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Accounting for the Effects of AIDS on Growth in Sub-Saharan Africa

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Abstract

In this paper, we, first, perform a quantitative assessment of the impact of the HIV/AIDS epidemic on growth. Second, we precisely account for the effects of the epidemic on income per capita through human and physical capital accumulations, population and labor force. That is, we disentangle the effect on the different sources of short and long run growth. Using a dynamic panel of 46 Sub-Saharan African countries over the period 1981-2007, we show that HIV/AIDS has negative, significant and long-lasting effects on demography and growth. According to the estimates presented, GDP per working age population will be 12% lower in the long-run for the average African country than it should be if the epidemic had not spread out. However, the impact is huge for the countries experiencing a high prevalence rate. To tackle the endogeneity issue of HIV/AIDS, we provide a new series of HIV prevalence rate build from the estimation of the propagation dynamic of the epidemic.

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1   Introduction

The UNAIDS 2008 "Report on the global AIDS epidemic" states that there were an estimated 33 million people living with AIDS in 2007 in the World. Sub-Saharan Africa remains the most heavily affected by HIV, accounting for 67% of all people living with HIV and for 72% of AIDS related deaths in 2007. In some countries, as Botswana, Lesotho and South Africa, HIV prevalence rate among pregnant women attending clinics is more than 30%. In Sub-Saharan Africa alone, the epidemic has orphaned nearly 12 million children under 18.

The dramatic humanitarian impact of the epidemic is largely recognized and has drawn most of the attention. This has helpfully mobilized the international community to set up powerful instruments and large financial funds to slow down the propagation of the epidemic and treat infected people. Thanks to these efforts, promising results have been reached, but enduring challenges remain.

Indeed the epidemic is likely to have very long lasting effects. The UNAIDS report declares that "the natural age distribution in many national populations in Sub-Saharan Africa has been dramatically skewed by HIV with potentially perilous consequences for the transfer of knowledge and values from one generation to the next". This declaration underlines some of the different channels through which the epidemic can affect growth: short run and long run impact, effects on population dynamics and on education.

In this paper, we, first, perform a quantitative assessment of the impact of the HIV/AIDS epidemic on growth. Second, we precisely account for the effects of the epidemic on income per capita through education, physical capital, population and labor force. That is, we disentangle the effect on the different sources of short and long run growth. Such an exercise may shed some light on the need to tackle the multidimensional impact of the epidemic.

The social and economic consequences of such epidemic are huge. Families are broken, sometimes with an important number of orphans. The burden, in families providing health care for infected people is heavy, and has consequences on savings and investment. It also have large effects on public policies as it diverts resources to health care from others under satisfied needs.

However, the assessments of the economic impact of the epidemic are very contradictory. In one hand, Bonnel (2000), Bell, Devarajan, and Gerbach (2003), McDonald and Roberts (2006), and Papageorgiou and Stoytcheva (2005) find some evidence of a negative effect of the epidemic. In the other hand, first estimation by Bloom and Mahal (1997) in a cross-country perspective found no evidence that AIDS impedes growth. Werker, Ahuja, and Wendell (2006) have also found a statistically insignificant effect of the HIV epidemic on growth.

Assessing the macroeconomic impacts of the HIV/AIDS epidemics is a very challenging exercise. There have been many reservations made on macroeconomic cross-country regressions. Durlauf, Jonhson and Temple (2005) provide an extensive review of the limitations and progress on the econometrics of growth. Most of the reservations comes from the lack of appropriate data and model uncertainty used to analyze growth. Therefore, they underline the necessity to draw lessons from these analysis with cautious. They also point out the difficulty to find out valid instruments in panel growth regressions. However, they find promising the strategy followed by Tavares and Wacziarg (2001), which consisted on estimating the effects of democracy on the long run determinants of growth (human capital accumulation, physical capital accumulation or government expenditures)

in order to net out its effect on growth.

Recently, Weil (2007) used microeconomics estimates of the effect of health on individual outcomes to construct macroeconomic estimates of the proximate effect of health on GDP per capita. This approach allows to circumvent the endogeneity bias encountered in structural estimates of growth determinant.

Our strategy is inspired by Weil (2007) accounting strategy[1]. We use instrumental variables, including circumcision rate as Werker, Ahuja, and Wendell (2006) to estimate the independent propagation dynamic of the epidemic, and then we construct a HIV prevalence series. These two complementary steps normally allow us to eliminate or reduce the endogenous bias problem.

Conceptually our approach is similar to the one done by Ashraf, Lester and Weil (2008). The reasoning is to estimate the impact of a variable of interest and to substract its impact in reference to the potential output the economy would reach without the shock due to the variable of interest. Moreover, their simulations of the impact of eradicating malaria or tuberculosis depict a very instructive functioning of the dynamic effects of epidemic disease directly and indirectly through education, life expectancy and productivity. As HIV/AIDS combines the distinctive patterns of these two diseases, that is hitting either the working adult age population and the young, their exercise help to explain its potential cumulative huge effects.

Our estimates show that for the average Sub-Saharan African countries, at the peak, the total effect of HIV/AIDS on growth is a reduction by 6% of income per capita; the effect through education account for 2.4%, the effect through physical capital account for 2%, the residual effect which account for the reduction of labour participation and technological progress account 1.5%, and the counterbalancing positive effect through capital intensity accounts for less than 0.1%. As the effects pile up, in the long run the cumulative permanent effect is a 12% loss in the level of GDP per capita.

Our results emphasize the necessity to tackle the impact of the epidemic not only through its prevention and the treatment of infected people, but also on human capital and physical capital accumulation.

The paper is organized as follows. Section 2 discusses the controversial literature and presents our conceptual framework. Section 3 presents the data sources and the construction of the independent HIV prevalence series. Section 4 is the econometric assessment of the impact of HIV/AIDS on per capita income and on the determinants of growth. Section 5 presents the accounting exercise we perform and the results. Section 6 discusses our results by comparing them to the literature and concludes.

2   The conceptual framework and controversial issues

This section presents the theoretical framework and the controversial issues in the disentanglement of the effects of HIV/AIDS on growth. Although there is no doubt that AIDS is a plague for Sub-Saharan African countries, the patterns of the economic outcomes is very controversial.

2.1   A non consensual literature

An evidence difficult to show   Several studies have tried to estimate the total effect

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[1]See Caselli (2005) for a survey of growth accounting analysis.

of AIDS on growth. Early estimates by Over (1992) and Cuddington and Hancock (1994) find evidence of a negative effect of AIDS on growth. But these estimates suffer from a lack of appropriate data. Bloom and Mahal (1997) run cross-country regressions of GDP per capita growth on HIV/AIDS prevalence and found no effect. However, their studies have been criticized because they used limited data[2]. More recently, McDonald and Roberts (2006) provide new estimates of the effect of AIDS on growth through its impact on health capital. Their study, based on data from 1984 to 1999, relies on the estimation of a Solow-augmented model. Using a panel estimation method applied to cross-country economic growth, they found a strong and significant negative effect of AIDS on growth. While, Werker, Ahuja, and Wendell (2006) show that there is no effect of the epidemics on growth of African countries. Corrigan, Glomm, and Mendez (2005) show calibration results that imply large negative effects of the epidemic on growth.

A room for a positive or negative impact   There are several channels through which

the AIDS epidemic can potentially harm economic growth[3]. First, as AIDS decreases life expectancy, it reduces the incentives to invest or accumulate physical or human capital. Moreover, as noted by Corrigan and al. (2005), to the extent that AIDS is more prevalent among highly skilled workers and that highly skilled labor is complementary to capital, the AIDS epidemic can lower the returns on investment and, in turn, savings and capital accumulation. In the same line, potentially large medical costs to treat HIV can divert private or public resources from education investment or capital accumulation. In addition to that, a decrease in life expectancy will reduce individuals or firms investment in education or training, reducing therefore workers productivity. Second, the disease may eventually hamper schools enrollment and the quality of education[4]. The presence of a large generation of orphans, specially in the context of African economies where the government can not compensate for missing parents, may induce a decrease in human capital investment. Finally, the disease can hamper the productivity of infected people, thus lowering average productivity. The disease could reduce the productivity of healthy people as well, as they may devote some of their time taking care of their sick relatives. All these effects, lower investment in physical and human capital, and productivity slowdown, may contribute cumulatively to a negative impact of HIV/AIDS on income.

However, there is still a room for a positive impact of AIDS/HIV epidemic on growth. The positive effect of AIDS on growth may occur through two closed mechanisms. The first one is the capital deepening effect occurring with the loss of working age population caused by epidemics as emphasized by Young (2007). Indeed, the short-run effect of the epidemic with the huge number of death of adult persons is, in a context or scarcity of capital, to increase the capital to worker ratio, and therefore induces an increase of productivity and growth. The second one is a long run effect depending on the impact of AIDS/HIV on fertility rate and ultimately on population growth. As emphasized by Young (2005), the AIDS/HIV epidemic with the adult mortality, the HIV infant mortality and the endogenous decreasing effect on fertility will lead to a lower population growth rate. Young (2005) argues that the positive effects of lower population growth are strong

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[2]Indeed, the time period of their estimates (1980-1992) is characterized by a growing, but still limited, AIDS epidemic.

[3]See Boucekkine, Diene and Azomahou (2006) for a survey.

[4]Indeed orphans due to AIDS are less likely to attend school. Moreover, the epidemic has not spared the teachers, increasing pupil-teachers ratios. Also the reduction of household earnings increases opportunity cost.

enough to offset the most pessimistic forecast of human capital losses, and therefore will bring about extra resources that will raise the per capita welfare of future generations.

A disputed issue : the impact on population growth through fertility   A long run positive effect of AIDS/HIV on growth depends ultimately on its impact on fertility. Theoretically, the impact can be either positive or negative. The first round effect of AIDS/HIV on fertility is negative coming from the direct biological impact of the disease, which lowers the fecundity of infected women. fecundity is reduced by HIV infection due to higher rate of miscarriage and stillbirth. There is an important and convergent literature confirming this negative direct effect[5]. For instance, Juhn, Kalemli-Ozcan and Turan (2008) find that being infected with HIV reduces births last year by approximately 21 percent.

The second round effect depends on behavioral response of individuals either infected and non infected. Thus, this behavioral response can offset or increase the negative first round effect, since it aggregates sexual behavior changes of all the women's population. As indicated by Young (2005), the rationals for a decrease of the fertility due to behavioral changes come directly from a reduction of the willingness to engage in unprotected sex, and indirectly, from the increase of wages due to scarcity of labor, which increase the opportunity cost of having a baby for women. However, the behavioral response of non-infected women may be to increase fertility, mitigating then the diseases' negative impacts on population. It follows from the link between mortality and fertility as exhibited by the historical pattern of demographic transition. There is no consensus on the impact of AIDS/HIV on fertility and individual behavioral response to the epidemic[6]. Young (2005) find that the HIV epidemic is lowering fertility in Sub-Saharan Africa. Kalemli-Ozcan (2009) using a restricted period of data (1990-1998) find that the effect of HIV prevalence on fertility is positive for South Africa. Ultimately, Forston (2009) and Fink and Linnemayr (2008) suggest that the AIDS/HIV epidemic had very little impact on fertility and is unlikely to have a significant effect on population size.

2.2   The theoretical model

We will denote N the working age population, L the equivalent number of full-time workers in the economy, K the aggregate stock of physical capital, S the average number of years of schooling among the workers, h the HIV prevalence rate and Y the total GDP. We consider an extended Solow-type growth model with both human and physical capital. The aggregate output is:

$$Y_t = F(A_t, K_t, L_t, S_t) = A_t K_t^{\gamma} L_t^{1-\gamma} e^{\xi(1-\gamma)S_t} \tag{1}$$

where $0 < \gamma < 1$. Total factor productivity A_t varies with time and can be affected by exogenous shocks such as civil conflicts for instance.

This framework allows to take into account the effects of the epidemic on the different sources of growth mentioned above. McDonald and Roberts (2006) consider a Solow augmented model that integrates directly health as a variable of the production function. In our specification, health is rather embedded in labor force participation and affects capital accumulation as well[7].

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[5]See Lewis and al. (2004) for a review.

[6]See Oster (2009a) and (2009b) for estimates of behavioral changes due to HIV prevalence.

[7]Indeed health is likely to have important effect on growth, see Bhargava and al. (2003).

In the long-run, fertility and thus the gross growth rate of population n may be endogenous as well. However, the changes in fertility is unlikely to have large effects on per adult income in the long-run. Indeed, although the change in fertility could affect the magnitude of the effects of HIV/AIDS on growth it is unlikely to modify their directions.

As infected adults have a shorter life expectancy, we model the net increase of the adult population as:

$$N_t = N_{t-1}\big(1 + n - vh_{t-1}\big) \tag{2}$$

Labour participation is supposed to be reduced by the epidemic. We assume therefore:

$$L_t = N_t(1 - uh_t) \tag{3}$$

We assume that physical capital depreciates at a constant rate δ and we introduce Inv the investment rate:

$$K_t = (1 - \delta)K_{t-1} + Inv(h)Y_t \tag{4}$$

Therefore the evolution of per adult income $y = \ln\left(\frac{Y}{N}\right)$, the variable we are interested in, only depends of the dynamics of the working age population $n_t = \ln(N_t)$, the labour force participation rate $l_t = \ln(L_t) - \ln(N_t)$, the lagged physical stock of capital $k_{t-1} = \ln(K_{t-1})$, the investment rate $inv_t = \ln(Inv_t)$ and the average years of schooling S_t:

$$y(h) = \ln(A_t) + l_t(h) + \gamma\Big(k_t(k_{t-1}, inv_t, y_t, n_t) - l_t - n_t\Big) + \xi S_t \tag{5}$$

To investigate how the epidemic affects the per capita income through the different channels, let us note $\tilde{x}_t$ the value the variable x_t would have taken if there had been no epidemic. The various effects of the epidemic on per capita income can be derived from the changes in the different factors x induced by the epidemic, $\Delta x_t = x_t - \tilde{x}_t$. The derivative of the production function gives:

$$\Delta y_t = (1 - \gamma)\Delta l_t + \gamma\left(\Delta k_t - \Delta n_t\right) + (1 - \gamma)\xi\Delta S_t \tag{6}$$

As the accumulation equation of physical capital is linear, we have to use a log-linear approximation at the first order to replace Δk_t. Introducing g the long-run growth rate of the economy[8]:

$$\Delta k_t \approx \frac{\Delta K_t}{K_t} \approx \frac{1 - \delta}{1 + g}\Delta k_{t-1} + \left(1 - \frac{1 - \delta}{1 + g}\right)(\Delta inv_t + \Delta y_t + \Delta n_t) \tag{7}$$

Denoting $\phi_K = \frac{1-\delta}{1+g}$, we could rearrange the equation (6) into four components:

$$\Delta y_t = \frac{\gamma\phi_K}{1 - \gamma(1 - \phi_K)}\left(\Delta k_{t-1} - \Delta n_t\right) + \frac{\gamma(1 - \phi_K)}{1 - \gamma(1 - \phi_K)}\Delta inv_t + \xi'\Delta S_t + \frac{1 - \gamma}{1 - \gamma(1 - \phi_K)}\Delta l_t \tag{8}$$

The first term measures the positive impact of the diminution of the labour force on per capita capital. The second and the third account for the effects of the disease on investment in both physical capital and human capital. The last one expresses the negative effect of HIV/AIDS on labour market participation. As the above system is linear, we know that there is a set of column matrix $\{A_i\}_{i=1}^t$ such that:

$$y_t = \tilde{y}_t + \sum_{i=1}^t A_i \begin{pmatrix} \Delta inv_i \\ \Delta S_i \\ \Delta n_i \\ \Delta l_i \end{pmatrix} \tag{9}$$

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[8]At the first order, we do not take into account the impact of the epidemic on the long-run growth rate here.

6

2.3 The accounting strategy

One may think to estimate the effects of HIV/AIDS on growth by looking separately at the effects on each components of the equation (9), that are investment, schooling, working age population and labour participation. The total effect of the epidemic on the per capita income would be:

$$\Delta y_t = \sum_{i=1}^{t} \left(A_i^{inv} \frac{\Delta inv_i}{\Delta h} + A_i^S \frac{\Delta S_i}{\Delta h} + A_i^n \frac{\Delta n_i}{\Delta h} + A_i^l \frac{\Delta l_i}{\Delta h} \right) \times h_i \tag{10}$$

However, only the first three multipliers, if significant, are likely to be measured directly using regressions. The last term however cannot be measured directly, as there is no available data on participation or on worked hours in most Sub-Saharan African countries.

A second strategy could be to estimate directly for the set of countries $\{i\}$ the following equation, where h_t^i is the epidemic's prevalence rate:

$$y_t^i = \tilde{y}_t^i + \alpha h_t^i + \varepsilon_t^i \tag{11}$$

This strategy is relevant only if the effects of the epidemic on capital accumulation have already materialized. If this is not yet the case, such an estimation is likely to underestimate the total effects of the epidemic, by capturing only the short-term effects, that are linked to labor participation.

To assert the relevance of this method, we have to calculate *a posteriori* the effects of the epidemic on both human and physical capital in order to assess whether or not the impact have already been substantial.

To measure all the effects of HIV/AIDS on growth both in the short and long-term, we propose in this paper the following strategy:

(i) In a first step, we estimate directly the effect of the epidemic on productivity.

(ii) In a second step, we estimate separately the effects of the disease on investment, education and the working age population by running the following regressions, where x is one of these variables:

$$x_t^i = \tilde{x}_t^i + \beta^x h_t^i + \varepsilon_t^i \tag{12}$$

To do so, we use control variables to instrument the unknown series $\tilde{x}_t^i$.

(iii) Using the equation (10) and the previously estimated multipliers $\frac{\Delta x_i}{\Delta h}$, we will calculate the evolution of the output per adult one should have observed if the epidemic has only influenced education, investment or the total adult population. If the sum of these three components represents a substantial part of the total effect estimated in the first step, then the regression (11) is unlikely to underestimate the total effect of the epidemic.

(iv) If the method appears to be valid, we finally estimate the remaining effect of HIV/AIDS, which is the impact on labour participation, by substracting the effects on education, investment and demography obtained from the direct estimation.

7

2.4 Common empirical framework

To run the previous regressions, one has to find satisfactory determinants of the variables $\tilde{z}_t$ being $\{x_t, y_t, y_t^*\}$. Let us assume that:

$$\tilde{z}_t^i = \rho^z \tilde{z}_{t-1}^i + \sum_{k=1}^{K} \beta_k^z Control_{t-1}^{ik} + u_i^z + \eta_t^z + \varepsilon_t^{zi} \tag{13}$$

where z_t^i is the dependent variable z at time t in the country i, u_i is the fixed effect of the country i, η_t is the year fixed effect and "CONTROL$_k$" represents the control variables.

To assess the effects of AIDS on demographic, education and macroeconomic variables, we therefore run the following panel within regressions with country and yearly fixed effects:

$$z_t^i = \rho^z z_{t-1}^i + \alpha^z h_{t-1}^i + \sum_{k=1}^{K} \beta_k^z Control_{t-1}^{ik} + u_i^z + \eta_t^z + \varepsilon_t^{zi} \tag{14}$$

The fixed effects take into account the heterogeneity between countries in terms of infrastructure, wealth or religion as well as the impact of global shocks such as crop or oil prices. To take into account autocorrelation problems we run GLS regressions with country fixed effects and with clustered errors within countries. To limit the issue of potential endogeneity of the HIV and other control variables, we only introduce lagged variables at the right hand side of equation (14). This estimation strategy is consistent with the one followed by McDonald and Roberts (2006) and Kalemli-Ozcan (2006).

2.5 Dealing with endogeneity

There are several good reasons to suspect that HIV prevalence could be affected by economic activity. If this variable were partially endogenous, simple estimations would be biased. Indeed higher growth could attract migrants or fasten urbanization and facilitate the diffusion of the disease, making then HIV increase with growth. Urbanization could also affect social control over sexual behaviors and contribute to the spread of the epidemic. It has also been claimed that industrialization, especially mining activities, could favor prostitution and in turn the spread of HIV. Otherwise, countries growing faster may also allocate more resources to health and treatments, reducing the mortality of HIV infected people. However, longer life expectancy of sick people induces higher prevalence rate among the population. To deal with endogeneity, we may think to instrument the series of HIV prevalence rate by external series.

However the epidemic feature of HIV/AIDS induces that prevalence rates are highly autocorrelated. They differ across time and from a country to another less because of initial conditions but rather because of particularities affecting the speed of diffusion of the disease. Therefore finding an exogenous series directly explaining the level of the epidemic within a country is difficult. It is more relevant to seek rather instruments explaining the speed of diffusion of the epidemic within a country.

Our strategy to tackle potential endogeneity issues is therefore to build a series of HIV prevalence rate from a theoretical dynamic equation where the speed of propagation of the disease is instrumented. The dynamic of the epidemic is derived from a theoretical model of propagation of HIV presented in the next section.

8

3 The data: sources, building and robustness

The data are from several international institutions and cover 46 countries of Sub-Saharan Africa on a yearly basis from 1970 to 2007. Data on HIV/AIDS epidemic are brought from three different sources:

- The UNAIDS (2003) epidemiological fact sheets provide raw data on HIV prevalence among pregnant women in rural or urban areas for most countries between 1987 and 2003. The methodology used is consistent over time and countries. However, it is likely to overestimate the extent of the epidemic among the labor force, as young adults are more likely to be infected by HIV. Moreover, as data were collected only on specific surveillance site, which could vary across time, these data are volatile and were not collected each year.

- The fact sheets[?] report also AIDS cases during the 80s and the early 90s. Collected data are less volatile but may underestimate the extent of the epidemic, as AIDS was not clearly identified as a specific disease in the 80s.

- Eventually the UNAIDS has recently released estimates of HIV prevalence among the adult population from 1990 to 2007. The UNAIDS used available data[9] to calibrate model-based simulations of the spread of the epidemic. This source provides therefore smoother series.

Unfortunately, using only one of these sources to run empirical estimate is not sufficient to identify the effects of HIV/AIDS on growth. First, because treatments and even diagnosis were poor before the 90s, then the disease, although less frequent, were likely to affect more productivity and fertility at that time. Second, the volatility of growth is substantial in African countries, because of conflicts, commodity prices volatility and weather hazard. Thus, long series are necessary to identify a convincing effect of HIV/AIDS on growth. Furthermore as the epidemic affected a limited share of the population in most African countries, the thought effect of HIV/AIDS on growth is likely to remain small in the short-run. To obtain robust results, we build *three different estimates of HIV prevalence rate*. The first two series are based on UNAIDS data, the last one is our own construction.

3.1 HIV prevalence estimates using prevalence among pregnant women

The data on HIV prevalence among pregnant women (hpw) have not been collected on a yearly basis. To build a comprehensive dataset, one can first use the correlation between urban and rural prevalence ($hpwr$) within a country to "predict" the national data. To build comparative data mostly between 1982 and 1990, one can use the correlation between AIDS case ($Case$) and HIV prevalence among women in urban areas ($hpwu$). AIDS cases were probably less underreported in urban areas, where health facilities are larger and better endowed.

The following equation, where u^i is a country fix effect, is estimated, using GLS regressions, to predict the missing data for HIV prevalence rate among pregnant women in rural area.

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[9] Raw data from the epidemiological UNAIDS fact sheets (2003) and also direct estimates of HIV prevalence among the population from household surveys conducted since 2003.

$$hpwr_t^i = \rho_r hpwr_{t-1}^i + \lambda_r hpwr_{t+1}^i + \gamma_r hpwu_t^i + u^i + \varepsilon_t^i \qquad (15)$$

Table A1 in appendix gives the correlations between HIV prevalence rate among pregnant women in rural areas and urban areas. It gives the coefficient used to predict HIV prevalence rate among pregnant women in rural areas. AIDS cases do not appear to be a good predictor of HIV prevalence rate in rural areas. The number of observations predicted using each regression is indicated in the last row "# predict". Predictions are calculated using the equation (15) and assuming that $\varepsilon_t^i = 0$. The different estimations of the table A1 are used to project HIV prevalence according to available data.

The following equation is used to estimate HIV prevalence rate among pregnant women in urban areas, using the same method. Results are reported in the table A2 in appendix.

$$hpwu_t^i = \rho_u hpwu_{t-1}^i + \lambda_u hpwu_{t+1}^i + \gamma_u hpwr_t^i + \alpha_u Case_{t-1}^i + u^i + \varepsilon_t^i \qquad (16)$$

HIV prevalence rate among pregnant women in total population is finally calculated as a weighting average of HIV prevalence rate among pregnant women in rural and urban population. However, pregnant women prevalence rates are available between 1987 and 2002 only. To complete the data set between 1982[10] and 1987, one can use reported AIDS cases and prevalence rate among the whole population (see table A3 in appendix).

$$\ln(hpw_t^i) = a\ln(Case_t^i) + u^i + \varepsilon_t^i \qquad (17)$$

This is the first series of prevalence rate.

3.2 Extension of UNAIDS estimates

UNAIDS estimates are not available for several countries in the early 90s. We use the lower and upper bound estimates to rebuild a main projection (see in appendix table A3, columns 4, 5 and 6).

$$\ln(hiv_t) = a^l \ln(hiv_t^l) + a^u \ln(hiv_t^u) + \ln(h_0) \qquad (18)$$

As linear HIV estimates are likely to be negative for numerous countries before 1990, we run an exponential regression model (see table A3).

$$\ln(hiv_t) = \ln(hiv_{t+1}) + a'\Big(\ln(Case_t) - \ln(Case_{t+1})\Big) + \ln(h_0) \qquad (19)$$

HIV prevalence data are projected backward using equation (19). But such a projection is not possible for countries that do not have estimates of HIV prevalence around 1990. Prevalence was indeed not sufficiently high to require the collection of data on AIDS in those countries. We use directly AIDS cases as a proxy of prevalence and run the regression (20) on countries with low prevalence rate, (see last column of table A3).

$$h_t = c_1 Case_t + c_2 Case_t^2 + c_0 \text{ for } h_t < 0.02 \qquad (20)$$

This gives the UNAIDS series reconstructed for the early 80's.

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[10]Prevalence rates of HIV are assumed to be null everywhere before 1982.

3.3 Building a new series of HIV prevalence rate based on propagation dynamics

As HIV prevalence series are evolving substantially across time, it is unlikely to find exogenous series allowing to instrument directly the profiles of the epidemic in several countries. Indeed Werker and al. (2006) managed to instrument HIV prevalence only in a cross-section of countries, but did not provide instrumented time series.

Our approach is to estimate the propagation dynamic of the epidemics from exogenous variables and variables very poorly correlated to growth[11]. We consider that the spread of propagation of HIV in a given country and the initial conditions are endogenous. We use therefore several variables arguably independent from growth as instruments. We proceed in three steps. First, we instrument the initial extent of the epidemic (initial conditions) using mostly geographical variables such as the distance to Congo Dem. Rep. (as Oster 2009). Second, we instrument (using a panel-data regression) the spread of propagation of the epidemic using mostly circumcisions rates, religion and road network. Although those variables are quite stable in time they do vary a little (except for circumcision rates because we only have one data for each country). Third, we simulate "instrumented" HIV prevalence profile for each country, starting from the "instrumented" initial infection, and using the estimated coefficient for our instruments. It is a projection of the speed of propagation of the epidemics.

As the values of the instruments varies from one country to another, the speed of propagation differs and as a consequence, the profiles are different. It is worth noting that as the propagation equation is quadratic, the long-term value of the prevalence (as well as the peak value) also depend on each country conditions. For instance, countries with more Muslims will experience lower HIV prevalence rate in the long-run according to our model.

3.3.1 Estimation of the propagation dynamics of AIDS

We build a simple dynamic model of epidemic propagation (see appendix for a full presentation). The model rests on the marital status of people (single or married), their health status (infected and non infected) and their location (which city). It allows mobility of people from one city to another. Combining these elements, we can deduce the following dynamic of HIV prevalence among the total population. A linear trend is allowed to take into account the global evolution of the HIV/AIDS epidemic.

$$h_{t+1} - h_t = \beta_1(\nu, \iota, \phi, \delta^a - \delta, \psi, m)h_t - \beta_2(\nu, \iota, \phi, \delta^a - \delta, \psi, m)h_t^2 + \beta_3 t + \beta_4 \qquad (21)$$

To identify this equation, we introduce the following instrumental variables :

- The share of unprotected intercourse ν can depend on time as people become more aware of the disease as HIV spreads across the continent. It may also decrease with the heterogeneity of the population as people might be more cautious with someone they do not know; it is measured by fractionalization indicators[12].

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[11]See in apendix a full description of the dynamic model of propagation of the epidemics and the fondations of the links between the instrumental variables and AIDS dynamics.

[12]Pongou and al. (2009) show in a network model the link between ethnic composition of the population and the spread of the HIV/AIDS epidemic.

- The probability of being contaminated by an unprotected intercourse ι has been proved to be lower for circumcised men[13]. We therefore introduce the share of circumcised men in the population as an instrument of the speed of propagation.

- Geographic mobility can be instrumented using the extent and the quality of the road network. We use the current number of kilometers of roads divided by the number of inhabitants in the country in 1980[14] and the share of paved roads for continental countries[15]. Mobility indicators are expected to increase prevalence.

- The differential of mortality between healthy and sick people may be impacted by civil conflicts and wars.

- We use the proportion of Muslims as an instrument of the share of faithful people.

- The rate of mixed unions, m could be also influenced by population fractionalization.

These instruments are poorly correlated with growth, which allows to treat them as good candidates as exogenous variables (see table 1).

Table 1: Partial Correlations (period 1970-2008)

	growth	musl.	ethnic	island	Lodja dis.	paved	roads
growth	1.00						
Muslims	-0.02	1.00					
ethnic fraction.	-0.09	-0.11	1.00				
island	0.01	-0.07	-0.40	1.00			
Lodja distance	-0.02	0.46	-0.00	0.34	1.00		
paved roads	0.05	0.08	-0.47	0.77	0.44	1.00	
road network	0.02	-0.23	-0.01	-0.11	-0.15	-0.18	1.00
circumcision	-0.02	0.55	0.42	-0.13	0.26	-0.14	-0.25

Rewriting equation 21 with the vector of instruments $Z_t^i = (1,\ ethnic_t^i,\ t,\ network_t^i,\ paved_t^i,\ muslm_t^i,\ civconf_t^i,\ civwar_t^i,\ circum_t^i)$ and γ_1 and γ_2 two vectors of parameters, the estimated propagation dynamic is:

$$h_t^i = h_{t-1}^i + \gamma_1' Z_{t-1}^i \times h_{t-1}^i + \gamma_2' Z_{t-1}^i \times (h_{t-1}^i)^2 + \gamma_T t + \gamma_0 + \epsilon_t^i \tag{22}$$

The results are reported in table 2 below.

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[13]See Weiss and all (2000) or Halperin and Bailey (1999) for a survey or Gray and alli (2007), for a recent randomized experience in Uganda.

[14]To avoid capturing demographic growth.

[15]Indeed the spread of the epidemics has been dramatically reduced by the isolation of island countries.

Dep. Var.	hiv	hiv	hiv
Table 2: Estimation of the dynamic of HIV prevalence			
hiv_{-1}	0.30*** (12.3)	0.30*** (13.1)	0.333*** (16.7)
$paved \times hiv_{-1}$	0.78*** (10.8)	0.77*** (10.8)	0.75*** (10.6)
$network \times hiv_{-1}$	1.60** (3.9)	1.54*** (4.1)	0.58*** (6.1)
$ethnic \times hiv_{-1}$	−0.18*** (5.3)	−0.17*** (13.5)	−0.18*** (14.3)
$mus \times hiv_{-1}$	−0.02 (0.3)	−	−
$circum \times hiv_{-1}$	0.06 ** (2.6)	0.06 ** (2.7)	0.05** (2.3)
$civc \times hiv_{-1}$	−0.11*** (5.7)	−0.12*** (5.9)	−0.12*** (6.4)
$civd \times hiv_{-1}$	−0.09** (2.5)	−0.11*** (7.1)	−0.11*** (7.3)
$t \times hiv_{-1}$	−0.01*** (21.1)	−0.01*** (21.3)	−0.01*** (21.1)
hiv^2_{-1}	−0.35** (2.9)	−0.34** (3.0)	−0.55*** (6.8)
$paved \times hiv^2_{-1}$	−1.84*** (4.6)	−1.81*** (4.8)	−1.16*** (4.2)
$network \times hiv^2_{-1}$	−7.28 (2.5)	−6.84 ** (2.6)	−
$ethnic \times hiv^2_{-1}$	6.74 (0.3)	−	−
$mus \times hiv^2_{-1}$	−0.89** (1.2)	−1.08** (2.4)	−1.19** (2.6)
$circum \times hiv^2_{-1}$	−0.52 *** (3.4)	−0.51 *** (3.6)	−0.48*** (3.4)
$civc \times hiv^2_{-1}$	0.31* (2.0)	0.32* (2.1)	0.39** (2.5)
$civd \times hiv^2_{-1}$	−0.13 (0.3)	−	−
t	−0.01** (2.8)	−0.01** (3.2)	−0.70*** (3.4)
$Intercept$	0.09*** (4.3)	0.09*** (4.3)	0.09*** (4.3)
# obs.	1279	1279	1279
adj. R^2	0.53	0.53	0.53

*,**,*** indicate respectively significance at the 5%, 1% and 0.1% level.

3.3.2 Simulation of the new series of HIV prevalence rate using the estimation of the propagation mechanism

We proceed in two steps to build a new series. First, the HIV prevalence rate is estimated for every country in 1990, which is the first date for which UNAIDS data is available. As prevalence rate are very small in most countries at this date, we use an exponential regression[16]. We take as instruments the share of the population being Muslims ($muslims$), the share of the population being catholic ($catho$), the distance D from the main city of the country to Lodja, the city located at the center of the Democratic Republic of Congo[17],

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[16]Indeed, linear estimates of HIV prevalence rate would be negative for a large number of countries. We impute the minimal value of 0.001 for the prevalence rate when actual UNAIDS estimates are below 0.01.

[17]As Democratic Republic of Congo is supposed to be the epicenter of the epicemic.

the rate of circumcised male (*circum*) and (*isl*) for islands. The results of this estimation are given in equation (23) below.

$$\ln\left(h^i_{1990}\right) = \left(\underset{(3.2)}{-1.3}\ln\left(D^i\right) - \underset{(3.9)}{2.2}\,musl^i\right)\times(1-Isl^i) - \underset{(4.6)}{14.2}Isl^i - \underset{(2.3)}{1.2}\,circ^i - \underset{(3.4)}{2.5}\,catho^i + \underset{(3.8)}{11.9} + \eta^i \tag{23}$$

To obtain an unbiased estimation of the prevalence rate in 1990, one should take into account the variance of the error term η. Denoting Z^i_0 the vectors of instruments used in equation 23 and γ_0 the vector of estimated parameters, we have:

$$\hat{h}^i_{1990} = E[h^i_{1990}] = E[e^{\gamma'_0 Z^i_0 + \eta^i}] = e^{\gamma'_0 Z^i_0} E[e^{\eta^i}] = exp\left(\frac{Var[\eta^i]}{2}\right)e^{\gamma'_0 Z^i_0} \tag{24}$$

which gives the estimation of HIV prevalence rate in 1990 for each country.

Second, the estimation of the propagation dynamic of HIV prevalence, given by table 2, is used to build the series forward, starting from the 1990 estimates and assuming null residuals. Therefore, this series does not contain any value of the actual series h^i_t. It is strictly constructed from the values of the instruments. The forward-looking series departing from 1990 is given by:

$$\hat{h}^i_t = \hat{h}^i_{t-1} + \gamma'_1 Z^i_{t-1} \times \hat{h}^i_{t-1} + \gamma'_2 Z^i_{t-1} \times (\hat{h}^i_{t-1})^2 + \gamma_T t + \gamma_0 \text{ if } t > 1990 \tag{25}$$

This process is reproduced backward using the reverse of the equation 22 to project the prevalence before 1990:

$$\hat{h}^i_{t-1} = \frac{\hat{h}^i_t - \gamma_0 - \gamma^T t}{1 + \gamma'_1 Z^i_{t-1} + \gamma'_2 Z^i_{t-1} h^i_t} \text{ if } t < 1991 \tag{26}$$

The following graph plots the average of HIV prevalence across Sub-Saharan Africa for each year and for the three different series, as well as the Oster's (2009) estimates. It appears that those series differ both by their level and dynamic. Our instrumented-simulated series becomes higher than expected since the early 2000 and stabilizes at a higher level. This gap could be explained by a change in behavior, that has not be taken into account by our instruments[18]. See figure 6 in appendix for the comparison between our instrumented series and the UNAIDS one for a set of countries.

It is worth noting that our instrumented-simulated series, thought that it is not based on observed AIDS cases, it compares fairly to UNAIDS and Oster series. It expands these series either in terms of the number of countries covered and of time-span. It allows us to perform our accounting exercise going back from early stage of the epidemics.

3.4 Sources of other data

Infant mortality data are available on a five-year average basis in the United Nations World Urbanization Prospects. We compute annual data from this source using linear projections. Fertility, life expectancy and demographic data, such as working age population[19] or urbanization are published on a yearly basis in the World Development

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[18]Although strong evidence supports such a change in behaviors since 2000, there is unfortunality no relevant data to measure it, which would be available and comparable for all coutries.

[19]The working age population, that is the share of population between 15 and 64 is not available for the Seychelles. We use therefore the total population for this country.

Figure 1: HIV prevalence series by year, average of all Sub-Saharan African countries.
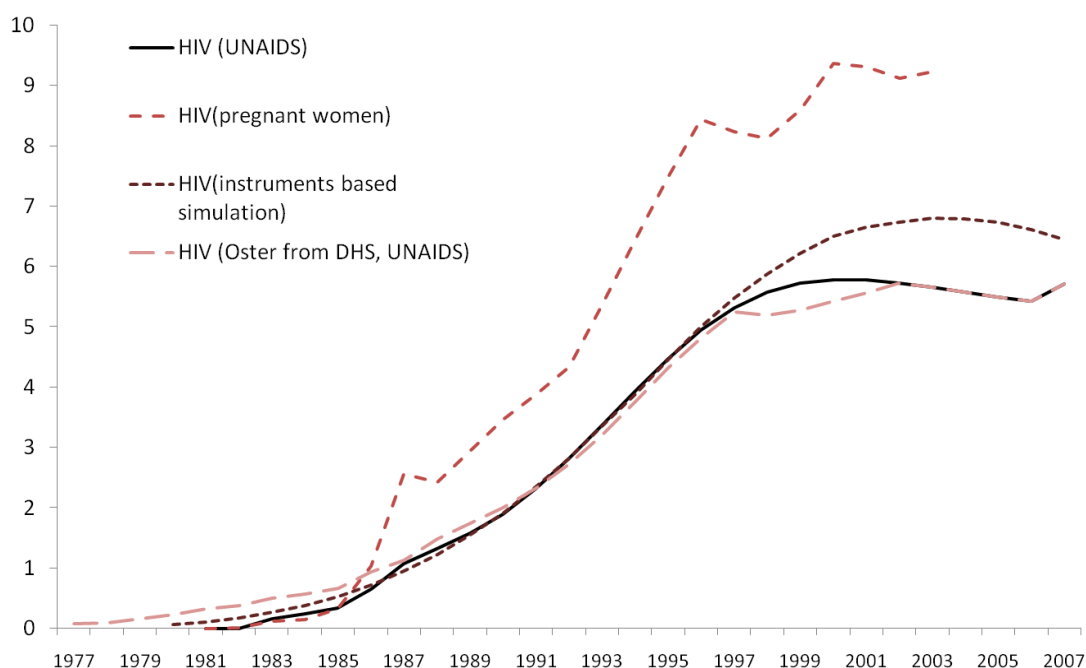
Indicators of the World Bank. Estimates of GDP and net formation of fixed capital (in constant US$), data on roads, development aid and assistance, openness to trade, foreign direct investments and education data including primary and secondary enrolment rate are pulled from the WDI as well. Data on conflicts are extracted from the third version of the Armed Conflict Dataset of the Uppsala Conflict Data Program, (UCDP/PRIO, 2007). This dataset contains annual observations of conflicts about all members of the international system, as defined by Gleditsch & Ward (1999), between 1946 and 2006. Data on Muslims and catholic are compiled using several sources such as census data from the UN, household surveys data from the Demographic and health surveys and the CIA fact book. Fractionalization data are from Alesina and al. (2003). Circumcision rates are provided by Williams and al. (2006). All measures of HIV/AIDS used in the following are prevalence rates.

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Table 3 : Descriptive statistics

Variables	# obs.	Mean	Std. Dev.	Min	Max	Source
GDP (constant 2000 US$)	1 662	1483	1933	98	19376	World Bank
Work. age pop. (thousands)	1 702	5715	8960	36	76656	World Bank
Fertility	1 726	6.0	1.3	1.9	8.5	World Bank
Life expectancy at birth	1 727	50.7	7.6	23.6	73.2	World Bank
Infant Mortality (for 1,000)	1 610	109.6	36.8	14.6	206.4	UN
HIV preg. women	1 498	0.03	0.061	0	0.417	UN Facts sheets
HIV	1 778	0.023	0.050	0	0.289	UNAIDS
Primary enrollment (%)	1 562	77.0	33.2	8.0	179.6	World Bank
Secondary enrollment (%)	1 536	21.6	18.4	1.1	114.0	World Bank
Urbanization (%)	1 739	29.0	14.3	2.4	84.1	World Bank
Civil conflict (occurrence)	1739	0.2	0.2	0	1	UCDP/PRIO
Civil war (occurrence)	1739	0.2	0.2	0	1	UCDP/PRIO

Notes: Variables are averaged over 1970-2006. Fertility is the total number of children per woman. HIV prevalence is the previously built variable. Enrollment in primary and secondary schools are gross. Urbanization is the share of population in urban areas. See Table A5 in appendix for partial correlations between variables.

4   The effects of HIV/AIDS on growth

4.1   Estimation of the direct effect of HIV/AIDS on productivity growth

A direct way to grasp the effect of HIV/AIDS on growth is to include the epidemic prevalence rate among the regressors. Following equation (14) which gives the common empirical framework for the different estimations, we run the following regression, where Y stands for the GDP, L for the working age population, HIV for the HIV prevalence rate and cc and cw respectively indicate the occurrence of a civil conflict or a civil war[20]:

$$\ln\left(\frac{Y_t^i}{L_t^i}\right) = \rho \ln\left(\frac{Y_{t-1}^i}{L_{t-1}^i}\right) + \alpha HIV_{t-1}^i + \beta^c Cv_t^i + \beta^w cw_t^i + u_i + \eta_t + \varepsilon_t^i \qquad (27)$$

By definition the ratio of GDP to working age population is a measure of the productivity per worker, therefore we will use indifferently growth or productivity growth. Table 4 below reports the different results. Recall that HIV denote the HIV prevalence rate from UNAIDS completed following the methodology described in section 3.2. HWP is the HIV prevalence rate among pregnant women constructed as described in section 3.1 with UNAIDS estimates of prevalence rate in rural and urban zone. And, HIVinst is the series we constructed completely following the model described in section 3.3 using instrumental variables.

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[20]By definition, a country experienced a conflict when at least 25 and at most 999 battle-related deaths occured within one year, and a war when at least 1,000 battle-related deaths occured within one year.

Table 4 : The effects of AIDS on productivity growth

Dep. var.	$\ln(Y/L)$	$\ln(Y/L)$	$\ln(Y/L)$	$\ln(Y/L)$	$\ln(Y/L)$	$\ln(Y/L)$	$\ln(Y/L)$
Method	Within†	Within†	Within	Within	Within	Within†	Within
$\ln(Y_{-1}/L_{-1})$	0.97*** (66.9)	0.96*** (34.9)	0.95*** (68.1)	0.96*** (34.0)	0.96*** (24.7)	0.96*** (35.1)	0.96*** (34.0)
HIV_{-1}	−0.14** (2.9)	−0.11* (2.4)	−0.12* (2.1)	−0.15* (2.2)	—	—	—
HWP_{-1}	—	—	—	—	−0.10* (2.1)	—	—
$HIVinst_{-1}$	—	—	—	—	—	−0.13* (2.2)	−0.13* (2.0)
Controls:	Civil conflicts & wars, intercept, year and country fixed effects						
Add. controls:	-	-	-	Reg YFE	-	-	Reg YFE
# obs.	1509	1174	1196	1174	1292	1179	1179
# countries	45	45	45	45	45	45	45
Period	70-07	81-07	70-00	81-07	81-04	70-07	81-07
adj. R^2	0.95	0.94	0.92	0.94	0.94	0.94	0.94

Notes: *,**,*** indicate respectively significance at the 5%, 1% and 0.1% level.

T-values reported between brackets are calculated by clustering errors by country.

Civil wars and civil conflicts are always negative and significant at least at the 1% level.

Additional controls are regional (Northern, Western and Eastern Africa) year fixed effects.

Breush and Pagan test rejects homoscedasticity in all configurations.

† indicates that the Hausman test confirms the relevance of country fixed effects.

Table 4 indicates that the lagged growth is the most important determinant and civil conflicts and wars have the expected negative and significant impact. It is worth noting that the effect of HIV prevalence on growth is neither identified nor significant when both civil disorders variables and yearly fixed effects are not added. This result underlines the necessity to control for the sources of volatility of growth in the short-run.

These estimates are robust to the introduction of regional year fixed effect and the use of any measure of HIV prevalence. We did not use Oster's prevalence series in our estimates due to its restriction to a fewer number of countries and time-span.

The epidemic prevalence rate has a negative and significant impact on growth. The results are not sensitive to the series used. The magnitude of the coefficient remains similar when using the series built directly from the UNAIDS estimates or the one constructed from the raw data on prevalence among pregnant women.

Moreover, the impact is similar when using the simulated series. As by construction this series is supposed to be more exogenous, the endogeneity bias appears to be limited. Finally, the results is not sensitive to the period (see line Period) of estimation.

Recalling equation (27), we can calculate $\widetilde{y_t}$, that is the dynamic of the logarithm of GDP per working age adult if HIV prevalence had remained null till the early 1980s.

$$\widetilde{y_t^i} = \rho\widetilde{y_{t-1}^i} + \beta^c Cv_t^i + \beta^w cw_t^i + u_i + \eta_t + \varepsilon_t^i \tag{28}$$

By substracting eq. (28) from equation (27), we can obtain the dynamic of the "AIDS gap", Δy_t, which is the difference between the actual level of GDP per adult and the would be GDP per adult if they were no HIV/AIDS.

$$\Delta y_t^i = y_t^i - \widetilde{y_t^i} = \rho\Delta y_t^i + \alpha HIV_t^i \tag{29}$$

In the short-run, the effect of HIV/AIDS on productivity is limited. A 1 point increase in prevalence rate induces a 0.13 point reduction of GDP per working age adult. But this

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negative effect cumulates over time and the "AIDS gap" becomes much larger. In the long-run, the effect of a permanent increase of HIV prevalence Δh on GDP per working age adult is:

$$\lim_{t \to \infty} \Delta y_t = \frac{\alpha}{1 - \rho} \lim_{t \to \infty} HIV_t \qquad (30)$$

Using the second regression (column 2, Period 81-07), a 1 point increase in prevalence induces indeed a 2.75 reduction in productivity in the long-run. Although likely to be negative, the magnitude of the effect remains nevertheless uncertain. Assuming that the distribution of the parameters α and ρ is normal, there is no easy analytical expression of the distribution of the ratio $\frac{\alpha}{1-\rho}$. Therefore, we use Monte-Carlo simulations to compute its distribution[21]. Figure 2 shows the distribution of the long-run effect of a 1 point HIV prevalence increase on productivity. A 1 point increase in HIV prevalence induces in average a 2.75 point decrease in GDP per working age adult.

Figure 2: Distribution of the effects of a 1 point increase of the prevalence rate
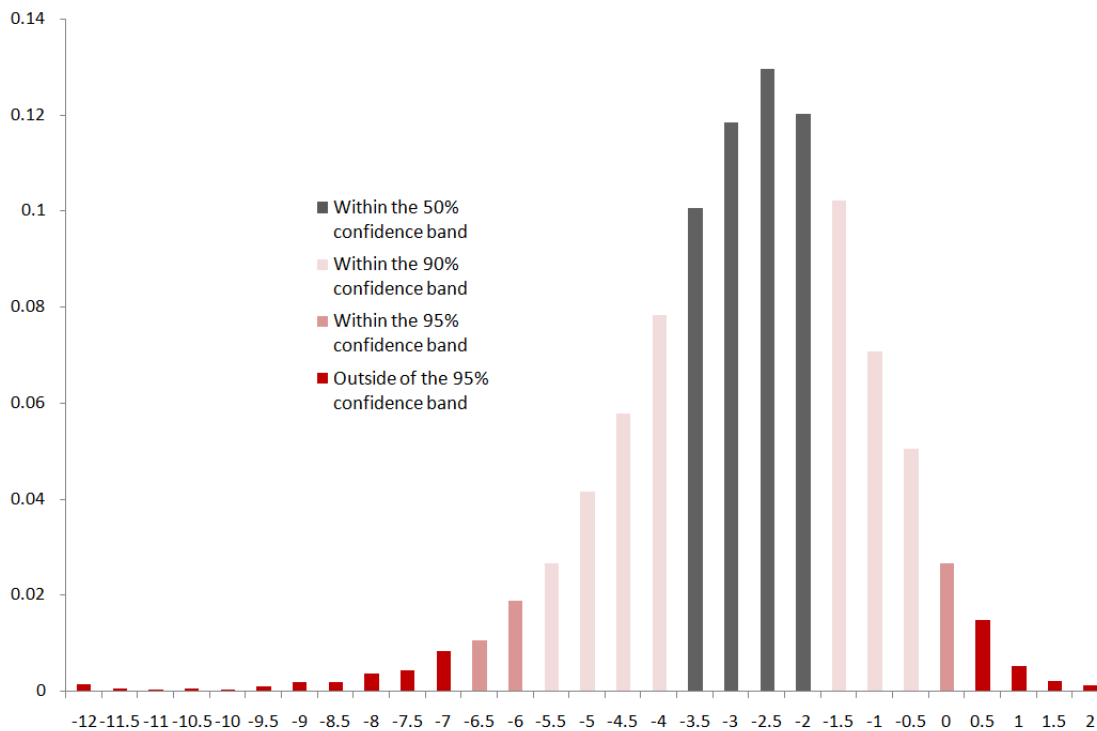
In the long-run, this means that the effect of HIV/AIDS on growth goes far beyond the diminution of the labour force[22]. This estimate is approximately half of what Bonnel (2000) obtained. And, it appears to be large compared to McDonald and Roberts's (2003)[23]. However, their method is likely to underestimate the effects of HIV/AIDS

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[21] We use a random sample of 10,000 observarions, assuming no correlation between α and ρ to plot this distribution. The distributions of α and ρ are assumed to be normal and are calibrated using the estimated expectancy and standard deviation of the regression 2 of table 4.

[22] If the epidemic just prevented the infected people to work, one should expect the multiplier to be around 1 in the long-run.

[23] According to their results, a 1 percent increase in HIV prevalence induce a drop of 0.6 points in per capita income in Africa in the long-run.

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on growth for two reasons. First, they do not take into account the effects of health on both physical and human capital accumulation[24]. Second, they measured the effects of HIV/AIDS on growth indirectly using infant mortality as a proxy of workers's health capital. But contrary to other endemic diseases such as malaria, HIV is well known to hit disproportionately adults, as the main source of infection is unprotected sexual intercourse. Our estimate is also approximately twice lower than Papageorgiou and Stoytcheva's (2005)[25].

To gauge the effect of the epidemics on the different channels considered in section 1, we investigate its direct effect on the different variables that determine short and long run growth.

4.2 Estimation of the effects of HIV/AIDS on the sources of growth

4.2.1 Effects of HIV/AIDS on working age population

The direct effect of the epidemic is to reduce life expectancy and demographic growth. Estimation of those straightforward effects of the epidemic reported in the table 5 confirm that all the presented measures of HIV prevalence are in the relevant interval. The effects appear to be negative and significant either on life expectancy or on working age population.

Table 5 : HIV prevalence rate effects on demographic variables						
Dep. Var.	LifeEx	LifeEx	LifeEx	$\ln(L)$	$\ln(L)$	$\ln(L)$
Method	Within†	Within	Within	Within†	Within†	Within†
$LifeEx_{-1}$	0.94^{***} (60.90)	0.98^{***} (49.2)	0.99^{***} (91.3)	—	—	—
$\ln(L_{-1})$	—	—	—	0.96^{***} (57.3)	0.94^{***} (46.7)	0.96^{***} (108.7)
HIV_{-1}	-10.57^{***} (6.6)	—	—	-0.08^{***} (3.7)	—	—
HPW_{-1}	—	-6.46^{***} (5.6)	—	—	-0.04^{*} (2.4)	—
$HIVinst_{-1}$	—	—	-3.93^{**} (2.9)	—	—	-0.07^{***} (3.7)
Controls:	Intercept, year and country fixed effects					
Controls:	$\ln(GDP/L)$			Civil conflicts and wars		
# obs.	1145	930	1145	1175	957	1180
# countries	45	43	45	45	45	45
Period	81-07	81-04	81-07	81-07	81-04	81-07
adj. R^2	0.98	0.98	0.98	0.99	0.99	0.99

Notes: *,**,*** indicate respectively significance at the 5%, 1% and 0.1% level.

T-values reported between brackets are calculated by clustering errors by country.

Breush and Pagan test rejects homoscedasticity in all configurations

† indicates that the Hausman test confirms the relevance of country fixed effects.

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[24]Bonnel's and our own estimates (see section 4.2) indeed indicate that HIV/AIDS affect significantly both investment rate and school enrollment.

[25]They reported that each AIDS case (over 100,000 people) induced a 0.3-0.4 points decrease of GDP per worker. With an average of 22 cases between 1979 and 2000, the epidemic would have costed during this period between 6.5 and 9.0 points of GDP per worker in Africa versus 4.0 pts according to our estimates.

4.2.2 Effects of HIV/AIDS on human capital

To assess the effects of the epidemic on human capital, we, first, estimate the effect on primary and secondary school enrollment rate, and second we compute the total effect on the stock of human capital.

Determining the effects of HIV/AIDS on enrollment rates requires also some work on the data. Indeed enrollment rates are reported by the World Bank on a five-year basis before the late 1990s for most of the African Sub-Saharan countries. Moreover, even on a five-year basis, data are not available for the same years across countries. To solve that problem, we propose two methods.

First, we run yearly regressions on existing data x_t^i using five-year lagged variables. When the five-year lagged figure x_{t-5}^i is not available, we use instead $\widehat{x_{t-5}^i}$ the closest lagged available data within the five-year span. This projected lagged value is defined as:

$$\widehat{x_{t-5}^i} = \begin{cases} x_{t-5}^i & \text{if} & x_{t-5}^i \text{ is defined} \\ x_{t-s}^i & \text{if} & s \in [1,4] \text{ and } x_{t-s}^i \text{ is defined} \end{cases} \tag{31}$$

Second, we use linear projection to complete the series. Let us consider the variable x_t^i which values are known only for $t \in K = \{t_k\}$. The projected series $\widetilde{x_t^i}$ is therefore define as:

$$\widetilde{x_t^i} = \begin{cases} x_t^i & \text{if} & t \in K \\ \frac{t_{k+1}-t}{t_{k+1}-t_k}x_{t_k}^i + \frac{t-t_k}{t_{k+1}-t_k}x_{t_{k+1}}^i & \text{if} & t \in]t_k, t_{k+1}[\end{cases} \tag{32}$$

Effects of AIDS on primary school enrollment We use our empirical framework (see equation 14) to identify the effects of HIV/AIDS on school enrollment rates. We introduce also, $Foreign\ Aid$, the share of total aid and assistance into GDP as a control variable. The first regression run is, denoting PGE the total[26] primary gross rate of enrollment as:

$$PGE_t^i = \rho^p \widehat{PGE_{t-5}^i} + \alpha^p HIV_{t-1}^i + \beta^c cc_t^i + \beta^w cw_t^i + \beta^a Aid_{t-1}^i + u_i + \eta_t + \varepsilon_t^i \tag{33}$$

Because of the lack of yearly data before 1990, the number of observations is limited. Regression results are reported in table 6. The equation estimated using projected data is the following.

$$\widetilde{PGE_t^i} = \rho^p \widetilde{PGE_{t-1}^i} + \alpha^p HIV_{t-1}^i + \beta^c cc_t^i + \beta^w cw_t^i + \beta^a Aid_{t-1}^i + u_i + \eta_t + \varepsilon_t^i \tag{34}$$

The epidemic seems to have a negative impact on gross primary enrollment. Although multipliers and standards errors vary widely with the specification, the correlation is significantly negative using both raw (PGE) and projected ($\widetilde{PGE}$) data. Moreover, estimates obtained using either the UNAIDS or our constructed series are similar.

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[26]Both boys and girls.

Table 6 : The impact of HIV/AIDS on primary school enrollment rate

Dep. Var.	PGE	PGE	PGE	PGE	PGE	$\widetilde{PGE}$	$\widetilde{PGE}$
Method	Within	Within†	Within†	Within	Within†	Within	Within
$\widehat{PGE_{-5}}$	0.42*** (7.2)	0.39*** (7.6)	0.36*** (6.8)	0.44*** (7.2)	0.40*** (7.5)	–	–
$\widetilde{PGE_{-1}}$	–	–	–	–	–	0.97*** (97.8)	0.97*** (80.0)
HIV_{-1}	−0.67** (3.1)	−0.70** (2.7)	−0.84* (2.4)	–	–	−0.18** (4.2)	–
$HIVinst_{-1}$	–	–	–	−0.68* (2.6)	−0.56* (1.8)	–	−0.22** (3.1)
Controls:	Foreign aid$_{-1}$, Civil conflicts and wars, intercept, country fixed effects.						
Add controls:	YFE	Trend	R trend	YFE	Trend	YFE‡	YFE‡
# obs.	565	565	565	560	560	1432	1123
# countries	46	46	46	45	45	47	46
Period	70-07	70-07	70-07	70-07	70-07	81-07	81-07
adj. R^2	0.51	0.42	0.45	0.50	0.41	0.96	0.95

Notes: *,*,**,*** indicate respectively significance at the 10%, 5%, 1% and 0.1% level.

T-values reported between brackets are calculated by clustering errors by country.

Breush and Pagan test rejects homoscedasticity in all configurations

† indicates that the Hausman test confirms the relevance of country fixed effects.

Additional controls are year fixed effects (YFE), linear yearly trend and regional yearly trend.

‡ Estimates remain similar and significant when using rather a yearly trend.

Deriving from equations 33 and 34, we can compute the long run effect of a permanent rise of HIV prevalence on primary school as:

$$\lim_{t \to \infty} \left(\frac{\Delta PGE_t}{\Delta HIV} \right) = -\frac{\alpha^p}{1 - \rho^p} \qquad (35)$$

Computing this figure with the coefficients of the first and fifth regressions (columns 1 and 5 in table 6) yields that a 1 point permanent increase of HIV prevalence induces a fall of respectively 1 and 6 points of the primary school enrollment rate in the long-run. The impact of the epidemic on schooling, although uncertain, appears to be very large. Given the uncertainty on the model, we will assume in the following that:

$$\lim_{t \to \infty} \left(\frac{\Delta PGE_t}{\Delta HIV} \right) \approx 3 \qquad (36)$$

Effects of AIDS on secondary school enrollment rate We follow the same methodology to estimate the effects of HIV/AIDS on the secondary school enrollment rate, except that we control for the evolution of the primary school enrollment rate. The estimated equation of the impact of HIV prevalence rate on secondary school enrollment rate is :

$$SNE_t^i = \rho^s SNE_{t-1}^i + \gamma PNE_{t-1}^i + \alpha^s HIV_{t-1}^i + \beta^c cc_t^i + \beta^w cw_t^i + u_i + \eta_t + \varepsilon_t^i \qquad (37)$$

Regression results are reported in table 7 below. The negative effect of HIV on secondary school enrollment is less pronounced than for primary education. There is no significant

effect of HIV prevalence when using the raw data. However when using projected data, HIV seems to have a negative effect on secondary school. But the uncertainty of such effect remains large. There is no significant effect using our instrumented measure. As the instrumented measure differs from the UNAIDS one mostly for the recent years, this may suggest that it is still too early to assert the consequences of HIV on secondary education.

Table 7 : The impact of HIV prevalence rate on secondary school enrollment rate

Dep. Var.	SGE	SGE	$\widetilde{SGE}$	$\widetilde{SGE}$	$\widetilde{SGE}$	$\widetilde{SGE}$	$\widetilde{SGE}$
Method	Within†	Within†	Within†	Within†	Within†	Within†	Within†
$\widehat{SGE_{-5}}$	0.40** (2.8)	0.40*** (3.0)	–	–	–	–	–
$\widehat{PGE_{-5}}$	0.08* (2.3)	0.08*** (2.3)	–	–	–	–	–
$\widetilde{SGE_{-1}}$	–	–	0.96*** (60.2)	0.96*** (61.8)	0.95*** (63.3)	0.95*** (60.4)	0.96*** (60.0)
$\widetilde{PGE_{-1}}$	–	–	0.02** (3.1)	0.02*** (3.6)	0.02*** (3.6)	0.02*** (3.6)	0.03*** (4.1)
HIV_{-1}	0.02 (0.0)	–	−0.07* (2.2)	−0.07* (2.2)	−0.10* (2.2)	–	–
$HIVinst_{-1}$	–	0.05 (0.2)	–	–	–	−0.07⋆ (1.9)	−0.06⋆ (1.8)
Controls:	Foreign aid$_{-1}$, Civil conflicts and wars, intercept, country fixed effects.						
Add controls:	YFE‡	YFE‡	YFE	Trend	RYFE	YFE	Trend
# obs.	504	500	1090	1090	1090	1089	1089
# countries	46	46	46	45	45	47	46
Period	70-07	70-07	81-07	81-07	81-07	81-07	81-07
adj. R^2	0.66	0.66	0.97	0.97	0.97	0.97	0.97

Notes: ⋆,*,**,*** indicate respectively significance at the 10%, 5%, 1% and 0.1% level.

T-values reported between brackets are calculated by clustering errors by country.

Breush and Pagan test rejects homoscedasticity in all configurations

† indicates that the Hausman test confirms the relevance of country fixed effects.

Additional controls are year fixed effects, linear yearly trend and regional yearly trend.

‡ Estimates remain similar and no significant when using other time specifications.

To assess the impact of the epidemic on secondary enrollment, one has to take into account the direct effect, and the indirect one, through the diminution of primary school enrollment rate. The derivative of from our linear specification in equation 37, the total effect of HIV on secondary education is given by

$$\frac{\Delta SGE_t}{\Delta HIV} = \rho^s \frac{\Delta SGE_{t-1}}{\Delta HIV} + \gamma \frac{\Delta PGE_{t-1}}{\Delta HIV} - \alpha^s \tag{38}$$

Therefore, in the long-run, the total effect of HIV/AIDS on secondary enrollment rates becomes:

$$\lim_{t \to \infty} \left(\frac{\Delta SGE_t}{\Delta HIV} \right) = -\frac{\alpha^s}{1 - \rho^s} - \frac{\gamma}{1 - \rho^s} \frac{\alpha^p}{1 - \rho^p} \tag{39}$$

As before, the evaluation of the multiplier depends of the model. Given the uncertainty, we choose:

$$\frac{\alpha_s}{1 - \rho^s} \approx 0.9 \tag{40}$$

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Taking the coefficients of the third regression (column 3, table 9) gives that, a 1 point increase of the HIV prevalence induces a permanent fall of 2.4 point of the secondary school enrollment rate. However, taking the maximal estimates of both α_p and α_s would lead to a far larger multiplier, about 4.8. The impact of HIV/AIDS on the investment in human capital may therefore be really high in the long-run.

Assessing the long-run effect of AIDS on human capital   We consider the average schooling years as the proxy of human capital. To assess the effects of HIV/AIDS on human capital, one has to transform the variations in enrollment rates into variations in the average schooling years. Theoretically, one can link enrollment rate and schooling years, if the average number of completed grades (D^j) by level $j = \{p, s\}$ is known, as follows:

$$\Delta S = D^p \Delta PNE + D^s \Delta SNE \tag{41}$$

However, the D^j figures are not precisely known and could also vary across time, as educational system betters. A crude approximation could be to assume that $D^j = \frac{\bar{D}^j}{2}$ where $\bar{D}^j$ is the official duration of the level j. To test the sensitivity of such an hypothesis, we use the available (although scarce) data on the years of schooling. Barro and Lee (2002) report such figures for 30 countries between 1985 and 2000. To this dataset, we add recent estimates for 42 countries, using household survey data[27]. Three specifications are used to regress years of schooling on gross enrollment rate. A more convincing regression would have to introduce lagged value of the years of schooling. However, there is not enough data to obtain significant estimates of the effects of enrollment on schooling using this specification. Because there is only a few years for each country, we do not introduce either country or years fixed effects. The regression is therefore:

$$S_t^i = b^p \widetilde{PGE_{t-5}^i} + b^s \widetilde{SGE_t^i} + S_0 + \varepsilon_t^i \tag{42}$$

This equation is estimated using a within regression:

$$S_t^i = \underset{(3.6)}{1.55} \widetilde{PGE_{t-5}^i} + \underset{(8.0)}{5.84} \widetilde{SGE_t^i} + + \underset{(1.6)}{0.51} + \varepsilon_t^i \;,\; adj.R^2 = 0.32 \;,\; \# \text{ obs.} = 149 \tag{43}$$

According to those results, a 1 point increase of HIV prevalence would induce a reduction of average human capital by 0.18 years of schooling.

Thus, considering that an additional year of schooling increases productivity by about 7% $\left(\text{as } \frac{\Delta \ln(Y/L)}{\Delta S} \approx 0.07\right)$, a 1 point increase of HIV prevalence would diminish productivity through human capital by approximately 1.3 point. Therefore approximately 45% of the long-run effect of HIV/AIDS on growth could be due to a permanent reduction of human capital.

4.2.3   Effects of HIV/AIDS on physical capital

As there is no data on physical capital stocks at the national level, we consider rather the effects of the epidemic on investment. The effects of HIV/AIDS on physical capital can indeed be approximated knowing the marginal effects of the epidemic on the investment rate.

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[27]See Cahu (2010) for data brought from MICS, DHS and LSMS household surveys.

Impact of AIDS on investment Investment rates can be computed using GDP and gross capital formation both in current or constant US$. We use the same framework (the theoretical background model and equation 14) to assess the effects of HIV/AIDS on investment. We denote inv the investment rate computed as the ratio of the annual gross capital formation to GDP. In the following regressions, civil conflicts appears to be never significant. Therefore, we only present the regressions without this variable. However, we introduce the openness to trade ratio, $open^{28}$, and the share of foreign direct investment in the GDP, fdi. Using both current and lagged values of those indicators allows to capture both short and medium run effects of openness to trade. Indeed investment is likely to be pro-cyclical and to overreact in the short-run to foreign activity.

$$
\begin{aligned}
\ln(inv_t^i) &= \rho^k \ln(inv_{t-1}^i) + \alpha^k HIV_{t-1}^i + \beta^o open_t^i + \beta_1^o open_{t-1}^i + \beta^f fdi_t^i + \beta_1^f fdi_{t-1}^i \quad (44) \\
&\quad + \beta^w cw_t^i + u_i + \eta_t + \varepsilon_t^i
\end{aligned}
$$

Regressions results are reported in table 8. Regressions indicate that HIV prevalence has also a negative impact on investment. As physical investment reacts more quickly than education investment, the negative effects of HIV/AIDS on physical capital affect the economy more rapidly. This effect although large varies however with the specification. Estimates depend of the measures and are only significant at the 10% level when using the pregnant women prevalence rate and the instrumented measure of HIV prevalence. This seems to indicate that if HIV is likely to dampen investment, its effect may depend on country specific conditions. One may think consequently that the adverse effect of the epidemic on physical capital accumulation might be mitigated, at least to some extent.

Impact of AIDS on physical capital To calibrate precisely the effect of the epidemic on productivity, one should estimate a Solow-type growth model. However, the lack of long-series for both human and physical capital has prevented such an exercise[29]. To assess the long-run effect of HIV/AIDS, let us recall the capital accumulation equation 4, from which we can deduce:

$$
\frac{K_t}{Y_t} = \frac{1-\delta}{1+g}\left(\frac{K_{t-1}}{Y_{t-1}}\right) + Inv_t \Rightarrow \lim_{t\to\infty}\frac{K_t}{Y_t} = \frac{\lim_{t\to\infty} Inv_t}{1 - \frac{1-\delta}{1+g}} \quad (45)
$$

As a consequence, we have in the long-run:

$$
K_t \propto Y_t \times Inv_t \Rightarrow \frac{\partial\ln(K)}{\partial HIV} = \frac{d\ln(Y)}{dHIV} + \frac{\partial\ln(Inv)}{\partial HIV} \quad (46)
$$

This allows to deduce the effect of HIV/AIDS on GDP through physical capital accumulation $\left(\frac{\partial\ln(Y)}{\partial HIV}\right)_K$:

$$
\left(\frac{\partial\ln(Y)}{\partial HIV}\right)_K = \frac{\partial\ln(Y/N)}{\partial\ln(K)} \times \frac{\partial\ln(K)}{\partial h} = \gamma\left(-\frac{\alpha^k}{1-\rho^k} - \frac{\alpha}{1-\rho}\right) \quad (47)
$$

As argued above, the parameter γ has not been estimated. We use here the historical average of the investment rate as a proxy of γ, that is $\left(\frac{\partial\ln(Y)}{\partial\ln(K)} = \gamma \approx 0.2\right)$. With such a

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[28] Openness is computed as the sum of the share of imports and exports into GDP.

[29] From the investment series, we have produced series for physical capital stock assuming constant depreciation rate over time. However, it was not possible to obtain neither plausible nor significant estimates of the marginal elasticity of capital to GDP.

value for γ, the long-run effect of a permanent 1 point rise of HIV prevalence on productivity through capital accumulation is about -0.9 when using the estimates of the first regression (column 1 table 8). Choosing a γ of 0.3 would increase this estimation up to -1.3. Therefore, physical capital accumulation explains approximately one third of the total effect of HIV/AIDS on productivity.

Table 8: The impact of HIV prevalence rate on investment

Dep. Var.	$\ln(inv)$	$\ln(inv)$	$\ln(inv)$	$\ln(inv)^{\S}$	inv	$\ln(inv)$	$\ln(inv)$
Method	Within†	Within†	Within	Within	Within†	Within†	Within†
$\ln(inv_{-1})$	0.63*** (16.4)	0.63*** (13.0)	0.54*** (8.0)	0.91*** (72.0)	—	0.59*** (7.9)	0.64*** (13.0)
inv_{-1}	—	—	—	—	0.54*** (4.5)	—	—
HIV_{-1}	−0.58** (3.0)	−0.60** (3.2)	−1.01* (2.4)	−0.19* (1.8)	−0.17* (2.2)	—	—
HPW_{-1}	—	—	—	—	—	−0.23* (1.8)	—
$HIVinst_{-1}$	—	—	—	—	—	—	−0.46* (1.8)
Controls:	Civil wars, *open*, *open*$_{-1}$, *fdi* and *fdi*$_{-1}$, intercept, year fixed effects						
Add. Controls:	Country fixed effects			—	Country fixed effects		
# obs.	1042	1225	737	794	1046	847	1047
# countries	45	45	45	36	45	45	45
Period	81-07	70-07	90-07	81-07	81-07	81-07	81-07
adj. R^2	0.59	0.62	0.49	0.73	0.51	0.56	0.58

Notes: *,*,**,*** indicate respectively significance at the 10%, 5%, 1% and 0.1% level.

T-values reported between brackets are calculated by clustering errors by country.

† indicates that the Breush and Pagan test rejects homoscedasticity and that Hausman test confirms the relevance of country fixed effects.

§ indicates that the regression has been run using the investment rate in constant (2000) US$ rather than current US$. Because of BP test rejection, no country fixed effects were added.

5 Computing the long run dynamic effects of the epidemic on growth

The epidemic affects productivity through four different channels: the demography, physical and human capital and labor participation/technical change[30], as pointed out by the following equation:

$$
\frac{d\ln(Y/N)}{dHIV} = \frac{\partial\ln(Y/N)}{\partial\ln(N)}\frac{\partial\ln(N)}{\partial HIV} + \frac{\partial\ln(Y/N)}{\partial\ln(K)}\frac{\partial\ln(K)}{\partial HIV} + \frac{\partial\ln(Y/N)}{\partial S}\frac{\partial S}{\partial HIV} \quad (48)
$$
$$
+ \frac{\partial\ln(Y/N)}{\partial\ln(L)}\frac{\partial\ln(L)}{\partial HIV}
$$

To calculate the effects of HIV/AIDS on the different components, we derive from equation (14) the cumulated effects of the epidemic on the variables z_t^i as Δz_t^i:

$$
\Delta z_t^i = z_t^i - \widetilde{z_t^i} = \rho^z \Delta z_{t-1}^i + \alpha^z HIV_t \; ; \; z = \{k, S, l, n\} \quad (49)
$$

---

[30]There is no data allowing to disentangle the effects of the epidemic on labor participation (hours worked) and technological change.

We have estimated so far the three first components and the total effect. As it is not possible to estimate the impact of HIV/AIDS on labor participation/technological change because of the lack of data, we will calculate this latter effect by substracting the three first one to the total effect. Recalling equation 7, the effect of HIV on productivity through labor participation/technological change is indeed:

$$\left(\frac{\partial \ln(Y/N)}{\partial HIV}\right)_L = \gamma \Delta l_t = \Delta y_t - \gamma(\Delta k_t - \Delta n_t) - (1 - \gamma)\xi \Delta S_t \tag{50}$$

The evolution of the working age population affects the GDP per adult through two opposite channels. On one hand, the diminution of the population, all other things remaining equal, increases physical capital per worker. On the other hand, the reduction of the size of the economy gradually reduces the level of investment and the stock of capital. In a Solow-type model, output per adult does not depend on the size of the population in the long-run. Therefore, the net effect of demography is null in the long-run. To disentangle the effects of demography and capital, let us define K_t^* the level the stock of capital would reach if it were not affected by the evolution of the adult population. From equation 8, we can obtain the dynamic of K_t^*:

$$\Delta k_t^* = \frac{\Delta K_t^*}{K_t^*} = \phi_K \Delta k_{t-1}^* + (1 - \phi_K)(\Delta inv_t + \Delta y_t) \tag{51}$$

The net effect of the demography on productivity is:

$$\left(\frac{\partial \ln(Y/N)}{\partial HIV}\right)_N = \gamma \left(\Delta k_t - \Delta k_t^* - \Delta n_t\right) \tag{52}$$

Consequently, the effect of HIV on productivity through physical capital becomes:

$$\left(\frac{\partial \ln(Y/N)}{\partial HIV}\right)_K = \gamma \Delta k_t^* \tag{53}$$

5.1 Illustration with an average country of the composition of the HIV/AIDS effects

We then can compute the dynamic effects of HIV/AIDS on the logarithm of per adult income through the four different channels. To illustrate the results we consider the average country which endures the average HIV prevalence of the all 46 African Sub-Saharan countries for which data are available. Between 1980 and 2010, AIDS would have already reduce GDP per worker by about 8.0 points. By comparison, the cumulated effects of civil conflicts and wars in Sub-Saharan Africa between 1980 and 2010 would have been a reduction in per worker GDP about 17 points in average[31].

For the same average country, the decomposition of HIV/AIDS effects on the GDP per capita growth rate is illustrated by the figure 4 below. It appears that the HIV/AIDS effect has already reached its peak around the year 2000. The long-term effects of HIV on growth, although huge, may be compensated by increased efforts in education and capital markets.

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[31]We use the same methodology to compute the cumulated effects of wars on growth, by switching civil wars and conflicts to HIV prevalence in equation (14).

Figure 3: Effects of HIV/AIDS on the level of GDP per adult in the average Sub-Saharan African country (Simulation using the estimated parameters).
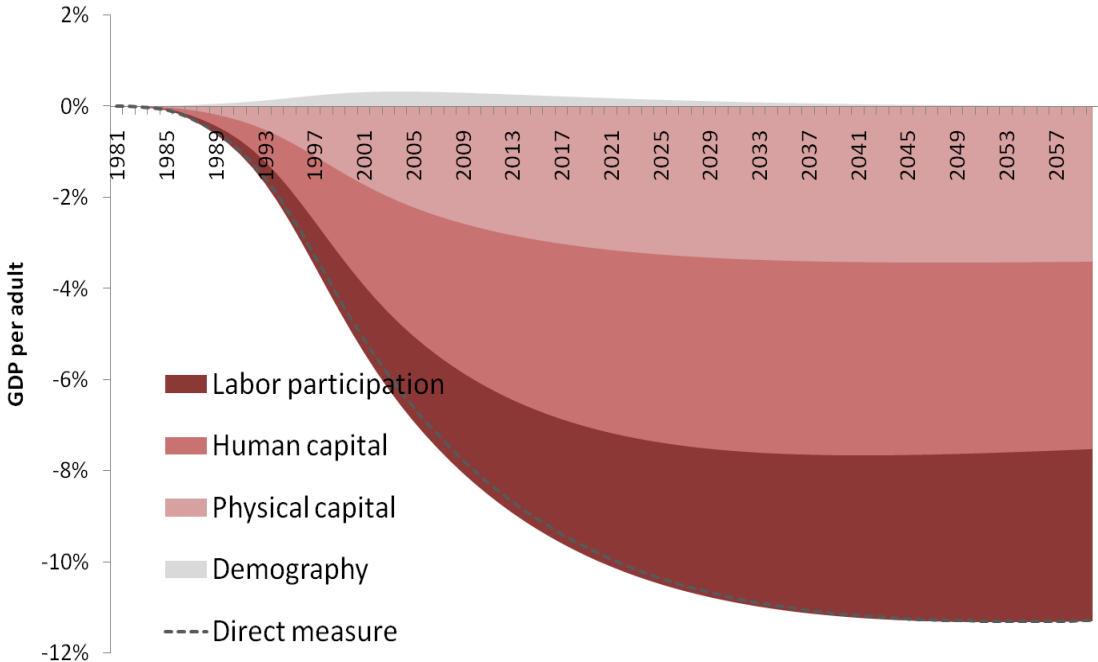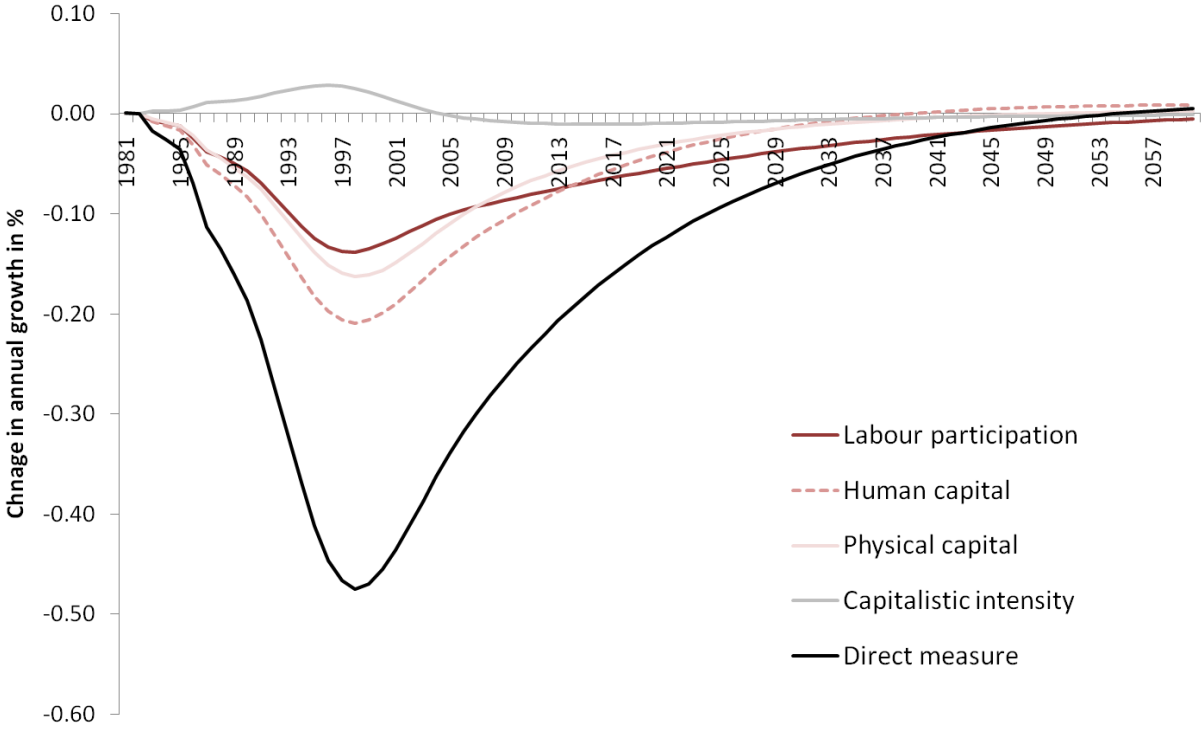

Figure 4: Effects of HIV/AIDS on GDP per adult annual growth rate in the average Sub-Saharan African country

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5.2 Decomposition of the effects of the epidemics on several countries

As an illustrative exercise, when we apply these estimated effects to the prevalence rate of specific countries, the impact appears to be very important for high prevalence rate countries. The effects of the epidemic on several Sub-Saharan African countries is presented in figure 5 below. The effects on the growth rate of the countries (South Africa and Lesotho) with high prevalence rate are huge. The cost has been higher than 2 points of growth during the last decade for Lesotho. For countries with a median HIV prevalence rate (Tanzania and Kenya for instance), the epidemic has cost between 0.5 and 0.75 point of growth each year.

The economic impact of such an epidemic is therefore strong and heavily persistent. Although the epidemic has already caused great damages to the hit countries, most of them will continue to experience lower growth because of it for several decades. Nevertheless, as most of the long-term effect of HIV/AIDS on growth go through physical and capital accumulation, public policies aiming to increase both investment and education enrollment rate might mitigate the burden of the epidemic.

Figures 7 and 8 in appendix plot the effects of HIV/AIDS on the different components of growth for the same countries. The cumulated cost of AIDS, the "AIDS gap" appears to be huge, especially for the Southern countries. Although dramatic these estimates are lower than most of the projections based on models simulations.

6   Discussion and conclusion

The relationship between HIV/AIDS and growth is likely to be bidirectional. Therefore, it is very challenging, in a macroeconomic framework, to disentangle the exact impact of HIV/AIDS on growth. We tried to do so in a simple and comprehensive way. To tackle the endogeneity issue, we built a series of HIV prevalence rate using instrumental variables. Although some of the instruments we use could not be arguably considered fully exogenous, they appear to be poorly correlated with growth in Sub-Saharan Africa (see table 1). We use nevertheless geographical variables and circumcision rates, which is seen more and more as a good instrument of risk infection in the literature, see Forston (2009b) and Ahuja, Wendell, and Werker (2006). Our methodology allows us to build a HIV prevalence rate series which compares fairly with both Oster (2009b) and UNAIDS series[32].

It is worth noting that our "instrumented" series does not incorporate actual UNAIDS estimates and has been dynamically simulated only using instrumental variables.

Based on our HIV prevalence rate series, our estimates show that the epidemics has a significant and negative direct effect on growth (see table 6). To assess the robustness of our estimates to measurement error in the data, we show the results using the UNAIDS series, the HIV prevalence rate among pregnant women. The direct effect of HIV/AIDS is negative and significant and this result holds whatever the time period considered, the measure of HIV used and the configuration of the regression. In the short run, the impact of the epidemics appears to be limited. A 1 point increase of the HIV prevalence induces a 0.13 point decrease of GDP per working age adult the following year. In the long run however, the (permanent) effect is much larger, a 1 point increase in prevalence causing

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[32]Oster (2009b) infers HIV prevalence rate from microeconomic mortality data.

Figure 5: The effects of the epidemics on the growth rate of several countries.
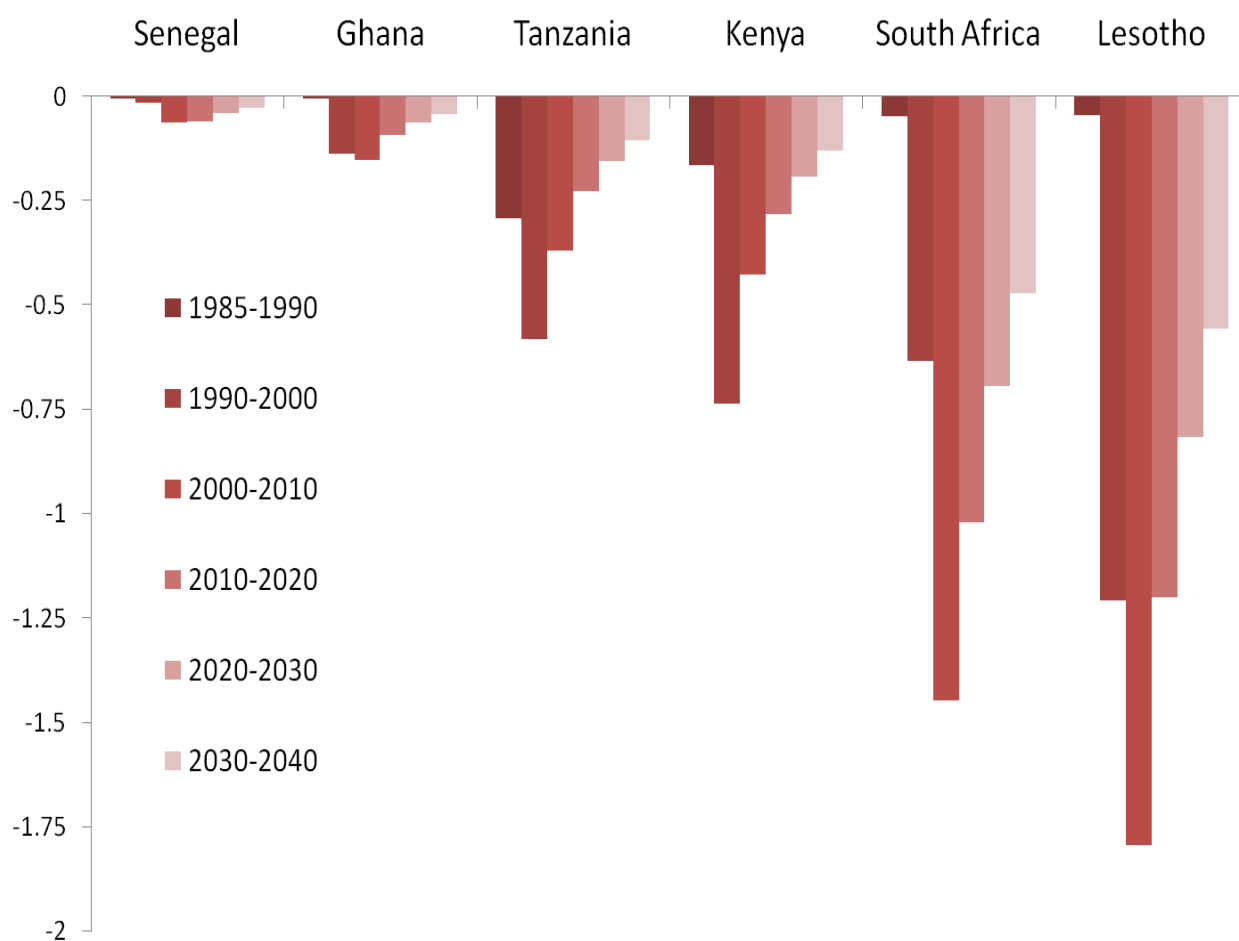
a reduction of GDP per worker about 2.75 points. Using a Monte Carlo simulation, we showed that the effect of HIV/AIDS is negative and statistically significant at the 5% level.

Our estimate stand between Bonnel's (2000), who regressed growth from 1990-97 on AIDS in 1994-97 for approximately 80 developing countries and find a negative and statistically significant effect, and McDonnald and Roberts's (2006), who in a dynamic panel regressions find that each point of HIV prevalence rate lowers per capita income by 0.59 percent. Our estimate are also consistent with the studies based on theoretical models simulations, as Arndt and Lewis (2000), Kambou and al. (1992) and Sackey and Raparla (2000; 2001a; 2001b).

To gauge precisely the impact of the HIV/AIDS epidemics on short run and long run growth, we performed an accounting exercise of the epidemics effects on the different determinants of growth: demography, human capital accumulation and physical capital accumulation. Our results exhibit a highly significant negative effect of HIV/AIDS on life expectancy. The impact on working age population is negative and significant but the magnitude is much lower. These results are consistent with the literature (see for instance Ahuja and al. 2006). As pointed by Young (2005), the reduction of working population in a context of capital scarcity may increase the total welfare. However, our computations show that although the capital intensity effect is positive in the short run,

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its magnitude is very limited, a 1 point rise in prevalence induces a rise in the yearly growth rate of the GDP per worker less than 0.1 percent. This capital intensity effect remains very small in the medium run and vanishes after 45 years, about two generations of workers. We did not report the results of our estimations of the impact of the epidemic on fertility. A converging literature find that either the impact is very small (Forston 2009b; Kalemli-Ozcan 2009; Fink and Linnemayr 2008) or is not significant (Boucekkine and al. 2009). Hence, we do not take into account this impact when considering the impact on demography.

The second channel we evaluate is the impact of the epidemics on human capital accumulation. Our estimations show a negative and significant impact of the HIV prevalence rate on primary school enrollment rate either using raw data (5 years interval) or projected annual data. A 1 point permanent increase of the HIV prevalence rate causes a one point decrease in primary school enrollment rate in the long run. The estimated impact is much higher when using annual data. However, the epidemics appears to have a limited negative effect on secondary school enrollment rate (see table 7). Only the estimations using the projected annual data exhibit significant effect. In the long run, a 1 point permanent increase of HIV prevalence rate induces a permanent fall of 2.4 points of the secondary school enrollment rate.

From these estimates, we derive the impact of the epidemic on the average years of schooling, our human capital variable. According to our results, a 1 point increase in HIV prevalence would induce a reduction in average human capital about 0.18 years of schooling. These results are comparable with Forston's (2009a), who estimated the relationship between regional HIV prevalence rate and the educational outcomes of successive birth cohorts in fifteen Sub-Saharan African countries. Using data from the Demographic and Health Surveys (DHS), she found that in areas with prevalence rate of ten percent, the post-1980 birth cohorts completed about 0.5 fewer years of schooling than pre-1980 cohorts. Her results confirm that HIV/AIDS is likely to have a negative impact on education.

From our estimates, we computed that a 1 point increase in HIV prevalence would diminish productivity through human capital by approximately 1.3 point in the long-run. Therefore, the human capital reduction accounts for more than 45% of the total long-run effect of HIV/AIDS on growth.

The third component we evaluate is the impact on physical capital accumulation. As already noted there are many channels through which the HIV/AIDS epidemics affect the incentives to invest or accumulate. We control for trade, foreign direct investment and civil wars in our regressions. We found a negative effect significant at the 5% level using UNAIDS estimate, but which is only significant at the 10% level using pregnant-women HIV prevalence or our "instrumented" series. Building from these estimates, we found that in the long run, the reduction of physical capital accumulation accounts for approximately one third of the long run effect of HIV/AIDS on growth.

According to this accounting exercise, human capital is likely to drive the most important part of the total effect. Physical capital accumulation plays a larger role in the short-run (see figure 3). Our estimates show that at the peak, around 2002-2003 in average, the total effect of HIV/AIDS is a reduction by 6% of income per capita; the reduction of human capital accounting for 2.4%, the reduction of physical capital accounting for 2%, labor participation (and technological change) accounting for 0.7% and the counterbalancing positive effect through capital intensity account for 0.1%. In the long-run, those effects are approximately double. They are consistent with Santaeulàlia-Llopis's results

(2008), based on a population model structured by age-specific groups and which takes into account the fertility process, the mortality and the aging process. The modelled economy experiences a development transition path from a Malthusian-agricultural sector to a neoclassical-industrial sector. When this model is calibrated to an African country, incorporating the different channels through which AIDS affects populations over time (i.e. reshapes the age distribution of the population, reduces population growth and life expectancy due to raising mortality rates of young adults and lower fertility rates), the author found that AIDS epidemic reduces per capita income as much as 12%.

However, Ashraf and al. (2008) perform a simulation exercise to explore the economic effects of an exogenous change in population health[33]. Their model allows for a direct effect of health on worker productivity , as well as indirect effects that run through schooling, the size and age-structure of the population, capital accumulation, and crowding of fixed natural resources. They show that eradicating specific diseases (malaria or tuberculosis) in a typical country in Sub-Saharan Africa would raise GDP per capita by only two percent in the long run. These results are very small compared to ours for HIV/AIDS effects, but the mechanisms their simulation shows are very instructive. First, as the two diseases they consider affect differently the different categories of the population (working-age adults and school-age population) their effect on capital per worker, productivity of workers and education are different. As tuberculosis hit more the working-age adults, its eradication causes an immediate decline of capital per worker and has a positive steep effect on productivity of workers. But, the effects on schooling years are small. In contrast, the eradication of malaria brings about a larger, but more delayed decline in capital per worker as the cohort of children who would have died from the disease enter the labor force. As malaria affects less the working age population, its eradication have a delayed effect on productivity, but a more immediate impact on schooling.

HIV/AIDS combines these different characteristics, it hits sharply the working-age adults, modifies the age distribution of the population, produces a great number of orphans and therefore brings about a negative impact on education. It combines a stock effect by killing a part of the population and a long lasting effect as affected people can live with the disease during a number of years. Therefore, it is likely that HIV/AIDS adds up the different indirect effects shown by Ashraf and al. (2008).

Acknowledgments

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[33]See also Acemoglu and Johnson (2007) for an assessment of the effect of disease on growth through life expectancy.

References

Acemoglu, D. and Johnson, S. (2007), Disease and development : The effect of life expectancy on economic growth. Journal of Political Economy, 115(6), 925-985.

Alesina, A., Devleeschauwer, A., Easterly, W., Kurlat, S., and Wacziarg R., (2003). Fractionalization. Journal of Economic Growth, 8(2), 155-94.

Arndt, C. and Lewis, J., (2000). The Macro implications of HIV/AIDS in South Africa: A preliminary assessment. South African Journal of Economics, 68 (5), 856-87.

Ashraf, Q. A., Lester, A., and Weil, D., (2008). When does improving health raise GDP?, NBER Macroeconomics Annual.

Barro, R., J., and Lee, J., W., (2002).International data on educational attainment: Updates and Implications, Oxford Economic Papers, 53(3), 541-563.

Bell, C., Devarajan, S., and Gersbach, H., (2003). The long-run economic costs of AIDS: Theory and an application to South Africa. World Bank Policy Research Working Paper No. 3152.

Bhargava, A., and Bigombe, B., (2003). Public policies and the orphans of AIDS in Africa, BMJ, 326, 1387-1389.

Bloom, D., and Mahal A., (1997). Does the AIDS epidemic threaten economic growth. Journal of Econometrics, 77, 105-124.

Bonnel, R., (2000). HIV/AIDS: Does it increase or decrease growth in Africa?, World Bank.

Boucekkine, R., Diene, B. and Azomahou, T., (2006). The Growth economics of epidemics. Discussion Papers 2006021, Université catholique de Louvain.

Boucekkine, R., Desbordes, R. and Latzer, H., (2009). How do epidemics induce behavioral changes?. Journal of Economic Growth, 14(3), 233-264.

Cahu, P., (2010). Human capital dynamics around the World. World Bank, mimeo.

Caselli, F. (2005). Accounting for Cross-Country Income Differences. In P. Aghion and S. Durlauf (ed.), Handbook of Economic Growth, North-Holland.

Central Intelligence Agency, The World Factbook, (2010).

Corrigan, P., Gloom, G., and Mendez, F., (2005). AIDS crisis and growth. Journal of Development Economics, 77(1), 107-124.

Cuddington, J., (1993). Modelling the macro effects of AIDS, with an application to Tanzania. The World Bank economic review, 7 (2), 173-189.

Cuddington, J. and Hancock, J. (1994). Assessing the impact of AIDS on the growth path of the Malawian economy. Journal of Development Economics, 4(3), 363-368.

Durlauf, S. N., Johnson, P. A., and Temple, J. R.W. (2005). Growth Econometrics. In P. Aghion and S. Durlauf (ed.), Handbook of Economic Growth (pp. 555-677), edition 1 (1).

Fink, G., and Linnemayr, S. (2008). HIV and fertility: Long-term Evidence from Sub-Saharan Africa. Harvard University, mimeo.

Forston, J. (2009a). Mortality risk and human capital investment: The impact of HIV/AIDS in Sub-Saharan Africa. Review of Economics and Statistics, forthcoming.

Forston, J. (2009b). HIV/AIDS and Fertility. American Economic Journal: Applied Economics, vol. 1(3), 170-194.

Gleditsch, K. S., and Ward, M. D. (1999). Interstate system membership: A revised list of independent states since 1816. International Interactions, 25, 343-393.

Gray, R. H., and al. (2007). Male circumcision for HIV prevention in men in Rakai, Uganda: a randomized trial. The Lancet, 369 (9562), 657-666.

Halperin, D.T., and Bailey, R. C. (1999). Male circumcision and HIV infection: 10 years and counting. The Lancet, 354 (9192), 1813-1815.

Juhn, C., Kalemli-Ozcan, S., and Turan, B. (2008). HIV and fertility in Africa: first evidence from population based surveys". NBER working paper 14248.

Kambou, G., Devarajan, S., and Over. M. (1992). The economic impact of AIDS in an African Country: simulations with a CGE Model of Cameroon. Journal of African Economies, 1, 109-130.

Kalemli-Ozcan, S. (2006). AIDS, "Reversal" of the demographic transition and economic development: evidence from Africa. NBER Working Papers 12181.

Kalemli-Ozcan, S. (2009). HIV and Fertility Revisited. University of Houston, mimeo, (2009).

Lewis, J., Ronsmans, C., Ezeh, A. and Gregson, S. (2004). The population impact of HIV on fertility in sub-Saharan Africa. AIDS, 18 (2), 35-43.

McDonald, S., and Roberts, J. (2006). AIDS and economic growth: A human capital approach. Journal of Development Economics, 80(1), 228-250.

Oster, E. (2009a). Routes of Infection: exports and HIV incidence in Sub-Saharan Africa. NBER Working Papers 13610.

Oster, E. (2009b). Estimating HIV prevalence and incidence in Africa from mortality data. mimeo, University of Chicago.

Over, M. (1992). The macroeconomic impact of AIDS in Sub-Saharan Africa. Population and Human Resources Department, The World Bank.

Papageorgiou, C., and Stoytcheva, P. (2005). What do we know about the impact of AIDS on cross-country income so far?. Working Papers 2005-01, Department of Economics, Louisiana State University.

Pongou, R., and Serrano, R. (2009). A dynamic theory of fidelity networks with an application to the spread of HIV/Aids. Working Papers 20090909, CEMFI.

Sackey, J., and Raparla, T. (2000). Lesotho: the development impact of HIV/AIDS - Selected issues and options. World Bank Report No. 21103-LSO.

Sackey, J., and Raparla, T. (2001a). Swaziland: selected development impact of HIV/AIDS. World Bank Report No. 22044-SW.

Sackey, J., and Raparla, T. (2001b). Namibia: selected development impact of HIV/AIDS. World Bank Report No. 22046-NA.

Santaeulalia-Llopis, R. (2008). Aggregate effects of AIDS on development. Mimeo, Washington University.

Tavares, J. and Wacziarg, R. (2001). How democracy affects growth. European Economic Review. 45(8), 1341-1378.

UCDP/PRIO (2007). Armed conflict dataset of the Uppsala conflict data program. Uppsala University and International Peace Research Institute (Oslo).

UNAIDS/WHO (2003). Epidemiological Fact Sheets.

UNAIDS (2008). Report on the global AIDS epidemic.

United Nations Population Division (2007). World Urbanization Prospects: The 2007 revision population database.

United Nations Population Division (2008). World Population Prospects: The 2008 revision population database.

Weil, D. (2007). Accounting for the effect of health on economic growth. Quarterly Journal of Economics, 122 (3), 1265-1306.

Weiss, H. A., Quigley, M. A., and Hayes, R. J. (2000). Male circumcision and risk of HIV infection in sub-Saharan Africa: a systematic review and meta-analysis. AIDS.14,

33

2361-2370.

Werker, E. D., Ahuja, A., and Wendell, B. (2006). Male circumcision and AIDS: the macroeconomic impact of a health crisis", HBS Working Paper.

Williams, B.G. , Lloyd-Smith, J. O., Gouws, E., Hankins C, et al. (2006). The Potential Impact of Male Circumcision on HIV in Sub-Saharan Africa. PLoS Medicine, 3(7).

World Bank (2010). World Development indicators, 2010.

Young, A. (2005). The Gift of the dying: the tragedy of AIDS and the welfare of future African generations. Quarterly Journal of Economics, 120 (2), 426-466.

Young, A. (2007). In Sorrow to bring forth children : fertility admidst the plaque of HIV. Journal of Economic Growth. 12(4), 283-327.

Appendix

A) Appendix related to data building

Estimations used to project prevalence among pregnant women in rural area

Table A1: Determinant of HIV prevalence among pregnant women in rural area				
Dep. var.	HIV rural	HIV rural	HIV rural	HIV rural	HIV rural
Method	GLS	GLS	GLS	GLS	GLS
HIV rural (-1)	0.77** (18.3)	—	—	0.53** (15.4)	—
HIV rural (-2)	—	—	—	—	0.29** (9.0)
HIV rural (1)	—	0.59** (7.9)	—	0.49** (15.3)	0.70** (25.2)
HIV urban	0.18** (5.2)	0.31** (4.6)	0.64** (17.7)		—
Intercept	0.21 0.4)	−0.88 (1.5)	0.72 (0.3)	−0.07 (0.3)	0.02 (0.1)
Country fix.	No	Yes	No	No	No
# obs	197	189	273	256	279
# Country	33	32	38	37	38
Adj. R^2	0.89	0.86	0.62	0.92	0.91
# predict.	24	19	51	46	28

*,** indicate respectively significance at the 5% and 0.1% level

Estimations used to project prevalence among pregnant women in urban area

Table A2: HIV prevalence among pregnant women in urban area				
Dep. var.	HIV urban	HIV urban	HIV urban	HIV urban	HIV urban
Method	GLS	GLS	GLS	GLS	GLS
HIV urban (-1)	0.88** (23.4)	—	—	0.53** (14.9)	—
HIV urban (-2)	—	—	—	—	0.37** (11.2)
HIV urban (1)	—	0.57** (9.9)	—	0.46** 12.8)	0.62** (20.5)
HIV rural	0.14** (3.3)	0.41** (7.4)	0.54** (10.1)	—	—
AIDS case (-1)	—		0.08** (8.6)	—	—
Intercept	0.86* (2.2)	1.55* (2.7)	4.33** (4.6)	0.14 (0.6)	0.16 (0.6)
Country fix.	No	Yes	No	No	No
# obs	197	191	241	256	279
# Country	33	33	38	37	38
Adj. R^2	0.9	0.88	0.6	0.94	0.93
# predict.	21	18	20	46	28

*,** indicate respectively significance at the 5% and 0.1% level

Estimations of prevalence rate before 1990

Dep. Var.	ln(hwp)	ln(hwp)	hiv	$\Delta\ln(hiv)$	hiv
Method	GLS	GLS	GLS	GLS	OLS
hiv^l	–	–	0.8 *** (79.1)	–	–
hiv^u	–	–	0.22*** (24.3)	–	–
ln($Case$)	–	0.2*** 5.2)	–	0.05*** (3.2)	–
ln($Case_{-1}$)	0.19*** (6)	–	–	−0.05 (−)	–
$Case$	–	–	–	–	0.13*** (15.9)
$Case^2$	–	–	–	–	−0.002*** (8.3)
Intercept	1.03*** (3.7)	0.97*** (3.6)	0.19*** (4)	−0.15*** (9.4)	–
Country fix.	Yes	Yes	No	No	No
Year fix.	Yes	Yes	No	No	No
# obs.	426	398	743	369	339
# countries	41	42	43	42	-
adj. R^2	0.39	0.38	0.99	0.99	0.52
# Predict	177	44	36	164	250

Table A3: Estimates of HIV prevalence before 1990 based on reported cases

*,** indicate respectively significance at the 5% and 0.1% level

B) Building a new series of HIV prevalence rate

A simple theoretical model of HIV dynamic

The model relies on the eight following assumptions:

(i) There are two types of individuals in the economy, having two different sexual behaviors. People of type F (faithful) has only intercourse with her legitimate spouse while type U has several sexual partners. One assumes that the proportion of faithful people is exogenous and equals ψ.

(ii) There is an exogenous fraction m of type-F people who marries randomly a type-U individual.

(iii) At each period, a fraction ν of the type-U people has unprotected intercourse with another random type-U people. This results in the contamination of the healthy partner with a probability ι. This probability may vary.

(iv) Moreover, one assumes that every legitimate spouse of an infected type-U individual is also HIV-positive.

(v) As the dynamic of a disease is closely linked to the movement of population, one has to take into account the geographical dimension of the epidemic. Therefore, let us assume that a country is constituted of N cities which are all populated by $\frac{1}{N}$ of type-U adults. Let us denote α_t^i the HIV prevalence rate of the type-U adults in the city i.

(vi) In addition to that, let us assume that at each period, an exogenous fraction ϕ of U-type individuals moves randomly to another city to find a sex partner.

(vii) Mortality rates of adults only depends of their HIV-status and are exogenous equalling δ^+ and δ^- respectively for HIV-positive and HIV-negative people.

(viii) Finally, a new generation of n HIV-negative young appears at the beginning of each period.

By definition, F-type people cannot contaminate everyone. Therefore, the dynamic of the epidemic only depends on the dynamic of the disease among the U-type people. According to assumption (ii), the prevalence rate of HIV among type-F people in the city i, $\alpha^{F,i} = m \times \alpha^i$. Therefore, the total HIV prevalence in the city i, h^i is:

$$h^i_t = \alpha^i_t\Big(1 - \psi + m\psi\Big) \tag{A1}$$

At the end of the period t, the newly infected U-type people A^i_t living in the city i could have been contaminated in their own city or in other places, either by locals or foreigners. The spread of the infection is an increasing function of the mobility of the sex partners. It also decreases with the spatial variance of the prevalence rate, $V(\alpha)$.

Dynamic of the epidemic among unfaithful individuals

The following array gives the probabilities of encounters by places and city of origin for a people from the city 1.

Table A4: cross probability meeting for partners from different cities

City of partner / City of encounter	1	2	...	N
1	$(1-\phi)^2$	$(1-\phi)\frac{\phi}{N-1}$	...	$(1-\phi)\frac{\phi}{N-1}$
2	$(\frac{\phi}{N-1})^2$	$\frac{\phi}{N-1}(1-\phi)$	...	$(\frac{\phi}{N-1})^2$
...	...	...	...	
3	$(\frac{\phi}{N-1})^2$	$(\frac{\phi}{N-1})^2$	...	$\frac{\phi}{N-1}(1-\phi)$

From this array, one can express the number of people from the city 1 who have been infected during the period t:

$$\Delta A^1_t = \nu\iota(1-\alpha^1_t)\left(\Big((1-\phi)^2 + \frac{\phi^2}{N-1}\Big)\alpha^1_t + \sum_{i=2}^{N}\left\{2(1-\phi)\frac{\phi}{N-1}\alpha^i_t + \sum_{j=2,j\neq i}^{N}(\frac{\phi}{N-1})^2\alpha^j_t\right\}\right) \tag{A2}$$

To simplify the previous expression, let us denote $\bar\alpha_t = \frac{1}{N}\sum_{i=1}^{N}\alpha^i_t$ the average prevalence rate among the U-type individuals.

$$\frac{\Delta A^1_t}{\nu\iota(1-\alpha^1_t)} = \Big((1-\phi)^2 + \frac{\phi^2}{N-1}\Big)\alpha^1_t + \frac{\phi}{N-1}\sum_{i=2}^{N}\left\{2(1-\phi)\alpha^i_t + \frac{\phi}{N-1}\Big(N\bar\alpha_t - \alpha^i_t - \alpha^1_t\Big)\right\} \tag{A3}$$

$$\frac{\Delta A^1_t}{\nu\iota(1-\alpha^1_t)} = \Big((1-\phi)^2 + \frac{\phi^2}{N-1}\Big)\alpha^1_t + \frac{\phi}{N-1}\Big(2(1-\phi)(N\bar\alpha_t - \alpha^1_t) + \phi N\bar\alpha_t - \frac{\phi}{N-1}(N\bar\alpha_t + (N-2)\alpha^1_t)\Big) \tag{A4}$$

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$$\frac{\Delta A_t^1}{\nu\iota(1-\alpha_t^1)} = \left(1 - \phi + \frac{\phi}{N-1}\right)^2 \alpha_t^1 + \left(1 + \frac{1}{N-1}\right)\phi\left(2 - \phi - \frac{\phi}{N-1}\right)\bar{\alpha}_t \tag{A5}$$

For a large number of cities in the country, the dynamic equation becomes:

$$\Delta A_t^1 = \nu\iota(1-\alpha_t^1)\left((1-\phi)^2\alpha_t^1 + \phi(2-\phi)\bar{\alpha}_t\right) = \nu\iota(1-\alpha_t^1)\left(\bar{\alpha}_t + (1-\phi)^2(\alpha_t^1 - \bar{\alpha}_t)\right) \tag{A6}$$

The total number of new infections is thus at the national level:

$$\Delta A_t = \frac{1}{N}\sum_{i=1}^{N}\Delta A_t^i = \nu\iota\bar{\alpha}_t(1-\bar{\alpha}_t) - \nu\iota(1-\phi)^2\frac{1}{N}\sum_{i=1}^{N}(\alpha_t^i - \bar{\alpha}_t)^2 \tag{A7}$$

One can deduce the dynamic of the prevalence rate among U-type people from the number of infected people:

$$\Delta\alpha_t = \frac{\nu\iota\alpha_t(1-\alpha_t) - \nu\iota(1-\phi)^2 V(\alpha_t) - (\delta^a - \delta + n)\alpha_t + (\delta^a - \delta)\alpha_t^2}{1 - \delta + n - \alpha_t(\delta^a - \delta)} \tag{A8}$$

Introduction of identification variables

Due to population mobility, the variance of HIV prevalence across space has a tendency to decrease with the spread of the epidemic. Thus, we assume the following form for the variance $V(\alpha)$:

$$V(\alpha) \equiv V_0\alpha_t(1 - v_1\alpha_t) \tag{A9}$$

Considering moreover that $\delta^+ - \delta^- << 1$, we obtain a quadratic equation for HIV among U-type people dynamic. Combining (A1), (A8) and (A9) we can deduce the dynamic of HIV prevalence among the total population. A linear trend is allowed to take into account the global evolution of the HIV/AIDS epidemic.

$$h_{t+1} - h_t = \beta_1(\nu, \iota, \phi, \delta^a - \delta, \psi, m)h_t - \beta_2(\nu, \iota, \phi, \delta^a - \delta, \psi, m)h_t^2 + \beta_3 t + \beta_4 \tag{A10}$$

To identify this equation, we have to introduce instrumental variables for the different parameters.

Comparison of our instrumented series and UNAIDS one for the different countries

Partial correlations

Table A5 : Partial Correlations of the different variables (period 1970-2008)								
	$\ln(Y/N)$	$\ln(N)$	$\ln(inv)$	PNE	SNE	HIV	Conflict	War	Muslims
$\ln(Y/N)$	1.00								
$\ln(N)$	-0.39	1.00							
$\ln(inv)$	0.36	-0.32	1.00						
PNE	0.61	-0.32	0.32	1.00					
SNE	0.74	-0.21	0.21	0.64	1.00				
HIV_{UNAIDS}	0.24	0.07	0.09	0.31	0.33	1.00			
Civil conflict	-0.16	0.17	-0.10	-0.17	-0.17	-0.06	1.00		
Civil war	-0.18	0.22	-0.33	-0.20	-0.18	-0.09	-0.12	1.00	
Muslims	-0.30	-0.05	-0.10	-0.55	-0.35	-0.39	0.07	0.01	1.00

Figure 6: Comparison country by country
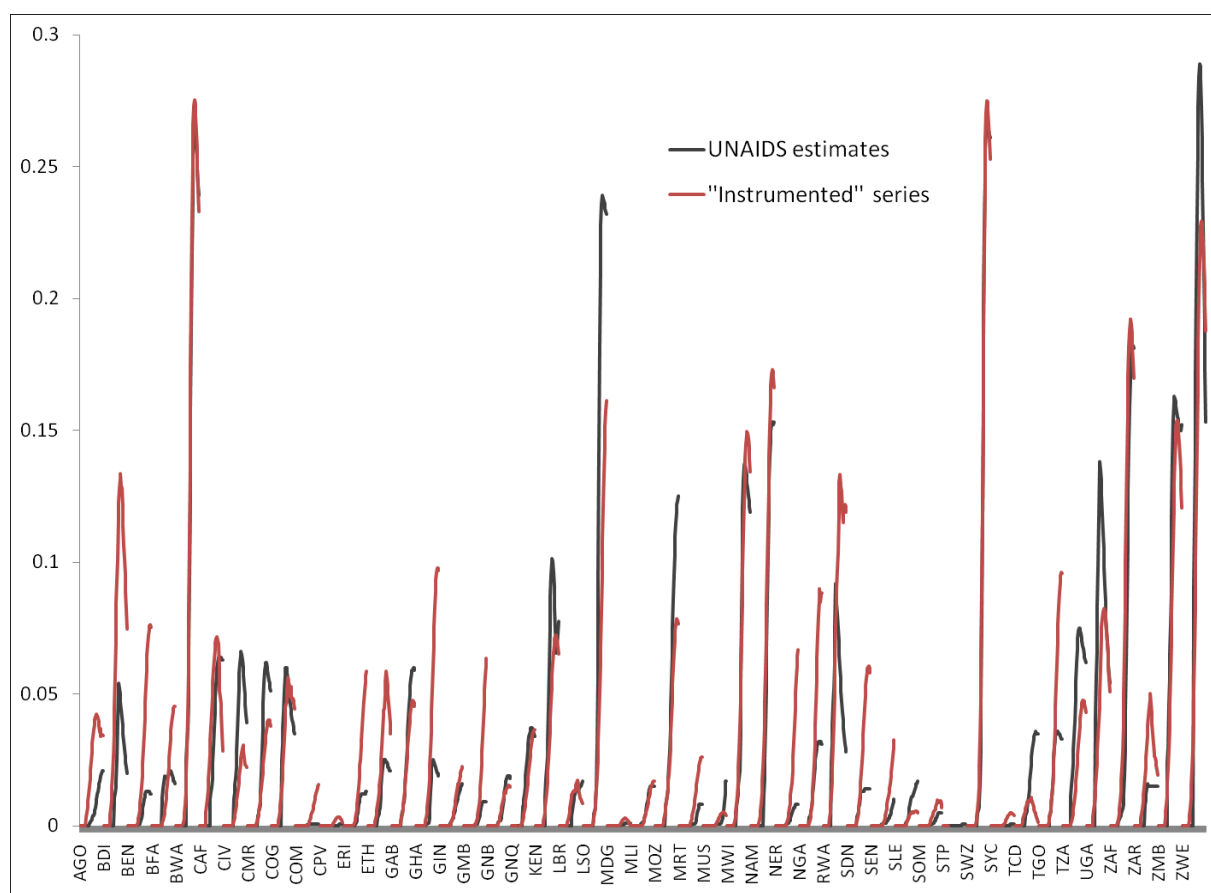
C) The impact of HIV prevalence on different countries

Table A6: Effects* of HIV/AIDS on physical and human capital and GDP per adult

	Peak year†			2009			2040		
	K	HC	Y/N	K	HC	Y/N	K	HC	Y/N
Senegal	-	-	-	-0.2	-0.6	-0.7	-0.6	-1.4	-2.0
Ghana	-0.4	-0.9	-1.2	-0.9	-2.1	-2.8	-1.5	-3.3	-4.8
Tanzania	-2.1	-4.8	-6.3	-3.4	-7.8	-10.4	-4.7	-10.4	-14.9
Kenya	-2.2	-4.9	-6.4	-3.8	-8.6	-11.4	-5.3	-11.8	-16.9
South Africa	-	-	-	-6.4	-14.1	-18.1	-12.1	-25.8	-35.1
Lesotho	-4.7	-10.6	-13.5	-9.1	-19.7	-25.2	-15.5	-32.1	-43.1

*Variations in percentage points compared to what would have happened without the epidemic:
GDP per adult would be 10.4 pp lower in 2009 in Tanzania to what it should have
experienced if HIV prevalence have been null.
†1998 for Tanzania and Kenya, 1999 for Ghana, 2001 for Lesotho while HIV prevalence
was still increasing in 2007 in Senegal and South Africa.
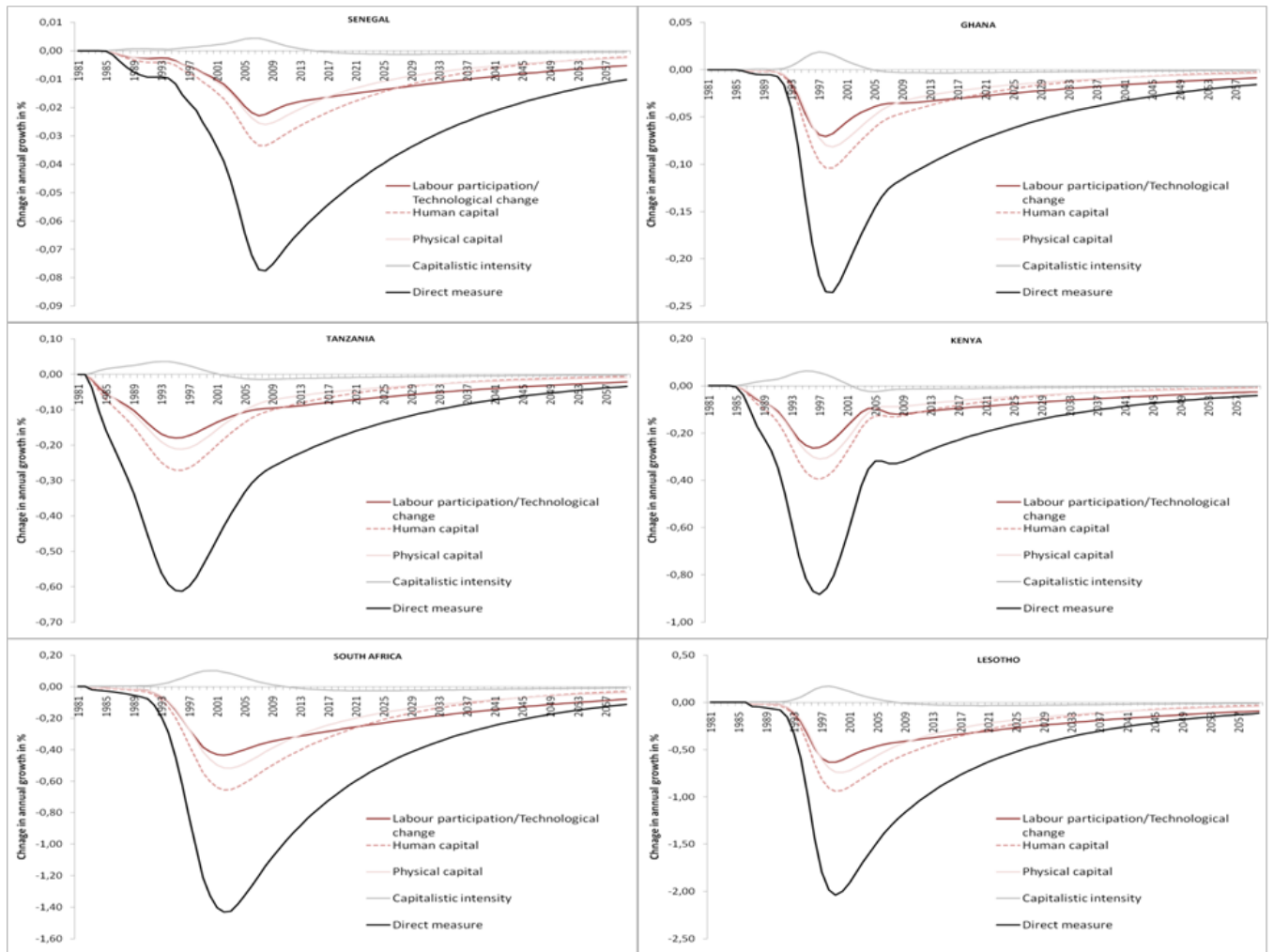K, HC and Y/N stands respectively for physical capital, human capital and GDP per adult.

Decomposition of the effects for some countries

Effects of HIV/AIDS on the components of the level of GDP per adult
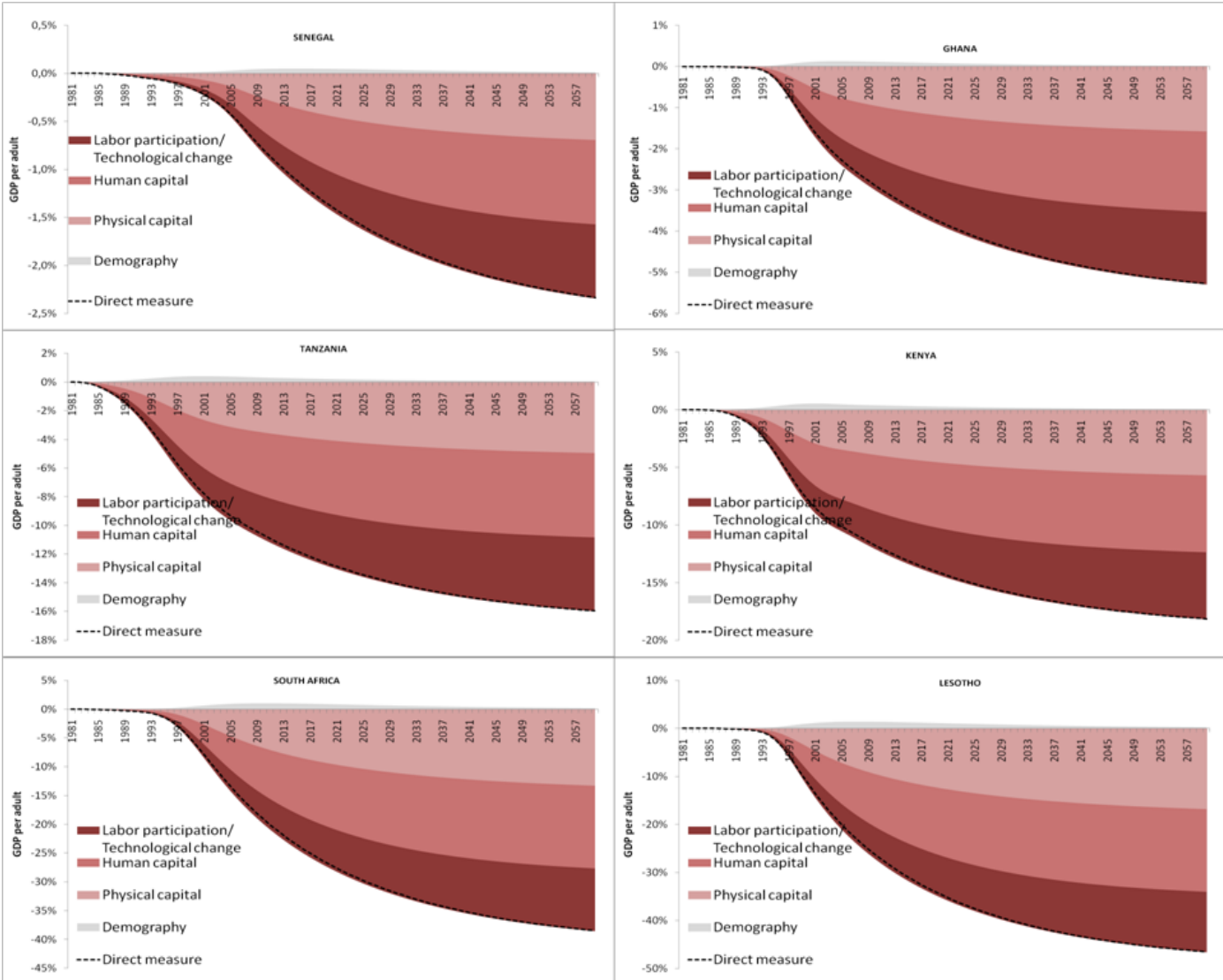
Level effects of HIV/AIDS on the components of the GDP per adult

Effects of HIV/AIDS on the components of the GDP per adult annual growth rate.

Growth rate effects of HIV/AIDS on the components of the GDP per adult