t+1}, l_{t+1}, c_{t+2})$ under the constraint (3). The resulting optimization problem is as follows. Call λ_{t+1} the Lagrange multiplier associated to (3). The first-order

[5] Of course, in case one of these elasticity parameters is equal to 1, the corresponding utility term becomes logarithmic.

conditions with respect to the four variables above in this order are:

$$c_{t+1}^{-\sigma_c} = \lambda_{t+1}, \tag{4}$$

$$\theta\ \alpha_2\ (1-\theta n_{t+1}-l_{t+1})^{-\sigma_l} = \alpha_1\ n_{t+1}^{-\sigma_n}, \tag{5}$$

$$\alpha_2\ (1-\theta n_{t+1}-l_{t+1})^{-\sigma_l} = \lambda_{t+1}\ w_{t+1}, \tag{6}$$

$$p\ \alpha_3\ c_{t+2}^{-\sigma_c} = \frac{p}{R_{t+2}}\ \lambda_{t+1}. \tag{7}$$

Characterizing optimal behavior amounts to solving the system (3) to (7) in the five variables $(c_{t+1}, n_{t+1}, l_{t+1}, c_{t+2}, \lambda_{t+1})$, all strictly positive, under the time resource constraint, $\theta n_{t+1}+l_{t+1} < 1$, and for given w_{t+1} and R_{t+2}. The first proposition shows that this problem has a unique solution.

Proposition 1 *The system (3) to (7) has a unique solution in* $(c_{t+1}, n_{t+1}, l_{t+1}, c_{t+2}, \lambda_{t+1})$, *all strictly positive, satisfying* $\theta n_{t+1} + l_{t+1} < 1$.

Proof: Combining (3), (4) and (7), one can find

$$c_{t+1} = \frac{w_{t+1}\ l_{t+1}}{1+p\ \alpha_3^{\frac{1}{\sigma_c}}\ R_{t+2}^{\frac{1}{\sigma_c}-1}}. \tag{8}$$

Now, combining (5) and (6), one gets

$$\alpha_1\ n_{t+1}^{-\sigma_n} = \theta\ \lambda_{t+1}\ w_{t+1},$$

which yields by (4) and (8)

$$\alpha_1\ n_{t+1}^{-\sigma_n} = \frac{\theta\ w_{t+1}^{1-\sigma_c}}{\left(1+p\ \alpha_3^{\frac{1}{\sigma_c}}\ R_{t+2}^{\frac{1}{\sigma_c}-1}\right)^{-\sigma_c}}\ l_{t+1}^{-\sigma_c}. \tag{9}$$

Now, it is straightforward to see that the proposition is done if we prove that the system (5)-(9) admits a unique solution in n_{t+1} and l_{t+1} satisfying $\theta n_{t+1}+l_{t+1} < 1$. Using (9) to express l_{t+1} as

a function of n_{t+1}, and substituting this function in (5), we get a single equation in n_{t+1}, which is fundamental to our purposes:

$$\alpha_1 \, n_{t+1}^{-\sigma_n} = \theta \, \alpha_2 \left[1 - \theta \, n_{t+1} - \Omega_{t+2}^{-\frac{1}{\sigma_c}} \, w_{t+1}^{\frac{1}{\sigma_c}-1} \, n_{t+1}^{\frac{\sigma_n}{\sigma_c}} \right]^{-\sigma_l}, \tag{10}$$

where $\Omega_{t+2} = \frac{\alpha_1}{\theta} \left(1 + p \, \alpha_3^{\frac{1}{\sigma_c}} \, R_{t+2}^{\frac{1}{\sigma_c}-1} \right)^{-\sigma_c}$. Denote by $\bar{n}_{t+1}$, the number of children satisfying the equality (implying zero leisure): $\theta \bar{n}_{t+1} + \Omega_{t+2}^{-\frac{1}{\sigma_c}} \, w_{t+1}^{\frac{1}{\sigma_c}-1} \, \bar{n}_{t+1}^{\frac{\sigma_n}{\sigma_c}} = 1$. On the interval $(0; \bar{n}_{t+1})$, the left-hand side of (10) is a strictly decreasing function from infinity to $\alpha_1 \, \bar{n}_{t+1}^{-\sigma_n}$ while the right-hand side is increasing from $\theta \, \alpha_2$ to infinity. Therefore, they should be equal at a single point comprised in the interval $(0; \bar{n}_{t+1})$. This ends the proof. □

We now study the impact of epidemics on the optimal decisions featured in Proposition 1, i.e. the effects on individual behavior of a drop in the survival probability p and an increase in the wage.

2.2 Optimal fertility and labor supply under epidemics

A quick look at equation (10) is enough to identify a special case: when the utility term with respect to consumption is logarithmic, that is when $\sigma_c = 1$, wages are no longer a determinant of optimal fertility and labor supply. In other words, the "wage effect" is no longer active in this case. This is by no means a surprise. Higher wages traditionally induce 2 well-identified, competing effects: a positive income effect, increasing both consumption and leisure (and thus decreasing labor supply), and a substitution effect stemming from the increase in the opportunity cost of leisure, favorable to labor supply. These 2 opposite effects have the same magnitude when $\sigma_c = 1$, and thus just offset each other in such a parametric case. Since labor supply is then unaffected, so is the fertility decision given the optimality condition (5).

In this case, the "mortality effect" is the only one left, and we are thus able to identify its specific impact. The next proposition hence provides the optimal response of both labor supply and fertility to a decreasing survival probability, p.

Proposition 2 *Assume $\sigma_c = 1$. Then optimal labor supply and fertility are independent of the wage, w_{t+1}. A decrease in the survival probability p always raises fertility n_{t+1} and reduces labor supply, l_{t+1}.*

Proof: The proof is rather simple, even for the second part of the proposition. Indeed, a change in p does not affect the left hand side of (10), it only affects the right-hand side through the term Ω_{t+2}. Because

$$\Omega_{t+2}^{-\frac{1}{\sigma_c}} = \left(\frac{\alpha_1}{\theta}\right)^{-\frac{1}{\sigma_c}} \left[1 + p\ \alpha_3^{\frac{1}{\sigma_c}}\ R_{t+2}^{\frac{1}{\sigma_c}-1}\right],$$

it follows that a drop in the survival probability p will decrease the right-hand side of equation (10). Because the left-hand side is unaffected, and the right-hand side is increasing in n_{t+1}, the equality (10) is re-established if and only if optimal fertility rises. To get the property relative to labor supply, we can use a similar argument. First obtain the corresponding single equation in l_{t+1} combining (5) and (9):

$$\frac{\alpha_1}{\Omega_{t+2}}\ w_{t+1}^{1-\sigma_c}\ l_{t+1}^{-\sigma_c} = \theta\ \alpha_2\ \left[1 - \theta\Omega_{t+2}^{\frac{1}{\sigma_n}}\ w_{t+1}^{\frac{\sigma_c-1}{\sigma_n}}\ l_{t+1}^{\frac{\sigma_c}{\sigma_n}} - l_{t+1}\right]^{-\sigma_l}, \tag{11}$$

which implies when $\sigma_c = 1$:

$$\frac{\alpha_1}{\Omega_{t+2}}\ l_{t+1}^{-\sigma_c} = \theta\ \alpha_2\ \left[1 - \theta\Omega_{t+2}^{\frac{1}{\sigma_n}}\ l_{t+1}^{\frac{\sigma_c}{\sigma_n}} - l_{t+1}\right]^{-\sigma_l}, \tag{12}$$

Then apply the same kind of reasoning as just above on (10). Focusing on equation (12), one can see that the left-hand side is shifted downwards when p drops. Since the function in the left-hand side is decreasing in l_{t+1}, this means that l_{t+1} has to decrease to re-establish equation (12) for an unchanged right-hand side. In contrast to the fertility analysis above, things are apparently more complicated because both sides of the equation (12) are indeed altered. A drop in p also causes the right-hand side to shift upwards, via the term $\Omega_{t+2}^{\frac{1}{\sigma_n}}$, inducing an additional downward move in l_{t+1} since the right-hand side is increasing in this variable. This ends the proof. □

Our results generalize those of Zhang and Zhang (2005) to a certain extent.[6] As in the latter paper, the reduction in survival probability diminishes the need for life-cycle consumption with respect to fertility, which would straightforwardly imply an increase in fertility and a decrease in labor supply **in the absence of disutility of working and rearing children**. In our model, the disutility of working and rearing children is present, and the properties still hold, which ultimately shows their robustness. Actually, as one can trivially infer from the proof of the proposition above, they also hold for any elasticity parameter σ_c.

[6] see their Proposition 3, page 50.

Proposition 3 *For any σ_c positive, a decrease in the survival probability p always raises fertility n_{t+1} and reduces labor supply, l_{t+1}.*

Things are apparently much trickier once we allow for the "wage effect" to be active, i.e. for $\sigma_c \neq 1$. The next proposition summarizes the associated properties.

Proposition 4 *For any σ_c positive and* **not equal to** 1*, an increase in the wage w_{t+1} raises labor supply l_{t+1} and reduces fertility n_{t+1}, if and only $\sigma_c < 1$.*

Proof: Recall equation (10):

$$\alpha_1 \, n_{t+1}^{-\sigma_n} = \theta \, \alpha_2 \left[1 - \theta \, n_{t+1} - \Omega_{t+2}^{-\frac{1}{\sigma_c}} \, w_{t+1}^{\frac{1}{\sigma_c}-1} \, n_{t+1}^{\frac{\sigma_n}{\sigma_c}} \right]^{-\sigma_l}.$$

While the left-hand side is unaffected by wages, the right-hand side is. One can trivially see that the direction of the shift induced by an increase in wages is entirely determined by the position of σ_c with respect to 1. If (and only if) $\sigma_c < 1$, an increment in wages increases the right-hand side, which leads to a drop in optimal fertility to re-establish equation (10).

Things are slightly more complicated for labor supply's optimal response since both sides of equation (11) are affected by increases in wages. Indeed recall this equation:

$$\frac{\alpha_1}{\Omega_{t+2}} \, w_{t+1}^{1-\sigma_c} \, l_{t+1}^{-\sigma_c} = \theta \, \alpha_2 \, \left[1 - \theta \Omega_{t+2}^{\frac{1}{\sigma_n}} \, w_{t+1}^{\frac{\sigma_c - 1}{\sigma_n}} \, l_{t+1}^{\frac{\sigma_c}{\sigma_n}} - l_{t+1} \right]^{-\sigma_l}.$$

Suppose the wage w_{t+1} is rising and $\sigma_c < 1$. The left-hand side is shifted upwards, which induces labor supply to increase to re-establish the equality (since, again, the left-hand side is decreasing in l_{t+1}). However, the right-hand side is also affected: it is actually shifted downwards, which again induces a further increase in labor supply since the right-hand side is increasing in l_{t+1}. We get the opposite picture if $\sigma_c > 1$. □

The obtained results deserve a careful interpretation. An increase in wages induces a classic positive income effect, which tends to increase consumption (in both periods), leisure and the number of children. However, an increase in the number of children and an increase in leisure are detrimental to each other : henceforth, the positive income effect has a non-trivial impact on fertility. On the other side, a higher wage increases the opportunity cost both of leisure and of rearing children. We thus get the typical opposition between income and substitution

effects *à la* Kremer and Chen (2002), which has in general an ambiguous effect on fertility.[7] A key departure from Kremer and Chen (2002) is the intertemporal nature of our model. This characteristic is crucial since it will readily enable us to understand the preeminent role of the preference parameter σ_c. Indeed, in our story, individuals can take advantage of this wage increase in $t+1$ **and only in** $t+1$: by increasing their labor supply to take advantage of higher wages in $t+1$, they can transfer consumption to their old age in $t+2$. Since the strength of intertemporal substitution in consumption is measured by $\frac{1}{\sigma_c}$, the lower σ_c, the more individuals will be willing to transfer consumption to $t+2$, the more they will work, and the lower leisure and fertility (via the needed reduction in the time devoted to rearing children). In our simple model, the "threshold" value for σ_c is just one, that is logarithmic preferences in consumption (in both ages): below this threshold, individuals work more and have less children, and above, we have the opposite picture.

Finally note that the simple result obtained (which itself derives from the simplicity of the model) does not mean that the other elasticity parameters are unimportant. One can for example notice that if σ_l, the elasticity of (marginal) utility of leisure with respect to the level of leisure, is increasingly large, the magnitude of the increase in labor supply will definitely get lower (in the case $\sigma_c < 1$). We will however not comment further, and focus now on the implications of our theoretical model concerning the HIV/AIDS fertility debate.

2.3 Summary and relation to the AIDS-fertility ambiguity

Our simple model neatly illustrates why an epidemic shock has an ambiguous effect on fertility. Indeed, if we interpret an epidemic shock as having direct consequences on both survival probabilities and wages (via the associated large cuts in labor supply), then our model shows that the **total** effect on fertility is *a priori* ambiguous. On one side, a drop in the survival probability has the same implications for fertility as the well-known insurance effect put forward by Kalemli-Ozcan (2002), i.e. a rationale for fertility to increase in response to epidemics. On the other side, under certain conditions, the increase in wages resulting from labor shortage has the exact opposite effect on fertility, i.e. decreasing it.

Whether the wage effect, neatly identified in our theory, can actually more than offset the mortality effect on fertility is a crucial question that should deserve the maximal attention. To go

[7] It should be noted that Kremer and Chen (2002) directly choose a quasi-linear utility function for the substitution effect to dominate the income effect.

even further, we do believe that disentangling the wage vs mortality effects on fertility is not only important to understand and quantitatively assess the economic and demographic impacts of the current HIV/AIDS crisis, but also to study accurately and meaningfully any other epidemic. In our empirical work, we shall thus also investigate the malaria case to bring out an additional insight into this approach. As we will see, this method, by shedding light on some possible divergences in the impact on individual behavior of two life-threatening diseases, will enable us to further comment on the two effects at work in an epidemic shock (i.e. the "wage effect" and the "mortality effect").

Last but not least, it is of utmost importance to note that one can perfectly extend the analysis conducted on fertility (and labor supply) to education. As already stated, if the typical trade-off quality vs quantity of children were introduced in the model, the wage effect of epidemics would have implied an increasing quality of children (that is, more education) as the number of children goes down. Hence, the increase in the opportunity cost of rearing children, directly induced by the rise in wages, should *increase* educational attainment along with *decreasing* the fertility rate. The mortality effect yields the opposite outcomes. Again, the total effect on education depends on the strength of the wage effect relative to the mortality effect. Our empirical section hence also investigates this crucial aspect of the ongoing debate on HIV/AIDS.

Hence, our empirical section will now be devoted to studying the impact of both malaria and HIV prevalence on the individual behavioral patterns that are fertility and education.

3 Empirical testing

3.1 Impacts of HIV and malaria on life expectancy, education and fertility

We first present the tested equations, variables of interest, control variables and specifications. We then check the robustness of our results before commenting them extensively.

3.1.1 Empirical model and data

In order to check for the behavioral effects of HIV and malaria prevalence, we estimate three equations separately with education, life expectancy and fertility rate being the three explained

variables:

$$\begin{aligned}\text{Life expectancy}_i^t &= \alpha_0 + \alpha_1 \text{ HIV prevalence}_i^t + \alpha_2 \text{Malaria prevalence}_i + \alpha_3 \text{Education}_i^t + \\ &\quad \alpha_4 \text{Ln(Real GDP per capita)}_i^t + \alpha_5 \text{Ln(Calories per day)}_i^t + \\ &\quad \alpha_6 \text{Ln(Calories per day)}^{2t}_i + \alpha_7 \text{Political competition}_i^t + \\ &\quad \alpha_8 \text{Conflict occurrence}_i^t + \alpha_9 \text{C}_i + \alpha_{10} \text{T}^t + \epsilon_i^t \end{aligned} \tag{1}$$

where C_i and T^t are respectively country-specific and time-specific fixed effects, while ϵ_i^t is the error term.

$$\begin{aligned}\text{Education}_i^t &= \beta_0 + \beta_1 \text{ HIV prevalence}_i^t + \beta_2 \text{Malaria prevalence}_i + \beta_3 \text{Life expectancy}_i^t + \\ &\quad \beta_4 \text{Ln(Real GDP per capita)}_i^t + \beta_5 \text{Political competition}_i^t + \\ &\quad \beta_6 \text{Women economic rights}_i^t + \beta_7 \text{Conflict occurrence} + \beta_8 \text{C}_i + \beta_9 \text{T}^t + \varepsilon_i^t \end{aligned} \tag{2}$$

where ε_i^t is the error term.

$$\begin{aligned}\text{Fertility rate}_i^t &= \gamma_0 + \gamma_1 \text{ HIV prevalence}_i^t + \gamma_2 \text{Malaria prevalence}_i + \gamma_3 \text{Life expectancy}_i^t + \\ &\quad \gamma_4 \text{Education}_i^t + \gamma_5 \text{Ln(Real GDP per capita)}_i^t + \gamma_6 \text{Conflict occurrence} + \\ &\quad \gamma_7 \text{C}_i + \gamma_8 \text{T}^t + \upsilon_i^t \end{aligned} \tag{3}$$

where υ_i^t is the error term.

αs, βs and γs only measure the direct effects of HIV prevalence, malaria prevalence, life expectancy and education, without taking into account interdependencies. For instance, the impact of HIV prevalence on life expectancy may be lower than α_1 if the spread of the HIV epidemic contributes to raising average educational attainment. After substituting equation (1) in (2) and *vice versa*, the total effects of HIV prevalence on life expectancy, education and fertility are respectively: $\frac{(\alpha_3\beta_1)+\alpha_1}{1-(\alpha_3\beta_3)}$, $\frac{(\beta_3\alpha_1)+\beta_1}{1-(\alpha_3\beta_3)}$ and $\gamma_1 + \gamma_3\frac{(\alpha_3\beta_1)+\alpha_1}{1-(\alpha_3\beta_3)} + \gamma_4\frac{(\beta_3\alpha_1)+\beta_1}{1-(\alpha_3\beta_3)}$. Similarly, the total effects

of malaria on life expectancy, education and fertility are respectively: $\frac{(\alpha_3\beta_2)+\alpha_2}{1-(\alpha_3\beta_3)}$, $\frac{(\beta_3\alpha_2)+\beta_2}{1-(\alpha_3\beta_3)}$ and $\gamma_3\frac{(\alpha_3\beta_2)+\alpha_2}{1-(\alpha_3\beta_3)} + \gamma_4\frac{(\beta_3\alpha_2)+\beta_2}{1-(\alpha_3\beta_3)}$.

Variables of interest

Life expectancy at birth indicates the number of years a new-born infant would live if prevailing patterns of mortality at the time of his birth were to stay the same throughout his life. Fertility rate is the number of children that a woman would have if she lived through all of her child-bearing years and experienced the current age-specific fertility rates at each age. Data on life expectancy and fertility come from the United Nations Population Division (2007). Education is measured by average schooling years in population aged 25 or over. Data come from Baier et al. (2006).[8] Since it is not available for consecutive years, missing values have been filled by linear interpolation. Data on HIV prevalence in adult population, the percentage of people aged 15-49 who are infected with HIV, have been confidentially obtained from UNAIDS. Though estimations of HIV prevalence in the adult population are mostly based on HIV prevalence in pregnant women, which may not be a representative sample, Walker et al. (2003) show that both prevalence rates are fairly close and Young (2005b) does not find that using antenatal infection rates instead of community infection rates causes a bias. Finally, malaria prevalence has been calculated by Gallup and Sachs (2001) using a 1994 World Health Organization map of the geographical extent of high malaria risk and detailed data on the world population distribution. It represents the fraction of the population living in high malaria prevalence areas in 1994.

Control variables

In terms of control variables, life expectancy is expected to depend on education, real income per capita, prevalence of undernourishment, political competition and conflict occurrence.

Well-educated individuals are more likely to adopt the behaviors that improve their health or the well-being of those under their responsibility, i.e. babies and children (Grossman, 2004; Cutler et al., 2006).

A high income supports the public and private per capita expenditures on goods and services which contribute to better health: public health services, private health care, medicines and shelter (Pritchett and Summers, 1996; Fuchs, 2004). Real GDP per capita in constant 2000 US$ from the Penn World Tables (Heston et al., 2006) is used since the purchasing power of US$1

[8] This dataset has been chosen in place of the most commonly used Barro-Lee (Barro and Lee, 2000) and Cohen-Soto (Cohen and Soto, 2007) datasets because it provides the highest spatio-temporal coverage. However it will be seen in the next section that results are robust to changes in the education variable.

widely differs across countries.

An adequate supply of calories should make an individual less vulnerable to sickness and, in the case of childbearers, it should foster a normal foetal development, which reduces the incidence of factors affecting mortality in adulthood (Fogel, 1994; Cutler et al., 2006). However, an excessive supply of calories may put health at risk (Hamoudi and Sachs, 1999a; U.S. Department of Health and Human Services, 2005). Hence, both the number of calories per day and the number of calories per day squared are introduced in order to take into account this quadratic relationship. Data come from the FAO Statistics Division[9] and since it is also not available for consecutive years, missing values have been filled by linear interpolation.

In democracies, voters and opposition parties who are unsatisfied with the policies of the current leader can withdraw their political support; at elections voters can replace him with a more attractive political opponent and during the political term, opposition parties can approve only consensual policies. Confronted to high political competition, a politic leader must thus satisfy a large fraction of the electorate to remain in power, which should favor spending on non-exclusive and universally demanded public goods, such as health or education (Pinto and Timmons, 2005; Stasavage, 2005). Political competition is measured by the Vanhanen competition index (Vanhanen, 2000).[10]It is calculated by subtracting the percentage of votes won by the largest party from 100[11] and ranges from 0, in the case of a non-elected government, to a cutting point of 70%.

An intra-territory war can reduce life expectancy by directly causing death or by destroying public infrastructures and economic activity (Collier et al., 2003). The armed conflicts dataset developed by Uppsala University (Eriksson and Wallensteen, 2004) provides a measure of the intensity and length of a territorial conflict. The conflict occurrence variable takes the value of *1* if a minor armed conflict occurs, i.e. at least 25 battle-related deaths per year and fewer than 1000 battle-related deaths during the course of the conflict, *2* if an intermediate armed conflict

[9] `http://www.fao.org/faostat/foodsecurity/`

[10] Other measures of political competition exist but they tend to be cruder. For instance the "PARCOMP" variable of the Polity dataset (Marshall and Jaggers, 2002) varies between 1 (oppositional activity is repressed) and 5 (competitive political participation). Both variables are nevertheless highly correlated, with a correlation coefficient of 0.80. Correlations with the combined Polity score or the Freedom House Political Rights indicator (Freedom House, 2005) are even higher: 0.85 and 0.82, respectively.

[11] In parliamentary elections "the largest party" refers to the party which received the largest single share of the votes or of the seats in parliament, and in presidential elections "the largest party" refers to the votes received by the presidential candidate who won the election. The index tries to correct for differences in electoral systems and political regimes, by taking into account the dominant governmental institutions (executive, legislature or both) and the electoral system (majority or proportional).

occurs, i.e. at least 25 battle-related deaths per year and at least 1000 battle-related deaths during the course of the conflict and *3* if a war occurs, i.e at least 1000 battle-related deaths per year. For ease of interpretation the variable has been re-scaled from range 0 to 1.

Besides HIV prevalence, malaria prevalence and life expectancy, probable determinants of education are real income per capita, economic rights of women, political competition and conflict occurrence. Access to public or private provision of education is likely to increase with real income per capita and expected economic growth, by raising the private return to investing in schooling, may induce greater schooling (Bils and Klenow, 2000). Even if theoretically they have access to education, women are unlikely to acquire new skills if they do not have any economic rights e.g. if they cannot work/choose freely their occupation without the consent of their husband or a male's relative and if they are otherwise highly discriminated in the workplace. The Cingranelli and Richards (2007) Women Economic Rights indicator provides a measure of gender inequality in access to work, varying between 0 and 3 (from none to strong). For ease of interpretation the variable has been rescaled from range 0 to 1. For reasons previously given, political competition may increase public provision of education whereas conflict occurrence can hinder access to education.

In addition to HIV prevalence, life expectancy and education, fertility should be influenced by real income per capita and conflict occurrence. The net costs of childrearing, e.g. housing, nursing and training costs, tend to be higher in more developed countries (Becker, 1992) and conflict occurrence is likely to decrease the willingness or the feasibility of having children since current and future upbringing conditions may not be (seen as) optimal (Agadjanian and Prata, 2001).

Specifications

Time and country effects are also included. Time dummies control for unobserved time-specific factors common to all countries. Country-specific effects capture factors which do not vary much over the period observed and which may have been omitted. Following standard practices and previous papers, most variables appear in their original form.[12] Box-Cox tests

[12]Variables measured in years, variables which are shares or percent and indexes tend to be untransformed as it facilitates interpretation of results. Variables strongly dominated by zero values, like HIV prevalence rate, should not be transformed. Variables which are large positive integer values usually appear in logarithms.

validate the choice of these specifications, especially for the fertility specification, and it facilitates interpretation of the results. However, not taking logs can increase the sensitivity of estimates to influential observations, especially when variables heavily skewed like HIV prevalence are included. Hence, thanks to a Cook's D test, influential outliers have been removed from the sample. The final sample is composed of 69 developing countries over the 1980-2004 period, with one observation per country, when available, every five year period.[13] The choice of quinquennia has been mainly driven by the fact that the U.N. population division only provides five year averages of some key variables such as life expectancy and fertility rate. Furthermore, the yearly variation of interpolated variables, like education or caloric intake, is relatively meaningless and taking averages may reduce the effect of short-run fluctuations. Summary statistics are given in table 1.

Variable	Mean	Standard Deviation	Minimum	Maximum
Life expectancy	59.23	9.84	39.2	78.10
Education	4.64	2.39	0.4	10.27
Fertility rate	4.79	1.72	1.24	8.5
HIV prevalence (%)	1.61	3.42	0	23.76
Malaria prevalence	0.6	0.43	0	1
Ln(Real GDP per capita)	7.81	0.92	5.68	9.74
Ln (Calories per day)	7.76	0.14	7.41	8.12
Political Competition	28.48	20.45	0	70
Conflict occurrence	0.17	0.31	0	1
Women economic rights	0.37	0.15	0	0.87
	Observations: 319			

Table 1: Summary statistics

Equations 2 to 3 explicitly account for circular causality between life expectancy and education, and all dependent variables may be endogenous to other explanatory variables. A likely candidate is income per capita. Income per capita tends to rise when the health and the education of the population improves and population growth slows down (Ahituv, 2001; Cohen and Soto, 2004; Bloom et al., 2004). The HIV prevalence rate may also be endogenous. For instance, educated people may know better the risks associated with unprotected sex (Kim, 2006), leading the coefficient of HIV prevalence to be negatively biased. To correct this simultaneity bias in the context of panel data models, when no obvious external instruments are available, internal instruments based on the lags of the instrumented variables can be used. This can be done through

[13] The five year periods are 1980-1984, 1985-1989, 1990-1994, 1994-1999 and 2000-2004.

the application of the generalized methods of moments (GMM) (Arellano and Bover, 1995; Blundell and Bond, 1998). In a nutshell, each equation is simultaneously estimated in first difference and level by using a system estimation.[14] To ensure consistent estimates of the coefficients, the equation in first-difference (level) is instrumented by the lagged levels (first-differences) of the endogenous variables. In order not to overfit the instrumented variables, which may bias the results towards those of feasible generalized least squares, the GMM instruments are restricted to period $t-2$ $(t-1)$lagged levels (first-differences) of each variable for the first-difference (level) equation.[15] Under the assumptions that these lagged values of the variables are not correlated with the error term and that the error term is not serially correlated, they should be appropriate instruments. The validity of the instruments will be tested through Hansen (1982) J tests of overidentifying restrictions and an Arellano and Bond (1991) test of serial correlation of the differenced error term.

All variables are instrumented in that way, besides malaria prevalence. Since this measure is time-invariant, external instruments are used. Quick reproduction of the malaria parasite and its vector, the *Anopheles* mosquito, requires a hot and humid stable climate (Bloom and Sachs, 1998). A series of first-stage regressions using Köppen Climate Classification data from CIESIN (2002) show indeed that malaria prevalence is higher in countries with a large percentage of land classified in tropical zones whereas the opposite tends to be true in countries with a large percentage of land classified in temperate zones.[16] In addition, Kiszewski et al. (2004) have developed an index of malaria ecology, highly significant in first-stage regressions, which combines in a single measure temperature, mosquito abundance, and vector specificity. Overall when both climatic measures and the more elaborate index of malaria ecology are included, first-

[14] The three equations can be seen as a system of simultaneous equations, implying that ϵ_i^t, ε_i^t, ν_i^t are correlated. In that case, simultaneously estimating them could provide more efficient estimates as correlation in the errors across equations are taken into account. A common system estimation method is three stage least squares. However, in order to deal with both the country-specific effects and the absence of obvious and time-varying instruments, only equations in differences, in which variables of interest are instrumented with their lagged level values, could be exploited, which may generate biased and inefficient estimates if past levels provide little information about future changes. Though "GMM-style" instruments could be constructed, using lagged values would reduce sample size, and by extension, efficiency. Control explanatory variables which are endogenous would have to be instrumented too, which increases the risk of inconsistency of the estimates if one equation is misspecified. Finally, using differences would preclude the inclusion of the time-invariant malaria measure. Hence, estimating each equation separately appears *in fine* a more robust and adequate estimation procedure.

[15] Roodman (2004) indicates that a rule of thumb is that the number of instruments should not exceed the number of countries in the regression. Note that instrumenting with lagged values requires at least three observations by country. Hence, countries not meeting this threshold have been dropped from the final sample.

[16] More specifically, only the following climatic zones were found significant: climatic zone of tropical, tropical monsoonal; climatic zone of tropical, tropical wet; climatic zone of temperate, marine west coast-mild with no dry season, warm summer; climatic zone of temperate, Mediterranean-mild with dry, hot summer. For each broad climatic zone, the two sub-climatic zones are combined into a single variable by adding them.

stage F tests and partial R^2 indicate that these instruments can be regarded as "strong" since they respectively equal 47, well above the Stock et al. (2002)'s rule of thumb of 10, and 0.30. In comparison to GMM instruments, these three variables are treated as exogenous standard instruments, with one column in the instrument matrix per variable since they are not lagged.

3.1.2 Results

Results are presented in table 2. In all regressions, the validity of the instruments cannot be rejected. In terms of control variables, most of them are significant and have the expected sign. Income per capita increases life expectancy and reduces fertility. However no significant impact is found on education, suggesting that high education of the population can be attained at different income levels. Though the supply of calories is not significant, the turning point beyond which the number of calories starts having a negative impact on health is coherent: 2400 calories per day, which is in the range of the number of calories required by active individuals to sustain their weight (U.S. Department of Health and Human Services, 2005). Political participation influences positively and significantly life expectancy but not education; in countries with the highest rate of political participation (70%), citizens tend to live 5 years more. Reduction of gender inequality in access to work could significantly enhance education since full women economic rights translate into an increase of 2.75 years in average years of schooling. Finally, occurrence of a conflict only seems to affect fertility: a war decreases fertility by 0.60 child. The number of casualties and the spatial coverage of most conflicts may not be high enough to influence life expectancy and education but may sufficiently disrupt the lives of many couples and darken the future.[17]

Turning to the variables of interest, though HIV prevalence and malaria prevalence are both found to decrease life expectancy (column 1), they exert diverging effects on education (column 2) and fertility (column 3): whereas HIV prevalence increases education and decreases fertility, malaria prevalence influences negatively education and does not seem to affect fertility. Education and life expectancy are significant in all columns and have the expected sign: both influence positively each other and lower fertility. As a consequence, for both infectious diseases, total effects on life expectancy, education and fertility differ from direct effects: lower in the case of HIV, higher in the case of malaria.

[17] Other variables, included in Hamoudi and Sachs (1999a) and Conley et al. (2006) have been tested: population density, urbanisation rate, area weighted Green Revolution modern variety crops. None was found to be significant in equations 2, 1 or 3.

	Life expectancy (1)	Education (2)	Fertility (3)
HIV Prevalence	-0.83^a	0.10^b	-0.07^b
	(0.09)	(0.04)	(0.03)
Malaria prevalence	-3.11^c	-2.05^a	0.38
	(1.80)	(0.47)	(0.32)
Life expectancy		0.08^b	-0.07^a
		(0.04)	(0.02)
Years of Schooling	1.70^a		-0.12^b
	(0.55)		(0.05)
Ln (GDP per capita)	3.12^a	0.58	-0.63^a
	(1.16)	(0.43)	(0.20)
Ln (Calories per day)	322.23		
	(279.73)		
Ln (Calories per day)^2	-20.70		
	(18.27)		
Political competition	0.07^a	0.00	
	(0.03)	(0.01)	
Women economic rights		2.75^a	
		(0.99)	
Conflict occurrence	0.15	0.29	-0.60^a
	(1.57)	(0.59)	(0.24)
Constant	-1226.05	-5.35^c	14.76^a
	(1073.59)	(3.23)	(1.37)
Observations	319	319	319
Countries	69	69	69
Instruments	50	44	38
Arellano-Bond test AR(1)	0.00	0.23	0.00
Arellano-Bond test AR(2)	0.30	0.88	0.58
Hansen overidentification test	0.25	0.80	0.85
Difference-in-Sargan test GMM	0.71	0.48	0.72
Difference-in-Sargan test IV	0.48	0.20	0.94
Total significant HIV impact	**-0.76**	**0.04**	**-0.02**
Total significant malaria impact	**-7.63**	**-2.66**	**0.85**

Notes: a, b, c denotes respectively significance at the 1, 5 and 10% level. Standard errors are in parentheses. All GMM standard errors are heteroscedasticity- and autocorrelation-robust and include the Windmeijer (2000) finite-sample correction. Unreported time dummies are included.

Table 2: The multifaceted impacts of HIV and malaria

Total significant impact	*Average years of Schooling*			
	Baier et al. Over 25	Cohen-Soto Over 15	Cohen-Soto Over 25	Cohen-Soto Not studying 16-64
HIV	0.07	0.17	0.20	0.19
Malaria	-0.42	-0.92	-1.08	-1.13
Countries	259	259	259	259
Observations	55	55	55	55
Total significant impact	*Gross enrollment rates*			
	Baier et al. Over 25	Primary enrollment	Secondary enrollment	Tertiary enrollment
HIV	0.16	1.24	-0.06	-0.33
Malaria	N.S.	-10.83	-6.03	-3.10
Countries	283	283	283	283
Observations	65	65	65	65

Notes: N.S.: not significant.

Table 3: Alternative schooling indicators

Before going any further in our interpretation of the results, finding a positive impact of HIV prevalence on education is novel enough to justify robustness checks by using alternative schooling data. In addition, the impact of HIV on education could be underestimated as it may occur with delay and average years of schooling concern only the over 25 years old population, i.e. people already at the end of their primary school when the HIV epidemic became rampant. In table 3, using the Cohen and Soto (2007) high quality educational attainment dataset, specifications (1) and (2) have been rerun with average years of schooling in three different fractions of the population: over 15, over 25, population 15-64 who is not studying. In a second stage, the impact of HIV prevalence on primary, secondary and tertiary gross enrollment rates has also been investigated since flows of schooling may better capture the short-term impact of HIV than stocks of human capital. Data on school enrollment come from the World Development Indicators (World Bank, 2007).[18] Outliers have been removed from the final samples, and for comparison, results with the Baier et al. (2006) variable are also provided.

[18]Due to the adoption by UNESCO of a new International Standard Classification of Education (ISCED97), post 2000 data are not directly comparable with old standard data (1970-2000). In order to take into account this change, series of school enrollment rates have been adjusted by regressing old standard data on available new standard data and replacing missing values for the period 2001-2004 by predicted values.

New estimations with the Cohen-Soto dataset are very comparable to those in table 2; coefficients of life expectancy, education and HIV prevalence are highly significant, whichever the schooling variable used. As expected, a slightly stronger impact of HIV prevalence is found when looking at the population aged over 15. In terms of magnitude, table 3 shows that the impact of HIV prevalence is more than twice larger than the one found with the Baier *et al.* dataset, keeping the sample constant. However, when the Baier et al. variable is not used, malaria prevalence never appears to influence directly education, which explains why its total impact on schooling is lower than what has been previously found. Estimations using school enrollment rates provide a more contrasted view. Coefficients of life expectancy, education and HIV prevalence are in most cases significant whereas no direct impact of malaria is again found. HIV prevalence appears to exert its strongest positive effect on primary school enrollment, which usually takes place between the ages of 5 and 13. Though HIV prevalence exerts a direct positive and significant impact on secondary school enrollment (between 14 and 17), the total effect is small and negative. Finally the direct impact of HIV prevalence on tertiary school enrollment (between 18 and 24) is positive but non-significant, resulting in a negative total effect. This pattern can be interpreted as proof that a full positive effect of HIV prevalence on education has only recently occurred, explaining why the impact becomes increasingly negative with the age of schooling. Alternatively, parents may invest more in the primary education of their children but the latter need to drop out of school to take care of their HIV-infected relatives or because they have become themselves infected (Shaeffer, 1994). In any case, this varying impact of HIV prevalence on different school enrollment rates is consistent with an overall positive impact of HIV prevalence on average years of schooling as long as the positive effect on primary school enrollment is overwhelming, as it seems to be the case. Overall, these robustness checks indicate that the positive relationship between HIV prevalence and education is robust to changes in dependent variable and sample.[19]

The results of table 2 and table 3 can nevertheless trigger two additional concerns. Since HIV prevalence and malaria prevalence are extremely high in Sub-Saharan Africa (SSA) relative to other developing regions, both may proxy for omitted structural factors specific to SSA. In

[19]Young (2005a) emphasizes that HIV-induced orphanhood can have a detrimental impact on general educational attainment. In unreported regressions, it is found that the share of orphans in the population of children does not exert a significant negative impact on schooling indicators any more if life expectancy is included among determinants, implying that the latter adequately captures the detrimental effect of orphanhood (data on the number of orphans over the 1990-2005 period come from UNAIDS (2002)).

addition, SSA countries represent about 50% of the sample: their inclusion may "dominate" the coefficient estimates. In other words, the coefficient of HIV and malaria may be different for non-SSA countries. These worries are investigated in table 4 by first including a SSA dummy among other determinants and by afterwards interacting HIV prevalence and malaria prevalence with a non-SSA dummy.[20] It appears that the SSA dummy is only significant when estimating the life expectancy specification (column 5); its coefficient implies that life expectancy is structurally three and a half years lower in SSA than in other developing regions. The fact that its inclusion slightly affects the magnitude and significance of other determinants suggests that some variables had previously picked up an omitted SSA effect, such as the effect of other infectious diseases. On the other hand, the hypothesis of slope differences between SSA and non-SSA countries can be strictly rejected since all interaction terms are not significant.

Taking into account the lower estimated impact of malaria prevalence and HIV prevalence on life expectancy induced by the inclusion of the SSA variable, these new estimates suggest that an additional 1% point in HIV prevalence reduces life expectancy by 0.68 years, increases average years of schooling by 0.06 and reduces fertility by 0.04 child or -0.83% at a sample mean. Likewise, in a country fully exposed to malaria, life expectancy, average years of schooling and fertility are respectively 5.74 years, 2.47 years lower and 0.62 child higher than in a malaria free country. These effects remain in the range of what studies have previously found. Overall, for a country like Zambia, with a HIV prevalence rate of about 17% in 2004, these results imply a loss of 12 years in life expectancy, a 1 year gain in average years of schooling and a fertility rate reduced by 0.70 child. Conversely, total eradication of malaria in this country, i.e. going from 100% to 0% of population at risk, would increase life expectancy and education by 5.74 and 2.47 years respectively while fertility would decrease by 0.62 child.

It is worth noting that these estimations are in line with the medical and economic literature. The estimations of the United Nations Population Division (2006) for life expectancy "with AIDS" and "without AIDS" suggest that a 1% rise in HIV prevalence is associated with a drop of 0.90 year in life expectancy. Concerning fertility, based on various studies and Demographic and Health Surveys (DHS) conducted in Africa, Lewis et al. (2004) and Young (2005b) find that a

[20]Interacting both variables with the SSA dummy yields the same results but highly increases multicollinearity between variables of interest (HIV prevalence, malaria prevalence, SSA dummy) and their interaction. In the case of HIV, the interaction term is instrumented by its lagged values. In the malaria case, the interaction term is instrumented by the external instruments interacted with the non-SSA dummy.

1% rise in HIV prevalence decreases fertility by about 0.37% and 1.20%, respectively.[21] With respect to education, Young (2005a) postulates that a positive relationship should exist between education and HIV prevalence, but finds in another paper an inconsistent influence of the HIV prevalence rate on measures of educational enrolment or achievement in Africa: depending both on the education variable and on control variables, sign and significance vary. Finally, only the results of Kalemli-Ozcan (2006) are in total contradiction with our study, since she concludes that AIDS incidence in Africa directly increases fertility and decreases school enrollment rates. Her findings suggest that the relationship between HIV prevalence rates and schooling or fertility may be sensitive to changes in sample or dependent variables.[22]

Concerning malaria, results reflect the literature. Using longitudinal demographic surveillance data from Ghana, Bawah and Binka (2003) calculates that life expectancy would increase by 6 years if malaria was eradicated. Several microeconomic studies have shown that beyond the effect of reduced longevity, infection by malaria causes neuro-cognitive impairment, both in the short- and long- term, and increases school absenteeism (Chima et al., 2003; Kihara et al., 2006). Finally Conley et al. (2006) find that the fertility rate is 0.90 child higher in a country fully exposed to malaria.

To put our results in a nutshell, a higher malaria prevalence *lowers* both life expectancy and education. The fact that no direct impact of malaria on fertility is found further suggests that life expectancy and education adequately capture the overall effects of this infectious disease.[23] On the other hand, a higher HIV prevalence rate is found to *lower* life expectancy and fertility, but to *increase* educational attainment.

[21] The fact that the elasticities of fertility to HIV found by Young (2005b) largely exceed the decline in total fertility attributable to the lower biological fertility of HIV-infected women, as calculated by Lewis et al. (2004), suggest that uninfected people, or people believing not to be infected, modify their fertility-related behaviors. In another paper, Young (2005b) then uses microdata of 57 DHS in 27 SSA countries and finds, after controlling for country-specific effects, time-specific effects and confounding factors that HIV prevalence leads to increased use of all forms of contraception, suggesting that these behavioral changes may not only reflect a willingness to reduce unprotected sexual activity but also to purposely control fertility.

[22] Using similar econometric techniques (weighted least squares with country fixed effects and and time effects), variables (infant mortality, primary and secondary school enrollment rates, AIDS incidence) and sample (only SSA countries over the 1985-2000 period), it is found in unreported regressions that AIDS incidence has a direct (non-) significant positive impact on (primary) secondary school enrollment rate and a highly significant direct negative impact on the fertility rate. In addition it is unclear how AIDS cases, which occur about 5-10 years after HIV infections (Walker et al., 2003), are an useful proxy of the contemporaneous spread of the HIV epidemic.

[23] Removing life expectancy or education from the fertility specification makes the coefficient of malaria become significant.

	Life expectancy			Education			Fertility rate		
	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
HIV Prevalence	-0.75^a	-0.76^a	-0.79^a	0.10^a	0.10^a	0.10^b	-0.07^b	-0.07^b	-0.04
	(0.08)	(0.08)	(0.07)	(0.03)	(0.03)	(0.04)	(0.03)	(0.03)	(0.02)
Malaria prevalence	-2.80^c	-2.64	-4.47^a	-2.13^a	-2.07^a	-1.35^b	0.33	0.14	0.94^a
	(1.67)	(1.84)	(1.50)	(0.45)	(0.43)	(0.63)	(0.39)	(0.44)	(0.29)
SSA dummy	-3.63^a	-3.03^b	-1.01	-0.40	-0.55	-0.79	0.27	0.37	-0.10
	(1.30)	(1.49)	(2.09)	(0.43)	(0.42)	(0.67)	(0.28)	(0.35)	(0.40)
HIV * No SSA dummy		0.06			-0.16			-0.10	
		(1.00)			(0.24)			(0.15)	
Malaria * No SSA dummy			4.16			-0.95			-0.99
			(3.05)			(1.11)			(0.72)
Life expectancy				0.06^c	0.07^b	0.07	-0.06^b	-0.06^b	-0.03
				(0.03)	(0.03)	(0.05)	(0.03)	(0.02)	(0.03)
Years of Schooling	1.19^a	1.42^a	1.39^a				-0.11^b	-0.10^b	-0.15^c
	(0.43)	(0.37)	(0.43)				(0.05)	(0.05)	(0.08)
Ln (GDP per capita)	2.33^c	2.81^a	1.88^b	0.55	0.48	0.79^b	-0.63^a	-0.67^a	-0.60^a
	(1.19)	(1.01)	(0.90)	(0.45)	(0.36)	(0.35)	(0.21)	(0.25)	(0.17)
Ln (Calories per day)	236.49	326.63	74.57						
	(262.60)	(310.78)	(231.09)						
Ln (Calories per day)^2	-14.89	-20.92	-4.68						
	(17.10)	(20.09)	(14.96)						
Political competition	0.05^c	0.05^c	0.08^a	0.00	-0.00	0.00			
	(0.02)	(0.03)	(0.02)	(0.01)	(0.01)	(0.01)			
Women economic rights				2.70^a	2.69^a	1.52			
				(0.98)	(1.02)	(0.95)			
Conflict occurrence	-0.88	-1.39	-0.92	0.18	0.20	0.11	-0.51^b	-0.33	-0.35
	(1.36)	(1.61)	(1.70)	(0.58)	(0.67)	(0.61)	(0.25)	(0.26)	(0.27)
Constant	-900.62	-1242.30	-258.01	-3.77	-3.48	-5.84	13.98^a	14.29^a	12.34^a
	(1008.46)	(1200.76)	(890.85)	(3.43)	(3.26)	(3.60)	(1.51)	(1.70)	(1.84)
Observations	319	319	319	319	319	319	319	319	319
Countries	69	69	69	69	69	69	69	69	69
Instruments	51	57	54	45	51	54	39	45	42
Arellano-Bond test AR(1)	0.00	0.00	0.01	0.18	0.16	0.00	0.00	0.00	0.00
Arellano-Bond test AR(2)	0.27	0.32	0.38	0.97	0.91	0.98	0.48	0.59	0.23
Hansen overidentification test	0.35	0.20	0.51	0.79	0.78	0.66	0.81	0.79	0.51
Difference-in-Sargan test GMM	0.64	0.37	0.96	0.34	0.38	0.72	0.71	0.62	0.53
Difference-in-Sargan test IV	0.49	0.09	0.58	0.30	0.45	0.47	0.96	0.77	0.78
Total significant HIV impact	**-0.68**			**0.06**			**-0.04**		
Total significant malaria impact	**-5.74**			**-2.47**			**0.62**		

Notes: a, b, c denotes respectively significance at the 1, 5 and 10% level. Standard errors are in parentheses. All GMM standard errors are heteroscedasticity- and autocorrelation-robust and include the Windmeijer (2000) finite-sample correction. Unreported time dummies are included.

Table 4: Testing for a SSA specificity

We can further notice that this direct effect of HIV on fertility exceeds what we can attribute to physiological reasons,[24] suggesting a modification of the fertility-related behavior of uninfected people. Indeed, similarly to Young (2005a), our elasticity of fertility reaction to HIV largely exceeds the one directly imputable to a lower biological fertility of HIV-infected women as calculated by Lewis et al. (2004).

Interpretation of the behavioral impacts of HIV found by our empirical study now enable us to bring a straightforward answer to the HIV/AIDS "fertility ambiguity" exemplified in our simple theoretical model, and comparing its effects those of malaria brings further insight in the two opposite forces at work. By lowering life expectancy, both infectious diseases exert a *similar* "mortality effect", i.e. *increasing* demand for children. Hence, if this were the unique direct effect of epidemics like HIV/AIDS or malaria on individual behaviors, they would systematically be found to increase the fertility rate, and through the previously evoked "quantity-quality" trade-off, diminish educational attainment. However, the two diseases are found to exert an *opposite* influence on education: higher malaria prevalence indeed lowers the educational attainment, whereas HIV has a positive impact on education and a negative influence on fertility. This suggests the existence of a second force, that could be either counterbalancing or reinforcing the "mortality effect" on the individual behaviors, depending on its direction. In the case of the HIV/AIDS epidemics, we can go further and state that this competing channel not only offsets, but even dominates the effects of the diminishing survival probability.

In our theoretical model, the force leading to a lower demand for children is a rise in the wages, induced by the labor shortage and resulting in an increase in the foregone value of time spent rearing children. In the case of the HIV/AIDS epidemics, this conjecture seems consistent with the *behavioral* nature of the HIV impact on fertility we exemplified, meaning that the drop is not only caused by physiological consequences of the infection, but indeed reflects a will to purposefully control fertility. In the case of malaria, however, one has to be more careful. First, the fact that under certain specifications we don't find any *direct* impact of this infectious disease on fertility and education casts doubts on the existence of a significant second force beyond the "mortality effect". Then, even in the cases where a direct effect is indeed found on education,

[24] As far as the biological changes are concerned, the fertility of HIV-positive women has been found to be about 30% lower than HIV-negative women in SSA, causing a population-attributable decline in total fertility of 0.37% for every percentage point of HIV prevalence (Lewis et al., 2004). This negative impact on fertility is usually explained by lower coital frequency due to ill health and epidemiological synergies between HIV and other sexually-transmitted infections, which reduce the ability to conceive and increase the risk of foetal loss (Gray et al., 1998).

one could always invoke the neuro-cognitive impairment induced by the disease, leaving few or no room to any behavioral channel behind the impact of malaria on educational attainment.

Hence, in order to ascertain our conjectures, we need now to control for the impact of HIV and malaria prevalence on the evolution of wages. The next section presents our results and concludes the interpretation.

3.2 HIV, malaria and wages

Following closely Rodrik (1999), the following specification will be estimated:

$$\begin{aligned} \text{Ln (Wages)}_i^t &= \phi_0 + \phi_1 \text{ HIV prevalence}_i^t + \phi_2 \text{Malaria prevalence}_i + \phi_3 \text{Ln(Productivity)}_i^t + \\ &\quad \phi_4 \text{Ln(GDP per capita)}_i^t + \phi_5 \text{Ln(Consumption price level)}_i^t + \\ &\quad \phi_6 \text{Political competition} + \phi_7 \text{C}_i + \phi_8 \text{T}^t + \mu_i^t \end{aligned} \tag{4}$$

where μ_i^t is the error term. Wages should be strongly related to labor productivity. In addition, GDP per capita controls for structural determinants associated with income, the price level of consumption takes into account cost of living differences and according to Rodrik (1999), stronger political participation may raise wages by conferring more bargaining power to workers. HIV prevalence (ϕ_1) should exert a direct positive influence on wages, by reducing the supply of labor.[25] The impact of malaria (ϕ_2) is however ambiguous as it depends on the overall effect of this infectious disease on the growth of the workforce, i.e. whether the mortality effect outweighs the fertility effect. In addition, its short-run impact may have dissipated since malaria prevalence is relatively stable since the end of the sixties, particularly in SSA (Hamoudi and Sachs, 1999b). On the other hand, the HIV epidemic has only started its spread since the beginning of the eighties and mostly kills economically active adults, whereas malaria mostly kills children.[26] Hence, over the period investigated (1980-2004), inter- and intra-country variations in HIV prevalence are most likely to affect the growth of labor force supply than differences in the level of malaria prevalence between countries.

[25] Indirect effects of HIV and malaria, e.g. impacts on health and schooling, should be captured by the labor productivity variable.

[26] In the world, 90% of the victims of malaria are less than 4 years-old. On the other hand, 81% of the victims of HIV are between 15 and 60 years-old. See `http://www.who.int/healthinfo/bodestimates/en/index.html`

Wages and productivity are respectively measured by average manufacturing wage per worker[27] and average manufacturing value added per worker. Current US$ values are converted into constant 2000 US$, by multiplying wages and productivity per worker by the ratio of constant GDP to current GDP, both values coming from the World Development Indicators (World Bank, 2007). Data on wages and productivity can be found in UNIDO (1997) and on UNIDO's website for the years beyond 1995.[28] Since data are not available for consecutive years, missing values have been filled by linear interpolation. Price level of consumption is the price level of a country's consumption basket in internationally comparable, purchasing-power-adjusted terms. Data come from the Penn World Tables (Heston et al., 2006). Other determinants have been previously described.

According to a Cook's D test, influential outliers have been removed. In terms of econometric methodology, equation 4 has been first estimated by applying the system GMM. However, regressors may not be endogenous and if explanatory variables are strictly exogenous, the system GMM estimators remain consistent but less efficient than a random effects panel estimator, for which no simultaneity and no correlation between explanatory variables and the country-specific effect are assumed. At the bottom of table 5, results of a Hausman specification test between the system GMM and random effects models suggest that taking into account SSA specificity is crucial as other determinants partially proxy for it, leading to an omitted variable bias. As long as a SSA dummy is included, the exogeneity of the regressors cannot be rejected. Hence, results obtained with the random effects panel estimator are given.[29] The sample includes 65 countries over the 1980-2004 period, with one observation, when available, by country every five years.

Results can be seen in table 5. Once a SSA dummy is included (column (14)), in line with the findings of Rodrik (1999), labor productivity is a strong determinant of wages. Price level of consumption may be picking up the effect of bargaining as workers are more interested in real consumption wages than in real product wages. The significant impact of income per capita may

[27] It is important to note that wages supposedly do not include employers contributions in respect of their employees paid to social security, pension and insurance schemes, as well as the benefits received by employees under these schemes and severance and termination pay. Hence, their variations should not reflect the impact of HIV on the health care costs and death benefits paid by employers, which can be fairly high. For instance, USAID (2001) reports that death benefits can equate as much as three years of salary, without including funeral-related expenses.

[28] `http://www.unido.org/doc/3474`. For some countries, 1995 values are available on the website. They are correlated at 0.97 with the UNIDO (1997) values.

[29] In line with this argumentation, GMM and fixed effects estimates are very close to the random effects estimates and the Hausman specification test between fixed effects and random effects tend not to reject the exogeneity of explanatory variables. Obviously, relying on within variations when using a fixed effects specification precludes the estimation of the impact of malaria as proxy variables are time-invariant.

	Ln (Wages per worker)					
	(13)	(14)	(15)	(16)	(17)	(18)
HIV Prevalence	0.016^a	0.013^b	0.013^b	0.013^b	0.015^b	0.013^b
	(0.006)	(0.006)	(0.006)	(0.006)	(0.007)	(0.006)
Malaria prevalence	0.021	-0.125	-0.117	0.027	-0.170	
	(0.125)	(0.107)	(0.106)	(0.124)	(0.124)	
1982 Malarious area						-0.200^b
						(0.102)
1982-1994 Δ Malarious area						0.215
						(0.140)
SSA dummy		0.334^a	0.330^a	0.188^c	0.374^a	0.315^a
		(0.080)	(0.081)	(0.107)	(0.092)	(0.076)
HIV * No SSA dummy			-0.026			
			(0.066)			
Malaria* No SSA dummy				-0.223		
				(0.169)		
Ln (Value added per worker)	0.747^a	0.741^a	0.735^a	0.732^a	0.726^a	0.745^a
	(0.046)	(0.047)	(0.047)	(0.049)	(0.048)	(0.046)
Ln (GDP per capita)	0.202^a	0.238^a	0.248^a	0.249^a	0.253^a	0.232^a
	(0.056)	(0.050)	(0.049)	(0.049)	(0.054)	(0.047)
Ln (Price level)	0.101^b	0.103^b	0.108^b	0.099^b	0.120^a	0.104^b
	(0.042)	(0.042)	(0.042)	(0.043)	(0.043)	(0.042)
Political competition	0.002	0.002^c	0.002^c	0.002^c	0.002	0.002^c
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Constant	-0.891^c	-1.192^b	-1.221^b	-1.150^b	-1.222^b	-1.088^b
	(0.514)	(0.506)	(0.495)	(0.499)	(0.536)	(0.499)
Observation	248	248	248	248	213	248
Countries	65	65	65	65	54	65
R^2	0.86	0.89	0.89	0.89	0.89	0.90
Hausman test GMM vs RE	0.00	0.74	0.09	0.53	0.85	0.91

Notes: a, b, c denotes respectively significance at the 1, 5 and 10% level. Standard errors are in parentheses. All standard errors are heteroscedasticity- and autocorrelation-robust. Unreported time dummies are included.

Table 5: Wages and infectious diseases: random-effects estimation

mean that the factor share of labor in manufacturing increases with the level of development or that GDP per capita captures changes in productivity not picked up by labor productivity. Finally, workers seem more likely to influence wages in a democratic political regime. The significant and positive coefficient of the SSA dummy suggests that some factors specific to this region raise wages above what can be expected in other developing regions. The data are not detailed enough to explore this issue further but this SSA premium would be consistent with a shortage of skilled manufacturing workers in SSA.

HIV prevalence exerts a significant and positive direct impact on wages; a 1% point rise in HIV prevalence would increase wages by about 1.3%, assuming, like Young does, that HIV-infected workers remain superficially healthy, i.e. their productivity does not decrease until their immune system totally breaks down. On the other hand, malaria prevalence exerts a negative impact on wages but its coefficient is not significant. Checking differences in slopes between SSA and other developing regions (columns 15 and 16) or limiting the sample to countries which belonged to the panel data of section 3 (column 17) does not affect these initial results. Taking these results at face value, wages in South Africa, thanks to a HIV prevalence rate of 19% in 2004, are 25% higher than they would have been in the absence of the HIV epidemic.

However, specification 4 does not take into account the short-run dynamics of malaria. Though the last revised data on population at risk of malaria is only available for the year 1994, a Harvard's Center for International Development (CID) dataset provides estimates of the percentage of a country's area infected with malaria in 1982 and 1994.[30] The 1982 value and the change in the percentage of malarious area over the 1982-1994 period are both included in column (18). Such specification, proposed by Gallup and Sachs (2001), can be roughly considered as a test of equality between the long-term impact and the short-term impact of higher malaria prevalence. It is found that an increase in malaria prevalence has a positive but not significant impact on wages whereas initial level of malaria exerts a strong negative effect on future wages as full prevalence of malaria reduces wages by about 20%. This finding can be interpreted as showing that an increase in malaria prevalence slightly decreases labor force supply, causing an insignificant increase in wages. However, the negative and significant impact of initial malaria prevalence may be explained by a lower labor demand if chronic malaria prevalence hinders economic activity.[31]

[30] `http://www.ksg.harvard.edu/CID/ciddata/geographydata.htm`

[31] Gallup and Sachs (2001) found that a country fully exposed to malaria in 1965, grew -1.3% less quickly than

Overall these results tend to support our conjectures. The opposite impact of HIV and malaria prevalence on education and fertility, even though those two diseases have a similar impact on life expectancy, is found to be accompanied by an opposite impact on wages. In the case of malaria, higher malaria prevalence in the short term does not sufficiently affect the labor supply to trigger a rise in wages. This is not surprising since malaria mostly kills children. However, in the long term, chronic malaria prevalence reduces both economic activity and labor demand, causing a *drop* in wages. The "mortality effect" and the "wage effect" go in the same direction, leading to a rise in fertility and a drop in educational attainment. In the case of HIV however, a higher HIV prevalence lowers life expectancy, but *increases* the level of wages in the short term. Since the overall effect of the epidemic is a lowered fertility rate and a higher education, associated to a rise in wages, we can conclude that the "wage effect" dominates the "mortality effect", bringing a clear answer to the ambiguity exemplified by our theoretical contribution.

One can finally notice that the evolution of wages determines the opportunity costs of rearing children and the quality-quantity trade-off in the demand for children in a nearly self-sustaining circle, since educational attainment will influence the level of future wages. Higher wages triggered by the spread of HIV epidemic leads to a lower fertility rate, more educational investment in a lower number of children and *in fine* higher wages. On the other hand, the low wages engendered by chronic malaria prevalence lead to a high fertility rate, less educational investment in a higher number of children and *in fine* lower wages. In addition to the cognitive and learning effects of malaria, this vicious circle may explain why educational attainment remains persistently low in countries chronically exposed to malaria. Finally, the positive but insignificant impact of malaria prevalence on fertility may reflect the stability of this infectious disease over time: after a certain amount of time, the circular effects of chronic malaria prevalence on wages disappear.

4 Conclusion

Theoretically, we demonstrated the existence of two opposite forces at work in the behavioral answer of individuals to an epidemic shock, identified as the mortality and the wage effects. The key empirical findings of this paper then enable us to provide a clear answer to the *a priori* ambiguous impact of HIV/AIDS on fertility and education. The rise in the wages triggered by

a similar but malaria-free country, over the 1965-1990 period.

reduced labor supply dominates the negative impact of the epidemics on life expectancy, and determines the HIV/AIDS behavioral consequences. Indeed, the increase in the opportunity cost of rearing children prevails, and causes a reduction in fertility and a shift in the demand for children towards quality instead of quantity. Opposite effects are found in the case of malaria in the long run, underlining the growth-detrimental effects of this infectious disease. Overall, we confirm the intuitions of Young (2005a) concerning the behavioral consequences of the HIV/AIDS epidemics for a much broader sample of countries.

It is however worth noting that our results do not imply that the HIV/AIDS epidemics is welfare-enhancing. Beyond the unfathomable effects of the dramatic cost in human lives, one can for example evoke the dire consequences of the growing number of orphans, and its detrimental effects on the level of inequalities in the economy (Boucekkine and Laffargue, 2007). However, our purpose in this work was to focus on a careful study of the behavioral consequences of epidemics such as HIV/AIDS, shedding light on the forces at play and their respective consequences, and opening the floor for use of such results in further studies on the overall economic impact of epidemics.

References

Agadjanian, Victor and Ndola Prata, "War and Reproduction: Angola's Fertility in Comparative Perspective," *Journal of Southern African Studies*, 2001, *27* (2), 329–347.

Ahituv, Avner, "Be Fruitful or Multiplay: On the Interplay Between Fertility and Economic Development," *Journal of Population Economics*, 2001, *14*, 51–71.

Arellano, Manuel and Olympia Bover, "Another Look at the Instrumental Variable Estimation of Error-Components Models," *Journal of Econometrics*, 1995, *68* (1), 29–51.

_ **and Stephen Bond**, "Some Tests of Specification for Panel Data: Monte Carlo Evidence and an Application to Employment Equations," *Review of Economic Studies*, April 1991, *58* (2), 277–97.

Baier, Scott L., Gerald P. Jr. Dwyer, and Robert Tamura, "How Important Are Capital and Total Factor Productivity for Economic Growth," *Economic Inquiry*, 2006, *44* (1), 23–49.

Barro, Robert J. and Jong-Wha Lee, "International Data on Educational Attainment: Updates and Implications," 2000. CID Working Papers, n°42.

Bawah, Ayaga A. and Fred N. Binka, "How Many Years of Life Could Be Saved If Malaria Were Eliminated from a Hyperendemic Area of Northern Ghana?," 2003. Population Council Policy Research Division Working Paper, n°203.

Becker, Gary S., "Fertility and the Economy," *Journal of Population Economics*, 1992, *5*, 185–201.

Bils, Mark and Peter J. Klenow, "Does Schooling Cause Growth," *The American Economic Review*, 2000, *90* (5), 1160–1183.

Bloom, David E. and Jeffrey D. Sachs, "Geography, Demography, and Economic Growth in Africa," *Brooking Papers on Economic Activity*, 1998, *1998* (2), 207–273.

_ , David Canning, and Jaypee Sevilla, "The Effect of Health on Economic Growth: A Production Function Approach," *World Development*, January 2004, *32* (1), 1–13.

Blundell, Richard and Stephen Bond, "Initial Conditions and Moment Restrictions in Dynamic Panel Data Models," *Journal of Econometrics*, 1998, *87* (1), 115–143.

Boucekkine, R. and J-P. Laffargue, "Medium term dynamics and inequalities under epidemics," 2007. CORE Discussion Paper 2007/37.

_ , B. Diene, and T. Azomahou, "The growth economics of epidemics: I. Theoretical considerations," *Mathematical Population Studies*, 2007. Forthcoming.

_ , D. Delacroix, and O. Licandro, "Vintage human capital, demographic trends and endogenous growth," *Journal of Economic Theory*, 2002, *104*, 370–375.

Brainerd, E. and M. Siegler, "The Economic Effects of the 1918 Influenza Epidemic," 2003. CEPR Discussion Paper, n°3791.

Chima, Reginald Ikechukwu, Catherine A. Goodman, and Anne Mills, "The Economic Impact of Malaria in Africa: A Critical Review of the Evidence," *Health Policy*, 2003, *63* (1), 17–36.

CIESIN, *National Aggregates of Geospatial Data: Population, Landscape and Climate Estimates (PLACE)*, New York: CIESIN (Center for International Earth Science Information Network), Columbia University, 2002.

Cingranelli, David L. and David L. Richards, *The Cingranelli-Richards (CIRI) Human Rights Dataset* 2007.

Cohen, Daniel and Marcelio Soto, "Growth and Human capital: Good Data, Good Results," 2004. Mimeo.

_ **and Marcelo Soto**, "Growth and Human Capital: Good Data, Good Results," *Journal of Economic Growth*, 2007, *12*, 51–76.

Collier, Paul, V. L. Elliot, Håvard Hegre, Anke Hoeffler, Marta Reynal-Querol, and Nicholas Sambanis, *Breaking the Conflict Trap: Civil War and Development Policy*, Washington: Oxford University Press, 2003.

Conley, Dalton, McCord Gordon C., and Jeffrey Sachs, "Africa's Lagging Demographic Transition: Evidence from Exogenous Impacts of Malaria Ecology and Agricultural Technology," 2006.

Cutler, David, Angus Deaton, and Adriana Lleras-Muney, "The Determinants of Mortality," *Journal of Economic perspectives*, 2006, *20* (3), 97–120.

Eriksson, Mikael and Peter Wallensteen, "Armed Conflict, 1989-2003," *Journal of Peace Research*, 2004, *41* (5), 625 – 636.

Fogel, Robert W, "Economic Growth, Population Theory, and Physiology: The Bearing of Long-Term Processes on the Making of Economic Policy," *American Economic Review*, June 1994, *84* (3), 369–95.

Freedom House, *Freedom in the World 2005: The Annual Survey of Political Rights and Civil Liberties*, Lanham: Rowman & Littlefield Publishers, Inc, 2005.

Fuchs, Victor R., "Reflections on the Socio-Economic Correlates of Health," *Journal of Health Economics*, July 2004, *23* (4), 653–661.

Gallup, John L. and Jeffrey D. Sachs, "The Economic Burden of Malaria," *American Journal of Tropical Medicine and Hygiene*, 2001, *64* (1-2), 85–96.

Gray, Ronald H., Maria J. Wawer, Maria J. Serwadda, Nelson Sewankambo, Chuanjun Li, Frederick Wabwire-Mangen, Lynn Paxton, Noah Kiwanuka, Godfrey Kigozi, Joseph Konde-Lule, Thomas C. Quinn, Charlotte A. Gaydos, and Denise McNairn, "Population-based Study of Fertility in Women With HIV-1 Infection in Uganda," *The Lancet*, 1998, *351* (9096), 98–103.

Grossman, Michael, "The Demand for Health, 30 years Later: A Very Personal Retrospective and Prospective Reflection," *Journal of Health Economics*, July 2004, *23* (4), 629–636.

Hamoudi, Amar A. and Jeffrey D. Sachs, "Economic Consequences of Health Status: A Review of the Evidence," 1999. Harvard CID Working Paper, n°30.

Hamoudi, Amar and Jeffrey D. Sachs, "The Changing Global Distribution of Malaria: A Review," 1999. CID Working Paper, n°2.

Hansen, L., "Large Sample Properties of Generalizes Method of Moments Estimators," *Econometrica*, 1982, *50* (3), 1029–1054.

Hazan, M. and H. Zoabi, "Does longevity cause growth? A theoretical critique," *Journal of Economic Growth*, 2006, *11*, 363–376.

Herlihy, D., *The Black Death and the transformation of the West*, Cambridge: Harvard University Press, 1997.

Heston, Alan, Robert Summers, and Bettina Aten, *Penn World Table Version 6.2*, Center for International Comparisons of Production, Income and Prices at the University of Pennsylvania, 2006.

Kalemli-Ozcan, S., "Does the mortality decline promote economic growth?," *Journal of Economic Growth*, 2002, *7*, 411–439.

Kalemli-Ozcan, Sebnem, "AIDS, Reversal of the Demographic Transition and Economic Development: Evidence from Africa," 2006.

Kihara, Michael, Julie A. Carter, and Charles R. J. C. Newton, "The Effect of Plasmodium Falciparum on Cognition: A Systematic Review," *Tropical Medicine and International Health*, 2006, *11* (4), 386–397.

Kim, J., "Literature Review on the Impact of Education Levels on HIV/AIDS Prevalence Rates," 2006. Publication of the World Food Programme.

Kiszewski, Anthony, Andrew Mellinger, Andrew Spielman, Pia Malaney, Sonia Ehrlich Sachs, and Jeffrey Sachs, "A Global Index Representing the Stability of Malaria Transmission," *American Journal of Tropical Medicine and Hygiene*, 2004, *70* (5), 486–498.

Kremer, M. and D. Chen, "Income distribution dynamics with endogenous fertility," *Journal of Economic Growth*, 2002, *7*, 227–258.

Lewis, James J.C., Carine Ronsmans, Carine Ezeh, and Simon Gregson, "The Population Impact of HIV on Fertility in Sub-Saharan Africa," *AIDS*, 2004, *18* (2), 35–43.

Mankiw, N.G., *Macroeconomics, 5th Edition*, Worth Publishers, 2005.

Marshall, Monty G. and Keith Jaggers, *Polity IV Project: Political Regime Characteristics and Transitions, 1800-2002, Dataset Users' Manual*, University of Maryland, 2002.

Pinto, Pablo and Jeffrey F. Timmons, "The Political Determinants of Economic Performance: Political Competition and the Sources of Growth," *Comparative Political Studies*, 2005, *38* (1), 26–50.

Pritchett, Lant and Lawrence H. Summers, "Wealthier is Healthier," *Journal of Human Resources*, 1996, *31* (4), 841–868.

Rodrik, Dani, "Democracies Pay Higher Wages," *Quarterly Journal of Economics*, 1999, *114* (3), 707–738.

Roodman, David, "The Anarchy of Numbers: Aid, Development, and Cross-country Empirics," 2004. Center for Global Development Working Paper, n°32.

Shaeffer, Sheldon, "The Impact of HIV/AIDS on Education: A Review of Literature and Experience," 1994. Publication of the UNESCO Section for Preventive Education.

Stasavage, David, "Democracy and Education Spending in Africa," *American Journal of Political Science*, 2005, *4* (2), 343–358.

Stock, James H., Jonathan H. Wright, and Motohiro Yogo, "A Survey of Weak Instruments and Weak Identification in Generalized Method of Moments," *Journal of Business and Economic Statistics*, 2002, *20* (4), 518–529.

UNAIDS, *Children on the Brink 2002, a joint report on orphan estimates and program strategies*, U.S. Agency for International Development, 2002.

UNIDO, *Industrial Development : Global Report*, Oxford: Oxford University Press, 1997.

United Nations Population Division, *World Population Prospects: The 2004 Revision, Volume III: Analytical Report*, New York: UN Publication, 2006.

_ , *World Population Prospects: the 2006 Revision*, New York: United Nations Publications, 2007.

U.S. Department of Health and Human Services, *Dietary Guidelines for Americans 2005*, U.S. Department of Agriculture, 2005.

USAID, "HIV/AIDS and Businesses in Africa," 2001. U.S. Agency for International Development (USAID). Population, Health and Nutrition Information Project.

Vanhanen, Tatu, "A New Dataset for Measuring Democracy, 1810-1998," *Journal of Peace Research*, 2000, *37* (2), 251–265.

Walker, Neff, Karen A. Stanecki, Tim Brown, John Stover, LazzarieStefano, Jesus Maria Garcia-Calleja, Bernhard Schwartländer, and Peter D. Ghys, "Methods and Procedures for Zstimating HIV/AIDS and Its Impact: the UNAIDS/WHO Estimates for the End of 2001," *AIDS*, 2003, *17* (15), 2215–2225.

Windmeijer, Franck, "A Finite Sample Correction for the Variance of Linear Two-Step GMM Estimators," 2000. IFS Working Paper, n°W00/19.

World Bank, *Confronting AIDS: Public Priorities in a Global Epidemic*, New York: Oxford University Press, 1997.

_ , *World Development Indicators* 2007. Online Database.

Young, Alwyn, "The Gift of the Dying: The Tragedy of AIDS and the Welfare of Future African Generations," *Quarterly Journal of Economics*, 2005, pp. 423–466.

_ , "In Sorrow to Bring Forth Children: Fertility amidst the Plague of HIV," 2005.

Zhang, Jie and Junsen Zhang, "The Effect of Life Expectancy on Fertility, Saving, Schooling and Economic Growth: Theory and Evidence," *Scandinavian Journal of Economics*, 2005, *107* (1), 45–66.