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Health and Growth: Causality through Education

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Abstract

**Purpose**--The paper theoretically and empirically investigates the impact on human capital investment decisions and income growth of lowered life expectancy as a result of HIV/AIDS and other diseases.

**Design/methodology/approach**--The theoretical model is a three-period overlapping generations model where individuals go through three stages in their life, namely, young, adult and old. The model extends existing theoretical models by allowing the probability of premature death to differ for individuals at different life stage, and by allowing for stochastic technological advances. The empirical investigation focuses on the effect of HIV/AIDS on life expectancy and on the role of health on educational investments and growth. We address potential endogeneity by using various strategies, such as controlling for country specific time-invariant unobservables and by using the male circumcision rate as an instrumental variable for HIV/AIDS prevalence.

**Findings**--We show theoretically that an increased probability of premature death leads to less investment in human capital, and consequently slower growth. Empirically we show that HIV/AIDS has resulted in a substantial decline in life expectancy in African countries and these falling life expectancies are indeed associated with lower educational attainment and slower economic growth world wide.

**Originality/value**--The theoretical and empirical findings reveal a causal link flowing from health to growth, which has been largely overlooked by the existing literature. The main implication is that health investments, that decrease the incidence of diseases like HIV/AIDS resulting on increases in life expectancy, through its complementarity with human capital investments lead to long run growth.

**Keywords**: HIV/AIDS, Africa, life expectancy, growth, overlapping generations.

**JEL classification**: I18;I20;O15
I. Introduction

Better health may lead to increased interest in education and education could also give rise to opportunities and choices that result in better health. Traditionally, economists have emphasized the second aspect of the relationship between health and education. This paper in contrast focuses on the impacts of health on education and economic growth. Investigating this relationship is especially illuminating in understanding development in Africa, where the HIV/AIDS epidemic has greatly reduced life expectancy.

The objective of this paper is two-fold. First we model theoretically how the HIV/AIDS epidemic, through its effects on the probability of premature death, impacts educational investment and growth. Then we empirically test the implications of our model and quantify the impacts of HIV/AIDS on educational attainment and growth. We develop a three-period overlapping-generation model with learning and technology adoption that links increased mortality and shortened life expectancy to the human capital investment decisions of representative agents. The propositions derived from this model are tested with a series of cross-sectional and panel analyses, explicitly accounting for potential endogeneity by using the instrumental variable approach based on predetermined variables.

In the following, we briefly review recent literature. Then we describe the theoretical model and report the empirical tests of hypotheses suggested by the model. In addition, we examine the effect of HIV/AIDS on life expectancy in African countries using an instrumental variable approach. The final part of the paper is a conclusion that summarizes the policy implications of the analysis.

II. Literature

Most discussions of the relationship between health and education assume that the main direction of causality has been from education to health (Grossman, 1973). For a

Only limited research has examined the role of health in economic growth, most of it theoretical. Ehrlich and Lui (1991) develop an overlapping-generations model of endogenous growth in which human capital is the engine of growth and the generations are linked through material and emotional interdependencies within the family. Swanson and Kopecky (1999) studied the effect of increasing life expectancies on human capital accumulation finding that increasing length of life not only encourages individuals to invest in more education prior to entering the work force but also to continue acquiring new knowledge and skills as adult workers. Kalemli-Ozcan, Ryder and Weil (2000) studied the relation between increased life expectancy and human capital investment during periods of economic growth in a continuous time, overlapping-generations model. They find that increasing life expectancies give rise to greater demand for education as well as increased consumption of other goods. Technology is assumed, however, to be stagnant in their model. Bils and Klenow (2000) investigate the empirical correlation between growth and education and suggest that part of the correlation between schooling and growth is accounted for by the effect of growth on education.

Kalemli-Ozcan (2002) analyzed theoretically the effects of declining mortality rates on fertility, education, and growth. She finds that economic growth is promoted as a result of declining mortality. Bloom and Canning (2000, 2005) argue that health is a form of human capital and therefore an input into the growth process, as well as an output. They find from their model that, improvements in longevity lower fertility, raise educational investment, and raise the long run growth rate of output. Bell et al. (2006) also examined the effect of an AIDS-type increase in premature adult mortality on the inter-generational transmission of human capital. Based on an over-lapping generation model, they show that an otherwise
growing economy could decline to a low-level subsistence equilibrium if hit with an AIDS-type increase in premature adult mortality.

There is a growing number of empirical cross-country studies on how HIV/AIDS affects macroeconomic variables. Bloom and Mahal (1997) failed to find that HIV/AIDS was a significant factor in slower growth rates in African countries during the period 1980-1992, when HIV/AIDS was considerably less widespread. In contrast, Bonnel (2000), using more recent data, found that this single disease resulted in a reduction in per capita growth in Africa of 0.7 percentage points. McDonald and Roberts (2006) estimate a Solow growth model with health and educational capital as well as physical capital and effective labor as explanatory variables. They found substantial impacts of HIV/AIDS on health and growth but did not find educational capital to be significant in their growth rate equation.

In this study, we model a particular mechanism through which HIV/AIDS affects growth rates, namely, the reduction in investments in human capital that is expected to result from lower returns to such investments when life expectancy declines. The theoretical model is a discrete-time, overlapping-generation model in which individuals learn from the previous generation and make schooling investment decisions, knowing that the human capital acquired from educational investments will facilitate technological adoption in a later period.

Our theoretical model departs from the previous work in three important ways. First, we build an extra period into the usual two-period overlapping generation model, which allows us to model the varying probability of premature death in human life cycles and the successive decisions of investing in education when one is young and adopting technologies when one starts to work. This is important for HIV/AIDS as HIV/AIDS tends to affect the life expectancies of children and adults in different ways. Secondly, human capital is two-dimensional in the model, reflecting both its quality and ease of adoption. Thirdly, the model includes exogenous technological advances.
The results show that a higher probability of premature death will reduce investment in human capital. This reduced investment means that economic growth is lower even though the rate of technical innovation is unchanged. We test the implications of the theoretical model in two steps. First, we test whether shorter life expectancy or higher probability of premature death leads to a decline in human capital investment and economic growth with both cross-sectional and panel regressions. In specifying the regression equations, we use health variables from 25 years ago that correspond to the health level when the current generation started to make lifetime human capital investment decisions. This approach also circumvents the potential endogeneity problem that would arise if current health variables are used. Second, we focus on African countries and examine how life expectancy in Africa is affected by the HIV/AIDS pandemic. We use the male circumcision rates compiled by Ahuja, Wendell, and Werker (2007) as instrumental variables to deal with the endogeneity of HIV/AIDS prevalence rates and life expectancy. The results clearly show that a falling life expectancy has significant adverse effects on human capital investment and the economic growth rate.

III. A Three Period Overlapping Generations Model.

Kim and Lee (1999) analyze the effects of technology change on growth rates of income and human capital. Their model includes two dimensions of human capital, referred to as width and depth. Human capital width represents flexibility, adaptability, and the influence of human capital on the adoption of new technologies. Width determines the adoption cost of a new technology. Human capital depth measures the quality of the human capital stock. They find that an increase in technological uncertainty decreases growth rates of income and human capital by lowering efficiencies both in creating new knowledge and in adopting new technologies.
In studying the impacts of health on educational investments, we extend Kim and Lee’s model to three periods and introduce the probability of dying prematurely. The extension to a third period can be justified by the likelihood that the probability of death is not constant throughout an individual’s life as shown by Kalemi-Ozcan, et al. (2000). In addition, there is abundant empirical evidence that AIDS results in higher probabilities of dying for young adults who are more sexually active than for other age groups. Hence we build a three-period model to allow for different age-specific probabilities of dying.

In this model, a representative agent lives at best for three periods, referred to as youthful, adult and mature. When young, the agent divides her time between work and education. As an adult, she allocates her available time to work and technology adoption. When mature, she devotes all of her time endowment to working.

The agent faces a probability of dying at the end of the 1st period and 2nd period, denoted by $P_Y$ and $P_A$ respectively. The relationship between the two probabilities is

$$(1) 1 - P_A = P(l \geq 2, l \geq t) \cdot (1 - P_Y) = P^* \cdot (1 - P_Y)$$

where $l$ denotes life expectancy of the agent, $t$ denotes one period of time, $P^*$ denotes the conditional probability of surviving to the end of the second period if the agent doesn’t die at the end of first period. Note that if $P^*=0$, the agent lives two periods at most.

A new and advanced technology A is assumed to occur with a probability of $p$ in each period. New technologies may differ in their characteristics. It is assumed that potential technologies can be arrayed along a continuum running from 0 to S and that a new technology might occur with characteristics that place it anywhere on this continuum.

Adult agents may adopt a new technology when a technology shock occurs. An agent may have several ‘knowledge points’ distributed along the technology space [0, S]. To adopt
a new technology, the agent must have a knowledge point located close to the point in \([0,S]\) which characterizes the technological innovation. The more knowledge points an agent has, the more likely it is that the technological innovation will lie close to one of his knowledge points, reducing the cost of adoption.

The initial structure of the agent’s human capital consists of two dimensions: width and depth. Width is represented by the number of knowledge points, \(N\), possessed by the agent in the interval \([0,S]\). The greater the width, the more knowledge points the agent has and the more likely it will be that the technological innovation will be adopted. Depth represents the quality of human capital. It is assumed that the depth of an agent’s human capital determines the level of technology that can be adopted. The agent cannot adopt a new technology if it is beyond the depth of her human capital, \(Q\).

An agent is assumed to need time \((l_A)\) to realize the technology adoption:

\[
(2) l_A = a |x - s| A
\]

where \(a\), the adoption cost, is a parameter indicating adoption efficiency, \(s\) denotes the location of an agent’s knowledge point closest to point \(x\) in \([0,S]\) which characterizes the innovation. The technical adoption, thus, requires more time as the level of the technology increases and as the distance between an agent’s own knowledge point and the location of the technology on \([0,S]\) increases. Because the agent’s human capital depth determines the level of technology she can embrace, the adoption time can be written as

\[
(2') l_A = a |x - s| Q
\]

To minimize expected adoption cost, the \(N\) knowledge points must be uniformly distributed over the knowledge space. The reason equal spacing represents the best strategy is...
because the characteristics of a technical innovation is a random variable uniformly distributed on the technological space \([0, S]\).

After an adult agent adopts a new technology with a level of quality of \(Q\), the depth or quality of the agent’s human capital becomes \(Q\).

\[
(3) \quad H_{at} = Q_t
\]

where \(H_{at}\) is the human capital stock of an adult at time \(t\) and \(Q_t\) is the agent’s depth of human capital at period \(t\). It is also assumed that there are spillover effects so that a certain fraction of the technology, once adopted and in use, can also be used by young agents without cost. Therefore, the young agents’ specific human capital at time \(t\) becomes

\[
(4) H_{yt} = \delta Q
\]

where \(\delta\) denotes the current adult generation’s amount of specific human capital and \(0 < \delta \leq 1\), is the spillover effect.

A youthful agent invests an amount of time \(l_E\) in education and builds his human capital stock of \(NQ\):

\[
(5) N_t Q_t = b \cdot l_E \cdot \overline{NQ}
\]

where \(l_E\) is the time the youthful agent devotes to education and \(\overline{NQ}\) is the human capital owned by the current adult generation. The parameter \(b\) captures the efficiency of the elder generation in passing its knowledge to the next cohort. This specification implies that youthful agents accumulate education by spending time in school and paying tuition to the adults if no new technology occurs for the current adult generation to compensate for the instructors’ opportunity cost. Here we assume that only the individuals with the highest available depth of human capital in a period can be instructors.
The next step is to determine the human capital stock of a mature agent. If the current mature agent adopted technology in his adult period and no new technology occurs during the period when he is mature, his human capital stock defines the highest level of human capital of the society. We therefore assume his human capital to be \((1 - \tau_1)Q\), where \(\tau_1\) is a depreciation rate in \([0,1]\). If he adopted in his adult period, but new technology occurs when he is mature, his human capital stock becomes \((1 - \tau_2)Q\), where \(\tau_2 < \tau_1\) is also a depreciation rate. If he had not adopted technology as an adult, but new technology occurred in his mature period, then he, like the youthful agent, enjoys a spillover effect from the human capital of the current adult. If no new technology occurred in either his adult or mature period, all three generations have to share the human capital stock that spilled over from the cohort that has passed away, i.e., \(\delta Q\). If new technology does not occur in his adult period but does occur in his mature period, his human capital is also the spillover effect of the current adult generation, \(\delta Q\). It is assumed that the spillover effect is not as large as the individual’s own human capital (depreciated). Therefore, the agent is always better off adopting his own new technology whether his technology is outdated by new technology or not.

The representative firm employs youthful, adult and mature workers together. The input is human capital only, and the technology is linear, which implies that the human capital of each of the three generations is a perfect substitute for that of the others:

\[
(6) \quad y_t = H_{yt} \cdot (1 - l_E) + H_{at} \cdot (1 - l_A) + H_{mt}
\]

where \(y_t\) is the total output of the economy at period \(t\), \(H_{yt}\) is the human capital possessed by the young generation at period \(t\), \(H_{at}\) and \(H_{mt}\) are that of the adult and mature generations respectively. A representative young agent’s maximization problem on the width and depth of her human capital is:
\[
(7) \quad \max_{N,Q} U(c_{yt}, c_{at}, c_{mt}) = \log c_{yt} + \frac{1}{1 + \rho} E(\log c_{at}) + \frac{1}{(1 + \rho)^2} E(\log c_{mt})
\]

where \( U \) is lifetime expected utility, which depends on consumption in the three periods, respectively denoted by \( c_{yt}, c_{at} \) and \( c_{mt} \). The solutions from the above equation for \( Q \) and \( N \) are given by (see Appendix 1 for details):

\[
(8) Q = \sqrt{\frac{2(1 - z^*)bp(1 - P_y)(1 + k)NQ}{(2 + 2\rho + (1 + k)p(1 - P_y))aS}}
\]

\[
(9) N = \sqrt{\frac{abp(1 - P_y)(1 + k)SNQ}{2(1 - z^*)[2 + 2\rho + p(1 - P_y)(1 + k)]}}
\]

where \( k = \frac{P_y}{(1 + \rho)} \), and \( z^* \) is the equilibrium ratio of depth to width given the characteristic space of the innovation that has occurred. It is a constant between \([0.42, 0.2]\) and it declines as \( k \) increases.

The comparative static results are:

\[
(10) \quad \frac{\partial N}{\partial (1 - P_y)} > 0 \quad \text{and} \quad \frac{\partial Q}{\partial (1 - P_y)} > 0
\]

\[
(11) \quad \frac{\partial N}{\partial P_y} = (1 - z^*)(2 + 2\rho) + (1 + k)[2 + 2\rho + p(1 - P_y)]\frac{\partial z^*}{\partial k} < 0
\]

\[
(12) \quad \frac{\partial Q}{\partial P_y} = (1 - z^*)(2 + 2\rho) - (1 + k)(2 + 2\rho + (1 + k)p(1 - P_y))\frac{\partial z^*}{\partial k} > 0
\]

In equilibrium, \( N, Q \) and income grow at the same rate. In this economy, only adults adopt new technology, therefore, all growth in production comes from the current adults’ human capital stock compared with their human capital stock in the youthful period. The equilibrium is a balanced growth path. In particular, \( \frac{N}{N} = \frac{Q}{Q} = \frac{y}{y} = 1 + g \), where \( g \) is the growth rate with technological advance, i.e., the income growth of an adult (relative to his youth) if he adopts a new technology. From Equation (5), the education time of young agents is...
Putting this expression into the first-order conditions of the maximization problem, we have

\[
1 + g = \sqrt{\frac{p (1 - P_y) b (1 + k)}{2 (1 + \rho) + p (1 - P_y)}}
\]

It is easy to see that the growth rate increases with the probability of surviving the youthful period (1-\(P_y\)) and with the conditional probability of living through the three periods if one survives the youthful period \(P^*\). The expected growth rate of output is,

\[
E (1 + g) = (1 - P_y) \left\{ p p \left[ 1 + g + P^* (1 - \tau_2) \right] + 2 (1 - p) \delta + p (1 - p) \left[ 1 + g + P^* (1 - \tau_1) \right] \right\}
\]

The expected adoption time is therefore:

\[
E(l_s) = \frac{p (1 - P_y)}{S} \int_0^s a Q_y dy = \frac{p (1 - P_y)}{2} (1 - z^*)
\]

Since (1-\(z^*\)) increases with the conditional probability of living to the mature period if the individual survives the youthful period \(P^*\) then the adoption time increases with the increases in probability of technological advance (p), the probability of surviving the youthful period (1-\(P_y\)) and the conditional probability of living through the three periods if one survives the youthful period \(P^*\). Thus the growth rate of income decreases as the probability of premature dying increases. Equation (14) and equation (10) provide the main theoretical implications of this model:

1. The income growth rate increases with higher life expectancy. Therefore, there can be persistently different growth paths for countries with different life expectancies, even if the occurrence of technological progress is the same for all the countries. This dimension of the relationship between health, human capital investments and growth may be more significant
in places such as Sub-Saharan Africa where the HIV/AIDS pandemic may lead to severe reductions in life expectancy.

2. Adoption time increases with the increases in the probability of technological advance, the probability of surviving the youthful period, and the conditional probability of living through the three periods if one survives the youthful period. Therefore the growth rate of income decreases as the probability of premature dying increases, establishing the main result of this paper. Note that the immediate effect of an epidemic or a persistent war that drastically shortens people’s life expectancy is to reduce income due to loss of labor. These effects are not discussed in the model. Rather, our results show that in long-run equilibrium, slower growth results from a reduction in individual investments in human capital due to a shorter life span.

III. Empirical Analysis

The empirical section of this study is relevant to the broader literature on the impact of health on education and income. At the macroeconomic level, it is not clear whether causality runs from health to increased wealth, the reverse, or in both directions. Although many believe that improving health leads to increased wealth and economic growth (e.g. World Health Organization, 2001; Bloom and Canning, 2005; Weil, 2005), Acemoglu and Johnson (2006) suggest that improving health may have a negative impact on GDP per capita because increased life expectancies lead to larger total populations and lower per capita income unless the population growth is matched by per capita productivity increases. This study also contributes to an understanding of one of the prominent debates in the literature on the economic impacts of the HIV/AIDS pandemic. On one hand, HIV/AIDS reduces the current human capital stock because it predominantly affects adults and, as we will argue, it also decreases the incentives for the young to invest in human capital, thereby decreasing income. On the other hand, the pandemic might increase income per capita through positive pressure on wages (Young, 2005).
The theoretical section stresses a channel largely ignored by previous research through which HIV/AIDS or other epidemics could affect growth. We showed in a three-period overlapping-generations general-equilibrium model that a decrease in life expectancy will result in a reduction in human capital investment and consequently a lower growth rate. It is worth noting that although we are especially interested in understanding the consequences of the HIV/AIDS epidemic for income growth in Sub-Saharan Africa, the implications of our theoretical model go beyond HIV/AIDS extending to other health conditions or even political conditions that may lower life expectancy significantly in a population or in a sub-population. Moreover, the existence of such a channel does not necessarily mean that we will find statistically significant effects of HIV/AIDS on growth or reduction in education, because the theoretical model does not include many other socio-economic factors and mechanisms that can potentially reinforce or offset the causal effects of HIV/AIDS on human capital investment. On the other hand, there are other channels through which HIV/AIDS might affect human capital investment and therefore growth. For example, if treating HIV/AIDS exhausts a family’s financial resources, then HIV/AIDS will result in a reduction in human capital investment even if education is still deemed desirable. The purpose of the empirical analysis is to explore whether HIV/AIDS affects life expectancy and whether decreased life expectancy leads to a reduction in human capital investment. Because of the significance of other factors, however, the results of the empirical analysis can only be interpreted as suggestive.

Our empirical strategies are the following. In the reduced form regression and working at the country level of aggregation, we first establish the relationship between probabilities of premature death and human capital formation and then we examine how HIV/AIDS has affected these probabilities.

The data set for this analysis is a country panel compiled from several sources. Data on the prevalence of HIV/AIDS are from the CIA World Fact Book (2007) which includes 2001 and 2003 estimates for 168 countries. Information on income, education and general
health indicators is from both the World Bank’s 2006 World Development Indicators (WDI) database and a database developed by Barro and Lee (2000). In Table 1 we list the source of each of the variables used in the regressions. Male circumcision rates compiled by Ahuja, Wendell, and Werker (2007) are used as instruments to establish the link between HIV and general health. The specific countries included in each of the regressions are listed in Table 7.

(1) Impact of Health on Education Attainment

The theoretical model is based on the distinction between human capital depth and width. There are no empirical measures of these dimensions of human capital so this analysis relies on more broadly defined human capital measures, such as years of education (Barro and Lee, 2000). The number of years of education could be taken to represent human capital depth. One of the behavioral relationships from the model described above (equation (A9) in appendix) establishes that the time an agent devotes to education and technology adoption is negatively related to her probability of dying prematurely.

We specify the following panel regression to examine the effect of the probability of premature death on educational attainment:

\[
Educ_{i,t,(t+25)} = \alpha_0 + \alpha Edu_{i,t} + \beta \log(Health_{i,t}) + \gamma X_i + \theta C_i + \varepsilon_{i,t,(t+25)}
\]

where \( Educ_{i,t,(t+25)} \) is country \( i \)'s average schooling years in the total population over age 25 in the year of 1985, 1990, 1995 and 1999, and \( Edu_{i,t} \) is the same variable 25 years earlier, or roughly one generation earlier, in the year 1960, 1965, 1970, and 1975. \( \log(Health_{i,t}) \) is the logarithm of the country’s health indicator 25 years earlier. We adopt three alternative health indicators, namely, infant mortality rate, life expectancy at birth and crude death rate. \( X_i \) is a vector of country specific covariates that can affect education, including the logarithm of GDP per capita 25 years earlier, the interaction term between it and the health indicator, and measures of openness and investment 25 years earlier. The
inclusion of these covariates controls for initial differences among the countries, other than
the initial level of education that will affect the efficiency of the younger generation’s
learning process. $C_i$ is the country-specific fixed effect used to control for time-invariant
country-specific heterogeneity, such as geography, climate and culture. In effect, the country
fixed effects and country-specific covariates are proxies for the parameter $b$ in the theoretical
specification.

The regression results are reported in Table 2. The results are consistent with the
hypothesis that the probability of premature death, as reflected by the infant mortality rate,
the crude death rate and life expectancy at birth, has a highly significant negative impact on
human capital investment. Although the signs are the same as the cross-sectional results, the
magnitudes of the coefficient estimates in the panel regressions are larger.

(2) Health impact on income growth

The theoretical model indicates that an increase in the probability of premature death
will slow income growth through its effects on human capital investment. In this section we
examine whether and to what degree the income growth rate is affected by health. The
estimated equation does not provide direct evidence related to the hypothesis because we are
not able to isolate the effect of health on income growth solely through its impact on human
capital investments. Rather, this exercise will capture the net effect of health on income
growth, whether through human capital investment or through other channels. Both cross-
sectional and panel data regressions of the various health indicators on the GDP per capita
growth rate were estimated.

Similar to the regression of educational attainment on health, the dependent variables
are the logarithm of the per capita GDP growth rate during the periods 1960-1985, 1965-
the regressions for educational attainment. Specifically, the independent variables are the
logarithms of the three health measures, the logarithms of GDP per capita in constant 2000
dollars, and the logarithms of the net secondary enrollment rate from the World Bank\(^2\) for the years 1960, 1965, 1970, 1975 and 2000. We use country fixed effects to control for country-specific heterogeneity. Table 3 shows the results. The growth rates are negatively correlated with initial GDP per capita, and also initial education levels. A plausible explanation for this is that the growth rates of high-income economies are slower than those of lower-income countries because of convergence. There is also the possibility of simultaneous equations bias as education and income might be simultaneously determined. Using initial education and initial income as controls, the results show that the healthier countries are growing faster than countries with poorer health. Specifically, a one percent increase in the infant mortality rate or the crude death rate at the beginning of the period reduces the growth rate of income by about a half percentage point. In summary, we have found empirical evidence using panel regressions that initial health contributes significantly to the growth rate of income in subsequent periods. This indirectly supports the theory, as established in equation (14) that an increase in the probability of premature death leads to slower income growth due to a reduction in incentives for human capital investments.

(3) The Effect of HIV/AIDS on life expectancy or mortality

Sub-Saharan Africa has been particularly affected by the HIV/AIDS pandemic and has seen a dramatic decline in life expectancy, notably in the countries in the southern cone. According to a UN report on population and AIDS (2005), a child born in Zimbabwe, where about a quarter of the adult population is living with HIV, would have a life expectancy of 64 if there had been no HIV/AIDS pandemic, but of only 37 given the actual prevalence of HIV/AIDS.

Using the data provided by this UN report, we plot in Figure 1 the difference in life expectancy at birth of the African countries with and without AIDS against the HIV prevalence rate. The strong relationship between HIV/AIDS prevalence and life expectancy

\(^2\) We use the secondary enrollment rate as a proxy for education instead of Barro & Lee’s average schooling years because the information is available for more countries than the Barro & Lee measure.
reduction is clearly shown. Although Figure 2 shows a clear relationship between life expectancy and HIV prevalence rates, it does not identify causality, or the direction of causality. More specifically, we have to deal with potential endogeneity or reverse causality problems in our investigation of HIV/AIDS effects on health outcomes. Reverse causality arises when the causality flows from health outcomes to HIV/AIDS prevalence, and endogeneity arises when health outcomes and HIV/AIDS prevalence rates move together due to a third variable, such as income.

[Figure 1 inserted here]

To address these two identification issues, we adopt an instrumental variable approach. A valid instrumental variable should be correlated with the HIV/AIDS prevalence, but uncorrelated with unobservable variables. Such a valid instrument overcomes the endogeneity issue by isolating the variation in HIV/AIDS prevalence rates due to exogenous variation in the instrumental variable. We follow Ahuja, Wendell and Werker (2007) and use male circumcision rates as our instrumental variables.

To explore the relation between life expectancy in Sub-Saharan Africa and the incidence of HIV/AIDS, we use panel data for 38 African countries in the years 1980, 1985, 1990, 1995, 2000 and 2004. Information on the prevalence of HIV in the early years of the pandemic is sparse. In addition, according to Donnelly (2004), UNAIDS has revised some of its estimates of the incidence of HIV in Africa in light of new information. An original estimate that 15.0 percent of adults in Kenya were HIV positive was subsequently revised down to 9.3 percent in 2004 after it was discovered that a 2003 household survey showed HIV prevalence as low as 6.7 percent. Donnelly reported that UNAIDS bases its estimates on the best available information and frequently revises them as more complete information becomes available. Because UNAIDS advises against comparing HIV/AIDS prevalence estimates of different years directly (UNAIDS, 2008), we use a single-year observation of HIV prevalence, interacted with a year dummy, as a proxy for the actual HIV/AIDS infection rates in the sample countries. Ahuja, Wendell and Werker (2007) used 1997 infection rates
but we have chosen to use more recent observations, 2001 or 2003 depending on which year is available in the CIA World Fact Book 2007. Interaction terms between this infection rate and dummy variables for 1990, 1995, 2000, and 2004 are included to reflect the rapid spread of the pandemic during those years (see Ahuja, Wendell and Werker (2007)). It is assumed that the HIV infection rate was zero for 1980 and 1985.

The following system of equations is estimated:

\[
L_{it} = a_i + b_t + \beta_1 h i v_i 1990_t + \beta_2 h i v_i 1995_t + \beta_3 h i v_i 2000_t + \beta_4 h i v_i 2004_t + \gamma_1 X_i 1990_t + \gamma_2 X_i 1995_t + \gamma_3 X_i 2000_t + \gamma_4 X_i 2004_t + \epsilon_{it},
\]

where \(L_{it}\) is life expectancy in year \(t\) in country \(i\), \(a_i\) and \(b_t\) are the country and year intercept effects, \(h i v_i\) is the CIA HIV prevalence rate estimate for country \(i\) in 2001 or 2003, \(X_i\) is a vector of country specific control variables, including initial life expectancy and initial GDP per capita. All right hand side variables are interacted with years so that different slope effects are obtained for each year. The last term is a stochastic error. We use male circumcision rates compiled by Ahuja, Wendell and Werker (2007) as an instrument for the prevalence of HIV. Though it is not entirely clear through what mechanism a higher male circumcision could reduce HIV prevalence, the medical literature has established a causal effect of male circumcision rate on HIV prevalence, through three randomized trials in three African countries, namely, South Africa (Auvert et al. 2005), Kenya (Bailey et al. 2007) and Uganda (Gray et al. 2007). In fact, the World Health Organization and UNAIDS recommended that an important strategy for the prevention of heterosexually acquired HIV is to promote male circumcision rates (UNAIDS/WHO 2007). Male circumcision rate exhibited a high degree of variation across countries in the Africa, and the variation seems to be associated with religious, linguistic and cultural differences across these countries.
Breirova (2002) also uses circumcision rates as instruments to examine the impact of AIDS on education in Kenya.

In order to show that male circumcision is a valid instrument, one still has to determine that it is uncorrelated with the error term in the regression that might cause HIV prevalence and health outcomes to move together. Although it is not possible to check whether male circumcision rates are correlated with all possible omitted variables, we can check whether they are correlated with some of the most important omitted variables which are generally considered as preconditions for country level health, such as initial income, initial health status, and initial education at country level.

We plot out these important economic preconditions against male circumcision rate as compiled by Ahuja, Wendell and Werker (2007) in Figure 2, Figure 3 and Figure 4.

[ Figure 2, Figure 3, Figure 4 inserted here.]

From these plots, there is no obvious correlation between these initial economic and health preconditions and male circumcision rates. Therefore, we conclude that the male circumcision rate can serve as a valid instrument in the regression equation (16).

Table 5 summarizes findings from this instrumented fixed-effect panel regression of life expectancy on HIV prevalence rate. We also report OLS and random effects two-stage least squares estimates for this relationship. The first stage F statistics of the instrumental variable regressions are more than 20, indicating that the instruments are not weak. The instrumented coefficients have smaller standard errors and larger magnitudes than the same coefficients estimated with OLS. The estimates seem robust as there is not much difference between estimates with the fixed effects and those with random effects. Generally, higher HIV infection rates have a negative effect on life expectancy. As time goes by, the effect becomes more and more prominent. For example, in the fixed effect instrumented regression,

---

3 The fixed effects model treats the differences across countries as parametric shifts of the regression functions while the random effects model views individual specific constant terms as randomly distributed across countries. Hence the fixed effects model might only apply to the countries in the sample while the random effects model would be appropriate if we believe that the countries were drawn from a large population (e.g., Green, 2000).
a 1% increase in the incidence of HIV decreases life expectancy by 0.3 years in 1990 and by one year in 2005.

In conclusion, we have linked HIV/AIDS prevalence with educational outcomes and GDP growth in two steps. We first showed that variables representing the probability of premature death have negative impacts on human capital investment as well as GDP growth using panel evidence. Then we established the link between the HIV/AIDS pandemic and life expectancy with instrumented regressions. The message is clear: HIV/AIDS leads to lower life expectancy or higher probability of premature death and lower life expectancy means that people are less likely to invest in human capital with the result that the country ends up with less human capital and slower economic growth in the long run.

IV. Conclusions

The empirical results reported in the preceding section are consistent with the analytical results derived from the overlapping-generations model. While it would be interesting to estimate an empirical model that more directly measures the impact of HIV/AIDS on economic growth, the lag between the effects of the disease on growth and the incidence as reflected in current data make it impossible to estimate meaningful relationships. Nevertheless, the analysis does provide substantial evidence that falling life expectancies in Africa as a result of the HIV/AIDS pandemic, as well as the widespread incidence of other diseases, is leading to reduced investments in human capital formation which in turn results in lower human capital stocks and slower growth. The implications of this result are extremely serious. If the spread of HIV/AIDS and other diseases leads to less economic growth in African countries, there will be fewer resources in these countries for use in combating the diseases. Through the mechanisms identified in this paper, as well as the more obvious connections between disease and economic growth, a vicious cycle could develop in

---

4 Alternatively, one might consider estimating a system of equations with simultaneous equations on education, GDP growth, life expectancy, and HIV prevalence rate. However, for identification there needs to be some exogenous variables that are in some of the equations but not in the others so in practice this would be very difficult.
which disease slows growth reducing the ability to control the disease, which becomes
more widespread slowing growth even further.

The Global Fund to Fight AIDS, Tuberculoses and Malaria was established in January
2002 by the United Nations to focus contributions from wealthy countries on the fight against
these diseases in low-income countries. According to the internet site for the Fund, some
$15.6 billion had been disbursed in 140 countries by mid-2009 (The Global Fund, 2009a).
According to a 2009 Progress Report (The Global Fund, 2009b), these efforts have had
substantial positive impacts in the fight against these diseases. The proportion of people
infected by the HIV virus who are receiving treatment, however, is predicted to fall from 31
percent to 21 percent as the amounts disbursed by the fund fall short of the growing needs. If
the analysis in this paper is correct, adequate funding and rapid implementation of the Global
Fund’s programs as well as of other programs targeted at these devastating diseases are
critical if the vicious cycle described above is to be short-circuited. The nature of HIV/AIDS
is such that it is very important to undertake effective preventive programs as soon as
possible in order to avert an explosion of cases in coming years. Reducing the incidence of
these diseases and raising life expectancies are clearly ends in themselves. But, in addition,
increased life expectancy has the instrumental value of providing incentives for greater
investments in the human capital that contributes significantly to economic growth and
human well-being.

References

on Economic Growth”. Cambridge, MA: MIT, mimeo


“Randomized Controlled Intervention Trial of Male Circumcision for Reduction of HIV

“Male circumcision for HIV prevention in young men in Kisumu, Kenya: a randomised


Table 1. Variables and Their Sources

<table>
<thead>
<tr>
<th>Variables</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Education</strong></td>
<td></td>
</tr>
<tr>
<td>Average schooling years in the total population over 25</td>
<td>Barro &amp; Lee (2000)</td>
</tr>
<tr>
<td>Net secondary enrollment rate (% children of secondary school age that are currently enrolled in secondary school)</td>
<td>WDI (2008)</td>
</tr>
<tr>
<td><strong>Health</strong></td>
<td></td>
</tr>
<tr>
<td>Crude death rate (number of deaths per 1,000 population /1,000)</td>
<td>WDI (2008)</td>
</tr>
<tr>
<td>Life expectancy at birth (years)</td>
<td>WDI (2008)</td>
</tr>
<tr>
<td>Infant mortality rate (number of deaths per 1,000 live birth/1,000)</td>
<td>WDI (2008)</td>
</tr>
<tr>
<td><strong>Income</strong></td>
<td></td>
</tr>
<tr>
<td>GDP per capita in 2000 constant dollars</td>
<td>WDI (2008)</td>
</tr>
<tr>
<td><strong>Country Specific Variables</strong></td>
<td></td>
</tr>
<tr>
<td>Openness (% GDP from imports and exports)</td>
<td>WDI (2008)</td>
</tr>
<tr>
<td>Investment (% GDP used for investment)</td>
<td>WDI (2008)</td>
</tr>
<tr>
<td><strong>HIV/AIDS</strong></td>
<td></td>
</tr>
<tr>
<td>HIV prevalence rate (%)</td>
<td>CIA (2007): The World Factbook</td>
</tr>
<tr>
<td><strong>Male Circumcision</strong></td>
<td></td>
</tr>
<tr>
<td>Male circumcision rate (share of male in the country that have been circumcised)</td>
<td>Wecker et al (2006)</td>
</tr>
</tbody>
</table>
Table 2. Panel Regression of Health on Schooling Years

I. Regression Results

<table>
<thead>
<tr>
<th>Dependent variable: Average schooling years in the total population over age 25 in year 85,90, 95 and 99</th>
<th>(a)</th>
<th>(b)</th>
<th>(c )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Average schooling years in the total population over age 25 in year 60,65,70 and 75</td>
<td>-0.046</td>
<td>-0.082</td>
<td>-0.032</td>
</tr>
<tr>
<td>Log (infant mortality rate in year 60,65, 70 and 75)</td>
<td>-2.558***</td>
<td>(0.324)</td>
<td></td>
</tr>
<tr>
<td>Log (life expectancy in year 60,65,70,75)</td>
<td>2.456***</td>
<td>(0.215)</td>
<td></td>
</tr>
<tr>
<td>Log (crude death rate in year 60,67,70 and 75)</td>
<td>0.730***</td>
<td>(0.126)</td>
<td>-2.40***</td>
</tr>
<tr>
<td>Log (GDP per capita in year 60,65,70 and 75)</td>
<td>0.011</td>
<td>(0.092)</td>
<td>1.488***</td>
</tr>
<tr>
<td>Interaction between log(health indicator) and log (GDP per capita)</td>
<td>0.243***</td>
<td>(0.035)</td>
<td>0.251***</td>
</tr>
<tr>
<td>Log (% openness)</td>
<td>-0.124***</td>
<td>(0.047)</td>
<td>-0.092**</td>
</tr>
<tr>
<td>Log (% investment)</td>
<td>0.023</td>
<td>(0.034)</td>
<td>0.004</td>
</tr>
<tr>
<td>Constant</td>
<td>-5.44***</td>
<td>(0.807)</td>
<td>-8.47***</td>
</tr>
</tbody>
</table>

# obs 225 220 240
Countries 89 86 91
F-statistic 37 58 38
R-sq: within 0.630 0.731 0.617
Between: 0.723 0.716 0.682
Overall 0.711 0.71 0.668

Notes: The numbers in parentheses are standard errors.

*** p<0.01, ** p<0.05, * p<0.1
## II. Summary Statistics

<table>
<thead>
<tr>
<th></th>
<th>(a)</th>
<th>(b)</th>
<th>(c)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Average schooling years in the total population over age 25</td>
<td>5.595 (2.836)</td>
<td>5.517 (2.866)</td>
<td>5.662 (2.823)</td>
</tr>
<tr>
<td>Average schooling years in the total population over age 25, 25 years ago</td>
<td>3.625 (2.573)</td>
<td>3.587 (2.627)</td>
<td>3.697 (2.578)</td>
</tr>
<tr>
<td>Infant mortality 25 years ago</td>
<td>0.088 (0.055)</td>
<td>0.088 (0.055)</td>
<td>0.088 (0.055)</td>
</tr>
<tr>
<td>Life expectancy 25 years ago</td>
<td>57.380 (11.424)</td>
<td>57.380 (11.424)</td>
<td>57.380 (11.424)</td>
</tr>
<tr>
<td>Crude death rate 25 years ago</td>
<td>0.014 (0.006)</td>
<td>0.014 (0.006)</td>
<td>0.014 (0.006)</td>
</tr>
<tr>
<td>GDP per capita 25 years ago</td>
<td>3200.296 (2914.735)</td>
<td>3200.341 (2976.803)</td>
<td>3240.383 (2895.797)</td>
</tr>
<tr>
<td>Openness</td>
<td>58.337 (87.174)</td>
<td>58.863 (89.396)</td>
<td>60.302 (87.935)</td>
</tr>
<tr>
<td>Investment</td>
<td>17.791 (12.724)</td>
<td>17.692 (12.902)</td>
<td>17.800 (12.674)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th># obs</th>
<th>(a)</th>
<th>(b)</th>
<th>(c)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>233</td>
<td>220</td>
<td>240</td>
</tr>
</tbody>
</table>

Notes: The numbers shown are means of the variables for each regression.
### Table 3. Panel Regression of Health on GDP Per Capita Growth Rate (Fixed Effects)

#### I. Regression Results

<table>
<thead>
<tr>
<th>Dependent Variable: log (GDP per capita growth rate of each of the 25-year periods: 60-85, 65-90, 70-95, 75-00, 80-05)</th>
<th>(a)</th>
<th>(b)</th>
<th>(c)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Log (Secondary net enrollment rate at the beginning of the period)</td>
<td>-0.099***</td>
<td>-0.118</td>
<td>-0.086***</td>
</tr>
<tr>
<td>(0.023)</td>
<td>(0.027)</td>
<td>(0.025)</td>
<td></td>
</tr>
<tr>
<td>Log (infant mortality rate at the beginning of the period)</td>
<td>-1.184 ***</td>
<td>0.703</td>
<td>-0.931 ***</td>
</tr>
<tr>
<td>(0.157)</td>
<td>(0.557)</td>
<td>(0.236)</td>
<td></td>
</tr>
<tr>
<td>Log (life expectancy at birth at the beginning of the period)</td>
<td>0.703</td>
<td>0.078 ***</td>
<td>0.075**</td>
</tr>
<tr>
<td>(0.557)</td>
<td>(0.096)</td>
<td>(0.034)</td>
<td></td>
</tr>
<tr>
<td>Log (crude death rate at the beginning of the period)</td>
<td>-0.931 ***</td>
<td>-0.931 ***</td>
<td>-0.121</td>
</tr>
<tr>
<td>(0.236)</td>
<td>(0.166)</td>
<td>(0.096)</td>
<td></td>
</tr>
<tr>
<td>Log (GDP per capita at the beginning of the period)</td>
<td>-0.685 ***</td>
<td>-1.19**</td>
<td>-0.296*</td>
</tr>
<tr>
<td>(0.072)</td>
<td>(0.419)</td>
<td>(0.166)</td>
<td></td>
</tr>
<tr>
<td>Interaction between log(health) and log(GDP per capita)</td>
<td>0.078 ***</td>
<td>0.121</td>
<td>0.075**</td>
</tr>
<tr>
<td>(0.016)</td>
<td>(0.096)</td>
<td>(0.034)</td>
<td></td>
</tr>
<tr>
<td>Log (openness at the beginning of the period)</td>
<td>0.026</td>
<td>0.105***</td>
<td>0.120***</td>
</tr>
<tr>
<td>(0.036)</td>
<td>(0.037)</td>
<td>(0.038)</td>
<td></td>
</tr>
<tr>
<td>Log (investment at the beginning of the period)</td>
<td>0.007</td>
<td>-0.028</td>
<td>-0.043*</td>
</tr>
<tr>
<td>(0.028)</td>
<td>(0.027)</td>
<td>(0.027)</td>
<td></td>
</tr>
<tr>
<td>Constant</td>
<td>3.918 ***</td>
<td>2.65</td>
<td>0.819</td>
</tr>
<tr>
<td>(0.482)</td>
<td>(2.415)</td>
<td>(1.103)</td>
<td></td>
</tr>
<tr>
<td># obs</td>
<td>404</td>
<td>438</td>
<td>437</td>
</tr>
<tr>
<td>Countries</td>
<td>104</td>
<td>106</td>
<td>105</td>
</tr>
<tr>
<td>F-statistic</td>
<td>65</td>
<td>53</td>
<td>52</td>
</tr>
<tr>
<td>R-sq: within</td>
<td>0.57</td>
<td>0.49</td>
<td>0.49</td>
</tr>
<tr>
<td>Between:</td>
<td>0.01</td>
<td>0.02</td>
<td>0.03</td>
</tr>
<tr>
<td>Overall</td>
<td>0.01</td>
<td>0.01</td>
<td>0.03</td>
</tr>
</tbody>
</table>

Notes: The numbers in parentheses are standard errors.

*** p<0.01, ** p<0.05, * p<0.1
## II. Summary Statistics

<table>
<thead>
<tr>
<th></th>
<th>(a)</th>
<th>(b)</th>
<th>(c)</th>
</tr>
</thead>
<tbody>
<tr>
<td>GDP per capita growth rate of each of the 25-year periods: 60-85, 65-90, 70-95, 75-00, 80-05</td>
<td>1.591</td>
<td>1.592</td>
<td>1.593</td>
</tr>
<tr>
<td></td>
<td>(0.942)</td>
<td>(0.964)</td>
<td>(0.965)</td>
</tr>
<tr>
<td>Secondary net enrollment rate at the beginning of the period</td>
<td>34.440</td>
<td>34.858</td>
<td>34.883</td>
</tr>
<tr>
<td></td>
<td>(28.786)</td>
<td>(28.661)</td>
<td>(28.685)</td>
</tr>
<tr>
<td>Infant mortality rate at the beginning of the period</td>
<td>58.157</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(11.567)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Life expectancy at birth at the beginning of the period</td>
<td>0.085</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(0.055)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Crude death rate at the beginning of the period</td>
<td></td>
<td>0.014</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>(0.006)</td>
<td></td>
</tr>
<tr>
<td>GDP per capita at the beginning of the period</td>
<td>4162.74</td>
<td>4113.79</td>
<td>4121.46</td>
</tr>
<tr>
<td></td>
<td>(6002.69)</td>
<td>(5910.98)</td>
<td>(5914.68)</td>
</tr>
<tr>
<td># obs</td>
<td>404</td>
<td>438</td>
<td>437</td>
</tr>
</tbody>
</table>

Notes: The numbers shown are means of the variables for each regression. Also shown in parentheses are standard errors.
Table 4. Panel Regression of HIV Prevalence on Life Expectancy

I. Primary Regression

<table>
<thead>
<tr>
<th>Dependent variable:</th>
<th>OLS (fixed effects)</th>
<th>IV (fixed effects)</th>
<th>IV (random effects)</th>
</tr>
</thead>
<tbody>
<tr>
<td>HIV*1990</td>
<td>-0.042 (0.063)</td>
<td>-0.294 (0.109)</td>
<td>-0.339 (0.125)</td>
</tr>
<tr>
<td>HIV*1995</td>
<td>-0.202 (0.063)</td>
<td>-0.546 (0.109)</td>
<td>-0.591 (0.125)</td>
</tr>
<tr>
<td>HIV*2000</td>
<td>-0.596 (0.063)</td>
<td>-0.850 (0.109)</td>
<td>-0.895 (0.125)</td>
</tr>
<tr>
<td>HIV*2004</td>
<td>-0.774 (0.063)</td>
<td>-1.024 (0.109)</td>
<td>-1.070 (0.125)</td>
</tr>
</tbody>
</table>

| Life expectancy at birth in 1980 *year | Yes | Yes | Yes |
| GDP per capita in 1980 *year           | Yes | Yes | Yes |
| Youth illiteracy rate in 1980 *year    | Yes | Yes | Yes |
| # obs                                 | 222 | 222 | 222 |
| # countries                           | 37  | 37  | 37  |
| First stage F-statistic               | --- | 29.15 | 23.64 |
| F-statistic                           | 23.71 | 15.69 | 11.89 |

Notes: The numbers in parentheses are standard errors. Bold numbers indicate that we fail to reject the null hypothesis at significance level of 0.05.

II. First Stage

<table>
<thead>
<tr>
<th>Dependent variable: HIV prevalence rate (CIA)</th>
<th>OLS cross-sectional regression</th>
</tr>
</thead>
<tbody>
<tr>
<td>Circumcision rate (Werker et al 2006)</td>
<td>-20.840 (3.658)</td>
</tr>
<tr>
<td>F-statistics of circumcision rate</td>
<td>32.46</td>
</tr>
<tr>
<td>P(&gt;F)</td>
<td>0</td>
</tr>
<tr>
<td># obs</td>
<td>37</td>
</tr>
<tr>
<td>R-square</td>
<td>0.481</td>
</tr>
</tbody>
</table>

III. Summary Statistics

<table>
<thead>
<tr>
<th>Variables</th>
<th># obs</th>
<th>Mean</th>
<th>Std. Dev.</th>
<th>Min</th>
<th>Max</th>
</tr>
</thead>
<tbody>
<tr>
<td>life expectancy at birth in 1980</td>
<td>37</td>
<td>50.919</td>
<td>6.193</td>
<td>39</td>
<td>62</td>
</tr>
<tr>
<td>HIV prevalence rate</td>
<td>37</td>
<td>8.365</td>
<td>10.36</td>
<td>1</td>
<td>38.8</td>
</tr>
<tr>
<td>Youth illiteracy rate in 1980</td>
<td>37</td>
<td>45.353</td>
<td>20.624</td>
<td>13.8</td>
<td>88.5</td>
</tr>
<tr>
<td>GDP per capita in 1980</td>
<td>37</td>
<td>755.703</td>
<td>936.57</td>
<td>135</td>
<td>4717</td>
</tr>
<tr>
<td>Circumcision rate</td>
<td>37</td>
<td>0.611</td>
<td>0.345</td>
<td>0</td>
<td>1</td>
</tr>
</tbody>
</table>
Table 5. Countries Included in Each Regression

Panel regressions of education on health

(a) Algeria, Argentina, Australia, Austria, Bangladesh, Belgium, Benin, Bolivia, Botswana, Brazil, Cameroon, Canada, Central African Republic, Chile, China, Colombia, Congo, Dem. Rep., Congo, Rep., Costa Rica, Cyprus, Denmark, Dominican Republic, Ecuador, Egypt, Arab Rep., El Salvador, Fiji, Finland, France, Gambia, Germany, Ghana, Greece, Guatemala, Guyana, Haiti, Honduras, Hong Kong, China, Hungary, India, Indonesia, Iran, Islamic Rep., Iraq, Ireland, Israel, Italy, Jamaica, Japan, Jordan, Kenya, Korea, Rep., Lesotho, Liberia, Malawi, Malaysia, Mali, Mauritius, Mexico, Mozambique, Nepal, Netherlands, New Zealand, Nicaragua, Niger, Norway, Pakistan, Panama, Papua New Guinea, Paraguay, Peru, Philippines, Poland, Portugal, Rwanda, Senegal, Serbia and Montenegro, Sierra Leone, Singapore, South Africa, Spain, Sri Lanka, Sudan, Swaziland, Sweden, Switzerland, Syrian Arab Republic, Thailand, Togo, Trinidad and Tobago, Tunisia, Turkey, Uganda, United Kingdom, United States, Uruguay, Venezuela, RB, Zambia, Zimbabwe

(b) Countries in (a) and Barbados, Malta

(c) Countries in (b) and Iceland

Panel regression of growth rate on health

(a) Albania, Algeria, Angola, Argentina, Australia, Austria, Bahamas, Bahrain, Bangladesh, Belgium, Belize, Benin, Bolivia, Botswana, Brazil, Bulgaria, Burkina Faso, Burundi, Cameroon, Canada, Central African Republic, Chad, Chile, China, Colombia, Comoros, Congo, Dem. Rep., Congo, Rep., Costa Rica, Cote d'Ivoire, Denmark, Dominican Republic, Ecuador, El Salvador, Estonia, Fiji, Finland, France, Gabon, Gambia, Ghana, Greece, Guatemala, Guyana, Haiti, Honduras, Hungary, Iceland, India, Indonesia, Iran, Islamic Rep., Ireland, Israel, Italy, Jamaica, Japan, Jordan, Kenya, Korea, Rep., Kuwait, Latvia, Lesotho, Liberia, Libya, Luxembourg, Madagascar, Malawi, Malaysia, Mali, Malta, Mauritania, Mauritius, Mexico, Moldova, Morocco, Mozambique, Nepal, Netherlands, New Zealand, Nicaragua, Niger, Nigeria, Norway, Oman, Pakistan, Panama, Papua New Guinea, Paraguay, Peru, Philippines, Portugal, Romania, Rwanda, Saudi Arabia, Senegal, Sierra Leone, Solomon Islands, Spain, Sri Lanka, Sudan, Suriname, Swaziland, Sweden, Switzerland, Syrian Arab Republic, Thailand, Togo, Trinidad and Tobago, Tunisia, Turkey, United Kingdom, United States, Uruguay, Venezuela, RB, Zambia, Zimbabwe

(b) Countries in (a), Cyprus, Georgia, and South Africa

(c) Countries in (b) except for Comoros

Panel regression of Life expectancy on HIV prevalence
Appendix 1. Derivation of the utility maximization problem

A representative young agent chooses width and depth of his human capital to maximize his lifetime utility:

\[
\max_{N, Q} U(c_{yt}, c_{at}, c_{mt}) = \log c_{yt} + \frac{1}{1 + \rho} E(\log c_{at}) + \frac{1}{(1 + \rho)^2} E(\log c_{mt})
\]

Where \( U \) is the lifetime expected utility, which depends on consumption of three periods, respectively denoted by \( c_{yt}, c_{at} \), and \( c_{mt} \). \( N \) is the width of human capital, or the number of knowledge points possessed by the individual through schooling and \( Q \) is the depth of human capital. \( \rho \) is the discount rate the agent faces. The consumption of each period depends on whether technology has occurred in the adult period, and whether the agent survives each period.

\[
c_{yt} = \delta Q(1 - l_E) = \delta Q(1 - \frac{NQ}{bNQ})
\]

\[
c_{at} = (1 - l_A)Q \quad \text{if he survives and technology occurs.}
\]

\[
c_{at} = \delta Q \quad \text{if he survives but technology doesn’t occur.}
\]

\[
c_{at} = 0 \quad \text{if he dies at the end of 1st period.}
\]

\[
l_A = \min_{x \in [0, S]} a \cdot x - s \cdot Q
\]

The adoption cost, measured by adoption time, \( l_A \), is assumed to be proportional to the distance between the occurring technology and the nearest knowledge point owned by the individual and the depth of technical innovation he adopts.

\[
c_{mt} = (1 - \tau_1)Q \quad \text{if he survives and the technology occurs in the adult period but not the mature period.}
\]

\[
c_{mt} = (1 - \tau_2)Q \quad \text{if he survives and a new technology occurs in both the adult and mature period.}
\]

\[
c_{mt} = \delta Q \quad \text{if he survives but no new technology occurs in his mature period.}
\]

In this case, the current adult generation shares the same human capital with the mature generation.

\[
c_{mt} = 0 \quad \text{if he dies at the end of 2nd period.}
\]

The mature agent enjoys higher consumption if she adopts technology in her adult period than otherwise. In other words, the spillover effect is assumed to be relatively small. The specifications imply that the level of the human capital of the current adults in the society determine the level of the human capital stock.

\[
E(\log c_{yt}) = p(1 - P_y) \frac{2N}{S} \int_{0}^{S} \log(Q(1 - aQx))dx + (1 - p)(1 - P_y) \log(\delta Q)
\]

\[
= (1 - P_y)l \int_{0}^{S} \log(Q(1 - aQ \frac{y}{2N}))dy + (1 - p) \log(\delta Q)
\]
Where \( p \) denotes the probability of technological advance, \( S \) stands for the scope of technological space where the technology occurs randomly following a uniform distribution.

The expected utility of the mature agent is the probability of living to the mature period multiplied by the sum of the expected utility under the different scenarios when technical change occurs or doesn’t occur in his adult period or mature period, that is:

\[
E(\log c_{m_1}) = P^*(1-P_y)[p(1-p) \log((1-\tau_p)Q)] + \nonumber \\
p p \log[(1-\tau_p)Q] + (1-p) p \log \delta Q + (1-p)(1-p) \log \delta Q \nonumber \\
= P^*(1-P_y)p \log Q + \text{constant} 
\]

Following Kim and Lee (1999), we assume that the utility of the second period with the technology adoption is always higher than that without it so that the adoption of the new technology is always certain. The first period maximization problem is:

\[
(A3) \quad \max_{\tau, Q} \log(1-\frac{NQ}{bNQ}) + \frac{p(1-P_y)}{1+\rho} \int_0^s \log(Q(1-\frac{aQy}{2N})) dy + \frac{P^*(1-P_y)p}{(1+\rho)^2} \log Q + \text{constant} 
\]

As long as \( p (1-P_y) \) is not zero, the individual expected utility would always be higher if he adopts technology when it occurs given the assumption above, therefore, an interior solution is guaranteed. The first order conditions are:

\[
(A4) \quad -\frac{Q}{bNQ-NQ} - \frac{p(1-P_y)}{1+\rho} \frac{1}{N-aQy} dy = 0 
\]

\[
(A5) \quad -\frac{N}{bNQ-NQ} + \frac{p(1-P_y)}{1+\rho} \frac{1}{Q} + \frac{2p(1-P_y)N}{(1+\rho)aQS} \log(N-\frac{aSQ}{2}) - \log N = 0 
\]

Multiplying both sides of (A4) by \( N \), and both sides of (A5) by \( Q \), and subtracting (A4) from (A5) yields:

\[
(A6) \quad (3+k)(z-1) = 2 \log z \quad \text{where } z = 1-\frac{aSQ}{2N}, \quad k = \frac{P^*}{(1+\rho)} 
\]

\( z \) is a linear function of the ratio of depth to width. Note that \( k=0 \) if \( P^* = 0 \), and \( k \) increases as \( P^* \) increases given the discount rate, where \( P^* \) is the conditional probability of surviving up to three periods if the agent survives the young period. Hence \( k \) can be interpreted as a time value coefficient of future consumption under uncertainty of life expectancy. Equation (A6) thus shows a relationship between the probability of premature death and the optimal depth-width ratio of human capital investment. When \( k=0 \), the equation is \( 3/2(z-1) = \log z \), as found by Kim and Lee (1999). The addition of a positive \( k \) is the result of adding a third period into the model. Since \( aSQ/2N \) is by definition not zero, \( z \neq 1 \). It is easy to see that for each definite value of \( k \) in \([0,1]\), there exists a unique solution for \( z \) between \((0,1)\) that satisfies equation (A6) since the left hand side can be depicted as a straight line through \((1,0)\) and \((0, 3+k/2)\) and the right hand side is a usual log curve through \((1,0)\).

An analytical solution of equation (A6) is not available since it is non-linear. Using simple computer simulation (EXCEL), we can solve for \( z \). Let \( z^* \) be the equilibrium \( z \) that satisfies equation (A6). Then, \( z^* \) gives the equilibrium ratio of depth to width given the characteristic space of the innovation that has occurred. The simulation shows that \( z^* \)
declines as k increases, and \( z^* \) is between [0.421, 0.2]. Therefore, as \( P^* \) increases, i.e., as one is more likely to have a longer life, the optimal ratio of depth to width of human capital acquisition increases. The result is expected since in the mature period, the agent is assumed to adopt no technology at all. We would expect a different result if the agent adopts new technology when mature.

Then (A7) \[
\frac{Q}{N} = \frac{2(1-Z^*)}{aS}
\]

Hence, the optimal ratio of depth to width decreases if the uncertainty about the characteristic of the technological advance increases. From (A4), (A6), and (A7), we can solve for \( Q \) and \( N \) as:

(A8) \[
Q = \frac{2(1-z^*)bP(1-P_y)(1+k)NQ}{(2+2\rho+(1+k)p(1-P_y))aS}
\]

(A9) \[
N = \frac{abP(1-P_y)(1+k)S\bar{N}\bar{Q}}{2(1-z^*)[2+2\rho+p(1-P_y)(1+k)]}
\]