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How do epidemics induce behavioral changes?

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Abstract This paper develops a theory of optimal fertility behavior under mortality shocks. In an OLG model, young adults determine their optimal fertility, labor supply and life-cycle consumption with both exogenous child and adult mortality risks. We show that a rise in adult mortality exerts an ambiguous effect on *both* net and total fertility in a general equilibrium framework, while child mortality shocks unambiguously lead to a rise in total fertility, leaving net fertility unchanged. We complement our theory with an empirical analysis using a sample of 39 Sub-Saharan African (SSA) countries over the 1980–2004 period, examining the overall effects of the child and adult mortality channels on both total and net fertility. We find child mortality to exert a robust, positive impact on total fertility but no impact on net fertility, whereas a rise in adult mortality is found to negatively influence both total and net fertility. Given the particular demographic profile of the HIV/AIDS epidemic (killing essentially young, active adults), we then conclude in favor of an unambiguous negative effect of the HIV/AIDS epidemic on net fertility in SSA.

Keywords Fertility · Mortality · Epidemics · HIV

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1 Introduction

Recently, a controversy has been taking place around the fertility impact of the HIV/AIDS epidemic. While the study of Young (2005) concerning the South-African case has concluded that the epidemic has led to a decrease in total fertility, Kalemli-Ozcan (2008a) has identified the opposite effect in a panel of African countries over the 1985–2004 period. At the heart of both authors' arguments lies the adjustment of fertility decisions to changes in the economic, as well as the mortality, environment. Indeed, Kalemli-Ozcan (2008a)'s empirical analysis suggests a positive effect on fertility decision of HIV/AIDS *despite* the negative impacts on total fertility of the biological and sexual responses to the epidemic,[1] while Young (2007) finds that higher HIV prevalence leads to a decline in fertility as a result of an increased use of *all* forms of contraception, hence illustrating a reaction going beyond a simple willingness to reduce unprotected sexual activity.

The claim shared by both authors, that households will *purposefully adapt* their fertility choices to the mortality shocks triggered by the HIV/AIDS epidemic, enables us to place their contribution within the existing extensive body of literature studying the link between mortality and fertility decisions. On one hand, Kalemli-Ozcan (2008a) relates the fertility response to *both* a shock on adult longevity and the survival probability of infants. She argues that increasing adult mortality raises the total fertility rate: this effect results from a quantity/quality trade-off à la Galor and Weil (1999); that is, confronted with a reduced lifespan, individuals end up having more children due to diminishing investment in both their own education and that of their offspring. As far as child mortality is concerned, her argument is tightly linked to the "hoarding effect" (Kalemli-Ozcan 2002); a precautionary demand for children might arise under uncertain survival to adulthood, provided that risk aversion with respect to the expected number of surviving offspring is greater than risk aversion with respect to consumption (Galor 2005). Young (2005), on the other hand, justifies his finding of a negative correlation between the epidemic shock and fertility decisions by asserting that the shock has raised the opportunity cost of rearing children. At the heart of his argument lies the idea that the increasing labor scarcity induced by epidemics of the type of HIV/AIDS essentially killing active adults should trigger a significant positive effect on wages, in a similar manner to the Black Death in the fourteenth century.[2] In a mechanism à la Becker (1981), the resulting substitution effect (lowering fertility through the rising opportunity cost of rearing children) might then end up dominating the related income effect (increasing fertility through a higher amount of resources available to bring up children) and lead to both an increase in female labor participation and a decline in fertility.[3]

We argue along with Young (2005) that the fertility impact of epidemics cannot be captured purely by considering the triggered decrease in survival rates. Fertility choices under epidemics are likely to be impacted by changes in the economic, as well as the mortality, environment. The aim of this paper is hence to capture both mortality *and* price effects on the

[1] 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).

[2] This positive effect on wages is not only emphasized by Young (2005) 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.

[3] It has however been demonstrated by Galor and Weil (1996) that for the substitution effect to dominate, women's *relative* wages need to increase.

fertility response to an epidemic shock in an intertemporal general equilibrium framework. In other words, we aim to study the influence of mortality shocks on fertility choices in an economic environment that is affected by changes in the mortality regime.

We shall proceed in two steps. First we present a theoretical contribution, developing an overlapping generations model that depicts individuals' response to epidemic shocks. Within this theoretical section, we proceed progressively, initially placing ourselves in a partial equilibrium framework where prices (basically the real wage and the interest rate) are given. This allows us to characterize optimal fertility, labor supply and saving decision in the face of transitory mortality shocks. We allow for and clearly differentiate between two possible mortality channels in the case of an epidemic such as the one of HIV/AIDS. The first channel involves a decrease in the life span (*adult* mortality shock) and the second a lower probability of child survival (*child* mortality shock). We show that both types of mortality shock induce an *increase* in total fertility and a lower labor supply, but that the underlying mechanisms are radically different. A positive shock on adult mortality will induce a positive income effect for the survivors, whose number has been reduced, hence increasing the demand for normal goods, and in particular, the desired number of offspring. The shock on infant mortality on the other hand increases the number of births parents need to have in order to reach the desired number of offspring, hence raising total fertility but leaving net fertility unchanged.

We then study how optimal fertility and labor supply are modified when we allow for prices (wages and interest rates) to move exogenously. This second stage can be considered as an intermediary step towards our final, general equilibrium analysis where prices are endogenous. A particular result is that, under certain parametric conditions, the increase in wages resulting from labor shortage can *reduce* fertility and raise labor supply. Hence we demonstrate the existence of a negative, indirect effect, that might eventually outweigh the direct, positive effect, of adult mortality shocks on fertility behavior. Our final step is to close the model and study a general equilibrium version where prices (wages and interest rates) are *endogenously* determined. We show that child mortality shocks have no impact on prices in general equilibrium, and hence the shocks unambiguously lead to a rise in total fertility while leaving net fertility unchanged. On the other hand, we find a positive effect on prices (both wages and interest rate) of a shock on adult mortality, therefore demonstrating the ambiguous effect of a rise in adult mortality on both net and total fertility in a general equilibrium setting.

In a second step, we complement our theoretical model with an empirical contribution to the AIDS-fertility debate. We conduct an empirical analysis on a sample of 39 Sub-Saharan African (SSA) countries over the 1980–2004 period, examining the overall effects of the adult and child mortality channels on optimal fertility behavior. While we find child mortality to exert a statistically significant positive effect only on total fertility, adult mortality is found to have a robust negative effect on *both* total and net fertility. Hence, given the absence of effect of child mortality on net fertility, and the very particular demographic profile of the HIV/AIDS epidemic (killing essentially young, active adults), we argue that it is likely that the HIV/AIDS epidemic has decreased net fertility in SSA.

This paper relates and contributes to an existing literature that has studied the links between mortality rates and fertility decisions. Most papers have focused on the impact on fertility choices of either an increasing infant mortality (Sah 1991; Kalemli-Ozcan 2002; Boldrin and Jones 2002; Doepke 2005) or a decreasing lifespan (Zhang and Zhang 2005; Hazan and Zoabi 2006), while our model encases both phenomena within a single framework, hence bearing a closer relationship to the models proposed by Galor and Weil (1999), Soares (2005) and Cervellati and Sunde (2007). Our model is also related to the literature studying the effects of income distribution on differential fertility, including papers such as Galor and Weil (1996),

Kremer and Chen (2002), and Delacroix and Doepke (2003). However, our paper is the first to examine the response to an epidemic in a model that accounts for both the mortality and wage effects on fertility behavior within a general equilibrium framework. Our paper can also help shed light on a particular, and ongoing, debate concerning the fertility impact of the HIV/AIDS epidemic. First, our theoretical model provides a general framework encasing all the effects identified by Young (2005, 2007) and Kalemli-Ozcan (2008a), and so enables a full decomposition of the mechanisms at work. Our empirical work then demonstrates that the data are consistent with the existence of both an increase in total fertility following a rise in child mortality *and* a negative influence on total as well as net fertility of a rise in adult mortality. This last result, along with the demographic profile of the epidemic, further enables us to conclude in favor of an unambiguous negative effect of the HIV/AIDS epidemic on net fertility, along with Young (2005, 2007).

The rest of the paper is organized as follows. Sections 2 and 3 present our theoretical model and set out its predictions concerning rational behavior under epidemics, first in a partial equilibrium setting with fixed prices (Sect. 2) and then moving to the general equilibrium results (Sect. 3). Section 4 presents our empirical exercise and reports the results obtained. Section 5 concludes.

2 Rational behavior under epidemics: partial equilibrium theory

In order to clearly identify the different forces at work in the influence of an epidemic shock on the fertility and labor supply behavior, we shall start with a partial equilibrium analysis where prices (basically the real wage and interest rate) are given.

2.1 The model

The model is a 3 periods, one good overlapping generations model. An individual born in period t has a probability q_t to survive to the young adult age in period $t+1$, and conditionally to this survival, he can live as a senior adult in period $t+2$ with probability p_{t+1}. In the first period of life (childhood), the individual spends all his time endowment (say one unit of time) having leisure. In period $t+1$, he becomes a young adult with (exogenous) probability q_t. A surviving 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 (provided he survives to this age). Only young adults work, there is no child labor, and we also disregard social security mechanisms. Having children is costly: in our model, raising children costs a certain amount of the overall time endowment. We assume a deterministic child mortality rate, with the rearing effort of parents being directed only at surviving children $q_{t+1}n_{t+1}$ and representing a proportion $\theta q_{t+1}n_{t+1}$ of the total time endowed, with $\theta > 0$. Such a modeling choice can be argued to be overly simplified, as one could be willing to add a specific cost per birth, as well as a stochastic infant mortality rate (Kalemli-Ozcan 2008b). The quantitative predictions of such refinements are however remarkably similar within Barro-Becker types of models (Doepke 2005), while the addition of such features would markedly complicate the algebra and discussion in our model. The young adult then becomes a senior adult with probability p_{t+1}. 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 $\frac{R_{t+2}}{p_{t+1}}$. It can be further noticed that we omit schooling and human capital

accumulation, classic features in the literature linking mortality rates, fertility choices and growth process. We however choose to stick to a simpler model, that will guarantee a precise identification of the effects at work concerning the fertility and labor supply decisions under epidemic shocks.

The preferences specification sticks closely to the spirit of Beckerian behavioral models, with the utility of a (surviving) young adult individual born in t being of the form:

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

where 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.[4]

Hence, consistently with Young (2005)'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. Also notice that utility depends on the number of surviving children, a feature which can also be found in evolutionary biology-based growth models à la Galor and Moav (2002). 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:

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

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

inducing the intertemporal budgetary constraint

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

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

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

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

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

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

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

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

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* $\theta\ q_{t+1}\ n_{t+1} + l_{t+1} < 1$.

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

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

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

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

which yields by (4) and (8)

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

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 $\theta\ q_{t+1}\ n_{t+1} + l_{t+1} < 1$. 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 purpose:

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

where $\Omega_{t+2} = \frac{1}{\theta}\left(1 + p_{t+1}\ R_{t+2}^{\frac{1}{\sigma_c}-1}\right)^{-\sigma_c}$. Denote by $\bar{n}_{t+1}$ the number of children satisfying the equality (implying zero leisure): $\theta q_{t+1} \bar{n}_{t+1} + q_{t+1}^{\frac{\sigma_n}{\sigma_c}}\ \Omega_{t+2}^{-\frac{1}{\sigma_c}}\ w_{t+1}^{\frac{1}{\sigma_c}-1}\ \bar{n}_{t+1}^{\frac{\sigma_n}{\sigma_c}} = 1$. On the interval $(0; \bar{n}_{t+1})$, the left-hand side of (10) is a strictly decreasing function from infinity to $\bar{n}_{t+1}^{-\sigma_n}$ while the right-hand side is increasing from $\theta\ q_{t+1}^{\sigma_n}$ to infinity. Therefore, both sides 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 probabilities p_{t+1} and q_{t+1} for given prices. We then also analyze how this optimal behavior is altered when the latter prices (exogenously) move.

2.2 Optimal behavior under mortality shocks

We consider an epidemic shock affecting *only* the generation born in period t. More precisely, and consistently with AIDS epidemiology, the epidemic first hits the young adults of this generation, lowering the survival probability p_{t+1}, then their children, causing the survival probability q_{t+1} to drop. Considering longer epidemic episodes makes the general equilibrium study analytically intractable as one can see in the next section. In this section, we also assume that the prices w_{t+1} and R_{t+2} are fixed.

Suppose the survival probability p_{t+1} goes down. Then, optimal fertility as given by Eq. 10 is necessarily altered via the term Ω_{t+2}. The same term will also modify labor supply and then savings, as reflected in the following proposition.

Proposition 2 *Under given prices* w_{t+1} *and* R_{t+2}, *a decrease in the survival probability* p_{t+1} *always raises total fertility* n_{t+1} *and reduces labor supply* l_{t+1} *and savings* s_{t+1}, *for any* σ_c *positive.*

Proof Let us start with optimal fertility response. Indeed, a change in p_{t+1} does not affect the left hand side of (10), it only affects the right-hand side through the term Ω_{t+2}. Because

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

it follows that a drop in the survival probability p_{t+1} will decrease the right-hand side of Eq. 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):

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

Then apply the same kind of reasoning as just above on (10). Focusing on Eq. 11, one can see that the left-hand side is shifted downwards when p_{t+1} 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 Eq. 11 for an unchanged right-hand side. However, in contrast to the fertility analysis above, both sides of the Eq. 11 are altered. Indeed, a drop in p_{t+1} 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.

It remains to depict how savings are altered. Using (1) and (8), one can express savings as:

$$s_{t+1} = \frac{p_{t+1}\, R_{t+2}^{\frac{1}{\sigma_c}-1}}{1 + p_{t+1}\, R_{t+2}^{\frac{1}{\sigma_c}-1}}\, w_{t+1}\, l_{t+1}. \tag{12}$$

It follows that savings drop when the survival probability goes down for two reasons. On one hand, as demonstrated above, labor supply diminishes following a drop in the adult survival probability, triggering a decrease in the overall available resources. On the other hand, p_{t+1} has a *direct* effect on savings via the term $\frac{p_{t+1}\, R_{t+2}^{\frac{1}{\sigma_c}-1}}{1+p_{t+1}\, R_{t+2}^{\frac{1}{\sigma_c}-1}}$, which is an increasing function of p_{t+1} for a given interest rate. This direct effect simply features that since young adults only save to consume in their senior age, a diminishing probability of survival should lower the incentives to save. Hence, savings unambiguously decrease. □

Our results complement those of Zhang and Zhang (2005) to a certain extent.[5] Indeed, in their model the reduction in survival probability diminishes the need for life-cycle consumption with respect to fertility, which implies an increase in fertility, a decrease in savings and a drop 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.[6] In our case, a reduced survival rate to old age increases fertility and reduces labor supply *via* a positive income effect, stemming from the

[5] See their Proposition 3, page 50.

[6] In Zhang and Zhang (2005), preferences are logarithmic, which has the further consequence of neutralizing the general equilibrium effects that might arise through an impact on equilibrium prices, as we shall neatly show in the next section. Hence, our partial equilibrium results can be compared to their general equilibrium effects.

presence of an actuarially fair annuity market (Chakraborty 2004; Zhang and Zhang 2005). Indeed, the adults surviving the epidemic and reaching seniority collect the insurance money of the deceased, and see their intertemporal income increase under a diminishing survival rate to old age. However, it is worth noting that were this feature of perfect annuity markets to be abandoned in our model, a positive shock on adult mortality would *still increase fertility*, through a *substitution* effect in favor of the normal goods enjoyed in first period (number of children, consumption and leisure) under a decreasing chance to reach old age.[7]

As far as child mortality is concerned, the results are reflected in the following proposition:

Proposition 3 *Under given prices w_{t+1} and R_{t+2}, a decrease in the survival probability q_{t+1} always raises total fertility n_{t+1} but leaves net fertility $q_{t+1}n_{t+1}$, labor supply l_{t+1} and savings s_{t+1} unchanged for any σ_c positive.*

Proof The proof is very simple. Recall Eq. 10:

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

It is straightforward to see that the previous equation can be rewritten as follows:

$$(q_{t+1}n_{t+1})^{-\sigma_n} = \theta\left[1-\theta\, q_{t+1}n_{t+1} - \Omega_{t+2}^{-\frac{1}{\sigma_c}}\, w_{t+1}^{\frac{1}{\sigma_c}-1}\, (q_{t+1}n_{t+1})^{\frac{\sigma_n}{\sigma_c}}\right]^{-\sigma_l} \tag{13}$$

It follows that a decrease in child survival probability q_{t+1} is "integrally" compensated by a proportional increase in fertility, leaving net fertility $q_{t+1}n_{t+1}$ unchanged. Moreover, Eqs. 11 and 12 giving optimal labor supply and savings are independent of child mortality q_{t+1}. □

Hence, the shock on infant mortality increases the number of births parents need to have in order to reach the desired number of offspring, and as a consequence raises *total* fertility. However, since the *desired* number of surviving children is not modified by the shock on q_{t+1}, net fertility is left unchanged.[8] Of course, this very simple picture is likely to be altered if we depart from some essential assumptions made in our model. The neutrality of child mortality as far as net fertility is concerned directly stems from the absence of specific birth costs (parents investing only in surviving offspring),[9] and more importantly from the deterministic nature of our infant mortality shock. As it has been demonstrated by Sah (1991) and Kalemli-Ozcan (2003), under stochastic survival rates a precautionary demand for children is likely to arise ("hoarding effect"), then also affecting net fertility rates. It has however been shown by Doepke (2005) that once the further feature of sequential fertility decisions is introduced (i.e. parents conditioning their fertility decisions on the observed survival of offspring born previously), the triggered "replacement effect" is quantitatively found to counteract the main bulk of the "hoarding effect". Even though we acknowledge that such a replacement ability is somewhat debatable in the case of an AIDS-like epidemic shock,[10] we chose to

[7] The computations concerning the non-annuity case have not been included in this version of the paper, but are available upon request.

[8] This result had already been pointed out by Doepke (2005) as a special case of the Barro and Becker (1989) model with continuous fertility choice and separate costs per birth and per surviving children.

[9] Introduction of such specific birth costs however leaves net fertility non-increasing along q_t, as demonstrated by Doepke (2005). Indeed, the complete cost of a surviving child is then of $\theta + \beta/q_t$, hence *increasing* with a drop in q_t: parents need to have a greater number of children for every surviving one.

[10] Heuveline (2003) has estimated age-specific fertility rates for women both HIV-positive and negative in East Africa, and found that while fertility rates are higher among HIV-positive women at early reproductive

abstract from further modeling refinements and stick to a somewhat simplified and canonical picture, since the aim of our model is essentially to study the influence of simple mortality shocks within a general equilibrium framework allowing for *price effects* to arise.

2.3 Impact of changing prices on optimal behavior

Before moving to general equilibrium we first study how the optimal behavior depicted above is modified when prices *exogenously* move. As mentioned in the introduction, the literature on epidemics often outlines the role of wages in the propagation of the initial mortality shocks. Our intertemporal framework further has the virtue of exemplifying the role of another possibly important price, i.e. the interest rate.

First, a quick look at Eqs. 10 and 11 is enough to identify a special case: when the utility term with respect to consumption is logarithmic, that is when $\sigma_c = 1$, neither the wage w_{t+1} nor the interest rate R_{t+2} matter in fertility and labor supply choices, and the second-round "price effects" are hence no longer active in this case. Indeed, in both Eqs. 10 and 11, all the wage terms disappear once we set $\sigma_c = 1$. In the case of the interest rate R_{t+2}, this result comes from the fact that R_{t+2} essentially operates through the term $\Omega_{t+2} = \frac{1}{\theta}\left(1 + p_{t+1}\, R_{t+2}^{\frac{1}{\sigma_c}-1}\right)^{-\sigma_c}$, which is trivially independent of R_{t+2} under logarithmic preferences for consumption. The interpretation of those results is straightforward. 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, hence 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). Similarly, when the interest rate R_{t+2} goes up, two effects emerge. The intertemporal substitution effect triggered by the fall of the relative price of future (senior) consumption with respect to present (young adult) consumption favors labor supply. At the same time, the positive income effect resulting from the relaxation of the intertemporal budget constraint goes against extra savings and labor supply. Again, both effects exactly compensate each other under logarithmic preferences for consumption.

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

Footnote 10 continued
ages (because of the selection effect induced by a lower proportion of sexually active individuals in the HIV-negative population), the reverse occurs at older ages. Setting aside any behavioral motive, those lower fertility rates of older HIV-infected women can be explained by the fertility impairment caused by the HIV infection (Gray et al. 1998; Lewis et al. 2004), the increasing morbidity of HIV-positive women, and finally the growing proportions of widows among them (provided they were infected by their husbands). Hence, the perception women have of their replacement ability might indeed be modified by the AIDS epidemics, both because of the biological effect of the disease on fecundability *and* the knowledge they have of a diminishing lifespan. However, a study of Gregson et al. (1997) on Zimbabwe investigated whether couples would seek to accelerate their childbearing for fear that they might not live throughout the normal reproductive lifespan (Setel 1995), and found that half of the women interviewed wanted to have fewer children because of AIDS and almost as many (46%) preferred to have their next child later. Even though such answers might reflect a more general and widespread desire to reduce fertility in Zimbabwe (where fertility was already decreasing before the onslaught of the AIDS epidemics), it still seems that a diminished replacement ability does not necessarily lead to an insurance-type behavioral answer.

Proposition 4 *For logarithmic preferences in consumption ($\sigma_c = 1$), the optimal fertility and labor decisions are insensitive to wages and interest rate. 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 total fertility n_{t+1}, if and only if $\sigma_c < 1$. A rise in the interest rate R_{t+2} has the same properties.*

Proof Let us start with wages. Recall Eq. 13:

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

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 Eq. 13. The optimal labor supply response is slightly trickier to characterize, since both sides of Eq. 11 are affected by an increase in wages:

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

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 $\sigma_c > 1$. As far as the interest rate is concerned, it can be readily established that when R_{t+2} increases, the term $\Omega_{t+2} = \frac{1}{\theta}\left(1 + p_{t+1}\, R_{t+2}^{\frac{1}{\sigma_c}-1}\right)^{-\sigma_c}$ goes in the opposite direction if and only if $\sigma_c < 1$. To conclude about the impact of higher interest rates on optimal fertility and labor supply, it is enough to observe that p_{t+1} and R_{t+2} have the same effect on Ω_{t+2} if and only if $\sigma_c < 1$. In the latter case, an increase in R_{t+2} lowers Ω_{t+2}, which generates the same effects on fertility and labor supply as an increasing p_{t+1}. By Proposition 2, and reversing its statement, we must have a decrease in fertility n_{t+1} and an increment in labor supply. □

Let us focus on the wage effect. 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, in our model 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, the other classic effect of higher wages is the substitution effect, increasing the opportunity cost of both leisure and rearing children. We thus get the typical opposition between income and substitution effects *à la* Becker (1981), which has in general an ambiguous effect on fertility. A key departure from previous similar models 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 will be (via the needed reduction in the time devoted to rearing children). In our 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. We have a similar picture in the case of the interest rate. [11]

Our simple model hence neatly illustrates why an epidemic shock has an ambiguous effect on fertility and labor supply decisions. Indeed, if we interpret an epidemic shock as having direct consequences on survival probabilities and prices (for example on wages via the associated large cuts in labor supply), then our model shows that the *total* effect on fertility choices is *a priori* ambiguous when $\sigma_c < 1$. On one side, a drop in both adult and children survival probabilities increases total fertility, while only adult mortality will impact net fertility. On the other side, under certain parametric conditions, the increase in wages resulting from labor shortage has the exact opposite effect on optimal fertility, i.e. decreasing it. If the interest rates also move, then a third effect has to be accounted for. As shown in Proposition 4, if the interest rates go up following an epidemic shock, then it will reinforce the wage effect described above, inducing a larger drop.

3 Rational behavior under epidemics: accounting for general equilibrium effects

We have so far only studied optimal labor supply and fertility responses to *exogenous* shocks in prices. We shall now take a step further by closing the model and studying a general equilibrium version in which both wages and interest rates are endogenously determined. The main result of this section will outline the contrasting implications of child Vs adult mortality in general equilibrium, which will provide a kind of theoretical synthesis of Young's and Kalemli-Ozcan's approaches.

In what follows, we place ourselves in the parametric case $\sigma_c < 1$, for which the wage effect has been shown to operate in the direction opposite to the mortality effects in the previous section. We assume a production function of the Cobb-Douglas type:

$$Y_t = K_t^{\alpha} \ (l_t \ L_t)^{1-\alpha},$$

where K_t is the stock of capital available in period t, L_t the size of active population (assumed homogenous) and α is the capital share. We shut down technological progress. We finally assume full capital depreciation in one period. At equilibrium, we then have the following equality:

$$K_{t+1} = L_t \ s_t. \tag{14}$$

Under perfect competition, the production factors are paid at their marginal productivities, which yields the two following price equations:

$$w_t = (1-\alpha) \ \left(\frac{K_t}{l_t \ L_t}\right)^{\alpha} \tag{15}$$

and

$$R_t = \alpha \ \left(\frac{K_t}{l_t \ L_t}\right)^{\alpha-1} \tag{16}$$

[11] Note that the simple results obtained (which themselves derive from the simplicity of the model) do 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, since the focus of our model concerns the implication of price shocks on optimal fertility behavior.

To close the model, we finally need to observe that active population evolves according to the following law of motion[12]:

$$L_{t+1} = q_t \; n_t \; L_t. \tag{17}$$

In our behavioral Eqs. 11 to 13, the relevant prices for the generation born in t are w_{t+1} and R_{t+2}. Using the Eqs. 14 to 17 within our general equilibrium extension, one obtains the following expressions for the two relevant prices:

$$w_{t+1} = (1-\alpha) \left(\frac{K_{t+1}}{l_{t+1} \; L_{t+1}} \right)^{\alpha} = (1-\alpha) \left(\frac{s_t}{q_t \; n_t \; l_{t+1}} \right)^{\alpha} \tag{18}$$

and

$$R_{t+2} = \alpha \left(\frac{K_{t+2}}{l_{t+2} \; L_{t+2}} \right)^{\alpha-1} = \alpha \left(\frac{s_{t+1}}{q_{t+1} \; n_{t+1} \; l_{t+2}} \right)^{\alpha-1} \tag{19}$$

Equations 18 and 19 are enough to gather a very important result concerning child mortality.

Proposition 5 *In general equilibrium, an increase in child mortality via a decrease in q_{t+1} has no impact on equilibrium prices, w_{t+1} and R_{t+2}. Therefore, a rise in child mortality does unambiguously raise total fertility and leave net fertility unchanged in general equilibrium.*

Indeed, a fall in q_{t+1} has not impact on $q_{t+1} \; n_{t+1}$, l_{t+1} and s_{t+1} by Proposition 3. From (18), it follows that it has no impact on the wage w_{t+1}. A priori, q_{t+1} may impact R_{t+2} via the term l_{t+2} in Eq. 19. Indeed, writing Eq. 11 one period ahead, one can see that l_{t+2} does depend on w_{t+2} , which itself depends on q_{t+1} according to:

$$w_{t+2} = (1-\alpha) \left(\frac{K_{t+2}}{l_{t+2} \; L_{t+2}} \right)^{\alpha} = (1-\alpha) \left(\frac{s_{t+1}}{q_{t+1} \; n_{t+1} \; l_{t+2}} \right)^{\alpha}$$

but again, here Proposition 3 is enough to conclude that such an effect is neutralized thanks to the presence of the product $q_{t+1} \; n_{t+1}$, which is independent of q_{t+1}. So l_{t+2} is unaffected by changes in the probability q_{t+1}, which leaves the interest rate R_{t+2} also unaffected. Therefore, child mortality has no general equilibrium price effect, and an increase in such a mortality unambiguously raises total fertility. As it can be seen from the demonstration above, the simplicity of this result is evidently linked to the absence of effects of child mortality on net fertility ($q_{t+1} n_{t+1}$) in our model, which we have already extensively commented upon in the previous section. We however deem this simple modeling choice to yield results quantitatively consistent with models displaying more complex refinements (Doepke 2005).

Things are definitely trickier for adult mortality, as reflected in the following proposition.

Proposition 6 *In general equilibrium, an increase in adult mortality via a decrease in p_{t+1} cause wages w_{t+1} to unambiguously go up. Therefore, a rise in adult mortality has an ambiguous effect on both net and total fertility in general equilibrium.*

[12] One could have expected that we take into account the impact on active population of young adults who get the disease early in their working life and die prematurely. This "extensive margin" has been studied by Cuddington (1993a,b) and Cuddington and Hancock (1994), who argue that AIDS-related morbidity and mortality do indeed decrease the size of the labor force. However, the importance of this effect has been contested by Bloom and Mahal (1997), who state that the surplus labor existing in many developing countries could mitigate the labor force reduction that might otherwise be associated with AIDS-related rising morbidity and mortality. We have hence chosen to restrict our analysis to the "intensive margin" captured in our model, which should exist despite any surplus labor, since the behavioral reaction to a increase in adult mortality concerns each and every worker active on the labor market.

The possible opposite directions of the wage and adult mortality effects on optimal fertility had already been exemplified in our partial equilibrium analysis, where positive shocks on prices were exogenously imposed. This property is here crucially shown to hold in general equilibrium. Indeed, by Eq. 18, one can immediately see that a drop in p_{t+1}, causing labor supply l_{t+1} to decrease under fixed prices (by Proposition 2), will further induce an increase in wages. Such an increment in wages plays against fertility, and this "second-round" effect may counteract and possibly outweigh the direct positive effect of the adult survival probability drop on the same variable.

We deem the results stated in proposition 5 and 6 as highly relevant for the ongoing debate about the overall impact on fertility choices of epidemics such as the one of HIV/AIDS. Indeed, showing that the impact of a rise in child mortality on optimal total fertility behavior is unambiguously positive through the absence of effect on prices (and particularly wages) at equilibrium (Proposition 5) shows that the opposition between mortality effect and wage effect does not hold in the case of child mortality. It hence demonstrates that the initial debate between Kalemli-Ozcan (2008a) and Young (2005) is somewhat flawed, since the "hoarding effect" channel had so far been argued to counteract the wage effect: we show that the two effects do not stem from the same type of mortality shock, since the first one is associated to child mortality, while the second only arises in the case of adult mortality (Proposition 6). In our view, disentangling and even isolating in such a way the two effects is a key result, once we consider that the mortality age profiles for major epidemics (Black Death, Spanish flu or AIDS) rather exhibit the preeminence of *adult* mortality.[13] The debate should then rather focus on the opposing positive *direct* effect of adult mortality on both total and net fertility behavior[14] and the negative, *"second-round"* effect triggered by the resulting endogenous increase in wages. Our model shows that if the wage effect associated to this kind of mortality shock is strong enough, then the optimal fertility decision might end up being reduced, and not augmented by the epidemic.

One should also notice that this last possibility is even more likely if the interest rate effect ends up reinforcing the wage effect. From Eq. 19, one can see that the interest rate is also likely to go up after the adult epidemic shock because a drop in p_{t+1} lowers savings, s_{t+1} and increases fertility, n_{t+1} by Proposition 2: Both moves increase the interest rate R_{t+2}, which by Proposition 4, reinforces the wage effect outlined above.

Unfortunately, it is not possible to bring out analytical results characterizing finely when the wage effect does dominate the direct mortality effect on fertility, neither in the short run nor in the stationary equilibrium. The model is certainly solvable in general equilibrium under logarithmic preferences, but as already outlined before this case is uninteresting as far as the question at hand is concerned, since it neutralizes the prices effects on optimal decisions (as properly shown in Proposition 4). Hence, in order to contribute to the ongoing debate concerning the overall impact of epidemic shocks on fertility, rather than looking at rough calibrations of the model we complement our theoretical analysis with an empirical exercise. We will see that in Sub-Saharan Africa, in which the HIV/AIDS epidemic has triggered large mortality shocks, the negative wage effect on fertility indeed seems to dominate the positive life-cycle effect in the case of an increase in *adult* mortality.

[13] Recent demographic projections (see, for example, the UNAIDS (2004) annual report) are nevertheless showing quite an alarming trend for HIV-related child 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.

[14] As well acknowledged by Kalemli-Ozcan (2008a) along with Soares (2005).

4 Empirical application

Our empirical model, using a sample of 39 SSA countries, will focus on dissociating the two possible different mortality shocks (child and adult mortality) that can arise from an epidemic episode, and on studying their respective impacts, both on total and net fertility. First and foremost, this identification of the overall effects will enable us to bring a clear empirical answer to the theoretically ambiguous direction of the adult mortality channel on fertility behavior. We will then as well be able to comment on the overall impact of the HIV/AIDS Sub-Saharan epidemic on fertility.

4.1 Empirical model and data

In addition to mortality, both total and net fertility should be influenced by education, real income per capita and conflict occurrence. A higher average number of schooling years is likely to increase the opportunity cost of childbearing for women, hence education is expected to have a negative impact on the fertility rate. Similarly, the net costs of childrearing, e.g. housing, nursing and training costs, tend to be higher in developed countries characterized by higher levels of real income per capita and greater urbanization (Becker 1992). Last, conflict occurrence may decrease the willingness or the feasibility of having children since current and future upbringing conditions may not be perceived as optimal (Agadjanian and Prata 2001). The following model will therefore be estimated:

$$\begin{aligned}\text{Ln(Fertility rate)}_i^t = {} & \beta_1 \text{Ln(Child mortality)}_i^t + \beta_2 \text{Ln(Adult mortality)}_i^t \\ & + \beta_3 \text{Ln(Real GDP per capita)}_i^t + \beta_4 \text{Education}_i^t + \beta_5 \text{Conflict occurrence}_i^t \\ & + T^t + \epsilon_i^t \end{aligned} \quad (1)$$

where T^t are country-invariant time-specific t fixed effects and $\epsilon_i^t = C_i + \upsilon_i^t$ is the overall error term, with time-invariant country-specific i fixed effects C_i and idiosyncratic shocks υ_i^t. As previously stated and in line with our theoretical model, total fertility is expected to be positively influenced by child mortality and negatively influenced by income per capita, education and the occurrence of a territorial conflict. On the other hand, the overall impact of adult mortality on both total and net fertility is ambiguous, while child mortality is expected to leave the latter unchanged.

The total 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 come from the United Nations Population Division (2007). Child mortality rate is the probability of dying before age 5 per 1000 live births, if subject to current age-specific mortality rates. Following Barro (1991), the net fertility rate is the number of children per women who live beyond the age of four. It is obtained by multiplying the total fertility rate by child survivability (1 - child mortality). Data come from the World Health Organization. Adult mortality rate is the probability of dying between 15 to 60 years per 1000 population, if subject to the age-specific mortality rates of the reporting year. Data on male and female adult mortality rates come from the World Bank (2007) and have been combined into an average mortality rate by weighing each mortality rate by the gender share in total population. Constant PPP GDP per capita and education, the latter measured by average schooling years in population aged 25 or over, come from Baier et al. (2006). Finally, 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, *2* if an intermediate armed

conflict occurs, *3* if a war occurs on the country's territory. For ease of interpretation the variable has been re-scaled from range 0 to 1.

Data on total fertility rates are reported as five year averages by the UNPD. Hence, a five-year period panel covering the apparition and spread of the HIV epidemic in Sub-Saharan Africa is constructed. The quinquennial periods are 1980–1984, 1985–1989, 1990–1994, 1995–1999, 2000–2004. However, besides the data on conflict occurrence, which are available for consecutive years and have been averaged over each five-year period, values of other variables are only available for the years 1980, 1990 and 2000. They have been linearly interpolated in order to obtain beginning of period values for 1985 and 1995.[15]

Concerns about the reliability of the data may be raised. The data generated by Baier et al. (2006) have been rarely used in the literature and mortality rates, especially those of the adult population, are certainly measured with substantial error. Therefore, as commonly done in the literature on the growth impact of education (see for instance Cohen and Soto (2007)), reliability indicators measuring the signal-to-total variance ratio of a variable have been constructed. They can be found in Appendix 1. They show that the informational content of the data series used in this paper is very high, even when expressed in changes, and that the spatio-temporal coverage of other data sources is much narrower.

The only two channels identified by our model through which a disease such as HIV should have an effect on fertility are the child mortality channel and the adult mortality channel. A simple way of testing such an hypothesis is to directly include a measure of HIV prevalence in Eq. 1. An absence of significance of its coefficient will be consistent with a model in which the effect of HIV is completely transmitted through the mortality variables. On the other hand, significance will imply that HIV prevalence exerts a direct effect on fertility, beyond its impact on child and adult mortalities.[16] Time-series data on HIV prevalence in adult population, the percentage of people aged 15-49 who are infected with HIV, have been obtained from Karen Stanecki, UNAIDS senior epidemiologist. The time series have been estimated in November 2006 with the use of the UNAIDS Estimation and Projection Package (EPP) 2005, which fits an epidemic model to all available estimates of HIV prevalence in order to produce a country-specific epidemic curve that describes the evolution of adult HIV prevalence rates over the 1980–2005 period.[17] Since the time-series for each country have been generated at the same time they are not plagued by temporal inconsistency caused by changes in assumptions, methodologies and data used. However, one major limitation of the EPP is related to the non-representative nature of the data used since estimates of HIV prevalence in adult population are mainly derived from data on the proportion of seropositive females among pregnant women attending urban or peri-urban antenatal clinics. Potential

[15] There is no substantial difference in our key results if we use decennial data.

[16] Such a direct effect might correspond to the potential negative physiological effects of a disease on fertility. Concerning HIV/AIDS, the fertility of HIV-positive women has been found to be about 30% lower than HIV-negative women in SSA (Lewis et al. 2004). Among the various biological factors discussed by the medical literature, it has been argued that HIV infection directly reduces the ability to conceive and increases the risk of foetal loss. In addition, it may also favor the transmission of sexually-transmitted diseases, e.g. syphilis, whose prevalence can have similar negative consequences on fertility (Gray et al. 1998; Lewis et al. 2004). However, the results of Ross et al. (1999) suggests that half of the negative impact of HIV infection on pregnancy can be explained by existing sub-fertility prior to infection with HIV. Hence, estimated impacts of HIV on fertility by the medical literature may suffer from a strong simultaneity bias.

[17] See Ghys et al. (2004) and Brown et al. (2006) for a clear presentation of the EPP. The approach is different for low-level or concentrated epidemics in which HIV is concentrated in groups with high-risk behaviors. For these epidemics, where the HIV prevalence rate is considered to be below 1% in pregnant women in urban areas, because transmission is assumed to occur mainly in groups at high risk of HIV infection, estimates for populations who are most exposed to HIV/AIDS are combined to produce an overall estimate of adult prevalence (Lyerla et al. 2006).

Table 1 Summary statistics

Variable	Mean	Std. Dev.	Min.	Max.
Total fertility$_{ln}$	1.79	0.19	1.03	2.14
Net fertility$_{ln}$	1.59	0.14	0.96	1.89
Average years of schooling	2.90	1.75	0.33	8.6
Child Mortality$_{ln}$	5.15	0.38	4.06	5.82
Adult Mortality$_{ln}$	6.05	0.18	5.42	6.54
Real GDP per capita$_{ln}$	6.75	0.59	5.74	8.75
Territorial conflict	0.19	0.32	0.00	1.00
HIV prevalence	2.82	4.80	0.00	28.73
HIV prevalence$_{ln}$	0.85	0.91	0.00	3.39
HIV prevalence adjusted	3.26	5.25	0.00	28.73

Notes: $_{ln}$: variable in logarithms. '1' has been added to the HIV prevalence rate in order to deal with zero values (15% of observations in 1980)

biases associated with the use of antenatal clinic data include the selection for sexual activity and absence of contraceptive use, the lower fertility of HIV-infected women, the joint determination of antenatal clinic attendance and HIV status, the under-representation of smaller rural sites in surveillance systems and how well prevalence levels among pregnant women represent those among men (Walker et al. 2004). Where countries' surveillance systems do not cover adequately rural areas, in which HIV prevalence is expected to be lower than in urban areas, the non-urban prevalence produced by the EPP is adjusted downwards by 20% to reflect this bias. Adjustments for expansion of surveillance systems into lower prevalence areas and for turnover in concentrated epidemics are also implemented. Finally and most importantly, when general population survey data are available, trends fitted from antenatal data have been recalibrated to adjust urban and rural HIV levels to those measured in the population-based survey. In Appendix 1, it is shown that these estimates of HIV prevalence appear reliable, certainly because the calibration of the data to national population surveys had occurred for most of the countries included in the data used in the reliability analysis.[18] Robustness of the results will be assessed by interacting the HIV variable with a dummy which takes the value of one when the prevalence rate has been adjusted.

Data are available for 39 SSA countries over the 1980-2004 period.[19] Summary statistics are given in Tables 1 and 2 while Figs. 1 and 2 show the evolution of fertility, HIV prevalence, child mortality and adult mortality over time. A visual inspection suggests that a rise in HIV prevalence tends to be associated with lower fertility and higher adult mortality.

Our estimation procedure needs to tackle three different issues. First, there may be unobserved time-invariant country-specific effects correlated with the regressors. Second, most explanatory variables are likely to be jointly determined with the fertility rate. Finally, measurement errors in both the dependent variable and in regressors are certainly present.[20] Estimation of Eq. 1 by OLS would generate biased and inconsistent estimators for all parameters. To address these issues, we employ the "system-GMM" estimator developed by Arellano and

[18] Nevertheless, note that Grassly et al. (2004) show that estimates based on antenatal sentinel surveillance provide a good approximation of HIV prevalence in adults in the local community and Young (2007), using antenatal and community data for 50 regions in 8 African countries, does not find a significant difference between community and antenatal infection rates.

[19] Influential outliers have been removed from the sample, according to a Cook's D test.

[20] In the absence of other endogeneity issues, OLS estimators remain unbiased and consistent but are less efficient if the measurement error in fertility rates is statistically independent of each explanatory variable. However, as it will be seen in Sec. 4.2, this may not be the case.

Table 2 Countries in the sample

Country	Child M. 1980	Child M. 1990	Child M. 2000	Adult M. 1980	Adult M. 1990	Adult M. 2000	Δ Child M. 1990–2000 (%)	Δ Adult M. 1990–2000 (%)	HIV Prevalence (%) 2000 (%)
Botswana	84	58	101	309	411	678	74	65	28
Lesotho	155	120	91	322	438	684	−24	56	24*
Zimbabwe	108	80	117	355	287	733	46	155	23*
South Africa	91	60	63	432	397	496	5	25	18*
Namibia	108	86	69	396	345	493	−20	43	17
Zambia	155	180	182	447	405	692	1	71	17*
Malawi	265	241	188	387	457	614	−22	34	14
Mozambique	220	235	178	413	367	534	−24	45	14
Central African Republic	189	180	180	480	431	618	0	43	11
Gabon	115	92	91	430	367	375	−1	2	8
Kenya	115	97	120	378	322	523	24	62	7*
Uganda	185	160	145	429	493	562	−9	14	7*
Tanzania	175	163	165	410	408	492	1	20	7*
Cameroon	173	139	166	451	395	472	19	19	6*
Congo	125	110	108	352	321	465	−2	45	6*
Côte d'Ivoire	172	157	188	385	324	454	20	40	5
Rwanda	219	173	203	455	450	543	17	21	4*
Angola	265	260	260	512	466	484	0	4	4
Guinea-bissau	290	253	215	526	539	426	−15	−21	4*
Nigeria	216	235	205	494	439	481	−13	10	4
Liberia	235	235	235	226	378	495	0	31	3
Burundi	195	190	190	443	418	526	0	26	3*
Chad	225	203	200	501	441	471	−1	7	3
Togo	175	152	142	415	355	329	−7	−7	3
Ghana	157	125	100	367	302	334	−20	10	2*
Benin	214	185	160	440	408	300	−14	−26	2
Burkina faso	247	210	207	415	384	426	−1	11	2*
Ethiopia	220	204	176	446	403	429	−14	6	2*
Gambia	231	154	128	524	480	313	−17	−35	2
Mali	300	250	224	408	392	344	−10	−12	2*
Sierra leone	336	302	286	533	546	411	−5	−25	2*
Sudan	142	120	97	500	431	308	−19	−28	2
Guinea	300	240	175	549	512	298	−27	−42	2*
Niger	320	320	270	508	465	354	−16	−24	1*
Senegal	218	148	139	550	443	298	−6	−33	1*
Somalia	225	225	225	456	427	392	0	−8	1
Mauritania	175	183	183	460	402	331	0	−18	1
Madagascar	175	168	137	315	405	310	−18	−23	0
Mauritius	40	25	20	214	187	173	−20	−7	0
Average	194	172	162	427	406	453	−3	14	7

Notes: *M*: Mortality. * indicates that HIV prevalence rates have been calibrated to national population surveys

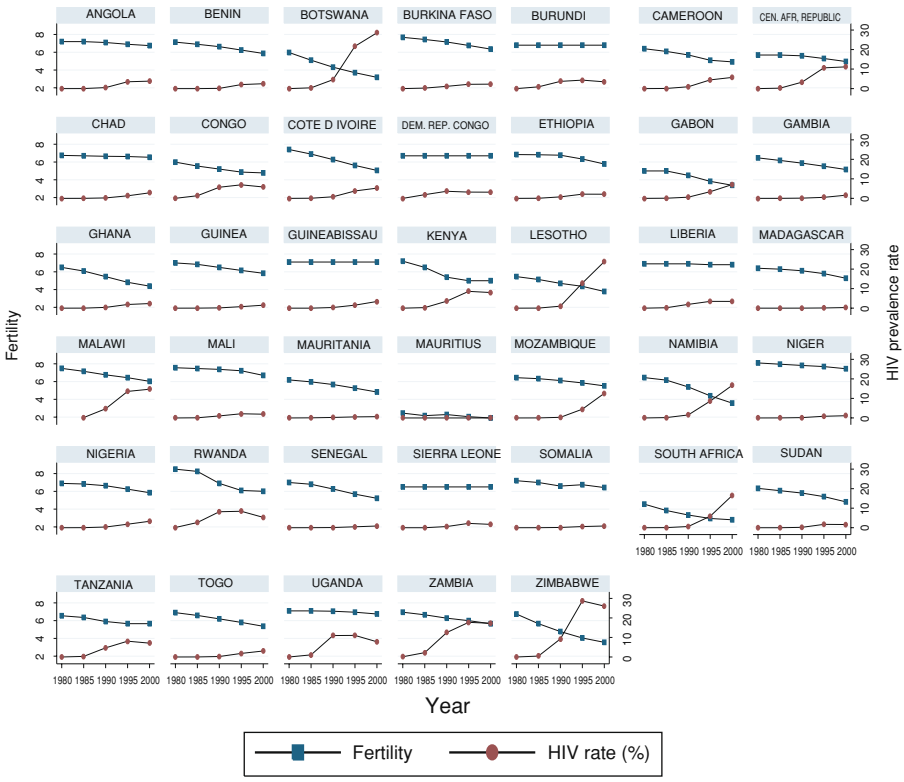

Fig. 1 Fertility and HIV prevalence

Bover (1995) and Blundell and Bond (1998), which is particularly appropriate for the analysis of our "small T, large N" panel with persistent time series. The country-specific effect is first eliminated by taking first-differences of Eq. 1. Instruments are then required to deal with the endogeneity of the explanatory variables and measurement errors.[21] Under the assumption that the error term is not serially correlated and that the explanatory variables are weakly exogenous (i.e. uncorrelated with future realizations of the error term), lagged values of the endogenous variables provide valid "internal" instruments. In other words, the Arellano and Bond (1991) "difference-GMM" estimator uses the following moment conditions:

$$E[X_{i,t-s}.(\upsilon_{i,t} - \upsilon_{i,t-1})] = 0 \quad for\ s \geq 2;\ t=3,...,T \tag{2}$$

where X are the determinants of fertility.

However this approach has conceptual and statistical drawbacks. First, it ignores the cross-country dimension of the data. Second, when the explanatory variables are persistent over time, lagged levels of the series are weak instruments as they are poorly correlated with subsequent first-differences. In that case, the GMM estimator obtained after first-differencing suffers from severe small sample biases and is very imprecise (Blundell and Bond 1998). These shortcomings can be confronted by exploiting information in levels through the use of the "system-GMM" estimator of Arellano and Bover (1995) and Blundell and Bond (1998),

[21] Note that measurement errors constant over time but specific to each country are absorbed into the time-invariant country specific effects and therefore removed by taking first-differences of Eq. 1.

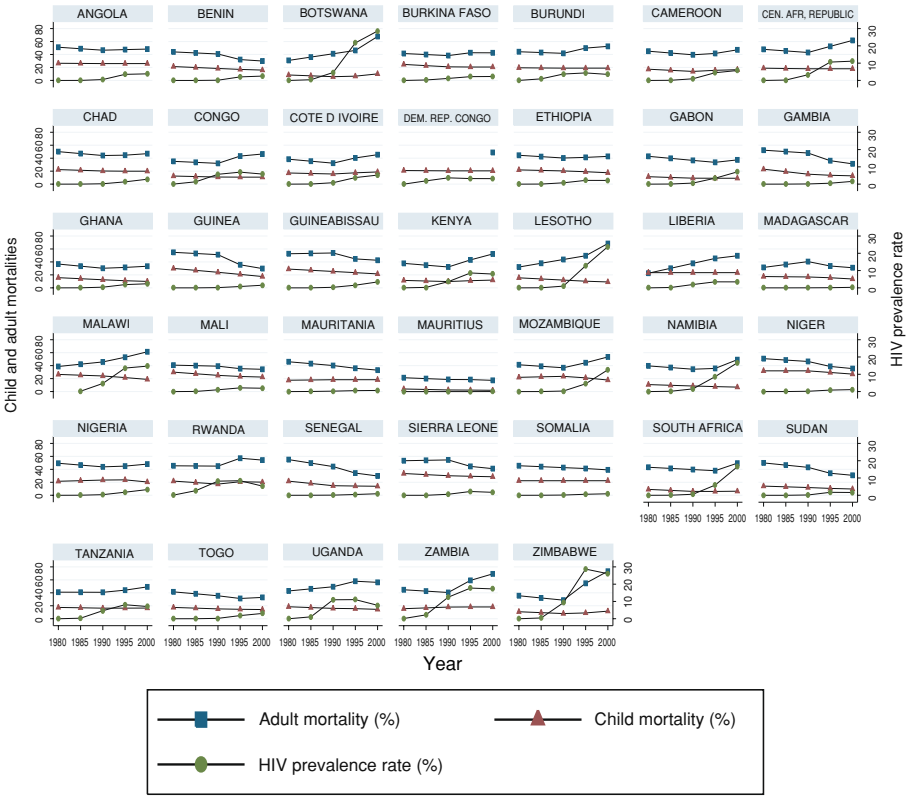

Fig. 2 Child and adult mortalities and HIV prevalence

which combines the equation in differences, instrumented with lagged levels of the regressors, with the equation in levels, instrumented with lagged differences of the regressors. The cross-country variation is preserved and past changes may be more predictive of current levels than past levels are of current changes. Lagged differences of the explanatory variables are valid instruments under the additional assumption that there is no correlation between the differences of these variables and the country-specific effect. This assumption results from the following stationarity property:

$$E[X_{i,t+p}.C_i] = E[X_{i,t+q}.C_i] \quad \textit{for all } p \textit{ and } q \tag{3}$$

and the additional moment conditions for the second part of the system (the equation in levels) are:[22]

$$E[(X_{i,t-s} - X_{i,t-s-1}).(C_i + \upsilon_{i,t})] = 0 \textit{ for } s{=}1 \tag{4}$$

Blundell and Bond (1998) and Blundell et al. (2000) show that the "system-GMM" estimator greatly improves on the performance of the first-differenced GMM estimator in terms

[22] The use of further lags beyond ΔX_{t-1} as instruments in the level equations can be shown to result in redundant moment conditions (Arellano and Bover 1995).

of bias and precision when the explanatory variables are persistent over time and the number of time periods of available data is small, which is the case we find ourselves in.

The consistency of the "system-GMM" estimator depends on the validity of the instruments, i.e. they must be uncorrelated with the current error term. Three specification tests, suggested by Arellano and Bond (1991) and Arellano and Bover (1995), are used to address this issue. The first test is a Sargan-Hansen test of over-identifying restrictions which tests the overall validity of the instruments. The second test is the "Difference in Sargan-Hansen test" which examines the validity of the subset of instruments employed for the equation in levels. Finally, it is tested whether the differenced error term is second-order serially correlated.[23] In order not to overfit the endogenous variables,[24] which can bias the results towards those of an uninstrumented regression and weaken the Sargan-Hansen tests (Roodman 2007), the GMM instruments are restricted to the latest valid lags, i.e. for the differenced equation, determinants of fertility are instrumented using the second lag of their levels while for the levels equation, their once-lagged differences are used. Robustness of the results to the number of instruments will also be investigated by "collapsing" the full instrument set, i.e. one instrument for each variable and lag distance will be created rather than one for each time period, variable, and lag distance (see Roodman (2007)).

4.2 Results

Before turning to our main results, we provide some justifications for our use of the "system-GMM" estimator by conducting several endogeneity tests. In columns (1) and (1') of Table 3, our empirical model is estimated by OLS. A robust Hausman test proposed by Arellano (1993) indicates that the explanatory variables are correlated with the country-specific unobserved effect, included in the overall error term. In columns (2) and (2'), we eliminate this first source of endogeneity (i.e. an omitted variable bias) by first-differencing the data. We then test for the strict exogeneity of each variable in the remaining columns of Table 3. We follow the procedure suggested by Wooldridge (2002) (p. 285),which consists in including the current level of a regressor as an additional explanatory variable in the first-differenced equation. Under the null hypothesis of strict exogeneity, the latter should not be correlated with changes in fertility and therefore its coefficient should not be statistically significant. Strict exogeneity is rejected for child mortality (col. 2A and 2'A), adult mortality (col. 2B and 2'B), GDP per capita (col. 2C and 2'C) and average years of schooling (cols 2D and 2D'). In the last columns (2F and 2'F), we test for the strict exogeneity of the HIV prevalence rate. We cannot reject it. Overall, these preliminary results indicate that an instrumental variables approach is warranted.

"System-GMM" estimates are presented in Table 4. Neither the Sargan-Hansen tests that evaluate the validity of the full set of instruments nor the Difference in Sargan-Hansen tests, that focus on the additional instruments used by the "system-GMM" estimator, can reject the null hypothesis that the instruments are exogenous, i.e. uncorrelated with the current

[23] The test is applied to the residuals in differences since the full disturbance contains the fixed effects. First-order serial correlation is expected since $\Delta\upsilon_t$ and $\Delta\upsilon_{t-1}$ are mathematically related through the shared υ_{t-1} term. Second-order serial correlation in differences implies first-order serial correlation in levels.

[24] Roodman (2004) indicates as a rule of thumb that the number of instruments should not exceed the number of countries in the regression.

Table 3 Child mortality, adult mortality and fertility, OLS and FD estimates

Determinants estimator	Total fertility							
	OLS (1)	FD (2)	FD (2A)	FD (2B)	FD (2C)	FD (2D)	FD (2E)	FD (2F)
(A) Child mortality$_{ln}$	0.30[a]	0.19[a]	0.07	0.18[a]	0.15[a]	0.15[b]	0.18[b]	0.19[b]
	(0.04)	(0.07)	(0.05)	(0.06)	(0.06)	(0.06)	(0.07)	(0.07)
(B) Adult mortality$_{ln}$	−0.11[b]	−0.05[c]	−0.02	−0.12[b]	−0.05[b]	−0.01	−0.05	−0.01
	(0.04)	(0.03)	(0.02)	(0.05)	(0.02)	(0.03)	(0.03)	(0.04)
(C) Real PPP GDP per capita$_{ln}$	−0.10[a]	−0.05[a]	−0.03[c]	−0.05[a]	−0.02	−0.07[a]	−0.05[a]	−0.05[b]
	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)
(D) Average years of schooling	−0.01	−0.05[a]	−0.01	−0.04[a]	−0.02	−0.02	−0.05[a]	−0.05[a]
	(0.01)	(0.01)	(0.01)	(0.01)	(0.02)	(0.02)	(0.01)	(0.01)
(E) Occurrence of a territorial conflict	0.01	−0.01	−0.02	−0.01	−0.01	−0.01	−0.01	−0.00
	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)
(F) HIV prevalence$_{ln}$								−0.01
								(0.01)
Exogeneity test p-value for regressor			(A)	(B)	(C)	(D)	(E)	(F)
			0.08[a]	0.09[a]	−0.04[a]	−0.01[a]	0.02	−0.00
			(0.01)	(0.03)	(0.01)	(0.00)	(0.02)	(0.02)
Estimator	Net fertility							
	OLS (1')	FD (2')	FD (2'A)	FD (2'B)	FD (2'C)	FD (2'D)	FD (2'E)	FD (2'F)
(A) Child mortality$_{ln}$	0.08	0.01	−0.13[b]	0.01	−0.03	−0.03	0.01	0.01
	(0.05)	(0.08)	(0.06)	(0.08)	(0.07)	(0.07)	(0.09)	(0.09)
(B) Adult mortality$_{ln}$	−0.13[a]	−0.06[c]	−0.01	−0.13[b]	−0.05[b]	−0.00	−0.05	−0.02
	(0.05)	(0.03)	(0.02)	(0.05)	(0.02)	(0.03)	(0.03)	(0.04)
(C) Real PPP GDP per capita$_{ln}$	−0.12[a]	−0.05[a]	−0.03	−0.05[a]	−0.02	−0.08[a]	−0.05[a]	−0.05[b]
	(0.03)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)
(D) Average years of schooling	−0.01	−0.06[a]	−0.01	−0.04[a]	−0.02	−0.02	−0.06[a]	−0.05[a]
	(0.01)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)
(E) Occurrence of a territorial conflict	0.00	−0.00	−0.01	−0.01	−0.00	−0.01	−0.01	−0.00
	(0.02)	(0.02)	(0.01)	(0.02)	(0.02)	(0.02)	(0.03)	(0.02)
(F) HIV prevalence$_{ln}$								−0.01
								(0.01)
Exogeneity test p-value for regressor			(A)	(B)	(C)	(D)	(E)	(F)
			0.09[a]	0.10[a]	−0.04[a]	−0.02[a]	0.01	−0.00
			(0.01)	(0.03)	(0.01)	(0.00)	(0.02)	(0.02)
Hausman test p-value	0.00	26						
Observations	180	145	145	145	145	145	145	144

Notes: [a,b,c] denotes respectively significance at the 1, 5 and 10% level. OLS: Ordinary Least Squares estimator. FD: First Differences estimator. Heteroscedasticity-autocorrelation robust standard errors are in parentheses. A constant and unreported time dummies are included. $_{ln}$: variable in logarithms. Exogeneity tests are successively performed for each variable

error term. Likewise no evidence of second-order serial correlation of the differenced residual is found, providing additional support for the use of appropriate lags of the explanatory variables as "internal" instruments. Overall, the validity of the instruments is never rejected and therefore they always appear to satisfy the necessary moment conditions for consistency of the "system-GMM" estimator. All control variables have the expected sign and most of

Table 4 Child mortality, adult mortality and fertility, GMM-SYS estimates

Determinants	Fertility$_{ln}$							
	Total (3)	Net (3')	Total (4)	Net (4')	Total (5)	Net (5')	Total (6)	Net (6')
Child mortality$_{ln}$	0.24[a]	0.06	0.23[a]	0.06	0.23[a]	0.05	0.17[c]	−0.01
	(0.06)	(0.07)	(0.06)	(0.08)	(0.05)	(0.07)	(0.10)	(0.12)
Adult mortality$_{ln}$	−0.18[a]	−0.20[a]					−0.17[a]	−0.16[b]
	(0.05)	(0.06)					(0.05)	(0.07)
Male adult mortality$_{ln}$			−0.20[a]	−0.22[a]				
			(0.05)	(0.05)				
Female adult mortality$_{ln}$					−0.16[a]	−0.17[a]		
					(0.05)	(0.06)		
Real PPP GDP per capita$_{ln}$	−0.12[a]	−0.13[a]	−0.11[a]	−0.12[a]	−0.12[a]	−0.13[a]	−0.15[b]	−0.16[b]
	(0.03)	(0.04)	(0.03)	(0.04)	(0.04)	(0.04)	(0.06)	(0.07)
Average years of schooling	−0.02[c]	−0.01	−0.02[b]	−0.01	−0.02[c]	−0.01	−0.02[c]	−0.02
	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.02)
Occurrence of a territorial conflict	−0.03	−0.01	−0.02	−0.01	−0.03	−0.01	−0.05	−0.05
	(0.04)	(0.04)	(0.04)	(0.04)	(0.04)	(0.04)	(0.05)	(0.05)
Constant	2.59[a]	3.48[a]	2.68[a]	3.59[a]	2.46[a]	3.34[a]	3.09[a]	3.80[a]
	(0.52)	(0.60)	(0.54)	(0.63)	(0.51)	(0.56)	(0.86)	(0.96)
Observations	180	180	180	180	180	180	180	180
Countries	39	39	39	39	39	39	39	39
Instruments	35	35	35	35	35	35	25	25
Arellano-Bond test AR(2) *p*-value	0.76	0.82	0.77	0.85	0.72	0.76	0.86	0.87
Hansen overidentification test *p*-value	0.38	0.28	0.43	0.31	0.34	0.26	0.25	0.19
Difference-in-Hansen test level GMM *p*-value	0.52	0.39	0.57	0.42	0.46	0.35	0.31	0.14
Difference-in-Hansen test IV *p*-value	0.53	0.48	0.62	0.56	0.44	0.40	0.50	0.50

Notes: [a,b,c] denotes respectively significance at the 1, 5 and 10% level. System-GMM estimator. Standard errors are in parentheses. All GMM standard errors are heteroscedasticity- and autocorrelation-robust and include the Windmeijer (2005) finite-sample correction. Unreported time dummies are included. $_{ln}$: variable in logarithms. In regression (3) and (3') the impacts of child and adult mortalities on total and net fertility are investigated. In regressions (4)–(4') and (5)–(5'), the average adult mortality rate is successively replaced by the male adult mortality and the female adult mortality rate. In regressions (6) and (6'), the robustness of the results to a "collapse" of the full instrument set is examined

them are statistically significant.[25] In line with our theoretical model, child mortality exerts a positive and statistically significant impact on total fertility (col. 3) but leaves net fertility unchanged (col. 3'). Adult mortality, on the other hand, is found to statistically reduce both total and net fertility. Interpreted along our theoretical framework, this result suggests that the negative, indirect wage effect outweighs the positive, direct effect of adult mortality on net fertility. The next four columns show that the impacts of male adult mortality rate (col. 4 and col. 4') and female adult mortality (col. 5 and col. 5') on total and net fertility are about

[25] Other variables, included in Conley et al. (2006) or Kalemli-Ozcan (2008a) have been tested: population density, urbanisation rate, area-weighted Green Revolution modern variety crops. Coefficients of all these variables were highly statistically insignificant and their inclusion did not affect our initial results.

the same. Finally, in the last columns (col. 6 and 6'), the sensitivity of results and specification tests to a reduction in the number of instruments is investigated by "collapsing" the full instrument set. Although statistical efficiency decreases, results are qualitatively unaffected by this robustness check.

In the epidemiological context of SSA, it could be argued that our results reflect the biological and mechanical effects of rising HIV prevalence on fertility instead of mortality-induced behavioral effects. Mortality of women who die before having reached the end of their childbearing years does not affect the estimation of fertility rates as long as the fertility of those who have died is not different from those who survive and report their fertility. However, given the potential sub-fecundability (Lewis et al. 2004) of the HIV-positive women who already died from HIV/AIDS, the biological consequences of a high HIV prevalence rate could mechanically lead to an inflation of the fertility rates based on fertility data of the more fecund living women. In that case, the positive correlation between the measurement error in fertility rates and the mortality rates associated with high HIV prevalence rates could produce an upward bias in the estimated impact of both mortalities on fertility. This upward bias, certainly stronger in the case of adult mortality given the demographic profile of the HIV/AIDS epidemic, would nevertheless not invalidate our conclusions since it would simply imply that the effects of child mortality are slightly smaller and those of adult mortality on total fertility slightly stronger than the ones we have found.[26] In fact, this should not even be the case as we have dealt with the endogeneity induced by measurement errors through our instrumental-variable procedure. Hence, the "selection bias" in the data underlying the estimation of fertility rates cannot explain a strong negative effect of rising adult mortality on fertility.

It might as well be the case that a disease does exert a *direct* effect on fertility, *beyond* the behavioral effects that we identify following a shock on child and adult mortalities. In the HIV/AIDS case, an obvious candidate is the previously discussed negative physiological effects of this disease on fertility, although it cannot be ruled out that a rise in HIV prevalence may generate other behavioral effects not directly related to adult and child mortalities. For instance, Kalemli-Ozcan (2008a) finds that the logarithm of the HIV prevalence rate[27] exerts a direct positive effect on total fertility, beyond a HIV-related rise in infant mortality. Hence, depending on the existence, direction and strength of this direct effect, our model may be underspecified and our conclusions on the most likely effects of a rise in HIV prevalence on fertility may be strengthened or, in the most extreme case, overturned. As indicated in the previous sub-section, a simple way of testing such an hypothesis is to directly include a measure of HIV prevalence in our empirical model. An absence of significance of its coefficient is consistent with a model in which the effect of HIV is completely transmitted through the mortality variables. On the other hand, significance implies that HIV prevalence exerts a direct effect on fertility, beyond its impact on child and adult mortalities. This hypothesis is investigated in Table 5. We then finally assess the "contemporaneous" total impact of a rise in HIV prevalence on fertility.

[26] Moultrie and Dorrington (2008) show that the presence of an HIV/AIDS epidemic may actually have a quasi-null impact on the estimation of the fertility rate when the technique most commonly used in Africa to estimate fertility from limited and defective census data, the Brass P/F ratio technique, is applied. For a description and an illustration of the Brass P/F ratio technique, see United Nations (1983).

[27] She uses the logarithm of the HIV prevalence for three reasons. First, it makes estimates less sensitive to outliers, i.e. extremely high prevalence rates in some SSA countries. Second, the estimated impact of HIV prevalence on fertility rates becomes insensitive to a common multiplicative error (region-wide under- or over-reporting). Finally, the fit of her first-stage regression (see footnote 30) is better when using the logarithm of the HIV prevalence rate. Note that in contrast with our empirical model, she does not include in hers any direct measure of adult mortality.

Table 5 Adult mortality, HIV prevalence and fertility

Determinants	Fertility$_{ln}$					
	Total (7)	Net (7')	Total (8)	Net (8')	Total (9)	Net (9')
HIV prevalence$_{ln}$	−0.01	−0.00	0.00	0.00	−0.04[c]	−0.04[c]
	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)
HIV prevalence$_{ln}$ * Dummy			−0.00	0.00		
			(0.03)	(0.03)		
Adjusted HIV prevalence dummy			0.01	−0.01		
			(0.04)	(0.04)		
Child mortality$_{ln}$	0.18	0.01	0.20	0.01	0.10	−0.07
	(0.12)	(0.15)	(0.13)	(0.15)	(0.16)	(0.15)
Adult mortality$_{ln}$	−0.17[a]	−0.18[b]	−0.17[a]	−0.18[a]		
	(0.06)	(0.07)	(0.06)	(0.07)		
Real PPP GDP per capita$_{ln}$	−0.15[b]	−0.15[c]	−0.12[c]	−0.13[c]	−0.16[c]	−0.15[c]
	(0.07)	(0.08)	(0.07)	(0.07)	(0.09)	(0.08)
Average years of schooling	−0.02	−0.02	−0.02[c]	−0.02	−0.02	−0.02
	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.02)
Occurrence of a territorial conflict	−0.05	−0.04	−0.05	−0.03	−0.14[c]	−0.12
	(0.03)	(0.04)	(0.04)	(0.05)	(0.07)	(0.08)
Constant	2.99[a]	3.77[a]	2.75[a]	3.62[a]	2.49[c]	3.15[b]
	(0.95)	(1.12)	(0.98)	(1.11)	(1.42)	(1.37)
Observations	179	179	179	179	179	179
Countries	39	39	39	39	39	39
Instruments	29	29	33	33	25	25
Arellano-Bond test AR(2) *p*-value	0.93	0.91	0.97	0.95	0.65	0.71
Hansen overidentification test *p*-value	0.33	0.17	0.18	0.15	0.27	0.21
Difference-in-Hansen test level GMM *p*-value	0.23	0.11	0.11	0.10	0.21	0.22
Difference-in-Hansen test IV *p*-value	0.66	0.55	0.48	0.40	0.71	0.55

Notes: [a,b,c] denotes respectively significance at the 1, 5 and 10% level. System-GMM estimator. Standard errors are in parentheses. All GMM standard errors are heteroscedasticity- and autocorrelation-robust and include the Windmeijer (2005) finite-sample correction. In all regressions, instruments are collapsed. Unreported time dummies are included. $_{ln}$: variable in logarithms. In regressions (7) and (7'), the direct effect of HIV prevalence on total and net fertility is tested. In regressions (8) and (8'), it is investigated whether the direct effect may be statistically different in countries for which HIV prevalence rates have been calibrated to national population surveys. In regressions (9) and (9'), the coefficient on the HIV variable is allowed to capture not only the direct impact of the disease on fertility, but also the effect of the HIV-induced rise in adult mortality, by omitting the adult mortality variable

First, keeping the instruments collapsed, the logarithm of the beginning-of-period HIV prevalence rate is included among the other determinants of fertility in columns (7) and (7').[28] Since, like other explanatory variables, HIV prevalence may be endogenous, the former var-

[28] '1' has been added to the HIV prevalence rate in order to deal with zero values (15% of observations for the period 1980-1984). One observation is lost as the 1980 value for Malawi is missing. For 20 countries, HIV prevalence rates have been calibrated to national population surveys.

iable is instrumented by its lagged values. Table 5 shows that no evidence of a direct effect is found as the coefficient on the HIV variable is close to zero and is highly insignificant. However, the reliability of HIV prevalence rates not calibrated to national population surveys is questionable. Robustness of these results are therefore examined by interacting the HIV variable with a dummy which takes the value of one when the prevalence rate has been adjusted (col. 8 and 8'). The absence of a direct impact in both sets of countries of a rise in HIV prevalence on fertility cannot still be rejected as neither the HIV variable nor the interaction term are statistically significant. Overall, these results suggest that the mortality channels adequately capture the total effect of this disease on fertility. They also provide some additional support to the results of Young (2007), who does not find much evidence that the effect of HIV on fertility reflects direct biological changes.[29]

Second, we assess the effect of a rise in HIV prevalence on fertility when omitting adult mortality, allowing for the coefficient on the HIV variable to capture not only the direct impact of the disease on fertility, but also the effect of the HIV-induced rise in adult mortality. This will enable us to compare more directly our qualitative results to those of Young (2005, 2007) and Kalemli-Ozcan (2008a). Columns (9) and (9') show that in the absence of adult mortality, the coefficient on the HIV prevalence rate becomes negative, large and significant at the 10% level. Using the average HIV prevalence in countries for which the HIV prevalence rate has been adjusted (Table 1), its estimate suggests that a 1% point rise in HIV prevalence decreases total fertility by about −1.1% and net fertility by about −1.2%.

It is worth mentioning that these estimates can be interpreted as roughly assessing only the "contemporaneous" adult mortality-related effect of a rise in HIV prevalence, i.e. about 2-3 years after the initial shock, given values for fertility rates are five-year averages and values for HIV prevalence rate are beginning-of-period. However, full progression from infection to AIDS death in the absence of competing causes of mortality can take much longer. It is estimated that it can take at maximum 15-20 years in the case of adults and 10 years in the case of children, which are not necessarily conceived at the time of maternal infection (Stover 2004). Hence, following a rise in HIV prevalence, total fertility may in future periods briefly rebound if a related increase in child mortality occurs,[30] although the demographic profile of the epidemic makes it very unlikely that this positive effect of child mortality will dominate in the long run. It is more likely that the "delayed" deaths of the main victims of HIV, the adults, will lead to an even stronger impact of a rise in HIV prevalence on total (and net) fertility in SSA than the one found contemporaneously. We can be more assertive in the case of net fertility. Following a rise in HIV prevalence in SSA, its fall at every future period is unambiguous since its evolution has been found to be totally unaffected by child mortality.

Finally, we provide in Table 6 an additional suggestive piece of evidence by looking at the impact of child and adult mortalities on the 2000 contraceptive prevalence rate, i.e. the percentage of women between 15–49 years who are practising, or whose sexual partners are practising, any form of contraception. Data come from World Bank (2007). Column (10)[31] shows that child mortality exerts a negative and statistically significant impact on the use

[29] He does not find any correlation between biological markers of sub-fecundability (amenorrhea and miscarriages) and average infection rate, suggesting that presence of sub-fertility in asymptomatic HIV-infected women may be in fact due to reverse causation or a behavioral response to knowledge of their infection.

[30] Compared with adult mortality, an increase in child mortality following a rise in HIV prevalence is much less likely to contemporaneously occur. Women becoming infected need first to be pregnant and of the 1/3 of children born to HIV-infected women who become themselves infected, only 1/3 of them die within one year of birth. On the other hand, 3/5 will die within four years of birth. See Stover (2004).

[31] Given that the dependent variable is a fraction, restricted to the unit interval [0,1], a fractional logit model is used (Wooldridge 2002).

Table 6 Contraceptive prevalence and child and adult mortalities

	Contraceptive prevalence rate in 2000			
	(10)	(11)	(12)	(13)
Child mortality$_{ln}$	−0.12[a]	−0.13[a]	−0.13[a]	
	(0.05)	(0.05)	(0.05)	
Adult mortality$_{ln}$	0.16[a]	0.12[b]		0.12[b]
	(0.05)	(0.06)		(0.06)
HIV prevalence$_{ln}$		0.01	0.03[a]	−0.01
		(0.01)	(0.01)	(0.01)
Real PPP GDP per capita$_{ln}$	−0.02	−0.02	−0.04[c]	0.02
	(0.02)	(0.02)	(0.02)	(0.02)
Average years of schooling	0.03[a]	0.03[a]	0.03[a]	0.04[a]
	(0.01)	(0.01)	(0.01)	(0.01)
Occurrence of a territorial conflict	−0.09[b]	−0.09[b]	−0.08	−0.09[c]
	(0.04)	(0.04)	(0.05)	(0.05)
Observations	39	39	39	39

Notes: [a,b,c] denotes respectively significance at the 1, 5 and 10% level. Fractional logit regression. Coefficients correspond to marginal effects evaluated at the sample mean. Heteroscedasticity robust standard errors are in parentheses. Standard errors are in parentheses. $_{ln}$: variable in logarithms. In regression (10), the impacts of child and adult mortalities on the use of contraception are tested. In regression (11), the direct effect of HIV prevalence on the contraceptive prevalence rate is investigated. In regressions (12) and (13), the adult mortality variable and the child mortality variable are successively omitted

of contraception whereas the opposite is true for adult mortality. In column (11), we cannot reject the absence of a direct effect of a rise in HIV prevalence on contraceptive prevalence, as the coefficient on the HIV variable is statistically insignificant. However, in column (12), if we allow for the coefficient on the HIV variable to capture the effect of the HIV-induced rise in adult mortality by omitting the adult mortality variable, we find that that an increase in HIV prevalence is statistically associated with an increased use of contraception. On the other hand, if we omit child mortality instead of adult mortality (column 13), the coefficient on the HIV variable becomes once again small and insignificant. First, those results further confirm the behavioral nature of the observed impact on fertility of mortality shocks, since contraceptive prevalence should not be impacted by the biological effects of a rising HIV prevalence. Second and foremost, the analysis of the impacts of child and adult mortalities on a proximate determinant of fertility confirms our previous findings and those of Young (2007). Child mortality and adult mortality have diametrically opposite effects on total fertility, respectively positive and negative, and the mortality channels, especially the adult mortality channel, adequately capture the total effect of the HIV disease on fertility behaviors.

As a last remark, although we have focused our paper on the effects of the HIV/AIDS epidemic, we could have used our theoretical model to analyze the impact of another disease in SSA, malaria. Given that 90% of its victims in Africa are children (World Health Organization 2008), it is likely that following a rise in malaria prevalence, total fertility would increase and net fertility would remain unaffected.

5 Conclusion

Theoretically, we have studied the influence of mortality shocks on fertility in a general equilibrium framework allowing for price endogeneity. To the best of our knowledge, our paper

is the first to provide a general equilibrium analysis of the behavioral effects of epidemic shocks affecting both infant and adult survival rates. After having identified positive *direct* effects of both the child and adult mortality shocks in a partial equilibrium environment with fixed prices, we have shown that the overall effects of these two types of mortality shocks differ in significant ways in a general equilibrium set-up. While the child mortality shock is found to exert an unambiguously positive overall effect on total fertility through the *absence* of price effect, we have demonstrated that there is an ambiguous impact of adult mortality on fertility once the "second-round", negative wage effect is considered.

The paper is complemented by an empirical application of our theoretical model using a sample of SSA countries, the majority of which have been deeply affected by a HIV/AIDS epidemic. Our empirical results show that the data are consistent with the predictions of our theoretical model concerning child mortality, i.e. child mortality shocks only exert a positive effect on total fertility and have no significant influence on net fertility. Our results also strongly suggest that the indirect negative wage effect on fertility following an adult mortality shock outweighs the direct, positive mortality effect, specifically we find that adult mortality decreases both total and net fertility. Given the absence of any significant effects of child mortality on net fertility, and the very particular demographic profile of the HIV/AIDS epidemic, we conclude in favor of an unambiguous negative effect of the HIV/AIDS epidemic on net fertility in SSA.

Finally, it is worth noting that our results do not imply that the HIV/AIDS epidemic 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 the detrimental effects of the resulting growing inequality 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, to shed light on the forces at play, and their respective consequences. In doing so, we have opened the floor for the use of such results in further studies on the overall economic impact of epidemics.

Appendix 1

Variables are frequently measured with errors and therefore their reliability needs to be carefully assessed. For instance, if X_1 is a noisy measure of Z, with $X_1 = Z + \epsilon$, its reliability λ_{X_1} can be defined as the signal-to-total variance ratio: $\lambda = \frac{Var(Z)}{Var(X_1)} = \frac{Var(Z)}{Var(Z+\epsilon)}$. However, the value of Z is frequently unknown. Reliability of X can nevertheless be approximated if a second noisy measure of Z, e.g. X_2, is available. If the errors of these two variables are uncorrelated with each other and with the true value, their covariance provides an approximation of the variance of the true value. In that case $\lambda_{X_1} \simeq \frac{Cov(X_1,X_2)}{Var(X_1)}$. Note that if the errors in X_1 and X_2 are positively correlated, the estimated reliability ratios will be biased upwards and may exceed the value of one. Hence, they should cautiously be interpreted as providing an upper bound on the reliability of the data series.

The formula used to calculate the reliability ratios coincides with that used to calculate the slope coefficient of a simple regression model. Hence, OLS regressions are run with each variable of interest alternatively acting as dependent or independent variable. Following Krueger and Lindahl (2001) and de la Fuente and Doménech (2002), all regressions include time dummies and are not only run in level but also in first-differences in order to assess whether changes in the data still convey some signal. Alternative measures of education, GDP per capita, adult mortality rates and HIV prevalence respectively come from Cohen and

Table 7 Data reliability

	Education BDT	Education CS	Δ Education BDT	Δ Education CS	$GDPPC_{ln}$ BDT	$GDPPC_{ln}$ PWT	Δ $GDPPC_{ln}$ BDT	Δ $GDPPC_{ln}$ PWT
Education CS	0.77[a] (0.19)							
Education BDT		**0.86**[a] (0.05)						
Δ Education CS			0.28 (0.19)					
Δ Education BDT				**0.23**[b] (0.10)				
GDP per $capita_{ln}$ PWT					0.77[a] (0.05)			
GDP per $capita_{ln}$ BDT						**1.10**[a] (0.07)		
Δ GDP per $capita_{ln}$ PWT							0.30[c] (0.16)	
Δ GDP per $capita_{ln}$ BDT								**0.41**[a] (0.07)
	Male AMR_{ln} UN	Male AMR_{ln} TJ	Δ Male AMR_{ln} UN	Δ Male AMR_{ln} TJ	Female AMR_{ln} UN	Female AMR_{ln} TJ	Δ Female AMR_{ln} UN	Δ Female AMR_{ln} TJ
Male AMR_{ln} TJ	0.28[a] (0.08)							
Male AMR_{ln} UN		**1.01**[a] (0.26)						
Δ Male AMR_{ln} TJ			0.30[a] (0.08)					
Δ Male AMR_{ln} UN				**0.74**[a] (0.13)				
Female AMR_{ln} TJ					0.37[a] (0.06)			
Female AMR_{ln} UN						**1.04**[a] (0.24)		
Δ Female AMR_{ln} TJ							0.34[a] (0.09)	
Δ Female AMR_{ln} UN								**0.59**[a] (0.16)

Table 7 continued

	HIV_{ln} UNAIDS	HIV_{ln} Oster	Δ HIV_{ln} UNAIDS	Δ HIV_{ln} Oster
HIV_{ln} Oster	0.71[a] (0.16)			
HIV_{ln} UNAIDS		**0.83**[a] (0.19)		
Δ HIV_{ln} Oster			0.22[c] (0.11)	
Δ HIV_{ln} UNAIDS				**0.88**[a] (0.20)

Notes: [a,b,c] denote respectively significance at the 1, 5 and 10% level. Heteroscedasticity-autocorrelation robust standard errors are in parentheses. All regressions include time dummies. $_{ln}$: variable in logarithms. GDPPC: GDP per capita; AMR: Adult mortality rate. BDT: Baier et al. (2006). CS: Cohen and Soto (2007) data available for 26 countries. PWT: Heston et al. (2006) data available for 39 countries. TJ: Timæus and Jasseh (2004) data available for 19 countries. Oster: Oster (2006) data available for 9 countries

Soto (2007), Heston et al. (2006), and Timæus and Jasseh (2004)[32] and Oster (2006). No second measure of child mortality rates was found. Their estimates are associated with about a ±15% range of uncertainty and are considered more reliable than the estimates of child mortality as the indirect demographic techniques used to estimate the former are more robust and less sensitive to assumptions about underlying mortality patterns than are the techniques used to estimate the latter (Ahmad et al. 2000).

The estimated reliability ratios are reported in Table 7. The data series on which the empirical findings of this paper are based appear fairly reliable. Focusing on adult mortality rates and HIV prevalence, the reliability ratios suggest that the informational content of these variables is very high, even when expressed in percent changes (growth rates). In the latter case it is not surprising as for most of the countries included in the sample, trends fitted from antenatal data have been recalibrated to adjust urban and rural HIV levels to those measured in population-based surveys, in order to reflect more accurately the national situation.

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