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AIDS and Economic Growth: A Human Capital Approach

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AIDS and Economic Growth: A Human Capital Approach

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Abstract

It is estimated that by 2001 20 million people had died from AIDS, which is now the world's fourth biggest cause of death. While the highest prevalence and death rates and number of infected persons are reported for sub-Saharan Africa, where life expectancies at birth are declining rapidly and infant mortality rates are increasing, there is evidence that the epidemic is accelerating in Asia and Eastern Europe. While the human and social costs of the HIV/AIDS epidemic are the major causes for concern, the econometric results reported in this paper indicate that the macroeconomic affects of the HIV/AIDS epidemic have been substantial; especially in Africa where the average marginal negative impact on income per capita of a one percent increase in HIV prevalence rate is 0.59 percent. Even in countries where the HIV prevalence rates are lower the marginal impacts are non trivial.

Keywords: AIDS; Growth; Human Capital; Panel Data.

JEL classification: O15; O47; I12; C23.

1. Introduction

> "Twenty years after the first clinical evidence of acquired immunodeficiency syndrome was reported, AIDS has become the most devastating disease humankind has ever faced. Since the epidemic began, more than 60 million people have been infected with the virus. HIV/AIDS is now the leading cause of death in sub-Saharan Africa. Worldwide it is the fourth-biggest killer." (UNAIDS/WHO, 2001)

It is estimated that in 2003 about 5 million more people acquired the HIV virus and that more 3 million people died from AIDS[1] bringing total deaths from the epidemic to more than 25 million (UNAIDS, 2003). While the highest prevalence and death rates and number of infected persons are reported for sub-Saharan Africa, there is evidence that the epidemic is accelerating in Asia and Eastern Europe and that complacency is causing an upturn in prevalence in North America and Western Europe. Wherever the epidemic strikes it imposes severe human and social consequences. Family life is severely disrupted as adults are rendered less able and/or unable to work, health care costs rise and children are forced prematurely onto the labour market and/or made orphans. These human and social consequences of the epidemic, especially in sub-Saharan Africa, are increasingly visible as the media devotes resources to covering the epidemic. Over and above these consequences are the economic costs of the epidemic. These include immediate costs, such as increased health care expenditures and reduced labour productivity, and long term costs, such as reduced levels of education, health, physical and social capital.

Since few economists would doubt the potential of the AIDS epidemic to create substantial economic costs, it is surprising that there have been relatively few studies into the economic, especially the macroeconomic, implications of the epidemic (see below).[2] In part this is probably a reflection of the limited recognition given to the impact of health upon macroeconomic performance and the relatively recent availability of data that allow systematic econometric evaluations of the epidemic. The results reported in this paper are based upon an augmented Solow model that incorporates both health and education capital. Importantly, the model is estimated as a system wherein health capital is partially determined by health status, in particular the prevalence of HIV infection, using panel data. The next section briefly reviews the literature on HIV/AIDS and development. Section 3 summarises

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1 Of the 37.8 million people estimated to be living with HIV at the end of 2003 17 million were women and 2.1 million were children under 15 (UNAIDS, 2004). The proportions of women and children infected in sub-Saharan Africa are greater.

2 The UK Parliament's House of Commons International Development Committee recently commented on the lack of information about the macroeconomic impact of HIV/AIDS (International Development Committee, 2001).

the derivation of the growth and health capital equations, and comments on the panel data methods. Section 4 starts with a discussion of the available data and how they influence the specification of the estimating equations, and then reports the results. The results are discussed in section 5 and the final section contains concluding comments.

2. HIV/AIDS and Development

Empirical analyses of the macroeconomic impact of the epidemic have been based largely upon standard neoclassical growth models. In the context of these models the epidemic would be expected to reduce productive efficiency, and hence output per worker, reduce the rate of growth of the labour force and lower the savings rate. Cuddington (1993, pp 178-1) demonstrates that in a simple neoclassical growth model the epidemic may cause per capita income and the capital-labour ratio to either increase or decrease because the labour force and savings rate effects operate in opposite directions. Hence it is arguable that the net impact of the epidemic upon economic growth is an empirical question.

Quantitative estimates in the early 1990s were compromised by a lack of appropriate data, and hence to a greater or lesser extent calibrated growth models were used to derive estimates of the macroeconomic effects of the epidemic. All known studies of this type have examined the impact of HIV/AIDS on individual countries. Over (1992) estimated that between 1990 and 2025 the 10 countries with the most advanced epidemics would see a reduction in growth per capita of around a third of a percentage point compared to a non-AIDS scenario. Similarly, Cuddington and Hancock (1994) suggest that between 1985 and 2010 Malawi could experience average real GDP growth up to 1.5 percentage points lower, while Cuddington (1993) estimates that in Tanzania per capita GDP could be up to 10% smaller. More recently two studies have focused on Botswana (BIDPA, 2000; and MacFarlan and Sgherri, 2001), with both using variants of calibrated neoclassical growth models. MacFarlan and Sgherri concluded that GDP growth in Botswana would decline from around 5.5 percent to between 1.5 and 2.5 percent per annum as a result of the epidemic, and would have 'non-negligible' impacts across production sectors, households and labour types while placing substantial burdens on the government budget.

However there have been very few cross-country statistical studies. Bloom and Mahal (1997) concluded, "there is more flash than substance to the claim that AIDS impedes national economic (income) growth." (p 120), while acknowledging that the negative impact on life expectancy may affect development. There are reasons related to the data to doubt the robustness of Bloom and Mahal's results. The empirical estimates were based on data for the period 1980-92, when prevalence estimates were typically one seventh to one fifth of those now produced. Furthermore, the AIDS epidemic was still in its early stages and hence its

impacts upon morbidity and mortality were still relatively restrained. Given the data limitations it is not surprising that Bloom and Mahal's sample was constrained to 51 countries, although it is worrying that countries with high infection rates were not included, e.g., South Africa, Botswana, Rwanda, Namibia and Swaziland.[3] There are also theoretical and statistical reasons to reevaluate Bloom and Mahal's results (see below).

More recently, Bonnel (2000) concluded, from a cross-country study using African data, that HIV/AIDS on average reduced Africa's per capita growth rate by 0.7 percent points, which is substantial given an average growth rate in the sample of 0.4 percent per annum per capita. Furthermore, if the countries were also affected by malaria, the growth rate was lowered by a further 0.3 percent points. These results were obtained using a cross-section/cross-country growth equation estimated in a system with equations for policy variables and HIV. It would therefore seem that Bonnel's work using more recent and comprehensive data are sufficient to cast doubt upon Bloom and Mahal's earlier study.

3. Growth and Human Capital

The analyses reported in this paper are derivatives of the empirical research into economic growth that has developed since the late 1980s. The contribution of human capital to the growth process has been central in this research (Mankiw *et al*., 1992; Barro and Lee, 1993; Benhabib and Spiegel, 1994, among many others). Mankiw *et al*., (1992, p 408) augmented a neoclassical growth model with a human-capital variable to counter the misspecification bias that will arise if both physical and human capital exists and human capital is omitted from the estimating equation. But it has long been recognised in the literature that human capital is a complex input that consists of more than education and productive skills. Part of this literature has emphasised the importance of health capital as a dimension of human capital, e.g., Schultz, (1961); Mushkin, (1962); Dasgupta (1993). Hence any misspecification bias will only be partially addressed if other dimensions of human capital are not included in estimating equations. Nevertheless estimates of the effects of health on growth are limited, e.g., Hicks (1979), Wheeler (1980), Knowles and Owen (1995), Bhargava *et al*. (2002) and McDonald and Roberts (2002). The latter three studies have confirmed the empirical significance of health capital as a determinant of economic growth.

It is still common in the empirical growth literature for estimating equations to be specified using the cross-country cross-section method, despite the well known potential weaknesses of this method. In particular, the need to impose the assumptions that there are common initial technologies, rates of technical progress and preferences across countries. Moreover the method discards potentially important information by collapsing dynamics.

3 Namibia is also excluded from the sample used in this study.

These issues can be addressed by adopting panel data methods and thereby explicitly testing the critical assumptions of the cross-section method by making use of information arising from variation across countries and over time. Most panel data studies relating to cross country economic growth have indicated that the results of cross-section studies may be suspect (Islam, 1995; Lee *et al*., 1997; Miller, 1996; Cellini, 1997; McDonald and Roberts, 1996; 1999; 2002).

An Augmented Solow Model[4]

Define the aggregate production function for the augmented Solow model as a Cobb-Douglas function with constant returns to scale and labour augmenting technical progress, and three types of capital as

$$Y_{it} = \left[A_{it} L_{it}\right]^{1-\alpha-\beta-\psi} K_{it}^{\alpha} E_{it}^{\beta} H_{it}^{\psi} \tag{1}$$

where Y is output, A is technology, L is labour, K, E and H are respectively physical, education and health capital, α, β and ψ are the elasticities of output with respect to the various types of capital, and the subscripts denote country (i) and time (t). Equation (1) can be re-written in intensive form as

$$y_{it} = k_{it}^{\alpha} e_{it}^{\beta} h_{it}^{\psi} \tag{2}$$

where y_{it} is output per 'effective' labour unit $\left(A_{it} L_{it}\right)$ in country i at time t, and k_{it}, e_{it} and h_{it} are respectively physical, education and health capital per 'effective' labour unit.

Assuming that the labour forces grow at country specific constant rates n_i, and technologies advance at period specific constant rates g_t, and that the physical, education and human capital stocks depreciate at the same constant rate, δ,[5] then it can be shown (see Appendix IV) that the augmented steady state output per capita (y_{it}^{*}) is

$$\begin{aligned} \ln y_{it}^{*} = \ln A_{i0} + g_t t - \frac{\alpha+\beta+\psi}{(1-\alpha-\beta-\psi)} \ln\left(n_i + g_i + \delta\right) \\ + \frac{\alpha}{(1-\alpha-\beta-\psi)} \ln s_i^{K} + \frac{\beta}{(1-\alpha-\beta-\psi)} \ln s_i^{E} + \frac{\psi}{(1-\alpha-\beta-\psi)} \ln s_i^{H} \end{aligned} \tag{3}$$

and where s_i^{K}, s_i^{E} and s_i^{H} are the savings rates devoted to physical, education and health capitals. Equation 3 is equivalent, except for the inclusion of a term for health capital, to Mankiw *et al.*'s equation 11 (p 417).

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4 This model was 'developed' in McDonald and Roberts (2002), which was itself a development of Knowles and Owen's (1995) model. Both models follow a lead established by Mankiw *et al*., (1992).

5 The use of a constant uniform depreciation rate follows Mankiw et al., (1992), who based their assumption upon estimates by Romer (1989); there remains inadequate data to estimate country specific rates.

Empirically (3) is difficult to implement because of an absence of disaggregated savings data. In general the available savings, or investment, data refer to physical capital, and estimates for education and health capital are likely to be missing. However underlying (3) is a steady-state condition in levels, which Mankiw *et al*., exploited to derive alternative formulations of the estimating equation according to whether the augmenting capital terms are recorded as rates of accumulation, as in equation 3, or as levels, e.g.,

$$\ln y_{it}^{*} = \ln A_{i0} + g_t t - \frac{\alpha}{(1-\alpha)} \ln\left(n_i + g_t + \delta\right) + \frac{\alpha}{(1-\alpha)} \ln s_i^K + \frac{\beta}{(1-\alpha)} \ln s_i^E + \frac{\psi}{(1-\alpha)} \ln h_{it}^{*} \tag{4}$$

where the asterisk indicates that the quantity of health capital per effective labour unit (h_{it}^{*}) is a steady state level. While (3) is implicitly derived from a steady state expression in terms of levels, the derivation of (4) does require the presumption of a steady state. The choice of whether an estimating equation is based on (3) or some variant of (4) will depend typically upon the available data, and consequently the extent to which the presumption of an underlying steady state is restrictive is largely determined by the available data.[6]

Linearising (4) around the steady-state level of income per effective unit of labour, y_{it}^{*}, following the method used by Mankiw *et al*., produces

$$\ln y_{it}^{*} - \ln y_{i0}^{*} = \left(1 - \exp^{\lambda t}\right) \ln A_{i0} + g_t t - \frac{\left(1 - \exp^{\lambda t}\right)\alpha}{(1-\alpha)} \ln\left(n_i + g_t + \delta\right) + \frac{\left(1 - \exp^{\lambda t}\right)\alpha}{(1-\alpha)} \ln s_i^K + \frac{\left(1 - \exp^{\lambda t}\right)\beta}{(1-\alpha)} \ln s_i^E + \frac{\left(1 - \exp^{\lambda t}\right)\psi}{(1-\alpha)} \ln h_{it}^{*} - \left(1 - \exp^{\lambda t}\right) \ln y_{i0}^{*}$$

. (5)

One substantive empirical advantage of the method used by Mankiw *et* al., is that it allows for the mixing of stock and saving/investment data for the capital components of the estimating equation. Solving (5) for $\ln y_{it}^{*}$, and using standard panel data notation, gives a general specification of the econometric growth model for which estimates are reported in this paper

$$z_{it}^{*} = \gamma z_{i0}^{*} + \sum_{j=1}^{4} \theta_j x_{it}^{j} + \eta_t + \mu_i + \nu_{it}^{1} \tag{6}$$

where

6 For instance McDonald and Roberts (2002) had estimates of the stock of education capital and therefore estimated a version of (4) where education capital was expressed in terms of the steady state levels.

$$z_{it}^{*} = \ln y_{it}^{*}$$
$$\gamma = \exp^{\lambda t}$$
$$z_{i0}^{*} = \ln y_{i0}^{*}$$
$$\theta_1 = -\theta_2 = \frac{\left(1-\exp^{\lambda t}\right)\alpha}{\left(1-\alpha\right)}$$
$$x_{it}^{1} = \ln(n_i + g_t + \delta)$$
$$x_{it}^{2} = \ln s_i^{k}$$
$$\theta_3 = \frac{\left(1-\exp^{\lambda t}\right)\beta}{\left(1-\alpha\right)}$$
$$x_{it}^{3} = \ln s_i^{E} \text{ or } \ln e_{it}^{*}$$
$$\theta_4 = \frac{\left(1-\exp^{\lambda t}\right)\psi}{\left(1-\alpha\right)}$$
$$x_{it}^{4} = \ln s_i^{H} \text{ or } \ln h_{it}^{*}$$
$$\eta_t = g_t t$$
$$\mu_i = \left(1-\exp^{\lambda t}\right)\ln A_{i0}$$

and the ν_{it}^{1} is the standard error term.

Equation (6) differs from a typical cross-section estimating equation in several important ways: it allows for cross-time variations in the rates of growth of technology, g_t, country specific initial states of technology, $\ln A_{i0}$, and uses the time series information by being estimated as a dynamic data panel with two-way fixed effects. In particular it facilitates testing of the identifying assumption for OLS estimation of the cross-section model, i.e.,

$$\ln A_{i0} = a + \varepsilon \tag{7}$$

where a is a constant, invariant across countries, that all country differences are accounted for by the random term, ε, and that the stochastic term is independent of all other explanatory variables. Panel estimates of (6) using data for 1960 to 1989 (McDonald and Roberts, 2002) indicate that health capital has a positive, and often significant, impact upon economic growth. But these results were obtained using a model that takes the health capital terms as being exogenously determined, and use data for a period that overwhelmingly precedes the substantive onset of the HIV/AIDS epidemic. Moreover, the estimation technique did not account for the presence of a lagged dependent variable on the right hand side of the growth equation (6), which may lead to bias in the OLS estimates.

The model in (6) differs appreciably from the estimating equations specified by Bloom and Mahal (1997) and Bonnel (2000). The variables included in the econometric growth equation are determined by the theoretical derivation of the model, which also provides the basis for a clear theoretical interpretation of the model that may be lost if additional explanatory variables are introduced. Most importantly, HIV/AIDS does not enter directly into the model. Rather, it is deemed preferable to identify relationships between the model's explanatory variables and the prevalence or incidence of HIV/AIDS. Hence it is hypothesised that the impacts of diseases enter into the determination of growth indirectly through their impact upon both the population growth terms and the health capital terms. This requires a model wherein health capital is explained.

Health Capital

Much of the research on the determinants of health capital stems from the household production function approach of Grossman (1972), which is a microeconomic formulation that is not readily applicable within a macroeconomic framework. Genberg (1992) has surveyed the literature on macroeconomics and health and concludes that "detailed relationships between macroeconomic change and health outcomes are not clearcut … and one is left with a set of associations relating the two sets of phenomena but no strong evidence of causality" (p.13).

Given the primary aim of estimating the effects of HIV/AIDS on economic growth via a theoretically supported macroeconomic growth model and the absence of any settled body of theory on the determinants of health capital that can be applied at the macro level, the second-best approach is to define a reduced form model for health capital. This reduced form postulates health capital as function of a set of exogenous (or predetermined) variables; the variables included are informed by the literature but do not represent a structural relationship between the chosen determinants and health capital.

Grossman's (1972) notion of health as a capital good suggests the inclusion of a lagged dependent variable, which requires a dynamic specification for the reduced form. There is evidence from both cross sectional studies (Preston, 1986; Hobcroft *et al*., 1984) and long time series (United Nations 1988; Hill 1987) that indicate that higher levels of income are associated with better health, and hence lagged per capita income is included as an explanatory variable.[7] Education capital and nutritional status are also included, since there are reasons to believe that these are important determinants of health, especially in developing countries. HIV prevalence and the proportion of the population at risk of malaria are included to account for the major diseases and are considered to be exogenous health shocks since the presence of malaria is defined primarily by climate and topography.[8] To account for possible lags and non-linearities in the relationship the following are also included: lagged values of the variables, interactions of each variable with per capita income, and squared terms on each explanatory variable.

Hence the reduced form health equation is:

$$x_{it}^4 = \varphi x_{it-1}^4 + \rho \mathbf{P} + \xi_t + \omega_i + v_{it}^2 \qquad (8)$$

7 A formulation that included current income, thereby specifying a simultaneous model with the growth equation, was examined. However current income was never significant in the empirical estimation of the health equation, hence this variable was omitted and consequently the system of growth and health equations are not simultaneously determined.

8 It is arguable whether HIV and malaria are exogenous determinants of the overall health outcome. Hence the choice of instruments for the instrumental variables estimation is tested in the empirical section of the paper; the test results suggest that we can have confidence in the overall set of instruments used.

where

$x_{it}^4 = \ln h_{it}$, the log of health capital per 'effective' labour unit;
P = vector of exogenous variables (set text above.);
ξ_t = time effects;
ω_i = country specific fixed effects; and
v_{it}^2 = standard error term.

As with the macroeconomic growth model, the equation for health capital does not encompass potentially important distributional questions. Simple national average measures of the explanatory variables may not be appropriate in circumstances where there are wide differences in the standard of living, access to health care systems and incidences of disease within countries. While these questions are relevant they are beyond the scope of this study.[9]

4. Analysis

Data

The main source of data was the World Bank (1999). The income data are real GDP per capita adjusted for purchasing power parity, which are an update of the Penn World Tables (PWT) (see Summers and Heston, 1988 and 1991) developed using data from the Global Development Finance & World Development Indicators databases. The population (POP) data are those reported in World Development Indicators (World Bank, 2001). The PWT series for real GDP per worker and number of workers series were not updated beyond 1990, and hence it was not possible to use such series. The data for investment rates were calculated from current price data on GDP and domestic investment reported in World Development Indicators (World Bank, 2001). Rates of growth for GDP and population were estimated by log linear regression of the GDP and population series. There are four education series, primary school enrolment rates, secondary school enrolment rates and tertiary enrolment rates, which were from World Development Indicators (World Bank, 2001), and estimates of the average years of schooling (Barro and Lee, 2000).[10]

Data for the health capital equation are limited in terms of both coverage of countries and length of time series. Two alternative variables are used as a proxy for health capital. The first is life expectancy at birth, which is defined as the mean age at death of a fictitious generation subject to the mortality conditions of the period considered; this is expressed as the shortfall of life expectancy relative to a nominal benchmark, i.e., LE = - ln(80 – life expectancy) . This

9 Arndt and Lewis (2001) and Arndt (2002) are studies that emphasise distributional issues.

10 Barro and Lee's (2000) series of average years of schooling made no substantive difference to the empirical results and consequently the results using the more commonly used enrolment data are reported.

proxy has been defended by Sen (1998), but can be criticised for making "no allowance for the quality of health beyond survival" (Knowles and Owen, 1995, p 102). In the present context it would be expected that changes in life expectancy would understate changes in health capital resulting from HIV/AIDS by not registering the morbidity affects. The second health capital proxy is the infant mortality rate, defined as the number of infant deaths before one year of age per 1,000 live births. This proxy can be defended on the grounds that it offers an indicator of the current health status of the population through those most susceptible to deterioration in the general level of health within the population. As such it partially captures the 'quality of health' dimension, but it may overstate changes in health capital since it is arguable that a mother's health may have a greater impact upon infant mortality and there is evidence of a greater prevalence rate among women in countries with higher overall prevalence rates. Neither proxy is ideal since both are likely to have involved some interpolation and hence may not fully reflect current health status and to a greater or lesser extent fail to capture 'quality' effects. On balance the preferred data set is infant mortality, but both were used in the estimation process although only the results for infant mortality are reported in detail. Both data series were obtained from World Development Indicators (World Bank, 2001).

Table 1 **HIV Prevalence Data** (unweighted percentage rates for populations 15-49)

About Here

Time series estimates for number of adults (15-49) who were HIV positive were obtained from UNAIDS (Walker, 2000). These figures for prevalence levels were derived using an epidemiological model (EPIMODEL, Chin and Lwanga, 1991); the key data used to calibrate the model for each country include: point prevalence estimates for 1994 and 1997; the year 'extensive spread' of infection started; the most likely shapes of the country specific epidemiological curves for the HIV prevalence over time; progression rates and age structure of the HIV-infected population. The method used and its strengths and weaknesses are discussed extensively in Schwartlander *et al*., (1999, pp 2455-2457). The HIV prevalence rates have been a subject of considerable recent research and are increasingly based on robust epidemiological data, e.g., blood samples taken at ante-natal clinics; indeed the "HIV sentinel surveillance systems are generally rather extensive when compared with surveillance systems for other communicable diseases" (Swartlander *et al*., 1999, p 2455). Consequently it is claimed that the current estimates of HIV prevalence are superior to estimates for the prevalence of other diseases (see Garnett *et al*., 2001), although clearly there are general issues about the quality of information about the prevalence of all communicable diseases. The data series were for all years from the start of the epidemic in each country up to 1999, and are expressed as prevalence rates among the adult population for use in the econometric

estimations. Summary statistics for the HIV prevalence data are reported in Table 1; these confirm the extent to which the epidemic is most pronounced in Africa and Latin America and the Caribbean, with all those countries in the latter sample with prevalence rates greater than one percent in 1999 being in the Caribbean basin.

The malaria data record the proportions of the population at risk from malaria (MAL) because they live in areas of the country where malaria is present (Hamoudi and Sachs, 1999). Data are only available for 4 points in time (1946, 1966, 1982 and 1994): hence the proportions of population at risk during the intervening years were interpolated and the interpolated series was used to derive 5 yearly averages. Protein supplies per capita, a proxy for nutritional status, were compiled using data from the FAO's FAOSTAT database (FAO, 2000). All the variables are in logarithms.

The database consists of time series data for 112 countries over the period 1960 to 1998, although for a number of variables the data are only available at 5 yearly intervals. There are five sub-samples; an 80-country developing world sample, a 21-country OECD sample, a 16-country Asia sample, a 39-country Africa sample and a 24-country Latin America and Caribbean sample. This last sample is arguably the least convincing of the five since it encompasses South America, Central America and the Caribbean islands, regions that contain countries that may be geographically proximate but are arguable economically diverse. Alternative samples were explored, although in no cases were countries added to or dropped from samples on the basis of the econometric estimates. The most notable omissions from the sub-samples are the former communist block eastern and central European countries and a few middle eastern economies. In both cases the sample sizes were too small to generate meaningful results.[11] The countries and the sub-samples are detailed in Appendix II.

Econometric Method

As explained in section 3 above, the model is comprised of a system of two equations, a structural growth equation based on the augmented Solow model (6) and a reduced form health equation (8) used to estimate the effect of HIV on the chosen measure of health capital. There are three main econometric issues for consideration. First, the health capital term $\left(x_{it}^{4}\right)$ is an endogenous variable in the growth equation (6) and thus is contemporaneously correlated with the errors $\left(v_{it}^{1}\right)$, resulting in biased and inconsistent estimates via OLS. Second, the health (8) and growth (6) equations are both dynamic models containing lagged dependent variables. The presence of a lagged dependent variable also results in biased and inconsistent parameter estimates via OLS. And third, given the panel nature of the data, individual country heterogeneity must be dealt with in the estimation procedure; failure to

11 Consideration was given to a European sample but it was concluded that this was more a geographic than an economic construct.

account for unobservable heterogeneity imposes invalid restrictions on the coefficient estimates. These three issues are interdependent; the approach adopted has the primary aim of producing consistent estimates of the effect of HIV/AIDS on growth, while avoiding unnecessary complexity in estimation and also retaining the theoretical interpretation of the parameters of the augmented Solow growth model.

Instrumental variables 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 dependent variables in both the growth and health equations. In the first stage a reduced form health equation (8) with health capital as a function of exogenous variables and a lagged dependent variable is used to obtain predicted values for the health capital variable $\left(\hat{x}_{it}^{4}\right)$. Instrumental variable regression is also employed at this stage to overcome the problems with the lagged dependent variable in the health equation. Here the lagged dependent variable is instrumented using the exogenous variables in the model and further lags on the health term. In the second stage the predicted values of the health capital variable ($\hat{x}_{it}^{4}$ from equation 8) are used to replace the health term $\left(x_{it}^{4}\right)$ in the growth equation (6), thus dealing with the endogeneity of health capital in this model. The growth equation is also estimated via the instrumental variable approach in order to deal with the lagged dependent variable.

Breusch-Pagan (BP) (Breusch and Pagan, 1980) and Hausman (H) (Hausman, 1978) tests are used to assess the need for country specific effects. If the Breusch-Pagan test is rejected time invariant country specific effects are included to account for unobservable heterogeneity. The Hausman tests confirm that fixed effects are the appropriate specification in each case. Where country specific effects are required the dynamic panel data model is estimated using the technique proposed by Arellano and Bond (1991). First differencing removes the time invariant country specific effects producing an equation that is estimable using instrumental variables. The Arellano and Bond generalised method of moments estimator employs lagged levels of the dependent variable and the predetermined variables and differences of the strictly exogenous variables to instrument the lagged dependent variable. This estimator has been shown to perform well in macro panels of this type (Judson and Owen, 1999). Where country specific effects are deemed unnecessary by the BP test, the data are pooled and the models are estimated by a standard instrumental variable approach. Again, lagged levels of the dependent variable and predetermined variables, as well as the strictly exogenous variables, are used as instruments for the lagged dependent variable.

It is not possible to test empirically whether the instruments are exogenous. However, given that we have more instruments than are needed to identify each equation we employ the Sargan (1958) test for over-identifying restrictions to test the conditional validity of the additional instruments. Failure to reject the null hypothesis means we can have ‘some

confidence' in the overall set of instruments used (Wooldridge, 2002: p123). In addition, for the standard instrumental variables regressions a generalised Hausman test is used to test for inconsistency of OLS estimates and thus confirm the need for the instrumental variable approach.

Given the strong theoretical basis underlying the growth model, variables have been retained in the growth equations even when the empirical results reveal them to be insignificant or where signs are contrary to theoretical expectations. Since the health equation is a reduced form the results reported here were derived using a general to specific approach to find the most parsimonious model that explains the chosen health capital variable in each sample, with variables retained only if the coefficients were significant at 5%. The full set of variables initially included lagged health (the lagged dependent variable, $\ln INF_{t-1}$), HIV prevalence (*HIV*), current income ($\ln YC$), lagged income ($\ln YC_{t-1}$), the proportion of the population at risk of malaria ($\ln MAL$), protein intake ($\ln PRO$), the secondary school enrollment rate ($\ln SEN$); in addition interactions of HIV prevalence, protein intake, risk of malaria, school enrollment rate with income and squared terms on each explanatory variable were also included. The health equations were estimated using both infant mortality and life expectancy as the dependent variable.

All the estimates were derived using Stata v.8. A table of variable definitions and data sources is given in Appendix I.

Results

The models are estimated using five yearly average panel data for six samples for the period 1960-1998 (eight time points). The results are reported in Table 2. The upper part of the table reports the results for the growth equation (6), where the dependent variable is the level of income per capita at the end of each five-year period ($\ln z_{it}$). The lower half shows the results for the reduced form health equation; the dependent variable in this case is the log of infant mortality ($\ln INF$). The predicted values from the health equation are then used as the health variable term in the growth equation (*pred* $\ln INF$).

Table 2 Results from growth models (using infant mortality)
About Here

Country specific fixed effects are included in the health and growth equations for the World and Developing World samples, as signified by the results of the BP and H tests. They are also included in the growth equation for the Asia sample; all other equations are estimated using pooled data. All equations include time specific effects where these were significant at 5 percent, but they are not reported here. All equations except the growth equation for the

World sample pass the Sargan test for over-identifying restrictions (although the result for the health equation for Latin America is marginal). For all of the pooled equations a generalised Hausman test shows that OLS is an inconsistent estimator in this context, which supports the use of the instrumental variable approach[12].

Consider first the reduced form health equations in the lower half of the table. The lagged dependent variable ($\ln INF_{t-1}$) is strongly significant in all cases. For the World, Asia and Africa samples lagged income ($\ln YC_{t-1}$) is also negatively related with infant mortality. In the Africa sample the proportion of the population at risk of malaria ($\ln MAL$) exerts a positive influence on infant mortality while protein intake ($\ln PRO$) has a negative influence. HIV prevalence is found to have a significant positive effect on infant mortality in the World, Developing World, Africa and Latin America; although the coefficient in this latter sample seems unfeasibly large.

The growth equation results return the expected positive and significant coefficients for the savings/investment variable ($\ln I$) in all samples except for Latin America & Caribbean, where it is insignificant. All the samples return positive and strongly significant coefficients on the lagged income term, which indicates that convergence is taking place, with the convergence rates given by the parameter, λ. The coefficient for 'capital widening'/'workforce growth' ($\ln(n+g+\delta)$) is positive and significant for the World and OECD samples, which is contrary to expectations, but negative and significant in the Asia sample; elsewhere it is insignificant. The coefficient on the education capital proxy ($\ln SEN$) is insignificantly different from zero in all cases. This finding is robust to the omission of the health capital term.

The effects of health capital on growth, through the predicted infant mortality term (*pred* $\ln INF$), are uniformly negative, and are strongly significant for the World, Developing World, Africa and Latin America samples. These results are robust to the omission of the education capital term, except for Latin America, where health capital is not significant when education capital is omitted from the growth equation. In all cases where health capital has a significant effect on growth the corresponding effects of HIV on infant mortality are significant and positive.

The marginal impact of HIV prevalence and malaria on growth can be estimated from the coefficients; in all cases the signs are consistent with expectations. These impact estimates are reported as the rows '*HIV Impact*' and '*Malaria Impact*' in Table 2; the interpretation is that the impact estimates are the percentage change in income per capita for a one percent change in the HIV prevalence rate or the proportion of the population at risk to malaria. Since HIV

12 No equivalent test is available for the dynamic panel models estimated using the Arellano and Bond procedure.

and malaria have no statistically significant effects on infant mortality for the OECD and Asia samples the impacts on growth are also zero. Elsewhere the HIV impacts are negative, with Africa recording an average reduction of 0.59 percent in income per capita for one percent increase in HIV prevalence, while the World and Developing World sample indicate negative impacts of 0.05 and 0.08 percent. The very large impact of HIV prevalence in the Latin American sample is overwhelmingly a consequence of the large coefficient on HIV in the infant mortality equation, whose reliability we have statistical reason to doubt. The malaria impact is also negative for Africa, where a one percent increase in the proportion of the population at risk has a marginal impact of 0.37 percent on income per capita.

The results using life expectancy (LE) as the health variable are not shown here. This alternative formulation resulted in no substantive differences to the results reported in Table 2. In particular, for the growth equations the estimated coefficients on *predLE* are almost equal and opposite to those reported for *pred*ln*INF,* hence the estimated marginal impacts of HIV prevalence are also very similar.

5. Discussion

The health capital and growth estimating equations confirm the importance of investigating heterogeneity across countries in macro econometric analyses. The results obtained during this analysis for the health capital equation are broadly consistent with expectations. The lack of any significant determinants of infant mortality other than lagged infant mortality in the OECD sample is consistent with an argument that above a certain income level the less important are the other/general economy wide factors for infant mortality.[13] In the other samples, where incomes per capita are lower, there is some evidence that income levels in previous periods do have positive effects upon infant mortality, which is also consistent with expectations, but this is made less clear cut by the lack of significant coefficients for the Developing World sample. This conclusion is further reinforced by the results for the poorest region – Africa – where the effect of nutrition upon infant mortality is strongly negative, which is again consistent with expectations. Somewhat less satisfying is the fact that the interaction terms between nutrition levels and malaria with HIV prevalence were not significant, which may be contrary to expectations. The literature indicates that susceptibility to HIV infection increases as the general health status declines but that the onset of full blown AIDS is faster the less good is the general state of health; these results do not disprove this since we would expect these two features of the relationship between general health states and HIV to produce counteracting effects upon infant mortality.

13 One dimension that would have been useful to include in the health equations is income distribution, but adequate data are not available. If the income distribution effects are time invariant country-specific effects they are removed by a fixed effects specification, this is only relevant for the World and Dev eloping World samples (and growth for Asia).

The impact of HIV upon infant mortality is positive and strongly significant in the World, Developing World and Africa samples but does not have significant effects in Asia or the OECD sample. It is expected that the infant mortality is more closely related to the HIV prevalence rate among women than the general population. Since the characteristics of the HIV epidemic are different in the OECD and Asia where the ratios of women to men infected are very much lower than in Africa, these results are consistent with expectations. The impact of malaria is also positive and strongly significant for the Africa, but for no other sample. This is consistent with expectations given the greater proportions of the population at risk to malarial infection and the lower income levels.

The weakest results are those for the Latin America and Caribbean sample. Neither the health capital nor the growth equation for this sample perform well compared to the equations for the other samples, and these results produce an unrealistic estimate for the marginal impact of HIV of 3.58. The origins of this apparently overlarge estimate are in the health capital equation where the lagged infant mortality effect is not ameliorated by the lagged income term and hence the impact of the large coefficient on HIV is not damped down as much as for the other samples. It is suspected that this is an inappropriate sample wherein the objective of grouping similar countries has not been achieved; indeed there are reasons to believe that this might be the case in the HIV data where all those countries with prevalence rates greater than 1 percent are in the Caribbean basin while rates in South America peak at 0.67 percent (Argentina). Moreover the fact that this sample only marginally passes the Sargan test suggests that the instruments may not be ideal. So as to check that this sample of countries did not have any adverse implications for the results from the World and Developing World samples, of which they are subsets, the equations for a second pair of World and Developing World samples that excluded Latin America and the Caribbean were also estimated. These are reported in Table AIII in Appendix III. The only notable effect is that the coefficient on the predicted infant mortality term in the growth equations for the second World sample (World 2) is not quite significant. Because of doubts about the Latin America and the Caribbean sample the subsequent discussion does not refer explicitly to the results for that sample.

The performance of the growth equation is interesting. In all cases, except for the Latin America and Caribbean sample, investment (I) in physical capital has a positive and significant effect that is roughly inversely related to income levels, which is in accordance with expectations and fully consistent with the evidence of convergence that is recorded by the positive signs on λ. The conditional convergence evidence is also consistent with expectations and the lagged income terms are all positive and significant. The degree of convergence is greater in the World and Developing World samples than in the OECD, Asia and Africa samples, which reflects the wider (relative) variations in incomes per capita in the

first two samples than in the latter three samples. However there is no evidence that investment in education capital through secondary school enrollment has the expected positive effect on growth, although this is a typical result for this variable in this type of cross country study (Bhargava *et al*. 2002; McDonald and Roberts 2002; Knowles and Owen, 1995; Islam, 1995). Despite this being a common result it is disturbing since it so strongly contradicts expectations; however it is a variable for which it is difficult to obtain truly satisfying data. Clearly education is not a perfect measure of the skill component of human capital and hence any education based measure is questionable, but it is very difficult to define precisely other measures of skills. It may also be argued that it is the skill stock of the current workforce that is relevant since this should directly influence the flow of skilled labour services currently available, whereas measures of investment in the skills of a future workforce, for which school enrolment rates are proxies, may be misleading. However use of a stock measure[14] in this case made no substantive difference to the results and hence the coefficients for the secondary school rates were reported.

The impact of predicted infant mortality on income is always negative, which is consistent with expectations that increases in health capital have positive implications for income, although the coefficients are insignificant for the OECD and Asia samples. But, there are reasons to believe that health capital may be less important in relatively wealthy economies and this is supported by the evidence that the lagged infant mortality and income variables returned significant coefficients in the health capital equations for the OECD and Asia samples while the coefficients on the income variables for the Asia sample were also significant. For the World, Developing World and Africa samples the negative infant mortality coefficients are all strongly significant, and, since the HIV variable returned significant effects in the health equations, it means that for these samples it is possible to estimate the marginal impact of HIV on incomes. Given the signs on the coefficients these are all negative effects. Moreover it is interesting to note that in all samples where HIV had a significant impact on life expectancy in the health capital equation, predicted infant mortality had negative and significant effects on income in the growth equation. *A priori* it would be expected given the HIV prevalence rates that the negative impact of HIV would be greater in Africa than the Developing World samples and greater in the Developing World than the World samples; these expectations are fulfilled by the results. Furthermore the estimated impact effects suggest that the marginal impact of HIV on incomes may increase with prevalence rates, which is an intuitively reasonable conclusion.

14 The stock measure was average years of schooling (Barro and Lee, 2000).

6. Concluding Comments

The pronounced impact of HIV/AIDS prevalence on health capital/infant mortality, and hence on incomes, suggests that the epidemic may now be entering a stage where the loss of life is starting to impact appreciably upon both social and economic interactions. These results suggest that the seemingly comforting conclusion that excess labour supplies in many developing countries would be sufficient to ensure that the macroeconomic impacts of the HIV/AIDS epidemic might be relatively muted does not appear to be holding. However, there are difficulties with this type of broad brush analyses; especially the dependence of the model specification upon the presumption of steady state relationships. In particular the results indicate that there are substantial differences between regions, which points to the importance of a careful consideration of samples and sub-samples in order to gain a fuller picture of the relationship between HIV/AIDS and economic performance. Among the least satisfactory aspects of the results is the absence of significant coefficients on the education capital terms in the growth equations; while this is common in the literature it continues to be unsettling. The results also indicate the need for further explorations of how the epidemic impacts upon economic relationships, so as to direct policy makers' attention to the economic implications of AIDS. In the longer term it is argued that there is a need for research that provides a greater understanding of the mechanisms by which sustained epidemics impact upon economic performance. Such a research agenda will necessitate the development of forward looking models that provide insights into how, *inter alia*, the large numbers of AIDS orphans will impact upon the accumulation of education capital (Bell *et al*., 2003, may provide a start for this type of research).

Nevertheless, these results produce useful additional information and indicate that the macroeconomic affects of the HIV/AIDS epidemic are substantial. In the countries of the Africa sample, where HIV and malaria are health problems on a scale that OECD countries might regard as catastrophic, the level of the marginal impacts of HIV prevalence on income pre capita are of orders of magnitude that suggest that a substantial proportion of the apparently poor economic performance of many of these economies over the past 10 to 20 years can be attributable to the HIV epidemic; a characteristic that has been omitted from many macroeconometric studies that have sought to explain economic performance in many developing countries. Moreover the marginal effects are sufficiently large to suggest that they threaten macroeconomic stability, which is generally most fragile in the poorer countries that are also those most prone to catastrophic escalations of the epidemic. If the legitimate aspirations of the inhabitants of these countries are to be achieved and poverty reduction strategies, and similar policies, in these countries are to be realised, the maintenance of macroeconomic stability is likely to be a prerequisite. All of which indicates that over and above the humanitarian case for supporting healthcare provision and services in countries

suffering from the HIV/AIDS pandemic, there is a strong case for providing more general economic support.

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Table 1 **HIV Prevalence Data** (unweighted percentage rates for populations 15-49)

		1984	1989	1994	1999
World	Average	0.09	1.08	2.17	3.26
	Std Deviation	0.39	2.16	4.32	6.49
	Max	3.52	12.04	24.09	36.13
Developing World	Average	0.13	1.51	3.03	4.56
	Std Deviation	0.46	2.45	4.89	7.36
	Max	3.52	12.04	24.09	36.13
OECD	Average	0.03	0.09	0.15	0.22
	Std Deviation	0.04	0.09	0.13	0.18
	Max	0.13	0.29	0.45	0.61
Asia	Average	0.00	0.08	0.18	0.28
	Std Deviation	0.00	0.17	0.35	0.52
	Max	0.00	0.70	1.40	2.10
Africa	Average	0.21	2.83	5.73	8.63
	Std Deviation	0.66	3.00	5.96	8.96
	Max	3.52	12.04	24.09	36.13
Latin America	Average	0.08	0.41	0.74	1.07
	Std Deviation	0.09	0.45	0.82	1.20
	Max	0.34	1.91	3.51	5.11

Source: Authors calculations from Walker (2000)

Table 2 Results from growth models (using infant mortality)

	World i = 112 n = 684	Developing World i = 80 n = 518	OECD i = 21 n = 109	Asia i = 16 n = 107	Africa i = 39 n = 259	Latin Am& Caribbean i = 24 n = 164
Income (ln*YC*)						
ln*I*	0.219*	0.238*	0.091*	0.245*	0.168*	-0.024
	(0.035)	(0.030)	(0.044)	(0.091)	(0.022)	(0.064)
ln*(n+g+d)*	0.182*	0.004	0.153*	-0.667*	0.001	0.180
	(0.087)	(0.094)	(0.079)	(0.254)	(0.103)	(0.170)
ln*SEN*	-0.012	-0.045	0.003	-0.147	0.016	0.073
	(0.037)	(0.024)	(0.072)	(0.098)	(0.018)	(0.051)
pred ln*INF*	-0.130*	-0.178*	-0.056	-0.131	-0.092*	-0.087*
	(0.059)	(0.059)	(0.037)	(0.094)	(0.046)	(0.043)
$\ln YC_{t-1}$	0.653*	0.631*	0.869*	0.829*	0.908*	0.966*
	(0.064)	(0.094)	(0.029)	(0.084)	(0.027)	(0.037)
λ	0.053	0.058	0.018	0.023	0.012	0.004
HIV Impact	-0.050	-0.082	0.000	0.000	-0.585	-3.582
Malaria Impact	0.000	0.000	0.000	0.000	-0.366	0.000
R^2	N/A	N/A	0.96	N/A	0.96	0.94
Sargan	39.64	27.72	2.247	11.37	0.174	5.44
	[.023]	[.148]	[.523]	[0.979]	[.676]	[.365]
BP	19.91	7.53	1.16	13.32	0.22	0.21
	[.000]	[.006]	[.282]	[.000]	[.638]	[.645]
H	65.04	60.49		200.40		
	[.000]	(.000)		[.000]		
Infant Mortality (ln*INF*)						
$\ln INF_{t-1}$	0.805*	0.888*	0.877*	0.998*	0.885*	0.920*
	(0.043)	(0.039)	(0.032)	(0.027)	(0.038)	(0.029)
$\ln YC_{t-1}$	-0.044*			-0.088*	-0.074*	
	(0.25)			(0.027)	(0.014)	
HIV	0.024*	0.019*			0.024*	0.112*
	(.004)	(.004)			(.004)	(.042)
ln*MAL*					0.015*	
					(.006)	
ln*PRO*					-0.119*	
					(0.037)	
R^2			0.96	0.98	0.94	0.94
Sargan	49.56	36.26	3.555	4.242	6.638	10.92
	[.333]	[.847]	[.615]	[.236]	[.084]	[.053]
BP	15.14	10.15	1.17	0.21	2.01	0.26
	[.000]	[.001]	[.280]	[.645]	[.156]	[.612]
H	69.12	174.42				
	[.000]	[.000]				

The variable *pred ln(INF)* in the growth equation are the predicted values from the health equation shown in the lower panel.
Where both Breusch-Pagan (BP) and Hausman (H) tests are rejected, country specific fixed effects are included. Where the BP test is not rejected the estimates are from simple pooled data.
Time specific effects are included if significant at 5%. Time specific and country specific effects are not reported here.
* denotes significance at 5%
Where individual effects are required the equations are estimated using the XTABOND procedure in STATA v.8. Pooled models are estimated using IVREG.

Appendix 1

Variable Definitions and Data Sources

Variable	Definition	Source
YC	Real GDP per capita	World Bank Macroeconomic Series – updated PWT data
I	Share of real GP invested	Global Development Finance & World Development Indicators
(n + g + d)	n = rate of growth of population g = rate of growth of technology d = depreciation rate of all capital stocks	World Bank Macroeconomic Series – updated PWT data
SEN	Secondary school enrollment rate (gross)	World Data, (World Bank 2001)
INF	Infant mortality	World Data, (World Bank 2001)
LE	Shortfall in life expectancy	World Data, (World Bank 2001)
HIV	HIV prevalence rate	Derived from UNAIDS figures by Walker (2000)
MAL	Proportion of population living in areas where malaria is prevalent	Data from Centre for International Development (Hamoudi and Sachs, 1999).
PRO	Protein per Capita per day (gms)	FAO

Appendix II Samples

Country	Groups	Country	Groups	Country	Groups
United Arab Emirates	DW, AS	United Kingdom	O	Namibia	DW, AF
Argentina	DW, LAC	Ghana	DW, AF	Niger	DW, AF
Australia	O	Gambia, The	DW, AF	Nigeria	DW, AF
Austria	O	Guinea-Bissau	DW, AF	Nicaragua	DW, LAC
Burundi	DW, AF	Greece	O	Netherlands	O
Belgium	O	Guatemala	DW, LAC	Norway	O
Benin	DW, AF	Guyana	DW, LAC	Nepal	DW, AS
Burkina Faso	DW, AF	Hong Kong, China	DW, AS	New Zealand	O
Bangladesh	DW, AS	Honduras	DW, LAC	Pakistan	DW, AS
Bulgaria		Haiti	DW, LAC	Peru	DW, LAC
Belize	DW, LAC	Hungary		Philippines	DW, AS
Bolivia	DW, LAC	Indonesia	DW, AS	Papua New Guinea	DW , AS
Brazil	DW, LAC	India	DW, AS	Poland	
Barbados	DW, LAC	Iran, Islamic Rep.	DW, AS	Paraguay	DW, LAC
Botswana	DW, AF	Iceland	O	Romania	
Central African Republic	DW, AF	Israel		Rwanda	DW, AF
Canada	O	Italy	O	Saudi Arabia	
Switzerland	O	Jamaica	DW, LAC	Sudan	DW, AF
Chile	DW, LAC	Japan	O	Senegal	DW, AF
China	DW, AS	Kenya	DW, AF	El Salvador	DW, LAC
Cote d'Ivoire	DW, AF	Korea, Rep.	DW, AS	Suriname	DW, LAC
Cameroon	DW, AF	Kuwait		Sweden	O
Congo, Rep.	DW, AF	Sri Lanka	DW, AS	Swaziland	DW, AF
Colombia	DW, LAC	Lesotho	DW, AF	Syrian Arab Republic	
Costa Rica	DW, LAC	Luxembourg	O	Chad	DW, AF
Cyprus		Latvia		Togo	DW, AF
Czech Republic		Morocco	DW, AF	Thailand	DW, AS
Denmark	O	Madagascar	DW, AF	Trinidad and Tobago	DW, LAC
Dominican Republic	DW, LAC	Mexico	DW, LAC	Tunisia	DW, AF
Algeria	DW, AF	Mali	DW, AF	Turkey	
Ecuador	DW, LAC	Malta	O	Uganda	DW, AF
Egypt, Arab Rep.	DW, AF	Myanmar	DW	Uruguay	DW, LAC
Spain	O	Mozambique	DW, AF	United States	O
Finland	O	Mauritania	DW, AF	Venezuela	DW, LAC
Fiji	DW, AS	Mauritius	DW, AF	South Africa	DW, AF
France	O	Malawi	DW, AF	Congo, Dem. Rep.	DW, AF
Gabon	DW, AF	Malaysia	DW, AS	Zambia	DW, AF
				Zimbabwe	DW, AF

DW – Developing World; O – OECD; AS – Asia; AF – Africa; LAC – Latin America and Caribbean.

Appendix III

Table AIII Additional Results From Growth and Health Capital Models

	World (2) i = 92 n = 532	Developing World (2) i = 56 n = 518		World (2) i = 92 n = 532	Developing World (2) i = 56 n = 518
Income			**Infant Mortality**		
ln(I)	0.249*	0.252*	$ln(INF)_{t-1}$	0.847*	0.977*
	(0.041)	(0.036)		(0.045)	(0.043)
ln(n+g+d)	0.216*	-0.016	$ln(YC)_{t-1}$	-0.046*	
	(0.096)	(0.107)		(0.27)	
ln(SEN)	-0.054	-0.060	*HIV2*	0.026*	0.020*
	(0.040)	(0.036)		(.004)	(.005)
pred ln(INF)	-0.117	-0.200*	*ln(MAL)*		
	(0.068)	(0.074)	*ln(PRO)*		
$ln(YC)_{t-1}$	0.643*	0.667*			
	(0.068)	(0.095)			
λ	0.055	0.051			
HIV Impact	-0.062	-0.522			
R^2			R^2		
Sargan	34.58	22.86	*Sargan*	45.39	23.16
	[.043]	[.351]		[.497]	[.281]
BP	8.10	3.71	*BP*	25.24	9.07
	[.004]	[.054]		[.000]	[.003]
H	140.89	157.96	*H*	65.43	28.20
	[.000]	(.000)		[.000]	[.000]

Appendix IV

If the labour forces grow at the constant rates n_i, and technologies advance at period specific constant rates g_t i.e., the growth rates of labour and technology are country- and time-period specific, and the physical, education and human capital stocks depreciate at the same constant rate, δ, then

$$\begin{aligned} K_{it} &= I^{K}_{i,t-1} + (1-\delta)K_{i,t-1} \\ E_{it} &= I^{E}_{i,t-1} + (1-\delta)E_{i,t-1} \\ H_{it} &= I^{H}_{i,t-1} + (1-\delta)H_{i,t-1} \end{aligned} \quad . \qquad \text{(A1.1)}$$

If savings are divided between physical, education and human capital accumulation, i.e., education and health capital accumulation is treated as an investment activity, such that

$$s_{it} = s_{it}^K + s_{it}^E + s_{it}^H = \frac{S_{it}}{Y_{it}} = \frac{I_{it}}{Y_{it}} = \frac{I_{it}^K + I_{it}^E + I_{it}^H}{Y_{it}}. \tag{A1.2}$$

then the rates of physical, education and health capital growth per unit of labour are defined as

$$\begin{aligned} \hat{k}_{it} &= s_{it}^K \hat{y}_{it} - (n_i + g_t + \delta)\hat{k}_{it} \\ \hat{e}_{it} &= s_{it}^E \hat{y}_{it} - (n_i + g_t + \delta)\hat{e}_{it} \\ \hat{h}_{it} &= s_{it}^H \hat{y}_{it} - (n_i + g_t + \delta)\hat{h}_{it} \end{aligned} \tag{A1.3}$$

and the steady state values of physical, education and health capital, indicated by asterisks, are

$$\begin{aligned} k^* &= \left[\frac{\left(s_i^k\right)^{1-\beta-\psi} \left(s_i^E\right)^{\beta} \left(s_i^H\right)^{\psi}}{n_i + g_t + \delta} \right]^{1/(1-\alpha-\beta-\psi)} \\ e^* &= \left[\frac{\left(s_i^k\right)^{\alpha} \left(s_i^E\right)^{1-\alpha-\psi} \left(s_i^H\right)^{\psi}}{n_i + g_t + \delta} \right]^{1/(1-\alpha-\beta-\psi)} \\ h^* &= \left[\frac{\left(s_i^k\right)^{\alpha} \left(s_i^E\right)^{\beta} \left(s_i^H\right)^{1-\alpha-\beta}}{n_i + g_t + \delta} \right]^{1/(1-\alpha-\beta-\psi)} \end{aligned} \tag{A1.4}$$

and therefore

$$\begin{aligned} \ln k_i^* &= \frac{1}{(1-\alpha-\beta-\psi)} \left[\ln\left(\left(s_i^k\right)^{1-\beta-\psi} \left(s_i^E\right)^{\beta} \left(s_i^H\right)^{\psi} \right) - \ln\left(n_i + g_t + \delta\right) \right] \\ \ln e_i^* &= \frac{1}{(1-\alpha-\beta-\psi)} \left[\ln\left(\left(s_i^k\right)^{\alpha} \left(s_i^E\right)^{1-\alpha-\psi} \left(s_i^H\right)^{\psi} \right) - \ln\left(n_i + g_t + \delta\right) \right]. \\ \ln h_i^* &= \frac{1}{(1-\alpha-\beta-\psi)} \left[\ln\left(\left(s_i^k\right)^{\alpha} \left(s_i^E\right)^{\beta} \left(s_i^H\right)^{1-\alpha-\beta} \right) - \ln\left(n_i + g_t + \delta\right) \right] \end{aligned} \tag{A1.5}$$

Incorporating physical, education and health capital the augmented steady state output per capita is

$$\begin{aligned} \ln y_{it}^* = \ln A_{i0} + g_t t &- \frac{\alpha+\beta+\psi}{(1-\alpha-\beta-\psi)} \ln\left(n_i + g_i + \delta\right) \\ &+ \frac{\alpha}{(1-\alpha-\beta-\psi)} \ln s_i^k + \frac{\beta}{(1-\alpha-\beta-\psi)} \ln s_i^E + \frac{\psi}{(1-\alpha-\beta-\psi)} \ln s_i^H \end{aligned} \tag{A1.6}$$

which is equation 3 in the main text.