How has the introduction of antiretroviral therapy (ART) affected the dynamics between AIDS and economic growth in Sub-Saharan Africa?

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How has the introduction of antiretroviral therapy (ART) affected the dynamics between AIDS and economic growth in Sub-Saharan Africa?

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Abstract

This paper quantifies the economic effect of the use of antiretroviral therapy as a frontline strategy to fight AIDS using data on 29 countries in Sub-Saharan Africa. To this purpose, I use two-stage least squares estimation defining both a health equation and an education and health capital-augmented structural Solow growth equation. Through the introduction of antiretroviral therapy (ART) and HIV prevalence in the health equation, I indirectly link HIV and ART to economic growth. The results show that the HIV/AIDS epidemic reduces GDP per capita by 0.175% per marginal increase in HIV prevalence. ART increases GDP per capita by 0.048% per 1% increase in ART provision. On average, this represents a 0.5% higher GDP per capita per year attributable to ART in highly affected countries (HIV prevalence>20%).

* I am indebted to my advisor Professor Mario Solís-García who provided extraordinary guidance and support during this process. I also want to thank Professor Sarah West, Dr. Christy Hanson and Professor Raymond Robertson for their invaluable input and advice. I want to thank my friends Rosie Mate, David Lopez, Daniel Volk, Mac McCreary, Chris Fowler, Dhritiman Murti, Qianyi Yang and all the others that supported me in many important ways. Finally, I want to thank my mother for always believing in me and helping me get to succeed in college.
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1) Introduction

The development of anti-retroviral therapy (ART) marked the transition of the AIDS epidemic from a deadly, highly morbid and costly disease, to a chronic condition with reduced health and economic implications.\(^2\) The sexual transmission mechanism of the disease affects mainly the working population (15-49 year-old adults), translating into negative economic outcomes. The reduction in productivity associated with AIDS morbidity and the shrinking of labor supply due to AIDS mortality could be potentially harmful for long run sustained growth (Dixon, McDonald, & Roberts, 2002). The increasing number of orphans, the negative effects on human capital accumulation and incentives to save and invest in education exacerbate the macroeconomic impact of AIDS. From the government’s perspective, AIDS mortality leads to a reduction of the tax base (considering that it mostly affects working age population) translating in reduced revenue collection and an increase in the fiscal burden of the epidemic through treatment provision and healthcare costs. Also, by increasing mortality of the working population, AIDS undermines the base that sustains children and the elderly, putting pressure on the remaining working population to sustain a larger amount of dependents.

The reduction of morbidity and mortality that ART has brought plays a crucial role in fighting the AIDS epidemic and its effects on growth and development. Recent scientific evidence suggests that the provision of antiretroviral therapy is of critical importance not only because it reduces AIDS related mortality and morbidity, but also as a way to reduce

\(^{2}\) The human Immune Deficiency Virus (HIV) is the pathogen that causes the Acquired Immune Deficiency Syndrome (AIDS). The morbidity effects of HIV are observable only when AIDS develops. Two parameters are used to diagnose AIDS: 1) the CD4 cell count drops below 200/cc, or 2) HIV related infections become symptomatic. The onset between initial infection and AIDS varies, averaging 9-11 years. Death usually takes place 1-2 years after the onset of AIDS related infections in the absence of ART (Santaeulalia-Llopis, 2008)
incidence. At a global scale, there has been a 19% decrease in AIDS-related mortality between 2004 and 2009, mainly due to the increase in provision of ART. ART has been associated with a 92% decrease in risk of sexual transmission between sero-discordant\(^3\) partners. The provision of ART has been associated with a 67% risk reduction of developing tuberculosis (TB) and a 95% reduction in the risk of TB associated death. ART has reduced the number of HIV-positive mothers’ deaths and it has decreased the risk of mother-to-child transmission (UNAIDS, 2010). All of this translates into a reduction in the burden placed on an already saturated healthcare system, a substantial reduction in healthcare expenses in the medium and long run and a decrease in the negative economic impacts of AIDS.

The importance of antiretroviral therapy in fighting the HIV/AIDS epidemic motivates this paper. Unlike other papers, I attempt to quantify and understand the effect that ART has had in the already weakened economy of some highly affected African countries. I use an augmented Solow Growth model augmented with human and health capital to indirectly link HIV prevalence and ART provision to economic growth. I find that an increase of 1% in HIV prevalence decreases GDP per capita by 0.175%. The effect of a 1% increase in ART provision is associated with an increment of 0.048% in GDP per capita. This means that in a country with 20% HIV prevalence, GDP per capita in a given year will be 3.6% lower due to the economic burden of the HIV/AIDS epidemic. According to the estimates the high HIV countries in the sample on average a 0.53% increase in GDP per capita per year due to ART provision since it massive distribution in 2004.

\(^3\) Sero-discordant means of different HIV status.
2) Overview of the Epidemic

It has been about 30 years since the first HIV case was identified. Over this period of time, 65 million people have been infected with the HIV virus and 25 million have succumbed to AIDS (a comprehensive review can be found in UNAIDS World AIDS Day report (2010)).\(^4\) By the end of 2010, 34 million people are living with HIV worldwide, which is equivalent to a 17% increase from 2001. This increase is both due to an increase in incidence and because of the increasing provision of ART, which significantly reduces mortality and morbidity and increases lifetime.

Figure 1.1 shows how the HIV epidemic affects Sub-Saharan Africa (SSA) more than any other region in the world. According to UNAIDS 2011 World AIDS Day Report, in 2010 68% of all people living with HIV were located in Sub-Saharan Africa (which accounts for 12% of the global population). Looking at the global 2010 incidence of HIV, SSA accounts for 70% of the new infections in 2010. This broad analysis is partially misleading because it does not indicate that although there is a large number of people living with HIV in the region, there has been a 26% decrease in new infections from the peak of the epidemic in 1997 to 2010 in the region.

The mid-term outcomes and major trends of the HIV epidemic have become discernible in some key demographic variables. For example, Figure 1.2 depicts the global number of AIDS-related deaths for both adults and children between 1990 and 2010. The graph shows how the epidemic has been growing relatively slowly, causing a higher number of deaths over time until 2005, when HIV related deaths peak.

\(^4\) This is half the number of deaths of the Second World War (1937-1945) that amounted to 55-60 million. The number also amounts to the 30 million deaths of the famine of the Chinese Great Leap Forward (1958-1961).
Figure 1.1

Adult (15-49) HIV Prevalence in 2010

- Sub-Saharan Africa: .9
- Caribbean: .6
- Latin America: .4
- South and South East Asia: .3
- Eastern Europe and Central Asia: .3
- North America: .2
- Wester and Central Europe: .2
- East Asia: .1

Figure 1.2

Number of adult and infant HIV related deaths (millions), 1990-2010

- 1990: .25
- 1991: .32
- 1992: .32
- 1993: .52
- 1994: .65
- 1995: .94
- 1996: .94
- 1997: 1.07
- 1998: 1.35
- 1999: 1.57
- 2000: 1.7
- 2001: 1.85
- 2002: 2.05
- 2003: 2.19
- 2004: 2.15
- 2005: 2.05
- 2006: 2.06
- 2007: 2
- 2008: 1.9
- 2009: 1.8
- 2010: 1.8
After this year, HIV-related deaths steadily decreased. UNAIDS attributes the drastic change in the demographic variables of the most affected countries to the increase in ART provision (number of people receiving treatment) and services that increase its effectiveness (increasing and supporting proper adherence and counseling).\(^5\)

Reductions in price of ART enabled the massive deployment of ART as a frontline strategy to fight HIV/AIDS. These price reductions are the results of the introduction of generic ART and the mediation efforts of massive purchases led by organizations like the Global Fund and the Clinton Foundation (Vasan, Mukherjeem Farmer, Rosenfield, 2006).

**3) Literature Review**

Since the start of the HIV epidemic in 1982, the highest demographic and economic impact has been felt in Sub-Saharan Africa. According to the Joint United Nations Programme on HIV/AIDS (UNAIDS, 2010), 34% of the people living with HIV in the world are concentrated in 10 countries in southern Africa. A disproportionately large part of the global sero-positive (HIV positive) population lives in this area and HIV-related deaths are the highest there due to exacerbating factors such as poor nutrition, lack of access to healthcare and HIV testing, and poverty.

The approaches to understanding and quantifying the relationship between HIV and growth are varied and propose different mechanisms. The most relevant research, McDonald & Roberts, 2006, investigates the effect of AIDS on economic growth using an augmented Solow model. Their augmented Solow model consists of a Cobb-Douglas

---

\(^5\) Adherence means following the treatment regime both in timing and dose. Adherence not only maximizes the benefits of ART by preventing the virus to replicate but is also necessary to prevent mutations and drug resistance (US. National Institute of Health, 2010)
function with labor enhancing technology, labor, physical, human and health capital. They use data for income, population, investment rates, and education from the World Bank’s Development Indicators and Global Development Finance database. The authors use a panel set of data for 112 countries over the period 1969 to 1998. They find that an increase of 1% in HIV prevalence decreases GDP per capita by 0.59%.

Ahuja, Wendell and Werner (2006) use male circumcision rates as an instrument for HIV prevalence in order to investigate the relationship between AIDS and economic growth. The use of male circumcision as an instrument for HIV prevalence is appropriate because it is not correlated with any potential omitted variable at the national level like initial income, initial life expectancy or modernity. The basis for their approach derives from the measurement error that is likely to plague estimates of HIV prevalence, the problem of reverse causality between HIV prevalence and many economic indicators of growth, and the possibility of omitted variable bias. Male circumcision rates are likely to face none of the problems of endogeneity that arise when using other macro-economic indicators. They measure economic growth and performance using crude death rate, infant mortality rate, GDP per capita, savings as percentage of GDP, crude birth rate, youth literacy rate and malnutrition rate.

---

6 The augmented Solow model follows a progression that starts with Mankiw, Romer and Weil who add human capital to the model (Mankiw, Romer & Weil, 1992). Knowles and Owen subsequently add health capital (Knowles & Owen, 1995).

7 The relationship between male circumcision and the decrease in HIV has been explained mainly by two theories. The first one is that uncircumcised males, within a less sanitary environment, are more likely to become infected with chancroid infections that ulcer. The ulcers increase blood exposure, increasing the likelihood of HIV transmission (Caldwell & Caldwell, 1996). The second theory proposes that there is a high concentration of Langerhans cells in the prepuce. Langerhans cells are known to be targets for HIV transmission, explaining why removing the prepuce would decrease the likelihood of transmission (Hussain & Lehner, 1995). Prevalence of other sexually transmitted infections also increases the likelihood of HIV transmission by the same ulcer mechanism.
Their first empirical strategy consists of two parts. First they divide a sample of 49 African countries into high 1997 prevalence (> 6%) and low 1997 prevalence (<6%) and low (<20%), medium (20-80%) and high (>80%) circumcision rates. They use HIV prevalence rate in 1997 as a proxy indicator for the average AIDS rate between 1990 and 2005. They proceed to graphically analyze how each economic outcome is affected by the AIDS rate through time by level of circumcision and high/low AIDS rate. They find the economic effect of AIDS on African economies to be minimal: AIDS does not significantly affect GDP per capita, savings rate and fertility. Contrary to the results of my research they do find that AIDS is reducing the rate at which youth literacy is increasing (Ahuja, Wendell & Welker, 2006).

The most direct attempt to link HIV prevalence to economic growth is done by Bonnel (2000). His model is based on three equations: one that links economic growth to macroeconomic policy, institutions and other determinants of growth; a second that links institutional variables to HIV; and a third that attempts to explain the determinants of HIV prevalence. Bonnel finds that the effect of AIDS on economic growth is significant for African countries with high HIV prevalence. He estimates that for countries with high HIV prevalence (20% or more) there is a 2.6% decrease in yearly economic growth and that for countries with low prevalence (less or equal to 8%) there is an associated 0.7% decrease in growth. The main concern with Bonnel’s work is that it a lacks theoretical framework. Additionally, here does not attempt to measure the effects of how ART might mitigate the effects of the AIDS epidemic.

Bell, Devarajan and Gersbach (2006) take a different approach to gauge the effect of AIDS on economic outcomes. They predict that AIDS will provoke an economic catastrophe
in the long run, triggered by a vicious cycle of premature death of parents and stagnation of human capital accumulation of the offspring. AIDS plays a role in reducing the combined human capital endowment of the parents (if one of them dies) and leaving the child as an orphan (if both parents die). This affects the ability of the offspring to transmit human capital (not only as education, but as care and nurture) to the coming generation. The increase in orphans and the vicious cycle described by their model implies that there will be growing income inequality across time.

Overall, the research that has been done regarding AIDS and its effect on the economy has focused on measuring the impact of the disease through the many different channels outlined. There have been no econometric attempts to gauge the effect of ART on the dynamics between the AIDS epidemic and economic growth. This paper attempts to fill this gap in the literature.

4) Theory

4.1 Augmented Solow Model

In this section I develop the structural growth equation that I will use to gauge the effects of ART provision on economic growth. The model development follows the chronological order of the literature, starting with the traditional model proposed by Solow (Solow, 1959), and the subsequent human capital augmented model developed by Mankiw, Romer and Weil (MRW, 1992). MRW find that adding human capital to the model improves how it fits existing data. They focus mainly on human capital as investment in education, particularly elementary schooling. They omit high school, higher education and investment
in health. In spite of this, the augmented model better reflects empirical evidence in comparison to the textbook Solow model.

Knowles and Owen (1994) propose the addition of human capital not only as investment in education, but also as investment in health. In 2005, McDonald and Roberts take the human-health capital augmented Solow model and define a relationship that allows them to explore the effect of AIDS on growth through the health capital parameter. They use a Cobb Douglas production function with labor-enhancing technology:

\[ Y_{it} = [A_t L_{it}]^{1-\alpha-\beta-\psi} K_{it}^{\alpha} E_{it}^{\beta} H_{it}^{\psi} \]  \hspace{1cm} (1)

where:

- \( Y_{it} \) is output of country \( i \) at time \( t \)
- \( A_t \) is technology at time \( t \) (assumed to be homogeneous throughout countries)
- \( L_{it} \) is labor of country \( i \) at time \( t \)
- \( K_{it} \) is physical capital of country \( i \) at time \( t \)
- \( E_{it} \) is education capital of country \( i \) at time \( t \)
- \( H_{it} \) is health capital of country \( i \) at time \( t \)
- \( \alpha-\beta-\psi \) are physical, human and health capital elasticities with respect to output

Dividing equation (1) by \( A_t L_{it} \) yields its intensive form:

\[ y_{it} = k_i^{\alpha} e_i^{\beta} h_i^{\psi} \]  \hspace{1cm} (2)

where the variables in lower case represent quantities per unit of effective unit of labor (for example \( y_{it} = Y_{it} / A_t L_{it} \)).

Assuming that labor grows at a country specific constant rate of \( n_i \), technology grows at a time specific rate of \( g_t \) and that physical, education and human capital
depreciate at a constant rate of $\delta$, we can show (see Appendix 1) that the augmented steady state output per capita\(^8\) ($y^*_{it}$) is given by:

$$
\ln y^*_{it} = \ln A_{i0} + g_{it} - \frac{\alpha + \beta + \psi}{(1 - \alpha - \beta - \psi)} \ln (n_i + g_i + \delta) + \frac{\alpha}{(1 - \alpha - \beta - \psi)} \ln s^K_i + \frac{\beta}{(1 - \alpha - \beta - \psi)} \ln s^E_i + \frac{\psi}{(1 - \alpha - \beta - \psi)} \ln s^H_i \tag{3}
$$

where $s^H_i$, $s^E_i$, $s^K_i$ are the savings rate for physical, education and health capital respectively. Equation (3) is equivalent to MRW’s equation except for the added health capital term, its elasticity with respect to output $\psi$ and the savings rate of health capital.

Estimating equation (3) is complicated by the fact that disaggregated investment data is not readily available and probably non-existent. Available investment data refers to physical capital, whereas human and health capital investments are likely to be missing. There is a way of overcoming this through the steady state condition in levels imbedded in equation (3). MRW used this condition to propose alternative formulations where the estimating equation is not in terms of rates of accumulation (refer to equation 3) or levels, for example:

$$
\ln y^*_{it} = \ln A_{i0} + g_{it} - \frac{\alpha}{(1 - \alpha)} \ln (n_i + g_i + \delta) + \frac{\alpha}{(1 - \alpha)} \ln s^K_i + \frac{\beta}{(1 - \alpha)} \ln s^E_i + \frac{\psi}{(1 - \alpha)} \ln h^*_{it} \tag{4}
$$

\(^8\)Steady state means that the growth of the per capita stock of capital is 0. This implies that investment is equal to depreciation and the change of per capita stock of capital remains unchanged given the current level of savings (Mankiw, 2009).
In Equation (4), $h^*_it$ is a steady state quantity of health capital per effective unit of labor. Since Equation (3) is derived under the assumption of a steady state, equation (4) does require the assumption of a steady state.

MRW’s method of formulating Equation (3) in terms of levels and not saving rates provides the empirical advantage of allowing to simultaneously use stock and savings data for the parts of the estimating equation. Using MRW’s method, I can linearize Equation (4) around the steady state level of income per effective unit of labor ($y^*_{it}$)

$$
\ln y^*_{it} - \ln y^*_{i0} = (1 - \exp^{\lambda t}) \ln A_{i0} + g_i t - \frac{(1 - \exp^{\lambda t}) \alpha}{(1 - \alpha)} \ln (n_i + g_i + \delta) + \frac{(1 - \exp^{\lambda t}) \alpha}{(1 - \alpha)} \ln s_{it}^E
$$

$$
+ \frac{(1 - \exp^{\lambda t}) \beta}{(1 - \alpha)} \ln s_{it}^E + \frac{(1 - \exp^{\lambda t}) \psi}{(1 - \alpha)} \ln h^*_it - (1 - \exp^{\lambda t}) \ln y^*_{i0}
$$

(5)

Solving for $\ln y^*_{i0}$ and using standard panel notation yields the general form of the estimating equation that I will use:

$$
z^*_{it} = y z^*_{i0} + \sum_{j=1}^{4} \theta_j x^*_{itj} + \eta_i + \mu_i + \nu_{it}
$$

(6)

Where:

- $z^*_{it} = \ln y^*_{it}$
- $\theta_3 = \frac{(1 - \exp^{\lambda t}) \beta}{(1 - \alpha)}$
- $y = \exp^{\lambda t}$
- $x^*_{itj} = \ln s_{it}^E$ or $\ln e^*_{it}$
- $z^*_{i0} = \ln y^*_{i0}$
- $\theta_4 = \frac{(1 - \exp^{\lambda t}) \psi}{(1 - \alpha)}$
\[
\theta_i = -\theta_2 = \frac{(1 - \exp^{\nu})\alpha}{(1 - \alpha)} \quad x_{it}^4 = \ln s_{it}^4 \text{ or } \ln h_{it}^* \\
x_{it}^1 = \ln(n_i + g_i + d) \quad \eta_i = g_i \\
x_{it}^2 = \ln s_{it}^c \quad \mu_i = (1 - \exp^{\nu}) \ln A_{it}
\]

and where \( \nu_{it} \) is the standard error term.

There are some particularities of Equation (6) that make it different from a typical cross-sectional estimating equation. First it allows for variation in the country’s rate of technology growth (\( g_i \)), the initial level of technology (\( \ln A_0 \)). Also, the estimating equation uses the time series information because it is being estimated as a two-way fixed-effects dynamic data panel.

The technology variances between countries can be accounted by:

\[
\ln A_{it} = a + \epsilon
\]

Equation (7) implies that all country differences depend in the random term \( \epsilon \). The term is a constant, invariant across countries.

### 4.2 Health Equation

The theoretical definition of health capital in a macroeconomic context that is suitable for this estimation is not clear-cut. The exploration of the determinants of health mostly comes from the micro-intensive derivation of the demand for health postulated by Grossman (1972). The determinants of the “shadow price” of health are not easily applicable to the macro estimating equation used in this paper. A more macro overview of the determinants of health is proposed by Genberg (1992), who concludes that
macroeconomic changes and health outcomes are not evidently clear and that there is not enough evidence of causality between the two.

Considering this, McDonald and Roberts (2005) propose that the second-best option to theoretical formulations of health determinants is to propose a reduced form health equation. The components of the health equation they used are “guided by literature but do not represent structural relationships” (McDonald & Roberts, 2005). The reduced equation they propose is a function of a set of exogenous variables. These variables include lagged income given that there is evidence that higher income populations have higher health outcomes. Education capital and nutritional capital are also included given that they are both related to better health outcomes as well, particularly in developing countries. HIV prevalence and the proportion of the population at risk of malaria are included as exogenous health shocks.

Through the health equation, I measure the impact of ART on reducing the effect of the HIV/AIDS epidemic on output per worker in the structural growth equation. I propose the introduction of more variables in the vector that McDonalds and Roberts (2006) developed. Antiretroviral provision is not only an effective way of reducing mortality and morbidity, but also reduces the probability of infection per unprotected sexual encounter.

The last addition to the health capital equation is DTP immunization provision. Following the procedure of McDonald and Roberts (2006) to account for non-linearities and possible lags, I also include lagged values of the variables, interactions of each variable with per capita income and the squared terms of each explanatory variable. The reduced form health equation is:

\[ x_{it}^4 = \alpha x_{it-1} + \rho P + \xi + \omega_i + w_{it} \]
where \( x_{it}^{\prime} = \ln h_{it} \). \( P \) is the vector of exogenous variables explained above, \( \xi_{i} \) stands for time effects, \( \omega_{i} \) stands for country specific effects and \( w_{i} \) is the error term. Time-specific and country-specific effects are necessary given that inherent time trends or country differences will distort the interpretation of my results and will need to be accounted for in the estimation.

Using infant mortality rate as the dependent variable will capture the variance of the HIV shocks that affect mothers and is transmitted to the newborn. Given the variables included in the health equation, it will capture fluctuations in the general health of the population and it might even overestimate it, given that women are often a vulnerable group in low-income settings. To pursue the direct impact of HIV on the working population, it would be ideal to have mortality by age cohorts, yet this would only capture the mortality and would not account for AIDS related morbidity.

5) Data

The main source of data for the structural growth equation is the World Bank World Development Indicators Database (World Bank, 2010).\(^9\) The panel includes 29 Sub-Saharan countries from 1983 to 2009 (see Appendix 2) Income data are real GDP per capita purchasing power parity adjusted. I calculate the investment rates from 2005 price data on GDP and domestic investment.

For human capital as education, there are three series: primary, secondary and tertiary school enrollment rates available. I use primary school enrollment given that it has the most data for most countries. In many countries there is inconsistency in the collection

of data for secondary and tertiary. Primary school also could capture the effects of improvements in health in the poorest populations given that the likelihood of a person completing secondary schooling are less than of completing primary schooling.

The data for the health equation are limited by availability and length of the time series. Following McDonald and Robert's (2005) approach, I use infant mortality rate. McDonald and Roberts (2006) argue that it would reflect the state of the health of the population by looking at the most vulnerable population. Infant mortality would be a better reflection of the quality aspect of health, but might overestimate shocks to the health capital that asymmetrically impact mothers. An ideal indicator would capture both the morbidity and mortality aspects related to the development of AIDS.

HIV prevalence between 15-49 years was obtained from the UNAIDS database AIDSinfo. The series includes the time period between 1990 and 2009. The method used to calculate these estimates is detailed in Schwartlander et al (1999).

Total ART coverage is available from UNAIDS starting from 2004. The use of antiretroviral therapy to fight HIV starts with the development of azidothymidine (AZT) in 1987. Prior to then, since no effective therapy existed it is safe to assume that ART provision was zero. Additionally, the cost of ART when it was developed was around 800 dollars per month in the United States in 1997. It is only with the introduction of generic nevirapine that the price of ART falls from 750 dollars per month in 2000 and 30 dollars per month in 2003 (Kumarasamy et al., 2005). This means that after 2000, ART became a

---

10 Obtained from: [http://www.aidsinfoonline.org](http://www.aidsinfoonline.org)

11 Nevirapine is a type of medicine called a non-nucleoside reverse transcriptase inhibitor (NNRT). It works by disrupting one of the early steps in the HIV life cycle, called reverse transcriptation (National Institute of Health, 2012)
cost-effective strategy in the fight against HIV and AIDS, when generic ART became available.

The price of ART that is used to instrument ART provision comes from the World Health Organization’s Global Price Reporting Mechanism.\(^\text{12}\) The price data is derived by adding up the price of lamivudine (3TC-150 mg.), nevirapine (NVP – 200 mg.) and stavudine (d4T-300 mg.), the most commonly used first line ART in low-income countries.

I obtain DPT (diphtheria, pertussis, tetanus) immunization, expressed as the percent of children between 12-23 months old vaccinated, from the World Bank’s Health, Nutrition and Population Statistics database.\(^\text{13}\) Protein intake supply in grams per person per day was obtained from FAO’s database FAOSTAT.\(^\text{14}\)

Table 1 below contains the summary statistics for the variables included in the regression analysis and from which calculations were made as well. ART provision is the one with the least available data given that its implementation has been relatively recent. HIV prevalence ranges from 0.1 to 26.5% throughout 1990-2009. This stark difference puts in perspective the difference between high and low HIV prevalence countries. This is more evident when comparing Table 2 (High HIV) and Table 3 (Low HIV).

\(^\text{12}\) Obtained from [http://apps.who.int/hiv/amds/price/hdd/index.aspx](http://apps.who.int/hiv/amds/price/hdd/index.aspx)

\(^\text{13}\) Obtained from [http://databank.worldbank.org/ddp/home.do](http://databank.worldbank.org/ddp/home.do)

## Table 1. Summary Statistics for all countries
**Time period 1983-2009**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Observations</th>
<th>Mean</th>
<th>Std. Dev.</th>
<th>Minimum</th>
<th>Maximum</th>
</tr>
</thead>
<tbody>
<tr>
<td>HIV Prevalence (% of 15-45 pop.)</td>
<td>580</td>
<td>6.29</td>
<td>7.10</td>
<td>0.1</td>
<td>26.50</td>
</tr>
<tr>
<td>ART Coverage (number receiving)</td>
<td>460</td>
<td>51006.36</td>
<td>11049.60</td>
<td>0</td>
<td>971556</td>
</tr>
<tr>
<td>DPT Immunization (% of children 13-23 months)</td>
<td>567</td>
<td>62.17</td>
<td>25.15</td>
<td>1</td>
<td>99</td>
</tr>
<tr>
<td>Population (number)</td>
<td>580</td>
<td>6447475</td>
<td>7646427</td>
<td>156983</td>
<td>4430000</td>
</tr>
<tr>
<td>GDP Per Capita (PPP adjusted 2005 dollars)</td>
<td>580</td>
<td>2886.77</td>
<td>4136.36</td>
<td>303.24</td>
<td>31738.23</td>
</tr>
<tr>
<td>Primary School Enrollment (%)</td>
<td>547</td>
<td>65.48293</td>
<td>22.08</td>
<td>17.47</td>
<td>99.73</td>
</tr>
<tr>
<td>Daily Protein Intake (grams)</td>
<td>580</td>
<td>56.73</td>
<td>26.97</td>
<td>30.34</td>
<td>674</td>
</tr>
<tr>
<td>Life Expectancy (years)</td>
<td>556</td>
<td>51.93</td>
<td>6.83</td>
<td>26.81</td>
<td>72.88</td>
</tr>
<tr>
<td>Infant Mortality (deaths/1000 births)</td>
<td>534</td>
<td>86.34</td>
<td>28.40</td>
<td>13.20</td>
<td>166</td>
</tr>
<tr>
<td>Investment (% of GDP)</td>
<td>576</td>
<td>20.61</td>
<td>11.27</td>
<td>1.57</td>
<td>113.57</td>
</tr>
<tr>
<td>Population Growth (%)</td>
<td>580</td>
<td>2.52</td>
<td>1.26</td>
<td>-7.53</td>
<td>9.77</td>
</tr>
</tbody>
</table>
Table 2. Summary Statistics for Mean Low HIV Prevalence countries (<6% in 1997)
Time period 1983-2009

<table>
<thead>
<tr>
<th>Variable</th>
<th>Years</th>
<th>Mean</th>
<th>Std. Dev.</th>
<th>Minimum</th>
<th>Maximum</th>
</tr>
</thead>
<tbody>
<tr>
<td>HIV Prevalence (% of 15-45 pop.)</td>
<td>20</td>
<td>2.10</td>
<td>0.54</td>
<td>0.96</td>
<td>2.62</td>
</tr>
<tr>
<td>ART Coverage (number receiving)</td>
<td>6</td>
<td>11246.02</td>
<td>8328.40</td>
<td>1630.56</td>
<td>23549.00</td>
</tr>
<tr>
<td>DPT Immunization (% of children 13-23 months)</td>
<td>27</td>
<td>54.86</td>
<td>13.61</td>
<td>26.91</td>
<td>73.44</td>
</tr>
<tr>
<td>GDP Per Capita (PPP adjusted dollars)</td>
<td>27</td>
<td>3097.43</td>
<td>842.28</td>
<td>2299.25</td>
<td>4950.46</td>
</tr>
<tr>
<td>Primary School Enrollment (%)</td>
<td>27</td>
<td>42.60</td>
<td>14.10</td>
<td>4.11</td>
<td>64.12</td>
</tr>
<tr>
<td>Daily Protein Intake (grams)</td>
<td>25</td>
<td>53.01</td>
<td>2.09</td>
<td>50.00</td>
<td>57.99</td>
</tr>
<tr>
<td>Life Expectancy (years)</td>
<td>27</td>
<td>51.60</td>
<td>2.55</td>
<td>48.60</td>
<td>56.19</td>
</tr>
<tr>
<td>Infant Mortality (deaths/1000 births)</td>
<td>27</td>
<td>92.55</td>
<td>11.54</td>
<td>73.33</td>
<td>111.75</td>
</tr>
<tr>
<td>Investment (% of GDP)</td>
<td>27</td>
<td>19.01</td>
<td>2.56</td>
<td>15.85</td>
<td>23.56</td>
</tr>
<tr>
<td>Population Growth (%)</td>
<td>27</td>
<td>2.54</td>
<td>0.37</td>
<td>1.70</td>
<td>3.03</td>
</tr>
</tbody>
</table>
Table 3. Summary Statistics for Mean High HIV Prevalence countries (>6% in 1997)  
Time period 1983-2009

<table>
<thead>
<tr>
<th>Variable</th>
<th>Years</th>
<th>Mean</th>
<th>Std. Dev.</th>
<th>Minimum</th>
<th>Maximum</th>
</tr>
</thead>
<tbody>
<tr>
<td>HIV Prevalence (% of 15-45 pop.)</td>
<td>20</td>
<td>12.20</td>
<td>3.50</td>
<td>4.07</td>
<td>15.17</td>
</tr>
<tr>
<td>ART Coverage (number receiving)</td>
<td>6</td>
<td>106794.50</td>
<td>71823.49</td>
<td>20060.50</td>
<td>212886.50</td>
</tr>
<tr>
<td>DPT Inmunization (% of children 13-23 months)</td>
<td>27</td>
<td>72.80</td>
<td>8.38</td>
<td>48.80</td>
<td>80.67</td>
</tr>
<tr>
<td>GDP Per Capita (PPP adjusted dollars)</td>
<td>27</td>
<td>2956.89</td>
<td>368.90</td>
<td>2622.26</td>
<td>3735.21</td>
</tr>
<tr>
<td>Primary School Enrollment (%)</td>
<td>27</td>
<td>42.22</td>
<td>12.62</td>
<td>23.00</td>
<td>64.00</td>
</tr>
<tr>
<td>Daily Protein Intake (grams)</td>
<td>25</td>
<td>57.86</td>
<td>1.51</td>
<td>55.51</td>
<td>60.68</td>
</tr>
<tr>
<td>Life Expectancy (years)</td>
<td>27</td>
<td>52.70</td>
<td>2.80</td>
<td>48.74</td>
<td>56.09</td>
</tr>
<tr>
<td>Infant Mortality (deaths/1000 births)</td>
<td>27</td>
<td>76.01</td>
<td>6.51</td>
<td>59.58</td>
<td>85.18</td>
</tr>
<tr>
<td>Investment (% of GDP)</td>
<td>27</td>
<td>20.90</td>
<td>1.96</td>
<td>18.23</td>
<td>24.45</td>
</tr>
<tr>
<td>Population Growth (%)</td>
<td>27</td>
<td>2.44</td>
<td>0.60</td>
<td>11.71</td>
<td>3.43</td>
</tr>
</tbody>
</table>
6) Graphical Analysis

This section focuses on a graphical analysis of the variables of interest throughout time. I follow Werker, Ahuja and Wendell (2006) and group countries in two categories: those with high HIV prevalence (1997 HIV prevalence>6%) and those with low HIV prevalence (1997 HIV prevalence<6%). I choose 1997 as the comparison year because this is often regarded as the year when the extensive spread of the epidemic took place. I then average the values for each year by the two groups described above for each of the variables of interest. The first variables of interest are mean GDP per capita and mean HIV prevalence, presented in Figure 2.a and 2.b.

Figure 2.a shows the time variation of GDP over time for both high and low HIV prevalence countries. The vertical times in the graphs represent two important years. The first line is 1997, the year that was chosen to divide the countries in high and low HIV prevalence. The second line is 2004, the year when UNAIDS starts recording data of ART provision.

Both groups have a similar starting point. High HIV prevalence countries have higher GDP per capita during 1983-1994. This is reasonable given that the effects of HIV are not noticeable in the short run and the effects of the new infections during this period would be felt on average 9-10 years after initial infection, when symptomatic AIDS related diseases appear. What is noticeable is that in the mid 1990s, both high and low HIV groups experience a consistent increase of GDP per capita. The change is noticeably larger for low HIV countries, which manage to close the gap around 2000 and reach approximately 5000 dollars per capita in 2009, doubling GDP per capita in around 15 years.
Figure 2.a

Figure 2.b

Figure 2.c

PPP GDP Per Capita

Mean HIV Prevalence

ART Provision
High HIV countries also experience growth, but it pales in comparison to the low HIV group. By 2009, the gap between high and low HIV countries has reversed and stands at about 1300 dollars per capita, favoring low HIV countries.

Figure 2.b shows the progression of the number of the mean sero-positive individuals between 15-45 years old separated by high HIV prevalence and low HIV prevalence. This graph indicates that countries with high HIV prevalence in 1997 did not only start with a higher HIV prevalence in 1990, but had a remarkable rate of increase during the 1990s, peaking at around 2000 and starting to decrease. The period of decrease of HIV prevalence seen starting during 2000 could potentially be attributed to ART provision and the implementation of prevention initiatives that reduce risky practices both sexual and in intravenous drug use.

Figure 2.c depicts the progression of ART over time. The graph indicates that countries with low HIV prevalence received overall substantially less ART than countries with high HIV prevalence. The two series start at different levels in 2004, and diverge from there. The two series diverge so noticeably because there is a massive increase in ART provision in countries with high HIV prevalence, while the low HIV prevalence ART provision does not increase substantially during this 5-year period. It is logical to think that the countries that are most afflicted would receive the highest quantity of antiretroviral therapy, both to slow down the rate of transmission and alleviate the effects of morbidity of AIDS. As shown in the following graphs, there is suggestive evidence that ART provision has had a noticeable effect both in the micro and macro levels.
Figure 3.a shows the progression of life expectancy at birth over time. This graph is also suggestive because it shows that life expectancy for low HIV countries has increased and overall improved over time without any noticeable drops. On the other hand, high HIV countries have a higher life expectancy than low HIV prevalence countries in 1983. Life expectancy peaks at around 1990 and decreases to 1983 low HIV levels. The drastic drop in life expectancy of high HIV countries supports the argument that HIV has substantially decreased the life prospects of the most afflicted nations.

Another important trend to notice is that the point at which life expectancy reaches its minimum seems to match with the period where ART began being intensively used to combat the contagion, morbidity and mortality of HIV/AIDS. According to UNAIDS, “at the end of 2009, 37% of the adults and children eligible for antiretroviral therapy were receiving it in the region overall, compared with only 2% seven years earlier” (UNAIDS, 2010).

Figure 3.b shows the progression of infant mortality rate over time. For both groups, there has been a steady decrease from the start of the series. There is a constant difference, with a lower infant mortality for high HIV prevalence countries. What is noticeable is that that in 1991, the infant mortality rate for the high HIV prevalence countries starts to increase, closing the gap of infant mortality rate between the two groups. The time period is suggestive that this greater infant mortality observed in high HIV countries could be caused by increased perinatal transmission (mother to child). The second important time period is 2000-2009, where ART was introduced as part of the strategy to eliminate mother to child transmission. According to UNAIDS, in 2009, an
estimated 370,000 children were newly infected with HIV, representing a 24% drop from the previous 5 years.

Figure 3.c indicates the progression of the population growth rate over time. Both high and low HIV countries experience a drop in population growth between 1987 and 1992. After this point, low HIV countries’ population growth rate increases and peaks in 2000, experiencing a slight decline after this year. The trend for high HIV countries after the initial drop is a plateau (in contrast to the increase of low HIV countries) and then it drops to its minimum between 2003 and 2004. After 2004, high HIV countries experience a slight increase in population growth rates.

From a theoretical perspective, it makes sense that AIDS would decrease the population growth rate. AIDS would do this in two ways: an increase in mortality and a decrease in fertility. Currently, AIDS related deaths account for 20.4% of the total deaths in Sub-Saharan Africa. This makes AIDS the leading cause of death in the region and the fourth cause of death worldwide (Jamison, Feachem, Makgoba, Bos, Baingana, Hofman & Rogo, 2006).

The decrease in fertility is supported by micro empirical evidence. Zaba and Gregson (1998) estimate that there is a 0.4% decrease in fertility associated with 1% increase in HIV prevalence, mainly due foetal loss associated with HIV infection and co-infection of other sexually transmitted diseases. Fertility also is reduced by the risk perception of HIV contraction. As HIV prevalence increases, the demand for children falls in response to the perceived increase in the hazard of infection (Young, 2005).

Figure 4.a shows the progression of protein intake throughout time. Protein intake is used in the included in the reduced health equation as a proxy for nutrition. In general,
protein intake has increased over time for both groups. What is noticeable is that there is a 10 grams per day difference in 1983 between high and low HIV countries. The difference between protein intake converges as time passes and reaches a 3 grams per day difference in 2009.

Figure 4.b shows the percent of children between 12-23 months provided with DPT immunization. The steepest increase for both groups is observed in the 1980s, noticing that high HIV prevalence countries have higher DPT immunization throughout the time period, and being the difference most stark in the 1980s. It is relevant to include a proxy for vaccination given that it most certainly increases the health capital of the population by reducing the incidence of preventable diseases particularly in the infant population.

Figure 4.c shows the progression of gross capital formation over time. High HIV countries start at a substantially higher level of investment than low HIV countries. There is a lot of volatility in the series for both groups. Both countries converge in the end of the series, with low HIV countries having higher gross capital formation from 2000 on. The volatility could be an indicator of other conditions that factor in the perception of risk of investing in the country (corruption, political and social unrest, lack of solid institutions, etc.). Although the plot of both groups over time is hard to interpret, it could be possible that the decline in investment of high HIV countries can be partially explained by a shift in risk preference of investors given both the morbidity and mortality effects of AIDS on the working population.
Something that must be noted is that all the previous graphs seem to indicate that prior to the development and widespread of the AIDS epidemic, countries with high HIV prevalence were in better conditions than low HIV prevalence countries. Countries with high HIV prevalence had a higher GDP per capita, higher life expectancy, higher, DPT immunization rates, higher protein intake per capita, investment and lower infant mortality rate during the start of the time series. The gap between the two groups got closer or even reversed particularly during the 1990s, where the epidemic was spreading at a higher rate and the effects of the new infections in the previous decades would be felt.

With the graphical analysis I have conducted, there are some interesting trends that match the peak of the epidemic and suggest that it has had an important effect both on the quality of living and in the rate of growth of countries with high HIV prevalence. Increases in ART seem to match increases in life expectancy, reductions in infant mortality rate and have managed to slow the rate of growth of the epidemic.

Although the graphs provide a good intuition of what HIV has done to health and growth, I cannot obtain a valuation of how much it has affected growth through the effects of mortality, morbidity and demographic change. The same applies to ART provision. This leads us to test formally for the effect of ART on reducing the impact of AIDS on the growth of the nations of the sample.

7) Econometric Method

The model developed by McDonald and Roberts (2006) is composed of two equations: the growth equation derived from the augmented Solow model and the reduced-form health equation. There are three econometric issues of concern. First, the health
capital variable is endogenous in the growth equation, making it correlated with the error term, which biases the estimation not only of the endogenous regressor, but also of all the other covariates included in the regression. The second concern is that both the health and growth equations represent dynamic models containing lagged variables. This also leads to biased and inconsistent estimates if I were to use ordinary least square estimation. The third concern is that given the panel nature of our data, individual heterogeneity must be accounted for to produce reliable estimates.

Instrumental variable estimation is used in two stages to deal with both the endogeneity of the health variable in the growth equation and the presence of lagged variables in both equations. The first stage obtains the health capital predicted values from the health equation. The lagged dependent variable is instrumented for using the exogenous variables in the model and further lags on the health term. The second step consists of using the predicted values obtained from the health equation in the growth equation. By doing this, I deal with the endogeneity of the health capital term on the growth equation. The growth equation is also estimated using instrumental variable estimation to deal with the lagged dependent variable.

Hausman (1978) tests are used to investigate the need of including country specific effects. The Hausman test is used to determine if the fixed effects inclusion is appropriate. Since country specific effects are appropriate, I estimate the dynamic model using pooled data through the instrumental variable method. I use lagged levels of the dependent variable, predetermined variables and exogenous variables are used as instrument for the lagged dependent variable.
Considering that the growth equation has strong theoretical support, the variables are maintained in the equation even if they are statistically insignificant or they have an unexpected sign. This does not hold for the health equation, where a general to specific approach was used to determine what variables are relevant. The variables I include in the health equation are the lagged dependent variable, HIV prevalence, antiretroviral provision, current income, lagged income, protein intake and primary school enrollment. Additionally, I include interactions between income and all the explanatory variables and the squared explanatory variables. Both infant mortality rate and life expectancy are used as dependent variables in the health equation.

7.1 Endogeneity Problems

The inclusion of lagged variables in the health equation justifies the use of instrumental variable regression. To understand the full implications of endogeneity, I proceed to define and describe why endogeneity is a problem and what the instrumental variable regression does to solve it.

Endogeneity occurs when the assumption that the errors are uncorrelated with the dependent variables is broken. This could happen because of measurement error, omitted variable bias or because of reverse causality between an explanatory variable and the independent variable. In this case the endogeneity problem arises with two variables: lagged infant mortality rate and ART coverage. I am trying to model the effect of the two last variables on infant mortality. It makes sense to think that countries that have higher infant mortality rate receive more of ART coverage to fight the higher incidence of perinatal transmission.
When endogeneity is present, ordinary least squares can produce biased and inconsistent estimates. The presence of one endogenous variable distorts all of the coefficients of the model. In order to correct for endogeneity we need to look for an alternative covariate as a proxy for the endogenous covariate. A common approach is to lag the endogenous variable by one period or more. This is what McDonald and Roberts (2006) do when including the lagged infant mortality rate in the reduced health equation. This is simple to implement yet it comes at the cost of ease in interpretation of the lagged coefficient and decreased precision in some cases. Unfortunately, there is not empirical method of gauging the severity of the endogeneity or the efficacy of introducing the lagged variable. This brings us to the second method of dealing with endogeneity, instrumental variable regression.

### 7.2 Instrumental Variable Regression – Two Stage Least Squares

An alternative for dealing with endogeneity is using instrumental variable regression. The method in essence consists of obtaining estimates of the potentially endogenous covariate using an instrumental variable. The essential part of this method is to find a genuinely exogenous instrumental variable that is correlated with the endogenous covariate but is only related to the dependent variable through the endogenous covariate. After obtaining the estimates, since they were derived from the exogenous instrumental variable, will not present the problem of endogeneity that the instrumented covariate had.

This method is more rigorous and transparent than lagging the independent variable and it allows for empirical tests for the validity of the instruments being used. If a strong instrument is used, the estimates can be interpreted similarly to OLS and be
consistent, unbiased and efficient. When using the TSLS (two stage least squares) models, three tests should be conducted to assess the severity of the bias due to the endogenous regressor, the relevance of the instrument used (whether it is sufficiently correlated with the endogenous regressor), and the exogeneity of the instrument (to what degree it is uncorrelated with the main equation residuals).

Since the estimation depends completely on the strength of the instruments used, I can regress the potentially endogenous variable on the exogenous instruments and check for their magnitudes and significance. They should have sensible signs and be significant for the instrument to be valid. A high F-statistic is desirable since it provides a test of joint significance.

The second concern that needs to be addressed is the degree of endogeneity that the suspect endogenous covariate has. I can use the Hausman test, where the null hypothesis is that the regressor is exogenous to assess for endogeneity. If the test rejects the null at the 10% level, there is need to be concerned about endogeneity of the suspect covariate. The intuition behind the Hausman test is equivalent to running the first regression where the endogenous covariates estimates are obtained and save the residuals. Then we should include the residuals as a covariate in the main equation and assess their significance. I am looking for the correlation between the dependent variable and the residuals of the first stage regression.

7.3 Fixed vs. Random effects – Accounting for country heterogeneity

The third econometric concern mentioned previously is that country specific differences need to be accounted for in order to obtain reliable estimates in my regressions. From a theoretical point of view, I should expect that the inclusion of country specific
effects in my model is sensible given the panel nature of the data and the heterogeneity in the countries that compose it. Fixed effects models should be used when each individual in the panel set of data has inherent characteristics that must be accounted for in order to obtain reliable estimates. In order to use a fixed effects model, I am assuming that the differences between countries are time invariant and uncorrelated between one another. These should be used when looking at different individuals over time. A random effects model assumes that the variation across entities is random and not correlated with the independent variables in the model. The random effects model assumes that the individual’s error term is not correlated with the predictors, allowing time invariant variables to play a role as explanatory variables.

In order to formally test if I should use random or fixed effects models, I can conduct a Hausman test where the null hypothesis is that the random effects model is preferable over the fixed effects model. In order to conduct the test, I run a fixed effects model then save the estimates, and proceed to do the same with the random effects model. The saved estimates are used to run the Hausman test.

8) Estimation

8.1 Health Equation

a) Testing for Instrument Relevance

The introduction of the price of ART as the instrument to obtain theoretically endogeneity free estimates of the endogenous regressor ART provision calls for some tests. The tests aim to evaluate the strength, relevance and validity that will assure that the TSLS estimation presents unbiased coefficients.
The first step is to test the relevance of the instruments being used. To do this, I run a regression of the endogenous regressor by all the exogenous regressors that will be used in the health equation. Table 4 presents the results of the regression.

**Table 4. Regression Results for Instrument Relevance Testing**  
*Dependent Variable: ln(ART Provision)*

<table>
<thead>
<tr>
<th>Variables</th>
<th>Estimate</th>
</tr>
</thead>
<tbody>
<tr>
<td>ART Price</td>
<td>-10.082 (-0.01)***</td>
</tr>
<tr>
<td>HIV Prevalence</td>
<td>-0.313 (0.03)***</td>
</tr>
<tr>
<td>ln(DPT)</td>
<td>0.301 (0.07)*</td>
</tr>
<tr>
<td>ln(Protein)</td>
<td>2.864 (0.07)*</td>
</tr>
<tr>
<td>Intercept</td>
<td>0.934 (0.12)</td>
</tr>
</tbody>
</table>

*p-values reported in parenthesis*  
*R-Squared: 0.56  Observations: 443*

The results of Table 4 show that the choice of ART price as an instrument is appropriate. The variable is significant and as expected has a negative sign. The magnitudes are sensible. The other exogenous variables are significant at the 5 and 10% level. The F-statistic of the model (12.33) indicates that the exogenous variables will serve as good instruments for the estimation.

**b) Fixed vs. Random Effects – Time Specific Effects**

The nature of the data I am using suggests that I should include country fixed effects to account for country heterogeneity. I can formally test this by running a Hausman test
between the fixed effects and the random effects model. The Hausman test’s p-value is 0.00, which ratifies the use of fixed effects as a sensible choice.

The next step is to assess the need for the inclusion of time effects in the fixed effects model. For this I can include the time dummies in the fixed effect equation and perform a f-test. The null hypothesis if this test is that the time dummies are not jointly significant. That means that with a p-value of less than 0.10, I reject the null hypothesis and conclude that time effects should be included in my model. The p-value for the test is 0.07, which supports the inclusion of time specific effects.

c) Health Equation Estimation and prediction of Health Capital

Given the specification of my model, the results of the health capital equation estimation are presented in Table 5 below. Only the coefficients that were statistically significant at the 10% level are presented.

The variables of interest have expected signs. ART provision therapy is negative and significant; the coefficient indicates that an increase of 1% in the provision of ART is associated with a decrease of 0.157% in infant mortality. As expected, increases in HIV prevalence are associated with increases in infant mortality. An increase of 1% in HIV prevalence is associated with a 0.21% increase in infant mortality. A percent increase in protein intake is associated with a 0.20% decrease in infant mortality. Similarly, DPT immunization decreases infant mortality by 0.3%. The coefficient for primary education is statistically significant at the 10% and was considered when estimating health capital for the structural growth equation estimation.
Table 5. Regression Results for Two Stage Least Squares Regression Health Capital Estimation
Dependent Variable: ln(Infant Mortality Rate)

<table>
<thead>
<tr>
<th>Variables</th>
<th>Estimate</th>
<th>Variables</th>
<th>Estimate</th>
</tr>
</thead>
<tbody>
<tr>
<td>ln(Infant Mortality_{t-1})</td>
<td>0.401 (0.00)***</td>
<td>ln(DPT)</td>
<td>-0.290 (0.05)**</td>
</tr>
<tr>
<td>HIV Prevalence</td>
<td>0.210 (0.04)**</td>
<td>ln(Protein)</td>
<td>-0.197 (0.03)**</td>
</tr>
<tr>
<td>ln(GDP)_{t-1}</td>
<td>-0.227 (0.02)**</td>
<td>ln(PrimaryEduc)</td>
<td>-0.085 (0.09)*</td>
</tr>
<tr>
<td>ln(ART)</td>
<td>-0.057 (0.03)**</td>
<td>Constant</td>
<td>3.891 (0.00)***</td>
</tr>
</tbody>
</table>

Instrumented: ln(Infant Mortality_{t-1})
Instruments: All explanatory variables + ln(ART Price) + ln(Infant Mortality_{t-3})

*significant at 10%  **significant at 5%  ***significant at 1%

P-values reported in parenthesis
R-Squared: 0.89  Observations: 443

The results in Table 5 can be compared to the results of a naïve regression that uses ordinary least squares and does not account for the endogeneity problem. The results of this regression are shown in Appendix 3. The problem of endogeneity is obvious given the coefficients observed in Appendix 3. The regression results of the naïve regression indicate that increases in GDP, DPT immunization, ART, education and protein intake are associated with an increase in infant mortality rate. The unexpected signs of the coefficients of the naïve regression, and the significance of some indicate that it is crucial to correct for the endogeneity that exists between ART provision and infant mortality rate.
8.2 Structural Growth Equation

After having estimated the predicted health capital from the health equation, the last component of the structural growth equation is available. The process of estimation of the structural growth equation is similar to the health equation because of the presence of lagged variables in the model and potential endogeneity concerns between the health capital term and income per capita. The results are presented in Table 6 below.

The structural growth equation estimation indicates that the coefficient for investment is positive and significant as expected. The positive and significant sign of the lagged income per capita term indicates that convergence is taking place, with convergence rate given by the parameter $\lambda$. The coefficient for “capital widening/workforce growth” $\ln(n+g+d)$ is positive but not significant. Unexpectedly, the primary school coefficient is not significant at the 5% level. The health capital is negative and significant at the 10% level and its significance does not change when excluding the health capital from the equation.

Once I have obtained the coefficients, it is possible to calculate the marginal impact of HIV on economic growth. From Table 5, the coefficient for HIV prevalence is 0.210, which means that a 1% increase in HIV prevalence is associated with a 0.21% increase in infant mortality. From Table 6, the coefficient for the estimated infant mortality is -0.820. Since the coefficient for HIV is the marginal increase in infant mortality rate, I can multiply the coefficient with the infant mortality estimate coefficient to obtain the reduction in GDP proportional to a 1% increase in HIV prevalence. This is the number shown in the HIV impact row of Table 6. The same principle can be applied to obtain the effect of a marginal increase in ART provision on GDP growth. The results show that an increase of 1% in HIV
prevalence decreases GDP per capita by 0.18%. An increase of 1% in ART provision increases GDP per capita growth by 0.048%.

### Table 6. Regression Results for Structural Growth Equation

**Dependent Variable: ln(GDP per capita)**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Estimate</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\ln(\text{GDP/\text{Capita}})_{t-1}$</td>
<td>1.033</td>
</tr>
<tr>
<td></td>
<td>(0.00)***</td>
</tr>
<tr>
<td>$\ln(\text{Investment})$</td>
<td>0.440</td>
</tr>
<tr>
<td></td>
<td>(0.01)***</td>
</tr>
<tr>
<td>$\ln(n+g+d)$</td>
<td>0.139</td>
</tr>
<tr>
<td></td>
<td>(0.23)</td>
</tr>
<tr>
<td>$\ln(\text{Primary School})_{t-1}$</td>
<td>0.330</td>
</tr>
<tr>
<td></td>
<td>(0.09)*</td>
</tr>
<tr>
<td>$\text{Health Capital Est.}$</td>
<td>-0.820</td>
</tr>
<tr>
<td></td>
<td>(0.03)**</td>
</tr>
<tr>
<td>$\lambda$</td>
<td>0.015</td>
</tr>
<tr>
<td>$\text{HIV Impact}$</td>
<td>-0.175</td>
</tr>
<tr>
<td>$\text{ART Impact}$</td>
<td>0.048</td>
</tr>
</tbody>
</table>

*significant at 10%  **significant at 5%  ***significant at 1%

p values reported in parenthesis

R-Squared: 0.98  Observations: 443

Hausman Test p value: 0.03 – Fixed effects are appropriate

The coefficient of HIV prevalence implies that a country with HIV prevalence of 20% has a 3.6% reduction in GDP per capita in a given year. Bonnel (2000) estimates the effect to be 2.6% using the same 20% prevalence benchmark. One potential reason for the
difference in the estimates is that I use different theoretical approaches. Also, I incorporated more recent data of HIV prevalence that better reflects the effects of the epidemic over time given the relatively slow progression of HIV to AIDS.

McDonald and Roberts (2006) estimate that a 1% increase in HIV prevalence decreases GDP per capita by 0.59%. It is possible that this is overestimating the effect of HIV on GDP per capita given the omission of both ART and DPT immunization in the health equation. My estimate of the effect of AIDS in the economy varies from the other literature because of the different theoretical approach that I take. It would be ideal to have more data on male circumcision rates to be able to obtain more accurate estimates of HIV prevalence, yet this information is extremely limited and not readily available.

9) Discussion

The results obtained from the health capital are consistent with what was expected. The decision to include DPT immunization provision and ART provision variables in the health equation seems to be sensible given the statistical significance and the expected signs of the variables. However, the magnitude of the ART coefficient could be misleading.

The statistical significance and expected negative sign of the lagged GDP indicate that increases in income are related to improvements both in financial and social determinants of health. It is important to mention that the interpretation of the lagged GDP per capita could be misleading because it does not address important income distribution issues. Asymmetric distribution of income should be considered as a potential limitation of the interpretation of the results presented. The insignificant coefficient of primary education in the health equation is surprising. To check for this, the estimation was also
done using secondary education. The coefficient using secondary education was also insignificant at the 5% level. As expected the positive sign of the education coefficient indicates that increases in primary schooling decrease infant mortality. This is supported by the increased income derived from human capital accumulation and its subsequent impact on the health of the individual.

Similarly, the coefficient for education human capital (ln(primary schooling)) is insignificant at the 5% level in the structural growth equation. The insignificance of the coefficient could be because of the choice of a Solow growth based economy. The developing economies are still constrained by agricultural production, which means that savings could not be an appropriate indicator of economic growth. Exploring this approach with a different model choice could produce interesting comparisons.

10) Conclusion

In order to quantify the impact of ART on economic growth, I defined a health capital equation that included lagged income, nutrition, education and DTP immunization to indirectly link HIV prevalence and ART to economic growth. Given the potential endogeneity problem between ART provision and infant mortality rate, I used price to instrument ART provision and included the endogeneity-free estimates in the health equation estimation. The health equation infant mortality estimates are then used as a proxy for human capital in a second structural growth, Solow-based growth equation. The structural growth equation includes physical, educational and health capital and it is used to estimate the effect how marginal increases in the different type of capitals affect growth. Through this mechanism, I calculate a marginal increase in HIV prevalence reduces GDP
per capita by 0.175%. Also, an increase of 1% in ART provision is associated with a 0.048% reduction in the effect that HIV has on GDP per capita. I argue that the magnitude of ART impact is not completely reflecting the positive effects it has when reducing the effect of the HIV/AIDS epidemic on economic growth.

The long run advantages of providing ART will be substantially greater because ART not only reduces immunodeficiency related illness but also decreases the probability of transmission. This means that we will not only continue to see a decrease in the incidence over time given sustained that sustained ART provision is held, but also an increase in the number of people living with HIV as AIDS related deaths decline.

11) Bibliography


Appendix 1 – Theoretical Derivation

Starting with the intensive form of the Cobb-Douglas production function

\[ y_{it} = A_{it}^{\alpha} L_{it}^{\beta} H_{it}^{\gamma} \]  

(A1)

Where the variables written in lower case stand for quantities per effective unit of labor.

We assume that the labor force grows at rate \( n_i \) (country specific) and that technology grows at a rate \( g_t \) (time specific). Both rates of growth are exogenously determined

\[ L_{it} = L_i \exp(n_i t) \]  

(A2)

\[ A_{it} = A_t = A_0 \exp(g_t t) \]  

(A3)

The accumulation of physical, education and health capitals is determined by:

\[ K_{it} = I_{it} + (1 - \delta)K_{i,t-1} \]

\[ E_{it} = I_{it} + (1 - \delta)E_{i,t-1} \]  

(A4)

\[ H_{it} = I_{it} + (1 - \delta)H_{i,t-1} \]

In the system of equations (A4) we assume that physical, education and health capital depreciate at the same rate \( \delta \). The equations above imply that the evolution of each type of capital is determined by the investment in each type of capital at period \( t-1 \) added to the already existing capital in the same period accounting depreciation.

Savings are divided between investment in capital, education and health. These activities are treated as an investment activity such that:

\[ s_{it} = s_{it}^K + s_{it}^E + s_{it}^H = \frac{I_{it}}{Y_{it}} = \frac{I_{it}^K + I_{it}^E + I_{it}^H}{Y_{it}} \]  

(A5)
Since Equation (A5) implies that savings is a fraction of total income. The evolution of output per unit of effective worker is then determined by:

\[
\hat{k} = s_{it}^K y_{it} - (n_i + g_i + \delta) \hat{k}_{it} \\
\hat{e} = s_{it}^E y_{it} - (n_i + g_i + \delta) \hat{e}_{it} \\
\hat{h} = s_{it}^H y_{it} - (n_i + g_i + \delta) \hat{h}_{it}
\]  

(A6)

Assuming the existence of a steady state \((\alpha + \beta + \psi < 1)\), system of equations (A6) would imply that:

\[
\hat{k}^* = \left[ \frac{(s_{i}^K)^{1-\beta-\psi} (s_{i}^E)^{\beta} (s_{i}^H)^{\psi}}{n_i + g_i + \delta} \right]^{1/(1-\alpha-\beta-\psi)}
\]  

(A7)

\[
\hat{e}^* = \left[ \frac{(s_{i}^K)^{\alpha} (s_{i}^E)^{1-\alpha-\psi} (s_{i}^H)^{\psi}}{n_i + g_i + \delta} \right]^{1/(1-\alpha-\beta-\psi)}
\]  

(A8)

\[
\hat{h}^* = \left[ \frac{(s_{i}^K)^{\alpha} (s_{i}^E)^{\beta} (s_{i}^H)^{1-\alpha-\beta}}{n_i + g_i + \delta} \right]^{1/(1-\alpha-\beta-\psi)}
\]  

(A9)

Where the asterisk stands for the steady state values of the different types of capital. Substituting (A7) to (A9) and (A3) in (A1) and taking the natural logarithms yields:

\[
\ln \hat{y}_{it} = \ln A_0 + g_i t - \frac{\alpha + \beta + \psi}{1 - \alpha - \beta - \psi} \ln (n_i + g_i + \delta) + \frac{\alpha}{1 - \alpha - \beta - \psi} \ln s_i^K \\
+ \frac{\beta}{1 - \alpha - \beta - \psi} \ln s_i^E + \frac{\psi}{1 - \alpha - \beta - \psi} \ln s_i^H
\]  

(A10)

Equation (A10) is Equation (3) in the main text.
## Appendix 2 – Countries used in the analysis

### Countries

<table>
<thead>
<tr>
<th>High HIV Prevalence (&gt;6%)</th>
<th>Low HIV Prevalence (&lt;6%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Botswana</td>
<td>Angola</td>
</tr>
<tr>
<td>Central African Republic</td>
<td>Benin</td>
</tr>
<tr>
<td>Kenya</td>
<td>Burundi</td>
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<td>Cameroon</td>
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<td>Chad</td>
</tr>
<tr>
<td>Malawi</td>
<td>Comoros</td>
</tr>
<tr>
<td>Namibia</td>
<td>Dijibouti</td>
</tr>
<tr>
<td>South Africa</td>
<td>Equatorial Guinea</td>
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<td>Swaziland</td>
<td>Eritrea</td>
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<td></td>
<td>Sudan</td>
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<tr>
<td></td>
<td>Ethiopia</td>
</tr>
</tbody>
</table>

12 17
Appendix 3 - Ordinary Least Squares Analysis

Given the model specification used by McDonald and Robertson (2006), the first naïve OLS regression below includes all the covariates, the lagged covariates, the squared covariates and the interactions of GDP with some of the covariates. The regression results using infant mortality rate are presented below. The table contains the regression using the main covariates without the lags, squared terms and interaction terms.

**Regression Results for Naïve OLS**

*Dependent Variable: \( \ln(\text{Infant Mortality Rate}) \)*

<table>
<thead>
<tr>
<th>Variables</th>
<th>Estimate</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \ln(\text{Infant Mortality}_{t-1}) )</td>
<td>1.057 (42.63)***</td>
</tr>
<tr>
<td>HIV Prevalence</td>
<td>-0.002 (2.84)***</td>
</tr>
<tr>
<td>( \ln(\text{GDP}) )</td>
<td>0.004 (0.56)</td>
</tr>
<tr>
<td>( \ln(\text{ART}) )</td>
<td>0.012 (2.74)**</td>
</tr>
<tr>
<td>( \ln(\text{CPT}) )</td>
<td>-0.0155 (-3.73)***</td>
</tr>
<tr>
<td>( \ln(\text{Protein}) )</td>
<td>0.006 (0.28)</td>
</tr>
<tr>
<td>( \ln(\text{DPT}) )</td>
<td>0.0155 (0.32)</td>
</tr>
<tr>
<td>( \ln(\text{SecEduc}) )</td>
<td>0.031 (1.98)*</td>
</tr>
<tr>
<td>Intercept</td>
<td>-0.488 (-1.73)*</td>
</tr>
</tbody>
</table>

*significant at 10%  **significant at 5%  ***significant at 1%
t-statistics reported in parenthesis
R-Squared: 0.99 Observations: 443