LUND UNIVERSITY
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Master in Economic Development and Growth

Population Health and Economic Growth: Panel Cointegration Analysis in Sub-Saharan Africa

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Abstract: The economic performance in Sub-Saharan Africa has been unimpressive, particularly in comparison with other developing regions like East Asia. Using a panel data for 30 Sub-Saharan African countries for the period 1970-2010, this study investigates the extent to which the health of the population affects the economic performance in the region. Employing the theoretical model based on an augmented Solow growth model; we estimate the relationship between population health capital and economic growth in SSA using the newly developed panel cointegration econometric strategy. We find that, health status of the population has not significantly driven economic performance. Accounting for the effect of HIV/AIDS, however resulted in a significant negative effect of population health on economic growth. We find further that, the obverse seems rather plausibly the case, as economic growth significantly increases life expectancy in the region. The results suggest that, other factors affecting growth in the region would indirectly improve on the health status of the population of SSA.

Key words: Population health, economic growth, panel cointegration

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DEDICATION

This work is in loving memory of my late father (Mr. Charles B. Frimpong) who passed away in the middle of this programme. It's sad you could not see me rise to this end. It is my utmost hope that, your lessons you taught me, inexhaustible encouragement to pursue my own dreams, and the unending opportunities to succeed will surely come to pass. May your soul rest in perfect peace!

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Suffice to sum up by requesting whoever accorded any form of assistance in the accomplishment of this study, to accept my thanks with utmost sincerity. Nevertheless, for any mistakes, inaccuracies and inadequacies, substantial or marginal which may be found in this work, I am fully answerable and I bear total responsibility.

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CHAPTER ONE

INTRODUCTION

1.1 Background

In recent years, the excruciating state of health in Sub-Saharan Africa (SSA) which has caught global attention has increased dramatically. Efforts have been made to combat major health problems in the region and some marked improvements have been made as a consequence. Nonetheless, the region is still lagging particularly on many of the Millennium Development Goals (MDGs), a set of goals agreed upon by world leaders at a UN summit in 2000 to reduce poverty, improve health, and foster economic development (United Nations, 2007). Despite the efforts to combat some major health problems, the health of the vast majority of people in SSA remains in jeopardy; as millions are still suffering from diseases that are relatively simple to treat.

Most African countries, especially those within the SSA region have registered dismal or negative economic growth as measured by changes in the gross domestic product (GDP) per capita. Table 1 presents a sample of SSA countries on a number of indicators for the period 1970 – 2010. These countries are included in the sample because they represent a number of significant economic structures. In other words, although they share patterns of early growth followed by periods of decline or stagnation, there is a large degree of heterogeneity in the overall growth performances.[1] As evident from a sample of SSA countries presented in Table 1, one-third of the sample experienced any reasonable average annual rate of growth in GDP per capita of over 1 percent a year during the 1970 – 2010 period. Also, one-third of the sample experienced negative growth within the same period while the remaining experienced creeping growth rates. Within the same period under consideration, many of these countries registered low human development index. Average life expectancy in SSA is currently 54 years, up from 44 years in 1970, which is still the lowest of any region in the world and far behind the average of nearly 70 years for the 135 countries in the Human Development Index (HDI) trends analysis (World Bank, 2011).

Furthermore, in the 1980s and 1990s, most countries in the region found it even more difficult to deal effectively with their poor economic performance. In fact, Angola, Burundi, Cameroon, Republic of Congo, Sierra Leone, Democratic Republic of Congo/Zaire, and Zambia registered

[1] These countries are considered in all empirical investigations in this study because data is also readily available on all variables used in the estimations.

Table 1: Key Economic and Human Development Indicators in Selected Sub-Saharan African Countries

Country	GDP per capita US$ 2000 in 2010	Average Annual rate of growth of GDP per capita, 1970-2010 (%)	Health Index	Human Development Index (2010)
Benin	377	0.5	0.562	0.425
Botswana	4188	6.2	0.525	0.631
Burkina Faso	276	1.8	0.552	0.329
Burundi	115	-0.2	0.472	0.313
Cameroon	709	1.0	0.49	0.479
Central African Rep.	240	-0.9	0.436	0.339
Chad	276	0.9	0.461	0.326
Congo DR	104	-2.8	0.583	0.528
Congo Republic	1253	1.8	0.443	0.282
Cote d'Ivoire	591	-1.1	0.547	0.401
Gabon	4176	1.2	0.667	0.67
Gambia	355	0.6	0.602	0.418
Ghana	358	0.6	0.692	0.533
Guinea Bissau	161	-0.1	0.437	0.351
Kenya	467	1.2	0.577	0.505
Lesotho	482	2.7	0.435	0.446
Madagascar	243	-1.2	0.733	0.481
Malawi	184	1.0	0.529	0.395
Mali	270	1.6	0.489	0.356
Mauritius	5175	3.5	0.839	0.726
Niger	180	-1.1	0.54	0.293
Rwanda	338	1.9	0.554	0.425
Senegal	561	0.1	0.615	0.457
Sierra Leone	268	0.2	0.432	0.334
South Africa	3745	0.5	0.509	0.615
Swaziland	1556	2.6	0.448	0.52
Togo	285	-0.3	0.578	0.433
Uganda	377	2.7	0.531	0.442
Zambia	432	-0.7	0.45	0.425
Zimbabwe	325	-1.1	0.473	0.364
Sub-Saharan Africa	640	0.4	0.536	0.46

Source: World Bank (2011). *World Development Indicators*. Washington, DC: The World Bank; UNDP (2011). *Human Development Report*. New York: UNDP.

relatively high rates of economic decline during the period 1987–1997 (Elu, 2000; World Bank (various years)). Several factors have been attributed to the poor economic performance in SSA such as: political violence, ethnic wars and diseases such as HIV/AIDS, lack of both human and physical capital, poor governance and corruption, poorly developed infrastructures, and non-competitiveness in international trade. Moreover, others have argued that, the economic policies by the World Bank and International Monetary Fund (IMF) have further aggravated African economic crisis – and are said to undermine the ability of SSA countries to participate effectively

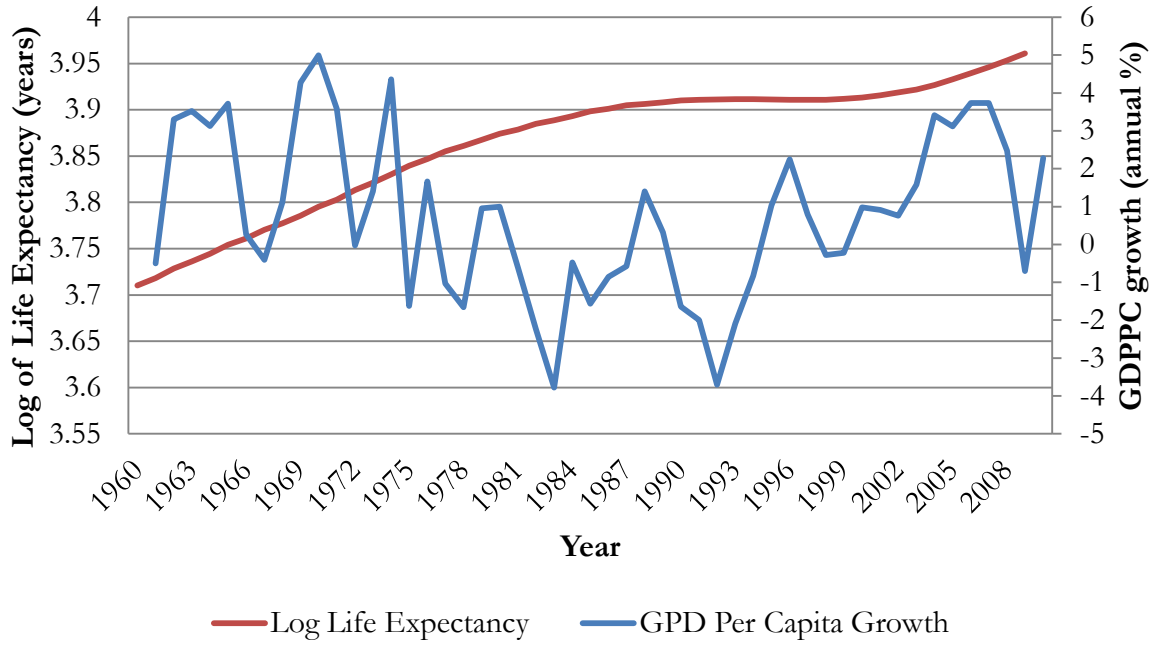
in the global economic set up (Mbaku, 1997; Mkandawire & Soludo, 1999). Many African countries have failed to achieve food security; most of them are today unable to meet the basic needs of their populations such as universal primary education and basic health guarantees. A majority of the citizens of these countries have become more impoverished, suffer from high rates of material deprivation, and face many uncertainties (Elu, 2000). "Growth in Sub-Saharan Africa rebounded sharply in 2010, supported by both the global recovery and developments on the domestic front: output is estimated to have expanded by 4.7 percent in 2010—up from the 1.7 percent in 2009—just shy of its 5 percent pre-crisis (2000-2008) average growth. Slower growth in the region's largest economy, South Africa (2.8 percent), dragged down overall regional growth in 2010. Excluding South Africa, GDP growth in Sub-Saharan Africa for 2010 is estimated at 5.8 percent, up from 3.8 percent in 2009, and above its pre-crisis average growth of 5.6 percent." (Africa's Pulse, 2011).

The analysis presented in this study is based mainly on the hypothesis that human capital as reflected in the health of the population is essential for economic growth in SSA countries. Health is one of the most important assets human beings possess, in the sense that it permits the development of human capabilities and potentialities. To be healthy is to be in a state of complete physical, mental and social well-being, and not merely the absence of infirmity or disease. Being healthy is one of the goals that are valued most by human beings. It is an undeniable fact that avoiding or alleviating illnesses, and developing and maintaining our physical and mental abilities are something that are considered an essential part of human welfare, both at the individual and social levels. Thus, improvement in health may be as important as improvement in income when thinking about development and human welfare. Though, good health is a goal in its own right, it also plays a substantial role in economic growth. Increasingly therefore, research is now showing that a healthy population is an engine for economic growth.

Figure 1 shows the trends in life expectancy and growth in GDP per capita over the period 1960 – 2010. The figure indicates that life expectancy prior to the early 1980s increased swiftly. The increase slowed down periods after 1980 and even stalled in the 1990s after which it began to pick up again. The obvious reason accounting for this trend perhaps is the incidence and prevalence of HIV/AIDS epidemic that has plagued SSA region severely. As a consequence of this epidemic as well as many other health factors, it can be conjectured that over the period under consideration, health improvements may not have affected economic growth positively as

evident in Figure 1.[2] Two main reasons for this tentative assertion seem apparent: 1) HIV/AIDS is a democratic disease in the sense that it can affect any person regardless of his/her social and economic status in a society. Thus, though no effective vaccine to cure the disease has been identified, there have been recent developments (at least in the mid 2000) of antiretroviral therapy which means those who would have afforded did not have access and consequently perished from the disease and; 2) since income inequality is much pronounced in the region, a large chunk of those who are affected by HIV/AIDS and are exposed to other diseases are involved in the subsistence sector while only a minority are formally employed or engaged in the cash crop sector to any significant extent.

Figure 1: Sub-Saharan African Life Expectancy and Real GDP per capita Growth (1960-2010)



Source: Author's construct based on data from African Development Indicators, World Bank, 2011

Figure 1 also shows vacillating trends in real GDP per capita growth in SSA within the period. However, a casual look at the two series indicates that, in periods where life expectancy was increasing sharply are associated with some pockets of high economic growth rates, albeit with fluctuations. On the converse, periods with stalled life expectancy generally are associated with low economic growth rates. However, this does not necessarily imply a causal relationship between them. The direction, strength and stability of the relationship between economic growth

[2] We test empirically this assertion in the subsequent chapters in a well-constructed theoretical model.

and population health play a significant role in designing different policies that are associated with the two variables. The direction and policy implications for the causal relationship between economic growth and population health can be classified as follows. If unidirectional causal relationship from population health to economic growth is found, it indicates that poor health of the population (which reduces labour productivity) would lead to a reduction in economic growth. On the other hand, if unidirectional causal relationship from economic growth to population health is found, then poor health has very little or no adverse impacts on economic growth. A bidirectional causal relationship implies that both variables are jointly determined and will be affected at the same time. If no causal relationship between these variables is found, the hypothesis of neutrality holds and indicates that poor health of the population will not work as an impediment to economic growth.

Four major difficulties are apparent in explaining the recent inclusion of health as a determinant of economic growth. This first is the possible existence of endogeneity between health and income. The second is the difficulties involved in finding comparable health indicators among countries that measure the multiple health dimensions of the population. The third is the issue of timing. There is a growing evidence of long-term effects of early childhood health on cognitive and physical development that affects productivity as an adult. As a consequence, health effects in the macro economy may have long time lags, given the average worker may have been born 40 or more years before, making the macroeconomic relationship difficult to estimate. The last difficulty is the differing effects of health on the economy, holding all other factors fixed, and the effect on a more general equilibrium framework where other factors respond to the improved health. Some studies measure the partial equilibrium effect whereas others attempt to capture the induced changes in other factors and the general equilibrium impact (Bloom and Canning, 2008).

1.2 Research Problem

The main engine of growth is the accumulation of human capital – of knowledge – and the main source of differences in living standards among nations is differences in human capital. Physical capital plays an essential but decidedly subsidiary role (Lucas, 1993; p. 270). Income differences are a peculiar feature across countries and even in SSA where income growth rates have been so poor over the years. For instance, according to the World Bank's data, in 2010, real GDP growth rate in Malaysia was 5.5 percent whiles it was 7.3 percent in India. In SSA, Guinea recorded with 1.9 percent, whiles it was 7.2 percent in Botswana. What factors explain such large differences in income growth rates among SSA countries? Though, the empirical literature suggest investment

in physical capital as a major source of the observed income variations in cross-country income growth rates, yet others factors could equally be important. Among these other factors, the nascent literature has emphasised on differences in endowments of human capital. Human capital, broadly defined, has several aspects, including health, education and training. Aside the direct payoffs that health improvements bring to individuals, it has also be identified to have indirect payoffs through accelerating economic growth (see e.g., Bloom and Sachs, 1998; Gallup and Sachs, 2001 and WHO, 2001). Gallup and Sachs (2001) for instance argue that eradicating malaria in SSA would increase that region's per capita growth rate by as much as 2.6 percent a year. Again, the report from the World Health Organisation states that "in today's world, poor health has particularly pernicious effects on economic development in Sub-Saharan Africa, South Asia, and pockets of high disease and intense poverty elsewhere" (WHO, 2001, p. 24) and "extending the coverage of crucial health services ... to the world's poor could save millions of lives each year, reduce poverty, spur economic development and promote global security" (ibid).

Essentially, this study explores the effects of human capital (population health) on the growth rate of per capita income in Sub-Saharan African countries during the period 1970 – 2010. Thus, by considering measures of various other controls and also by acknowledging that the joint determination of health and income can be yield incorrect results, the present study attempts to investigate a comprehensive cross-country-time-series assessment of the effects of population health on economic growth in Sub-Saharan Africa. We do so by estimating an expanded neoclassical growth equation with population health as an added regressor.

1.3 Aims and Scope

The primordial objective of the study is to investigate empirically the impact of population health on economic growth in Sub-Saharan African countries for the period 1970 to 2010. In doing so, the study specifically seeks to achieve the following:

- To examine the relative impacts of other forms of capital on the economic performance in SSA.
- To ascertain the direction of causality between health and economic growth.

1.4 Hypotheses

The study explores the following hypotheses.

Hypothesis 1: Population health may have significant positive effects on economic growth.

To test this hypothesis, we need to estimate a regression equation with real GDP per capita as the dependent variable, and a proxy variable for population health – life expectancy – and other control variables as explanatory variables. Prior to the regressions, we have to conduct panel unit root and panel cointegration tests to detect the existence of nonstationarity and of long-run equilibrium relationship and to apply proper estimation techniques for panel cointegration.

Hypothesis 2: In contrast, there may be a reverse causation or feedback effect such that economic growth causes or improves the health status of individuals.
To test this hypothesis, performing the panel cointegration estimation of a regression model with a proxy for population health as a dependent variable and real GDP per capita as an explanatory variable is required.

Hypothesis 3: There could be bi-directional relationship between population health and economic growth such that economic growth improves the health of the population and the health further stimulates economic growth.
Testing this hypothesis will involve carefully comparing the regression results from testing of the above two hypotheses.

1.5 Outline of the Study

The rest of the study proceeds as follows: chapter two will review the possible linkages between health and economic growth; and also the empirical study relating to the topic. In the last section of this chapter, we present the theoretical model explaining the impact of human capital on economic growth. In the third chapter, we thoroughly present the alternative econometric strategies used in estimating the relationship as well as the data used. Chapter four presents, analyses and discusses the empirical results whiles chapter five concludes the study with some policy recommendations.

CHAPTER TWO

LITERATURE REVIEW

2.1 The Link between Health and Economic Growth

Conceptually, health can affect economic growth through at least three mechanisms: 1) the direct link between health status and individual earnings; 2) indirectly, through the effect of health on the levels of education and; 3) through physical capital investments. Figure 2 summarizes these mechanisms through which health affects economic growth.

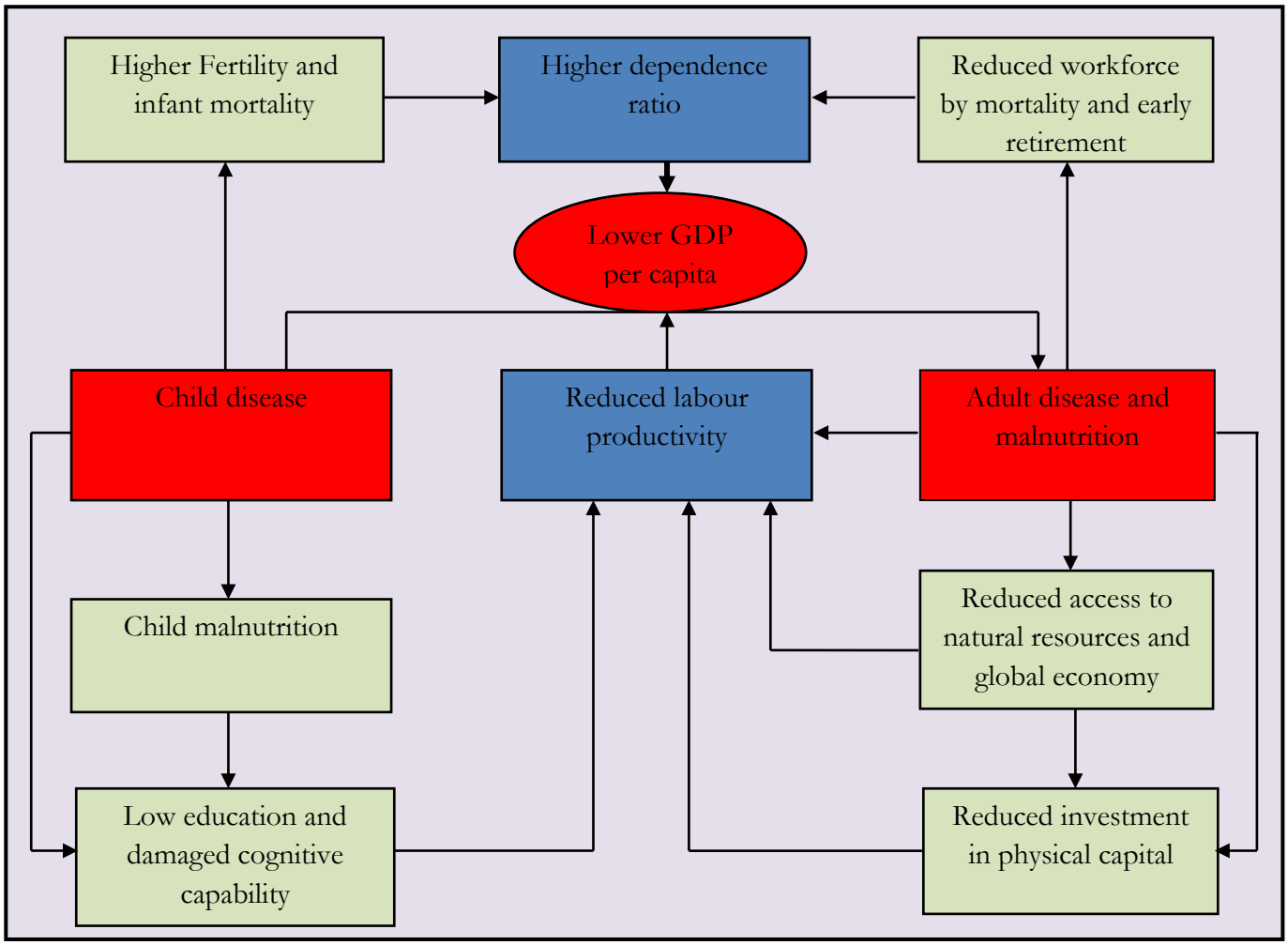
A deteriorating health status causes considerable losses in individual income by decreasing labour productivity, the number of hours worked and participation in the labour force (Luft 1975). These losses can affect the population's level of wealth and may result in lower levels of economic growth, therefore contributing to decreased social well-being.

The second mechanism operates indirectly through the levels of education. On the one hand, there is a contemporaneous effect of health on educational level since individual health status affects individuals' school attendance and learning abilities (Cutler and Lleras-Muney, 2006; Sachs, 2001). On the other hand, the relationship between health and human capital stock can even be analysed through the dynamic aspect of this relationship. The depreciation rate of human capital stock is directly related to the population's health status, and both are higher or lower depending on the technological level that contributes to improved medical interventions, access to healthcare services, demographic profiles, living and consumption habits, and criminality rates, and other factors. In societies with higher depreciation rates (for example, high mortality rates or lower life expectancy), the level of investments in education also tends to be lower, since the costs involved cannot be offset (Falcão and Soares 2005; Cutler and Lleras-Muney, 2006). From an individual perspective, lower health status decreases educational returns and thus affects family expenditures on education.

Another approach to the second mechanism emphasizes on the relationship between mortality rates and fertility rates and their effects on educational levels. According to Sachs (2001), families with higher infant mortality rates tend to also show higher fertility rates, in order to compensate for the deaths of earlier children. In general, due to risk aversion, fertility rates can even surpass mortality rates and thus reduce the amount of resources available to parents for investing in

further education. The relationships among health, education and economic growth may become even stronger by linking them to technological progress: the lower the level of human capital the less is spend on research (Howitt, 2005).

Figure 2: Links between Health and Income



Source: Prah et al., 2001 pp 619

The third mechanism relating health to economic growth has to do with the effect of health on investments in physical capital. This pathway could operate either through the savings rate (since poor health conditions shorten people's life expectancies) or through health externalities, also known as diffuse externalities, since individual health levels also depend on the average health conditions in a society (Sachs, 2001). This is the case of contagious diseases, including those that are avoidable through immunization and basic sanitation. Such externalities decrease investment levels and particularly more evident in less developed countries, where health and poverty are closely related. Countries in Africa with high rates of HIV/AIDS are examples of how health externalities can undermine economic growth. Among other factors, business investments are

very costly in these countries, partly because high rates of disease increase both the turnover of labour and rates of absenteeism and these factors raise training costs (Sachs, 2001).

2.2 Empirical Review

Grossman (1972) who provided the first formal contribution to the objective study of the determinants of individuals health status, argues that it is possible to observe a positive relationship between education and health, in particular, education has a positive impact on the likelihood of enjoying a good health because it increases the production of health technology, altering both productive efficiency and allocative efficiency. Stated alternately, he develops the demand for health model, in which it allows health capital formation, seen as a capital good, to be able to work, to earn money, and to produce domestic goods. He shows that, increase in the quantity of health capital reduces the time lost of being sick. The underlying assumption of the model is that individuals have initial endowments of health which depreciates with age and appreciates with investment in health. Some of the key findings were that, the principal determinants of health capital accumulation and the demand for medical services are wages, age and the level of education. Also, it was found that, the productive nature of health is produced when a good state of health allows a more effective performance in the job and study.

The inclusion of health as a component of human capital in economic growth specification was first suggested by Mankiw et al. (1992) who extended the Solow model. Later, a number of authors developed models to include human health capital. Barro (1996) develops a growth model including physical capital inputs, level of education, health capital, and the quantity of hours worked in a Ramsey growth setting/fashion. He finds an increase in health indicators raises the incentives to invest in education and a rise in health capital lowers the rate of depreciation of health, adding there are diminishing returns to investment in health (Gallego, 2000).

Bloom et al. (2001) also extended the Solow growth model to include human capital. They used the two stage least squares technique and found a significant relationship between health capital and economic growth. However, physical capital and schooling which are also key variables were found to be insignificant thus rendering the results doubtful. For Latin America, there are series of technical research documents of public health developed by the Pan-American Health Organisation, which find a strong correlation between economic growth and the regional health, estimating regressions similar to Barro's (1996) where health is much more robust than education (Mayer et al. 2000).

Furthermore, Bloom and Canning (2000) have described how healthy populations tend to have higher productivity due to their greater physical energy and mental clearness. Likewise, Strauss and Thomas (1998) review the empirical evidence of the relationship between health and productivity, establishing correlations between physical productivity and some health indicators. They focus particularly on those related with nutrition or specific diseases.

The endogenous causality between health and income has been extensively studied in the field of health economics. For instance, Luft (1978) gives an informal explanation of this causality as "a lot of people who otherwise wouldn't be poor are, simply because they are sick; however, a few people who otherwise would be healthy are sick because they are poor".[3] Smith (1999) uses life cycle models, which link health condition with future income, consumption and welfare to explain the direction of the causality of the impact of health over income. Accordingly, Bloom and Canning (2000) explain this direction of causality with education, indicating healthy people live more and have higher incentives to invest in their abilities since the present value of the human capital formation is higher. Higher education creates higher productivity and consequently, higher income.

Knowles and Owen (1995, 1997) showed that there was a significant statistical relationship between health and growth with education having a modest role. In contrast, Webber (2002) reached different conclusions and argued that growth oriented policies should focus on investments in education over health. Yet in another study, McDonald and Roberts (2002) conducted a panel data analysis of 77 countries and their findings seem to be consistent with those of Knowles and Owen (1995).

Hamoudi and Sachs (1999) also analysed the relationship between health and economic growth and established an endogenous relationship between them. They argue that, there are exogenous factors which determine the health conditions of a person. Recently, Li and Liang (2010) used the extended Solow model to investigate the relationship between health, education and economic growth in East Asia. They found that, the impact of the stock of health and education on economic growth is statistically significant and that the statistical impact of health on growth is stronger than that of education.

[3] Luft, H. (1978). Poverty and Health: Economic Causes and Consequences. Cambridge, MA: Ballinger Publishing Company.

Acemoglu and Johnson (2006) used innovative instrument for life expectancy of predicted mortality based on cause of death data, and showed that, between 1940 and 1980, improvements in life expectancy did not contribute to the increase in GDP per capita growth across this same period.

None of the studies mentioned above specifically investigates the empirical relationship between health and economic growth in SSA. Second, none of these studies employs the panel cointegration technique to estimate such relationship. We attempt to address these issues in this study. Given the findings of the above studies reviewed, one could expect that if the health of the population in SSA improves considerably, labour productivity would increase, life expectancy increases and consequently increases economic growth in the long-run. In particular, reductions in child mortality, eradicating some basic diseases, having right attitude towards HIV/AIDS prevention would suffice to develop the human capital in SSA.

2.3 Model and Theoretical Framework

The theoretical framework of this study follows the procedures of Knowles and Owen's (1995), Islam (1995) and Li and Liang (2010). However, in the extended Solow model in this study, we assume that human capital is quintessentially health and education capital and we model it in a panel data framework. Romer (1990), Barro (1991) and Benhabib and Speigel (1994) have argued that, perhaps the most important factor determining economic growth is human capital. The model is the Cobb-Douglas production function with Harrod-Neutral technical progress and assumes constant returns to scale for country *i* and time *t* and is given as:

$$Y_{it} = K_{it}^{\alpha} E_{it}^{\beta} H_{it}^{\gamma} \left(A_{it} L_{it}\right)^{1-\alpha-\beta-\gamma},\ 0<\alpha,\beta,\gamma<1,\ \alpha+\beta+\gamma<1 \tag{2.1}$$

where Y is output, K is physical capital, E is human capital of education, H is human capital of health, L is labour, and A is the level of technology. L and A grow at rates n_{it} and g_{it}:

$$L_{it} = L_{i0} e^{n_{it} t} \tag{2.2}$$

$$A_{it} = A_{i0} e^{g_{it} t} \tag{2.3}$$

Knowles and Owen (1995) further assumed that the growth rates of labour (n_{it}) and the level of technology (g_{it}) are exogenously given, that is, $n_{it} = n_i$ (assumed to be the same over time for country *i*) and $g_{it} = g$ (assumed to be same for all countries and over time). Thus, the growth rate of effective labour unit ($A_{it} L_{it}$) is given as $n_i + g$. The rates of savings, population growth and technical progress are constant and are exogenously given in the model.

Furthermore, we define s_{ki}, s_{ei} and s_{hi} as the constant fractions of output that is invested in physical, education and health capitals respectively for economy *i*. Again, we define $\bar{k}$, $\bar{e}$ and $\bar{h}$ as the stocks of physical, education and health capital per effective unit of labour respectively, that is, $\bar{k} = K/AL$, $\bar{e} = E/AL$ and $\bar{h} = H/AL$. Similarly, define $\bar{y} = Y/AL$ as the level of output per effective unit of labour. Therefore the output per effective unit of labour can be written as:

$$\bar{y}_{it} = \bar{k}_{it}^{\alpha} \bar{e}_{it}^{\beta} \bar{h}_{it}^{\gamma} \tag{2.4}$$

The dynamics of $\bar{k}$, $\bar{e}$ and $\bar{h}$ are given as follows:

$$\dot{\bar{k}}_{it} = s_{ki}\bar{y}_{it} - (n_i + g + \delta)\bar{k}_{it} = s_{ki}\bar{k}_{it}^{\alpha}\bar{e}_{it}^{\beta}\bar{h}_{it}^{\gamma} - (n_i + g + \delta)\bar{k}_{it} \tag{2.5}$$

$$\dot{\bar{e}}_{it} = s_{ei}\bar{y}_{it} - (n_i + g + \delta)\bar{e}_{it} = s_{ei}\bar{k}_{it}^{\alpha}\bar{e}_{it}^{\beta}\bar{h}_{it}^{\gamma} - (n_i + g + \delta)\bar{e}_{it} \tag{2.6}$$

$$\dot{\bar{h}}_{it} = s_{hi}\bar{y}_{it} - (n_i + g + \delta)\bar{h}_{it} = s_{hi}\bar{k}_{it}^{\alpha}\bar{e}_{it}^{\beta}\bar{h}_{it}^{\gamma} - (n_i + g + \delta)\bar{h}_{it} \tag{2.7}$$

where δ is the rate of depreciation (assumed to be constant over time for all economies). This implies that $\bar{k}$, $\bar{e}$ and $\bar{h}$ converge to their steady-state values $\bar{k}_i^*$, $\bar{e}_i^*$ and $\bar{h}_i^*$ where

$$\bar{k}_i^* = \left(\frac{s_{ki}^{1-\beta-\gamma} s_{ei}^{\beta} s_{hi}^{\gamma}}{n_i + g + \delta} \right)^{1/\theta} \tag{2.8}$$

$$\bar{e}_i^* = \left(\frac{s_{ki}^{\alpha} s_{ei}^{1-\alpha-\gamma} s_{hi}^{\gamma}}{n_i + g + \delta} \right)^{1/\theta} \tag{2.9}$$

$$\bar{h}_i^* = \left(\frac{s_{ki}^{\alpha} s_{hi}^{1-\alpha}}{n_i + g + \delta} \right)^{1/\theta} \tag{2.10}$$

where $\theta = 1 - \alpha - \beta - \gamma$ and lets define $p_i = n_i + g + \delta$. Now substituting Equations (2.3), (2.8)-(2.10) into (2.4) and taking logs, we obtain the implied steady-state income per capita:

$$\ln y_{it} = \ln A_{i0} + gt + \frac{\alpha}{\theta}\ln s_{ki} + \frac{\beta}{\theta}\ln s_{ei} + \frac{\gamma}{\theta}\ln s_{hi} - \frac{1-\theta}{\theta}\ln p_{it} \tag{2.11}$$

where $y = Y/AL$ is the per capita output.

Following the ideas of linearization from Mankiw et al. (1992), Equation (2.11) can be converted into the growth equation since the main purpose of this study is to investigate the effect of population health alongside other determinants of economic growth. They define $\bar{y}_i^*$ as the steady

state level of income per effective unit of labour and $\bar{y}_{it}$ as its value at any time *t* for economy *i*. The rate of convergence is therefore given as:

$$\frac{d \ln \bar{y}_{it}}{dt} = \lambda_i \left[\ln \bar{y}_i^* - \ln \bar{y}_{it} \right] \tag{2.12}$$

where $\lambda_i = (n_i + g + \delta)(1 - \alpha - \beta - \gamma) = (n_i + g + \delta)\theta$. Equation (12) implies

$$\ln \bar{y}_{it_2} = \left(1 - e^{-\lambda_i \tau}\right) \ln \bar{y}_i^* + e^{-\lambda_i \tau} \ln \bar{y}_{it_1} \tag{2.13}$$

where $\tau = t_2 - t_1$. Therefore, we can easily get:

$$\ln \bar{y}_{it_2} - \ln \bar{y}_{it_1} = \left(1 - \exp(-\lambda_i \tau)\right)\left(\ln \bar{y}_i^* - \ln \bar{y}_{it_1} \right) \tag{2.14}$$

Substituting Equation (9) into (12) yields:

$$\ln \bar{y}_{it_2} - \ln \bar{y}_{it_1} = \left(1 - \exp(-\lambda_i \tau)\right)\left(\frac{\alpha}{\theta} \ln s_{ki} + \frac{\beta}{\theta} \ln s_{ei} + \frac{\gamma}{\theta} \ln s_{hi} - \frac{1-\theta}{\theta} \ln p_{it} - \ln \bar{y}_{it_1} \right) \tag{2.15}$$

Since $\bar{y}_{it}$ is the income per effective labour and we are interested in income per capita, we can substitute $\bar{y}_{it} = \ln y_{it} - \ln A_{0i} - gt$ into Equation (2.13) and get the growth equations:

$$\begin{aligned} \ln y_{it_2} = {} & e^{-\lambda_i \tau} \ln y_{it_1} + \left(1 - e^{-\lambda_i \tau}\right) \frac{\alpha}{\theta} \ln s_{ki} + \left(1 - e^{-\lambda_i \tau}\right) \frac{\beta}{\theta} \ln s_{ei} + \left(1 - e^{-\lambda_i \tau}\right) \frac{\gamma}{\theta} \ln s_{hi} \\ & - \left(1 - e^{-\lambda_i \tau}\right) \frac{1-\theta}{\theta} \ln p_{it} + \left(1 - e^{-\lambda_i \tau}\right) \ln A_{0i} + g\left(t_2 - e^{-\lambda_i \tau} t_1\right) \end{aligned} \tag{2.16}$$

Equation (2.16) investigates the effects of investment ratios in human capital on economic growth. However, since our interest in the level of human capital stock on economic growth, we can convert Equation (2.8) to express s_{hi} in terms of $\bar{h}_i^*$, and substitute the converted expression into Equation (2.14) to yield:

$$\begin{aligned} \ln y_{it_2} = {} & e^{-\lambda_i \tau} \ln y_{it_1} + \left(1 - e^{-\lambda_i \tau}\right) \frac{\alpha}{1-\alpha-\gamma} \ln s_{ki} + \left(1 - e^{-\lambda_i \tau}\right) \frac{\beta}{1-\alpha-\gamma} \ln \bar{e}_i^* + \left(1 - e^{-\lambda_i \tau}\right) \\ & \times \frac{\gamma}{1-\alpha-\gamma} \ln s_{hi} - \left(1 - e^{-\lambda_i \tau}\right) \frac{(\alpha+\gamma)}{1-\alpha-\gamma} \ln p_{it} + \left(1 - e^{-\lambda_i \tau}\right) \ln A_{0i} + g\left(t_2 - e^{-\lambda_i \tau} t_1\right) \end{aligned} \tag{2.17}$$

$$\begin{aligned} \ln y_{it_2} = {} & e^{-\lambda_i \tau} \ln y_{it_1} + \left(1 - e^{-\lambda_i \tau}\right) \frac{\alpha}{1-\alpha-\beta} \ln s_{ki} + \left(1 - e^{-\lambda_i \tau}\right) \frac{\beta}{1-\alpha-\beta} \ln s_{ei} + \left(1 - e^{-\lambda_i \tau}\right) \\ & \times \frac{\gamma}{1-\alpha-\beta} \ln \bar{h}_i^* - \left(1 - e^{-\lambda_i \tau}\right) \frac{(\alpha+\beta)}{1-\alpha-\beta} \ln p_{it} + \left(1 - e^{-\lambda_i \tau}\right) \ln A_{0i} + g\left(t_2 - e^{-\lambda_i \tau} t_1\right) \end{aligned} \tag{2.18}$$

$$\begin{aligned} \ln y_{it_2} = {} & e^{-\lambda_i \tau} \ln y_{it_1} + \left(1 - e^{-\lambda_i \tau}\right) \frac{\alpha}{1-\alpha} \left[\ln s_{ki} - \ln p_{it}\right] + \left(1 - e^{-\lambda_i \tau}\right) \frac{\beta}{1-\alpha} \ln \bar{e}_i^* \\ & + \left(1 - e^{-\lambda_i \tau}\right) \frac{\gamma}{1-\alpha} \ln \bar{h}_i^* + \left(1 - e^{-\lambda_i \tau}\right) \ln A_{0i} + g\left(t_2 - e^{-\lambda_i \tau} t_1\right) \end{aligned} \tag{2.19}$$

Islam (1995) advocates a dynamic panel data approach to estimate Equations (2.16)-(2.19). The main usefulness of the panel data approach lies in its ability to allow for differences in the aggregate production functions across economies. Note that in a dynamic time series model, the savings rates s_{ki} and s_{hi} are varying for different time periods. Consequently, Equation (2.16) can be rewritten in the form of a dynamic panel data model, which is the baseline regression model in the econometric specification of this study:

$$\ln y_{it} = \pi_i \ln y_{i,t-\tau} + \beta_{i1} \ln s_{kit} + \beta_{i2} \ln s_{eit} + \beta_{i3} \ln s_{hit} + \beta_{i4} \ln p_{it} + \eta_i + \mu_t + \varepsilon_{it} \quad (2.16^{/})$$

where $\varepsilon_{it} \sim iid\left(0, \sigma_{\varepsilon}^2\right)$, i =1, 2, …, N and $t = 2\tau, 3\tau \cdots, T\tau$. For the annual data, $\tau = 1$. In this regression model, y_{it} is the per capita real GDP, s_{kit}, s_{eit} and s_{hit} are the percentages of GDP saved and invested in the physical capital, education and health respectively and p_{it} is the effective labour force. The regression coefficients are defined as:

$$\pi_i = e^{-\lambda_i \tau} \quad (2.20)$$

$$\beta_{i1} = \left(1 - e^{-\lambda_i \tau}\right) \frac{\alpha_i}{\theta_i} \quad (2.21)$$

$$\beta_{i2} = \left(1 - e^{-\lambda_i \tau}\right) \frac{\beta_i}{\theta_i} \quad (2.22)$$

$$\beta_{i3} = \left(1 - e^{-\lambda_i \tau}\right) \frac{\gamma_i}{\theta_i} \quad (2.23)$$

$$\beta_{i4} = \left(1 - e^{-\lambda_i \tau}\right) \frac{1 - \theta_i}{\theta_i} \quad (2.24)$$

$$\eta_i = \left(1 - e^{-\lambda_i \tau}\right) \ln A_i(0) \quad (2.25)$$

$$\mu_t = g\left(t - e^{-\lambda_i \tau}(t - \tau)\right) \quad (2.26)$$

The implied value for λ_i is $-\tau^{-1} \ln(\pi_i)$ which according to Equation (2.20) is the convergence rate. A larger value of λ_i would imply a faster convergence rate toward the steady state. Similarly, we can apply the same idea to convert Equation (2.17)-(2.19) into (2.17/)-(2.19/) as:

$$\ln y_{it} = \pi_i \ln y_{i,t-\tau} + \beta_{i1} \ln s_{kit} + \beta_{i2} \ln e_{it} + \beta_{i3} \ln s_{hit} + \beta_{i4} \ln p_{it} + \eta_i + \mu_t + \varepsilon_{it} \quad (2.17^{/})$$

$$\ln y_{it} = \pi_i \ln y_{i,t-\tau} + \beta_{i1} \ln s_{kit} + \beta_{i2} \ln s_{eit} + \beta_{i3} \ln h_{it} + \beta_{i4} \ln p_{it} + \eta_i + \mu_t + \varepsilon_{it} \quad (2.18^{/})$$

$$\ln y_{it} = \pi_i \ln y_{i,t-\tau} + \beta_{i1} \ln s_{kit} + \beta_{i2} \ln e_{it} + \beta_{i3} \ln h_{it} + \beta_{i4} \ln p_{it} + \eta_i + \mu_t + \varepsilon_{it} \quad (2.19^{/})$$

where e_{it} and h_{it} is the average proxies for education and health capital stock.

It might seem relatively dubious to apply this theoretical model in the context of SSA, having outlined some of the inherent problems the region is facing with regard to the health levels of the population. Nonetheless, it is seemingly innocuous to apply it for the greater number of countries within the region where data is readily available on all the variables in the model, whiles controlling for the major disease affecting the region. Moreover, a number of studies (see e.g., McDonalds and Roberts, 2002, Li and Liang, 2010) have applied this model to include both developed and developing countries.

Equation ($2.19^{/}$) is taken to be the baseline equation in our empirical study considering the proxies we have for health and education capital. However, we modify this equation by controlling for trade openness. Also, since HIV/AIDS epidemic has had serious debilitating effect on the health of individuals within the region, we account for that in the estimation of the model.

CHAPTER THREE

DATA AND ECONOMETRIC FRAMEWORK

3.1 Data Description and Sources

This empirical study relies on panel dataset collected from different data sources from 1970 to 2010 on 30 Sub-Saharan African countries[4]. The criterion used to create the sample was based upon the availability of data, where the candidate countries should have available data on physical capital, health and schooling variable for the periods 1970 to 2010. Additionally, data on real GDP per capita, trade openness and population growth were obtained from World Development Indicators (2011). Finding appropriate proxies for health and education capital was relatively based on some general rules following Li and Liang (2010): (1) the proxy variables must be comparable across the economies in SSA; (ii) they must address the characteristics of health and education status in SSA; and (iii) they must be estimable with data available. Based on these criteria, following previous literature and searching the available data sources, the following proxy variables were employed for both health and education.

Regarding health capital, we settle on life expectancy at birth (LE). Knowles and Owen (1995) have argued that, using life expectancy and mortality rate as proxies for health in developing countries is seemingly innocuous. Since SSA is widely regarded as a developing or at worst poor region, using life expectancy at birth as a proxy variable to indicate health capital is also in order.

For education human capital, we use pupil-to-teacher ratio for primary education (PTRATIO) as a proxy variable.[5] This variable will serve as a reflection of the quality of education on economic growth. In most SSA countries, primary education forms the most important and fundamental part of the whole educational system. Hence, it is not incongruous to consider the pupil-to-teacher ratio in primary level as a measurement of the fundamental education level for a region like SSA. This measure of education is also a reflection of the outcome of education investment. It is thus important to see whether increasing the share of investment in education capital will stimulate economic growth.

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[4] The countries included in the sample are listed in the appendix.

[5] Data is relatively exiguous and hence limited a wide range of other proxies to serve as robust checks in the estimations and also to estimate the actual investments in education on economic growth. Also as in other studies, using health care expenditures or body mass index (BMI) would have been good proxies for health.

With reference to the theoretical model, we have employed the following variables in the growth equation: real GDP per capita (RDGPPC), physical capital proxied by gross fixed capital formation as a percentage of GDP (GFCF), the summation of population growth rate, technological progress and depreciation rate ($n+g+\delta$), which is sometimes called "workforce growth (PGRW)" and trade as a percentage of GDP (OPEN). Following Mankiw et al. (1992), we assume of that the sum of technological progress and depreciation rate ($g+\delta$) is 0.05 (5 percent) and is the same for all the countries in the sample and in all the years. A dummy variable to account for HIV/AIDS epidemic in SSA is created. The periods between 1980 and 2002 are given values of 1, and all other periods zero. This is because after 2002, there were developments in antiretroviral drugs which have consequently tolled down the deaths associated with the disease and hence has the potential of prolonging the life span of affected individuals. The period prior to 1980 has no record of HIV cases. This would suggest that the relationship between health and economic growth could be different between these two different sub-periods.

All data were gleaned from the World Bank's World Development Indicators, United Nation's EDStat and other augmenting sources from the respective countries' Statistical Services and other departments.[6]

3.2 Econometric Methodology[7]

This study uses linear panel data regression methods to evaluate how the health status of Sub-Saharan African countries has impacted on their economic performance. The cross-section includes Sub-Saharan African countries, for which annual observations of a number of variables were collected. There are a number of advantages and disadvantages of using panel data.[8] However, the focus here is on the aspects that contrast macro panels to time series regressions. Some of the advantages include: (i) controlling for individual heterogeneity;[9] (ii) more informative data, variability, degrees of freedom and efficiency, as well as less collinearity among the variable; (iii) allowing the construction and testing of more complicated behavioural models; and (iv) panel unit roots tests that have more power and less standard asymptotic distributions.

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[6] Summary Statistics of the variables are presented in Table A1 in the appendix.
[7] The empirical methodology of this study follows closely that of Martin (2011) who used panel cointegration analysis to investigate aid absorption and spending in Africa.
[8] Baltagi (2008: pp 6 – 11) provides a good summary of the advantages and disadvantages of using panel data.
[9] Unobserved heterogeneity or time-invariant variables that are correlated with explanatory variables (such as history, institutions and political regimes) may cause omitted variable bias in time series regressions.

Some of the serious limitations are: (i) the 'poolability' (homogeneity) assumption, although there are formal tests to evaluate its validity; (ii) potential cross-sectional dependence, which complicates the analysis; (iii) some tests and methods require balanced panels; and (iv) cross country data consistency. Having acknowledged the advantages and limitations of using panel data, we now proceed to the presentation of two important methodological approaches – dynamic panel data methods and panel cointegration analysis.

3.2.1 Dynamic Panel Data

Economic relationships often possess some degree of dynamic behaviour. To capture this feature, dynamic panel data models – which include a lagged dependent variable – are usually considered (Baltagi, 2008):

$$y_{it} = \phi y_{it-1} + \beta x_{it} + u_{it} \tag{3.1}$$

where ϕ is a scalar, x_{it} is a $1 \times k$ vector of explanatory variables and β is a $k \times 1$ vector of coefficients. For the purpose of illustration, assume that u_{it} is a one-way error component model:

$$u_{it} = \mu_i + v_{it}$$

where $\mu_i \sim iid\left(0, \sigma_u^2\right)$ and $v_{it} \sim iid\left(0, \sigma_v^2\right)$ independent of each other and among themselves. The dynamic panel model is characterized by two sources of persistence over time: (i) autocorrelation due to the lagged dependent variable; and (ii) individual effects capturing country heterogeneity (Baltagi, 2008). Estimating dynamic panel models with both fixed and random effects is fraught with some problems. For example, the lagged dependent variable is correlated with the disturbance term (since y_{it-1} is a function of μ_i), even if the v_{it} is not serially correlated (Green, 2003). The OLS estimator is biased and inconsistent in finite samples, especially if T is small. In fact, the coefficients of the explanatory variables will be subject to a downward bias in absolute terms (i.e., biased towards zero). Even for T=30 the fixed-effects (FE) estimator can present a significant bias (Baltagi, 2008). To overcome these problems, we use instrumental variables (IV) regressions or generalized method of moments (GMM) estimators (Green, 2003).

Arellano and Bond (1991) developed one-and two-step GMM estimators for dynamic panels ('difference GMM'). They obtain additional instruments by using orthogonality conditions between the lagged dependent variables and the disturbance terms. The difference GMM does

not require any prior knowledge of the initial conditions or even the distribution of v_{it} and μ_i. However, if the dependent variable is very persistent (close to a random walk), then the lagged levels are poor instruments for the first-differences and difference GMM performs poorly. Blundell and Bond (1998) thus develop a 'system GMM' estimator for dynamic panel data models to solve the problem of 'weak instruments'. The system GMM estimator combines moment conditions for the model in first-differences with those for the models in levels. The procedure uses lagged differences of y_{it} as instruments for the equation in first-differences. Moreover, it requires a stationary restriction on the initial conditions process (Baltagi, 2008). The validity of the moment conditions imposed is usually assessed by a test of over-identifying restrictions (either Hansen's or Sargan's).

The main advantages of these GMM estimators relate to their perceived robustness to heteroskedasticity and non-normality of the disturbances. Moreover, the use of instrumental variables helps address biases arising from reverse causality. Nonetheless, there are some remaining concerns about the efficiency of such methods. The violation of moment conditions (e.g. presence of non-stationarity), will yield inconsistent estimates. Moreover, Roodman (2009) argues that the number (and quality) of instruments generated by difference and system GMM methods can affect the asymptotic properties of the estimators and specification tests. In samples with large *T*, instrument proliferation can be particularly serious, inducing two main types of problems: (i) overfitting endogenous variables; and (ii) imprecise estimates of the optimal weighting matrix. Greene (2003) provides another strong criticism. He argues that introducing a lagged dependent variable to an otherwise long-run (static) equation will significantly change its interpretation, especially for the independent variables. In the case of a dynamic panel data model, the coefficient on x_{it} merely represents the effect of new information, rather than the full set of information that influences y_{it}. Finally, it is often argued that while dynamic panel data methods are appropriate for panels with a small *T*, but when *T* is sufficiently large other methods should be preferred. Hence, we now turn to panel data methods that were specifically developed for a relatively long panel.

3.2.2 Panel Cointegration

Traditional panel data econometrics rests on micro panels that usually include thousands of households or hundreds of firms (large *N*), which are tracked over a few survey rounds (small *T*). This study, however, uses macroeconomic variables that are collected for several Sub-Saharan

African countries over a significant number of years. Using panel datasets with large *N* and large *T*, thus presents new challenges to researchers. Since macroeconomic variables are often characterized by non-stationarity, panels with a significant time dimension are subject to spurious relationships. According to Baltagi (2008), the accumulation of observations through time generated two strands of ideas: (i) the use of heterogeneous regressions (one for each country) instead of accepting coefficient homogeneity (implicit in pooled regressions), e.g. Pesaran et al (1999); and (ii) the extension of time series methods (estimators and tests) to panels in order to deal with non-stationarity and cointegration, e.g. Kao and Chiang (2000) and Pedroni (2000).[10]

Cointegration analysis in a panel data setting is analogous to the steps usually employed in time series analysis: (i) unit root testing; (ii) cointegration testing; and (iii) estimation of the long-run and short-run relationships.

3.2.2a Unit Root Tests

Panel unit root testing is the first step in the cointegration analysis which requires analyzing the stationarity properties of the variables since it is believed that most macroeconomic variables exhibit trends in them. Panel unit root tests are often grouped into two main categories: (i) first-generation tests, which assume cross-sectional independence – for example, Maddala and Wu (1999), Choi (2001), Levin et al. (2002) and Im et al. (2003); and (ii) second-generation tests, which explicitly allow for some form of cross-sectional dependence – for example, Pesaran (2007). As a starting point, consider the following autoregressive (AR) process for panel data:

$$y_{it} = \rho_i y_{it-1} + \phi_i Z_{it} + u_{it} \tag{3.2}$$

where ρ_i is the AR coefficient and the error term u_{it} is assumed to be independent and identically distributed. Moreover, Z_{it} includes individual deterministic effects, such as constants (fixed effects) and linear time trends, which capture cross-sectional heterogeneity.

Levin et al. (2002) propose a test (LLC) that can be seen as a panel extension of the augmented Dickey-Fuller (ADF) test:

[10] Moreover, the estimators for panel cointegrated models and related statistical tests are often found to have different asymptotic properties from their time series counterparts (Baltagi, 2008:298). An important contribution is Phillips and Moon (1999, 2000), who analyse the limiting distribution of double indexed integrated processes.

$$\Delta y_{it} = \gamma y_{it-1} + \sum_{j=1}^{p_i} \theta_{ij} \Delta y_{it-j} + \phi_i Z_{it} + \varepsilon_{it} \quad (3.3)$$

Since the lag length of the differenced terms (p_i) is unknown, Levin et al. (2002) suggest the following three-step procedure: (i) carry out separate ADF regressions for each individual and generate two orthogonalised residuals;[11] (ii) estimate the ratio of long-run to short-run innovation standard deviation for each individual; (iii) compute the pooled *t*-statistics, with the average number of observations per individual and average lag length. In this test, the associated AR coefficient is constrained to be homogenous across individuals (i.e. $\gamma_i = \gamma$ for all i). Hence, the null hypothesis assumes all cross-sections are non-stationary ($H_0: \gamma = \rho - 1 = 0$) against the alternative hypothesis that each time series is stationary ($H_1: \gamma < 0$). The authors show that the pooled *t*-statistic has a limiting normal distribution under the null hypothesis (i.e., when $N \to \infty$ and $T \to \infty$). They suggest for example that in Z_{it} , one can include trend, cross-section specific effects as well as time specific effects. This test is often recommended for moderate sized panels, especially for $N>10$ and $T>25$. Thus, the test has poor power when T is small and also when using fixed effects.

Im et al. (2003) extend the LLC test by allowing heterogeneity on the autoregressive (AR) coefficient. In practice, the test entails the estimation of individual ADF regressions, and then combining this information to perform a panel unit root test. The ADF regression equation is given as:

$$\Delta y_{it} = \gamma_i y_{it-1} + \sum_{j=1}^{p_i} \theta_{ij} \Delta y_{it-j} + \phi_i Z_{it} + \varepsilon_{it} \quad (3.4)$$

This approach allows for different specifications of the coefficients (γ_i for each cross-section), the residual variance and lag-length (Asteriou and Hall, 2007). They propose a *t*-bar statistic, based on the average of the individual unit root (ADF) test statistics. Let denote:

$$\bar{t} = \frac{1}{N} \sum_{i=1}^{N} t_{\gamma_i} \quad (3.5)$$

[11] Here, the lag order of the differenced terms (pi) is allowed to vary across individuals and is usually determined by a lag selection criterion (to correct for serial correlation).

where t_{γ_i} is the individual t-statistics for testing γ_i=0. This statistic evaluates whether the coefficient γ is non-stationary across all individuals (H_0: $\gamma_i = 0$ for all i), against the alternative hypothesis that at least a fraction of the series is stationary (H_1: $\gamma_i < 0$ for at least one i). The standardized IPS *t*-bar statistic is given by:

$$t_{IPS} = \frac{\sqrt{N}\left(\bar{t} - 1/N\sum_{i=1}^{N} E\left[t_{iT}\,|\gamma_i = 0\right]\right)}{\sqrt{N^{-1}\sum_{i=1}^{N} \mathrm{var}\left[t_{iT}\,|\gamma_i = 0\right]}} \qquad (3.6)$$

Both LLC and IPS tests require N to be small enough relative to T, whilst the LLC test also requires a strongly balanced panel (Baltagi, 2008). Fixed effects can cause loss of power of this test.

Breitung (2000) uses Monte Carlo experiments to show that the power of the LLC and IPS tests statistics is sensitive to the specification of the deterministic components, such as the inclusion of individual specific trends (Baltagi, 2008). He proposes a test statistic based on modifications to the LLC steps to overcome these difficulties. Breitung's test statistic assumes a common unit root process and is also shown to be asymptotically distributed as a standard normal. The test is often suggested for samples of around N=20 and T=30.

Maddala and Wu (1999) and Choi (2001) suggest the use of nonparametric Fisher tests. The main feature of these tests is that they combine the probability limit values (*p*-values) of unit root tests from each cross-section rather than average test statistics, that is:

$$P = -2\sum_{i=1}^{I} \ln\left(p_i\right) \sim \chi^2_{2I} \qquad (3.7)$$

when $T \to \infty$. Fisher tests are usually implemented using individual ADF or Phillips-Perron unit root tests, and their asymptotic distribution follows a chi-square (*P*-test). Choi (2001) also proposes an alternative Fisher-type statistic that follows a standard normal distribution (*Z*-test). *T* must be relatively large enough to apply this test. Both IPS and Fisher-type tests combine information of individual unit root tests, but simulation studies suggest that Fisher tests have better power properties than the IPS test which in turn is better than LLC test. The disadvantage of Fisher-type tests relates to the need to derive *p*-values which takes time to bootstrap if we have cross sectional dependence.

Hadri (2000) proposes a residual-based Lagrange multiplier (LM) test, which is in fact a panel generalisation of the KPSS test (Baltagi, 2008). As highlighted by Maddala and Wu (1999), IPS and LLC tests are sensitive to the choice of lag lengths in the ADF regression. Furthermore, both tests assume cross-section independence. Thus results of the tests can be misleading if this assumption does not hold. In empirical works however, cross-section independence assumption is a strong and often unrealistic assumption. Thus, Hadri test uses the residuals from individual OLS regressions of y_{it} on deterministic components (constant and trend) to compute the LM statistic given by:

$$LM = \frac{1/N\sum_{i=1}^{N} 1/T^2 \sum_{t=1}^{T} s_{it}^2}{\hat{\sigma}_{\varepsilon}^2} \qquad (3.8)$$

This test also differs from the previous in the sense that it is a stationarity test. The null hypothesis assumes no unit root in any of the time series (all panels stationary), against the alternative of non-stationarity for, at least, some cross-sections. Hadri's test can also allow for a general form of dependence over time and for the disturbance component to be heteroskedastic across *i*.

The main drawback of the first-generation tests described above relates to the assumption that the data is independent and identically distributed across individuals (cross-section independence). In practice, this means that the movements of a given variable through time are independent across countries. This restrictive assumption has often been challenged by empirical studies, and it should be evaluated on a case-by-case basis. Some cross-sectional dependence tests include Pesaran (2004) and a Breusch-Pagan LM statistic (for $T>N$). Banerjee et al (2005) show that in the presence of cross-section dependence, first-generation tests tend to have serious size distortions and therefore perform poorly. This often leads to the over-rejection of the null hypothesis (unit root) when the sources of non-stationarity are common across individuals.

These findings led to the development of unit root tests for panels with cross-sectional dependence (second-generation tests). Pesaran (2007) suggests a simple method to remove the influence of cross-sectional dependence, which involves augmenting standard ADF regressions with the cross-section averages of lagged levels and first-differences of the individual series. These individual cross-sectionally augmented Dickey-Fuller (CADF) statistics (or the corresponding *p*-values) can then be used to develop modified versions of standard panel unit root tests – such as IPS's *t*-bar, Maddala and Wu's *P*, or Choi's *Z*. The tests are applicable for

both when $N>T$ and $T>N$, and are shown to have good size and power properties, even when N and T are relatively small (e.g. 10). However, the t-bar statistic (CIPS) can only be computed for balanced panels. For unbalanced panels, the modified Z test can be reported.

Table 2: Characteristics of Panel Unit Root Tests

Test	Null	Alternative Hypothesis	Deterministic Components	Autocorrelation Correction	Cross Section Dependence	Unbalanced panel (Gaps)
LLC	UR	No UR	None, F, T	Lags	demean	No (-)
Breitung	UR	No UR	F, T	Lags	robust[1]	No (-)
IPS	UR	Some CS without UR	None, F, T	Lags	demean	Yes (No)
Fisher	UR	Some CS without UR	None, F, T	Lags/Kernel	demean	Yes (Yes)
Hadri	No UR	Some CS with UR	F, T	Kernel	robust[1]	No (-)
Pesaran	UR	Some CS without UR	F, T	Lags	robust	Yes (No)

Notes: UR = unit root, CS = cross-sections, None = no exogenous variables, F = fixed effect, T = individual effect and individual trend. [1]Stata's 'xtunitroot' command computes robust versions that account for cross-sectional dependence.

Source: Compiled from QMS (2007:110, corrected) and Stata's 'xtunitroot' command help.

3.2.2b Cointegration Tests

The order of integration of the variables can be assessed by the panel unit root tests proposed above. Consequently, if the main variables are found to be integrated on order one, then we should use panel cointegration tests to determine whether a long-run equilibrium relationship exists among the non-stationary variables in level form.

Pedroni (1999, 2004) provides cointegration tests for heterogeneous panels based on the two-step cointegration approach of Engle and Granger (1987). Though the test allows for heterogeneity, there are different versions of the test and the within-group test assumes homogeneity. To allow for heterogeneity, however, we must use the between-group test. The test is based on the regression:

$$y_{it} = x_{it}\beta_i + \delta_i + \theta_i t + \varepsilon_{it} \qquad (3.9)$$

and $\varepsilon_{it} = \rho_i \varepsilon_{it-1} + \varphi_{it}$. Pedroni uses the residuals from the static (long-run) regression and constructs seven panel cointegration test statistics: four of them are based on pooling (within-dimension or 'panel statistics test'), which assumes homogeneity of the AR term, whilst the remaining are less restrictive (between-dimension or 'group statistics test') as they allow for heterogeneity of the AR term. The assumption has implications on the computation of the second step and the specification of the alternative hypothesis. The v-statistic is analogous to the

long-run variance ratio statistic for time series, while the rho-statistic is equivalent to the semi-parametric 'rho' statistic of Phillips and Perron (1988). The other two are panel extensions of the (non-parametric) Phillips-Perron and (parametric) ADF t-statistics, respectively. These tests allow for heterogeneous slope coefficients, fixed effects and individual specific deterministic trends, but are only valid if the variables are I(1). Pedroni (1999) derived critical values for the null hypothesis of no cointegration.

Kao (1999) proposes residual-based DF and ADF tests similar to Pedroni's, but specifies the initial regression with individual intercepts ('fixed effects'), no deterministic trend and homogeneous regression coefficients. The test is based on the following:

$$y_{it} = x_{it}\beta_i + \phi_i + \varepsilon_{it} \qquad (3.10)$$

and $\varepsilon_{it} = \rho\varepsilon_{it-1} + \varphi_{it}$. Kao's tests converge to a standard normal distribution by sequential limit theory (Baltagi, 2008). Both Kao and Pedroni tests assume the presence of a single cointegrating vector, although Pedroni's test allows it to be heterogeneous across individuals.

Maddala and Wu (1999) propose a Fisher cointegration test based on the multivariate framework of Johansen (1988). They suggest combining the *p*-values of individual (system-based) cointegration tests in order to obtain a panel test statistic. Moreover, Larsson et al (2001) suggest a likelihood ratio statistic (LR-bar) that averages individual rank trace statistics. However, the authors find that the test requires a large number of temporal observations. Both of these tests allow for multiple cointegrating vectors in each cross-section.

Basically, this study relies on the test developed by Westerlund (2007) who suggests four cointegration tests that are an extension of Banerjee et al (1998). These tests are based on structural rather than residual dynamics and allow for a large degree of heterogeneity (for example, individual specific short-run dynamics, intercepts, linear trends and slope parameters). All variables are assumed to be I(1). Suppose we have a data generating process in the form:

$$\Delta y_{it} = \delta_i' d_t + \alpha_i \left(y_{i,t-1} - \beta_i' x_{i,t-1} \right) + \sum_{j=1}^{p_i} \alpha_{ij} \Delta y_{i,t-j} + \sum_{j=0}^{p_i} \gamma_{ij} \Delta x_{i,t-j} + e_{it} \qquad (3.11)$$

where $t = 1, \ldots, T$ and $i = 1, \ldots, N$ index the time-series and cross-sectional units, respectively, while d_t contains the deterministic components, for which there are three cases. In the first case, $d_t = 0$ so (3.11) has no deterministic terms; in the second case, $d_t = 1$ so Δy_{it} is generated with a

constant; and in the third case, $d_t = (1, t)'$ so Δy_{it} is generated with both a constant and a trend. For simplicity, we model the K-dimensional vector x_{it} as a pure random walk such that Δx_{it} is independent of e_{it}, and we further assume that these errors are independent across both *i* and *t*. We will handle any dependence across *i* by means of bootstrap methods.

The parameter α_i measures the speed of adjustment, that is, the speed at which the system returns to its equilibrium after a sudden shock in one of the model variables. We require the parameter α_i to be negative to have a cointegration relationship among the variables. If the parameter α_i is equal to zero there is no cointegration.

Westerlund tests for cointegration using the null hypothesis of no cointegration with the alternative hypothesis which depends on the assumption about the homogeneity of α_i. Unlike other panel cointegration tests, Westerlund's test does not impose any common parameter constraint. According to the alternative hypothesis one can distinguish between group-mean tests and panel tests.

The between group-mean tests can be calculated by:

$$G_T = \frac{1}{N}\sum_{i=1}^{N}\frac{\hat{\alpha}_i}{SE(\hat{\alpha}_i)} \tag{3.12}$$

$$G_\alpha = \frac{1}{N}\sum_{i=1}^{N}\frac{T\hat{\alpha}_i}{\hat{\alpha}_i} \tag{3.13}$$

The panel statistics are given by:

$$P_T = \frac{\hat{\alpha}}{SE(\hat{\alpha})} \tag{3.14}$$

$$P_\alpha = T\hat{\alpha} \tag{3.15}$$

Moreover, bootstrapping provides robust critical values in cases of cross-section dependence. The tests assess the null hypothesis that the error correction term in a conditional ECM is zero – i.e. no cointegration (Baltagi, 2008).

3.2.2c Estimation of the Long-Run

In the presence of cointegrating non-stationary variables, it becomes relatively curious to estimate efficiently the long-run economic relationships between them. Thus, a number of panel estimators have been suggested in the literature. An important difference is that the panel OLS estimator of the (long-run) static regression model, contrary to its time series counterpart, is inconsistent (Baltagi, 2008).

Kao and Chiang (2000) propose a panel dynamic OLS estimator (DOLS) which is a generalisation of the method originally proposed by Saikkonen (1991) and Stock and Watson (1993) for time series regressions. The regression equation is:

$$y_{it} = \alpha_i + \beta' X_{it} + \sum_{j=-q}^{q} c_{ij} \Delta X_{i,t+j} + \varepsilon_{it} \tag{3.16}$$

where X_{it} is a vector of explanatory variables, β the estimated long-run impact, q the number of leads and lags of the first-differenced data, and c_{ij} the associated parameters. The estimator assumes cross-sectional independence and is asymptotically normally distributed. The authors provide Monte Carlo results suggesting that the finite-sample properties of the DOLS estimator are superior to both fully-modified OLS (FMOLS) and OLS estimators.

Pesaran et al (1999) suggest a (maximum-likelihood) pooled mean group (PMG) estimator for dynamic heterogeneous panels. The procedure fits an autoregressive distributed lag (ARDL) model to the data, which can be re-specified as an error correction equation to facilitate economic interpretation. Consider the following error correction representation of an ARDL(*p*, *q*, *q*,…, *q*) model:

$$y_{it} = \phi_i y_{i,t-1} + \beta_i' X_{it-1} + \sum_{j=1}^{p-1} \lambda_{ij} \Delta y_{i,t-j} + \sum_{j=0}^{q-1} \delta_{ij}' \Delta X_{i,t-j} + \mu_i + \varepsilon_{it} \tag{3.17}$$

where X is a vector of explanatory variables, β_i contains information about the long-run impacts, ϕ_i is the error correction term (due to normalisation), and δ_{ij} incorporates short-run information. The PMG can be seen as an intermediate procedure, somewhere between the mean group (MG) estimator and the dynamic fixed-effects (DFE) approach. The MG estimator is obtained by estimating N independent regressions and then averaging the (unweighted) coefficients, whilst the DFE requires pooling the data and assuming that the slope coefficients

and error variances are identical. The PMG, however, restricts the long-run coefficients to be same ($\beta = \beta_i$ for all i), but allows the short-run coefficients and error variances to vary across countries (Pesaran et al, 1999). This approach can be used whether the regressors are I(0) or I(1) (Pesaran et al, 1999).

CHAPTER FOUR

EMPIRICAL RESULTS AND DISCUSSION

This section undertakes a comprehensive econometric exercise to evaluate the impact of population health on the economic performance of Sub-Saharan Africa. The main empirical analysis of the study concerns the estimation of the growth model specified in Equation (2.19′) while controlling for openness to trade using various estimators as outlined in the preceding chapter. The basic growth model includes the logarithm of lagged income (real GDP per capita), population health capital, education capital as well as other control variables in line with the empirical growth literature. The basic strategy therefore, is to predict growth of Sub-Saharan African countries using health and education indicators and vice versa.

Since we are dealing basically with macroeconomic variables that spans over a relatively long period, and hence are often found to be nonstationary, we first take panel unit root tests to evaluate their order of integration. Next, we apply panel cointegration tests to ascertain whether there are long-run relationships amongst the variables of interest. In the final step, we estimate the long-run and short-run relationships using the relevant and efficient techniques.

4.1 Panel Unit Root Testing

We begin the empirical analysis with the application of panel unit root tests to verify whether or not the variables are nonstationary. As thoroughly explained the various types of panel unit root tests in the previous chapter, several authors have proposed unit root tests based on different sets of assumptions. In this study however, we settle on five distinct panel unit root tests on the variables over 1970-2010: Levin-Lin-Chu's (LLC) t*, Breitung's t, Hadri's Z, Im-Pesaran-Shin's W, and Maddala and Wu's χ^2 statitics. Among these tests, LLC, Breitung and Hadri's tests are based on the common unit root process assumption that the autocorrelation coefficients of the tested variables across cross sections are identical. However, the IPS and ADF-Fisher χ^2 tests rely on the individual unit root process assumption that the autocorrelation coefficients vary across cross sections. In all the test specifications, we include deterministic time trend. In the LLC, IPS and ADF-Fisher tests, cross-sectional means are subtracted in order to minimise problems arising from cross-sectional dependence. However, Hadri and Breitung tests used in this study allow for cross-sectional dependence. The Schwarz-Bayesian information criterion (BIC) is used to determine the country-specific lag length for the ADF regressions, with a maximum lag of 3 regarding the LLC and the IPS tests. Further, the Bartlett kernel was used to estimate the long-

run variance in the LLC test, with the maximum lags determined by the Newey-West bandwidth selection algorithm. The test results are presented in Table 3.

Table 3: Panel Unit Root Tests Results

	Tests Assuming a Common Unit Root Process			Tests Assuming Individual Unit Root Process	
Series Name	**LLC t*-stat:**	**Breitung t-stat:**	**Hadri Z-stat:**	**IPS W-t-bar stat:**	**ADF-Fisher χ^2:**
	H_0: Unit root	**H_0: Unit root**	**H_0: No Unit root**	**H_0: Unit root**	**H_0: Unit root**
			Level		
ln*RGDPPC*	0.1348	1.3058	57.4286***	0.1981	1.7010**
	[0.5536]	[0.9042]	[0.0000]	[0.5785]	[0.0445]
ln*LE*	9.9701	2.9751	86.8690***	5.6913	-2.0851
	[1.0000]	[0.9985]	[0.0000]	[1.0000]	[0.9813]
ln*PTRATIO*	-1.7752	0.7369	51.5415***	-0.3702	0.8776
	[0.0379]	[0.7694]	[0.0000]	[0.3556]	[0.1901]
ln*GFCF*	-1.0608	-2.5512***	38.5436***	-1.6784**	2.4205***
	[0.1444]	[0.0054]	[0.0000]	[0.0466]	[0.0078]
ln*OPEN*	-1.0109	-4.2105***	33.4589***	-2.0428**	1.3581*
	[0.1560]	[0.0000]	[0.0000]	[0.0205]	[0.0872]
$(n+g+\delta)$	2.4636	-0.3802	50.9363***	-0.6456	-0.2984
	[0.9931]	[0.3519]	[0.0000]	[0.2593]	[0.6173]

Notes: *, ** and *** represent significance at the 10%, 5% and 1% levels respectively.

Generally, the test results show evidence of nonstationarity in all the variables under consideration. The LLC test confirms that all the variables are nonstationary. The IPS, Breitung and ADF-Fisher tests also indicate that all the variables except GFCF and OPEN are nonstationary. Thus, in comparison with the LLC, the evidence on the nonstationarity of these variables is mixed. Thus applying the Hadri test which has a different null hypothesis (stationary) provides strong evidence that all the variables including GFCF and OPEN have unit roots.

The evidence provided, thus, may suggest the variables contain unit roots. It must however, be emphasised that, although the cross-sectional averages were subtracted from each series (demeaning) prior to applying the LLC, IPS and ADF-Fisher tests, the original versions of Hadri and Breitung tests were also applied, which are not robust to cross dependence and similar conclusions were drawn.

4.2 Panel Cointegration Testing

In order to avoid the spurious regression problem, a cointegration test is required. Although we have ascertained the fact that the variables are nonstationary, a valid inference can be made if a

stable equilibrium relationship is found amongst the variables. This is particularly the case when a linear combination of the variables produces stationary error terms. Table 4 presents three variants of panel cointegration in this study. The Pedroni and Kao tests use the Bayesian information criterion (BIC) to automatically select the appropriate lag length.[12] Moreover, spectral estimation is undertaken by the Bartlett kernel with the bandwidth selected by the Newey-West algorithm. Whilst the Pedroni and Kao tests are based on the residuals of the long-run static regression, the Westerlund test assesses the significance of the adjustment coefficient in the ECM specification. Deterministic time trends are included in all specifications. All tests are derived under the null hypothesis of no cointegration.

The results indicate that the Pedro and Kao tests do not provide any significant evidence of cointegration. Thus, attention is shifted on Westerlund's (2007) technique of panel cointegration test. Before testing for cointegration using Westerlund technique, there is the need to ascertain whether or not there is cross-sectional independence in the residuals. This is to ascertain whether to bootstrap robust critical values for the test statistics or to use the original critical values provided. To test for cross-sectional independence, it requires that T > N (in our case T = 41 and N = 30). Given that the time series are relatively short and some periods are lost in the calculation of differenced variables and lags, we tested only for independence of the first twenty cross-sectional units. Assuming the same short-run dynamics for all series (with a single lag and lead, pi = qi =1), we obtain the test for cross-sectional independence from Table A2 in appendix.[13] The Breusch-Pagan LM test of independence $\chi^2(171)$ is 214.924 with probability value of 0.0128 indicates the presence of common factors affecting the cross-sectional units. As a consequence we bootstrapped robust critical values for the test statistics for testing for cointegration.[14] Also since the results could be affected by the Kernel width chosen, we conduct the test using three different Kernel windows.

In line with the hypotheses set in the first chapter, we test for cointegration considering both the logarithm of real GDP and life expectancy as dependent variables as a precursor to ascertain their direction of causality. The results thus evaluate whether logarithm of real GDP and its covariates as well as logarithm of life expectancy and its associated covariates share a common stochastic trend. The results as far as the Pedroni's and Kao's tests statistics are concerned do not provide

[12] Maximum lag length selected in the cointegration test is 9.
[13] We use Stata command xttest2 to test for cross-sectional independence in the residuals.
[14] The bootstrapped robust critical values are only for Westerlund's technique as the presence of cross-sectional dependence might bias the results of the cointegration test.

Table 4: Panel Cointegration Tests Results

	Panel Cointegration tests	Dep. var. of coint. reg.	Dep. var. of coint. reg.
		ln*GDPPC*	ln*LE*
Pedroni	Panel-v	0.409	4.375***
		(0.341)	(0.000)
	Panel-rho	1.792	5.264
		(0.963)	(1.000)
	Panel-PP	-1.110	4.031
		(0.133)	(1.000)
	Panel-ADF	-2.486***	2.102
		(0.007)	(0.982)
	Group-rho	3.126	7.245
		(0.9991)	(1.000)
	Group-PP	-1.215	5.644
		(0.1122)	(1.000)
	Group-ADF	-3.643***	3.341
		(0.0001)	(0.9996)
Kao	*T*	-1.208	-2.986***
		(0.114)	(0.001)
Westerlund	G_T	-3.356***	-3.918***
		[0.005]	[0.000]
	G_α	-3.733	-6.781***
		[1.000]	[0.000]
Kernel 1	P_T	-17.043***	-15.063***
		[0.006]	[0.000]
	P_α	-3.725	-3.328***
		[1.000]	[0.000]
	G_T	-3.356***	-3.918***
		[0.000]	[0.000]
	G_α	-3.282	-7.610***
		[1.000]	[0.003]
Kernel 2	P_T	-16.167**	-15.051***
		[0.013]	[0.000]
	P_α	-3.221	-3.761***
		[1.000]	[0.000]
	G_T	-3.356***	-3.918***
		[0.004]	[0.000]
	G_α	-3.042	-8.404***
		[1.000]	[0.001]
Kernel 3	P_T	-15.518**	-14.854***
		[0.034]	[0.000]
	P_α	-2.949	-4.155***
		[1.000]	[0.000]

Notes: Test results were generated by Eviews and 'xtwest' Stata module. Pedroni's Panel statistics as well as all of Westerlund's are weighted. Dep. var. of coint. reg. = dependent variable of the cointegrating regression. Values in [ ] are robust p-values generated through bootstrapping because of cross-sectional dependence in the residuals. *, **, and *** indicates significance at 10%, 5% and 1% respectively.

any strong support for the presence of cointegration, particularly when logarithm of real GDP was taken as the dependent variable. As we take into account cross-sectional dependencies, the Westerlund's test results, however, provide evidence of cointegration further indicating the possibility of a somewhat bi-directional long run equilibrium relationship between real GDP and a proxy for health human capital (life expectancy) and are consistent across the different kernel widths. However, it seems as though, real GDP causes life expectancy greatly other than the reverse, as all the various test statistics reject the null hypothesis of no cointegration. Overall the results appear to suggest that the variables are cointegrated.

4.3 Estimation and Interpretation of the Long-Run Relationship

Having established a unique cointegrating relationship amongst the variables of interest, we now use panel data estimation methods to investigate the impact of population health on economic growth in SSA. Potential reverse causality between economic growth and population health is also addressed. Furthermore, as argued in the introduction, HIV/AIDS may have had a deleterious effect on economic growth in SSA and as a consequence, we account for this in another specification besides the baseline results. To ensure robustness of the analysis, we present the results of alternative estimation strategies. To reduce biases and inconsistencies associated with the presence of a lagged dependent variable, we first apply the system GMM (SYS-GMM) estimator instead of fixed effects (FE) or difference GMM. Nevertheless, the relatively large time series in this study would also require an estimator which will provide more efficient estimates of the coefficients. Thus, aside the system GMM estimator, we use the dynamic OLS (DOLS) and the maximum-likelihood estimator (MLE) for the error correction model. The DOLS approach involves estimating a static long-run relation augmented by leads and lags of the first-differenced explanatory variables.[15] Though this strategy improves the efficiency of the long-run estimates, it does not capture the short-run behaviour. As a consequence, we apply the pooled mean group (PMG) estimator which uses the panel extension of the single equation autoregressive distributed lag (ARDL) model.[16] One advantage of using this strategy is that the error correction representation in the ARDL provides information about the contemporaneous impacts and the speed of adjustment towards equilibrium following a shock. Moreover, while the long-run coefficients are assumed to be homogeneous (that is, identical across panels), the short-run coefficients are allowed to be heterogeneous (that is, country-specific).

[15] We included two lags and two leads in the specification of each equation and reported the robust standard errors.
[16] We use the Stata command "xtpmg" in estimating the MG, PMG and DFE while "xtabond2" is the command used for the SYS-GMM estimator.

Table 5: Panel Cointegration Estimation Results for lnRGDPPC

	(1)	(2)	(3)	(4)	(5)
	SYS-GMM	DOLS	PMG	MG	DFE
Dependent variable:	ln*RGDPPC*	ln*RGDPPC*	Δln*RGDPPC*	Δln*RGDPPC*	Δln*RGDPPC*
Convergence	0.99***		-0.13***	-0.37***	-0.05***
coefficients	(0.002)		(0.03)	(0.00)	(0.00)
Long-run coefficients					
ln*LE*		3.16***	0.37***	-0.37	-0.23
		(0.18)	(0.10)	(0.91)	(0.51)
ln*GFCF*		0.39***	0.40***	0.13**	0.46
		(0.05)	(0.03)	(0.06)	(0.17)
ln*PTRATIO*		-0.52***	0.28***	-0.15	0.14
		(0.07)	(0.06)	(0.13)	(0.21)
ln*OPEN*		0.31***	-0.04	0.24*	0.33
		(0.06)	(0.05)	(0.14)	(0.18)
$(n+g+\delta)$		-0.18***	-0.005	0.02	0.07
		(0.03)	(0.007)	(0.05)	(0.05)
Short-run coefficients					
ΔlnLE	0.03		0.64	1.11	0.11
	(0.02)		(0.55)	(1.37)	(0.14)
ΔlnGFCF	0.03***		0.02*	0.01	0.04**
	(0.01)		(0.01)	(0.01)	(0.02)
ΔlnPTRATIO	-0.01		-0.001	0.05	0.01
	(0.01)		(0.03)	(0.03)	(0.02)
ΔlnOPEN	0.004		-0.03*	-0.06***	-0.05
	(0.01)		(0.02)	(0.02)	(0.04)
$\Delta(n+g+\delta)$	0.002		-0.02	-0.13**	0.0004
	(0.003)		(0.02)	(0.06)	(0.004)
Hausman test (χ^2)			18.18***	18.18***	
			[0.0027]	[0.0027]	
Hansen test (χ^2)	23.64				
	[1.00]				
No. of countries	30	30	30	30	30
No. of observations	1200	1140	1200	1200	1200

Notes: All equations include a constant country-specific term. Values in ( ) and [ ] are standard errors and probability values respectively. For DFE estimates, the standard errors are heteroskedatic consistent. SYS-GMM generates 1141 instruments for 1200 observations. The speed of adjustment for SYS-GMM equals one minus the coefficient on the lagged dependent variable (0.01). Hansen test is the test of overidentifying restrictions (the null hypothesis is that the instruments are valid).***, ** and * indicate significance at the 1%, 5% and 10% levels respectively.

We also use the mean group (MG) estimator as an alternative to the PMG estimator, which allows the long-run parameters to vary, and then test the PMG's poolability assumption through a Hausman test. Finally, we also report the dynamic fixed-effects (DFE) estimator, which assumes short- and long-run parameter homogeneity. The results are presented in Table 5.

The long-run and short-run estimates based on different estimation strategies are reported in each column of the Table 5. The magnitudes of the long-run coefficients denote the elasticities of output with respect to each variable in the model. The SYS-GMM estimates of the coefficients

are usually taken to represent short-run impacts, while the long-run impacts are approximated by the short-run coefficients divided by one minus the coefficient on the lagged dependent variable. The results suggest that with the exception of physical capital (lnGFCF), all the other variables have no significant impact on the economic performance in SSA, albeit they obtained the anticipated signs. However, the coefficient on the lagged value of lnRGDP has the expected sign and highly significant. It seems the SYS-GMM underestimates the short-run impacts of health on economic growth and overestimates its long-term effects, in comparison with the other estimators. The second column reports the results from the dynamic OLS (DOLS) specification, which only provides information on the long-run. The coefficients of all the variables are with the expected sign and also highly significant. In particular, the results indicate that health capital (lnLE) significantly improves the economic performance of SSA by approximately 3 percent as the former increases by a percentage point. The last three columns provide the pooled mean group (PMG), mean group (MG) and dynamic fixed-effects (DFE) estimates.

The PMG estimation shows a positive impact of health on economic growth in both the long-run and the short-run period, although the latter is insignificant. The long-run results indicate that, an increase in life expectancy from say 40 to 50 years (25%) will increase economic growth rate by 3.7 percent ceteris paribus. Moreover, the speed of adjustment is negative as expected but the magnitude is somewhat small. This implies that the model does not return immediately to its equilibrium after a shock pushes it away from the steady state. The fact that the error correction term (convergence coefficient) is statistically significant provides further evidence of the existence of a long-run relationship. Furthermore, physical as well as education capital also significantly affect economic growth in the long-run, however, the sign of the latter is reversed. Nonetheless, the short-run estimates provided the correct signs for each of these variables but at the expense of their statistical significance. The short-run impact of health estimated is 0.6 as against the long-run estimate of 0.4 for the PMG estimates. Moreover, the MG estimates are presented as a two-equation model: the normalized cointegrating vector and the short-run dynamic coefficients. In comparing the PMG and MG estimators, we note that the estimated long-run health and education elasticities are no longer significant as far as the latter estimator is concerned. However, the MG estimates of the health elasticities (long-run and short-run) appear to be larger than the estimate from PMG (0.37: 0.64 and -0.37: 1.11 respectively). The same cannot be said of the estimates of the education elasticities (0.28: -0.001 and -0.15: 0.05). The speeds of adjustment estimates from each of the model imply significantly different short-run dynamics (compare $\hat{\phi}$ = -0.13 from PMG and $\hat{\phi}$ = -0.37 from MG). Another intriguing revelation is that, including both

education and health variables simultaneously in the regression reduces the impact of both variables on economic growth.[17] This reiterates the complementary nature of the human capital variables as one reinforces the other. Mention can also be made to the statistical significance of these variables, as they apparently assume the same significance levels across the different estimators.

In the preceding chapter, it was noted that the PMG estimator constrains the long-run elasticities to be equal across all panels. Stated alternately, whilst the PMG constrains the long-run coefficients to be identical across countries (i.e., homogenous), the MG allows the long-run effects to be country-specific (and reports the averaged responses). This "pooling" regarding the PMG across countries yields efficient and consistent estimates when the restrictions are true. If the true model, however, is heterogeneous, the PMG estimates are inconsistent; the MG estimates are consistent in either case. We therefore, use the Hausman test to test the difference in these model or to test the validity of the "pooling" assumption to decide on the preferred specification.[18] The test assesses whether the differences in long-run coefficients are not systematic (null hypothesis), and follows a chi-square distribution with 5 degrees of freedom. The test results reported in Table 5 indicates a rejection of the null, thus refuting long-run homogeneity. Preference is therefore given to the MG estimates since the parameters are consistent, though not efficiently estimated.

The dynamic FE (DFE) estimator, like the PMG estimator, restricts the coefficients of the cointegrating vector to be equal across all panels. However, the former allows intra-group correlation in the calculation of the standard errors. The DFE model further restricts the speed of adjustment coefficient and the short-run coefficients to be equal. The results though are quite similar to the PMG and MG in terms of the signs and magnitudes, statistical significance is hugely compromised as virtually all coefficients are insignificant. FE models are subject to a simultaneous equation bias from the endogeneity between the error term and the lagged dependent variable (Baltagi, Griffin and Xiong, 2000). We use the Hausman test to test for the extent of this endogeneity. The results indicate that the simultaneous equation bias is a problem for these data, and as a consequence, the MG model is preferred over the DFE model. Thus, the

[17] The regression results of including health and education variables separately are not reported in this work and is available upon request.

[18] Stata's hausman test offers a sigmamore option. This option forces the variance-covariance matrix from the efficient model (PMG here) to be used in calculating the test statistic. This is what is presented here. See Baum, Schaffer, and Stillman (2003) for more details.

estimates of the DFE are neglected and we do not conduct any further investigations relating to them.

We also take into account the effect of HIV/AIDS on the economic performance in SSA. The results are reported in Table A3 in the appendix. The long-run results based on the MG estimator indicate a strong negative effect of health on economic growth within the period. The coefficient on AIDS also indicates the deleterious effect the disease has had on the economic performance in the region. This is consistent across the alternative estimators, perhaps suggesting the relative severity of the effect AIDS has on economic development. The stigma that comes along with the disease alone has the potential of wrecking affected individuals, which affects their productivity and consequently on economic growth. Overall, accounting for HIV/AIDS did not significantly change the baseline results in terms of magnitude and significance.

Table 6 also presents the results from specifying the model by making the logarithm of life expectancy the dependent variable, in order to establish the issue of reverse causality between economic growth and health in line with hypothesis 2. Our interest is in basically on the coefficient of the logarithm of real GDP. The results suggest that the coefficient is consistently positive across the alternative estimators and also highly significant. It thus appears that, economic performance rather improves the health conditions of the population in SSA. Also, the coefficient on the education is mixed in terms of signs and statistical significance. As far as the PMG estimator is concerned, quality of education has a significantly positive long-term impact on life expectancy in SSA. The other estimators (MG and DFE) indicate no statistical relationship between the variables, though they are positively correlated. Considering that life expectancy in SSA did not significantly improve over the period 1970 – 2010 (i.e., 44 – 54), it seems to suggest the MG and DFE estimators are to be given reverence. Indeed, despite efforts to improve education in the region, millions of people still die of basic diseases like malaria which is easily preventable. This somehow has hindered improvements in life expectancy and consequently affected the economic performance in SSA over the period under consideration. Hence, the results achieved here lend support for the deleterious effect of life expectancy on economic growth in the region.

Table 6: Panel Cointegration Estimation Results for lnLE

	(1)	(2)	(3)	(4)	(5)
	SYS-GMM	DOLS	PMG	MG	DFE
Dependent variable:	lnLE	lnLE	ΔlnLE	ΔlnLE	ΔlnLE
Convergence	0.98***		-0.02*	-0.02	-0.03***
coefficients	(0.01)		(0.01)	(0.01)	(0.001)
Long-run coefficients					
ln*RGDPPC*		0.08***	0.18***	2.17***	0.16**
		(0.004)	(0.01)	(0.70)	(0.08)
ln*GFCF*		0.03***	0.05***	0.73	0.11
		(0.008)	(0.01)	(0.74)	(0.08)
ln*PTRATIO*		-0.05***	0.52***	1.45	0.08
		(0.01)	(0.04)	(1.67)	(0.17)
$(n+g+\delta)$		0.04***	0.13***	0.22	0.12***
		(0.004)	(0.006)	(0.14)	(0.03)
Short-run coefficients					
Δln*RGDPPC*	0.0004		-0.00003	0.05***	0.01***
	(0.001)		(0.006)	(0.004)	(0.004)
Δln*GFCF*	0.002		0.001	-0.001	-0.001
	(0.002)		(0.002)	(0.002)	(0.002)
Δln*PTRATIO*	-0.003		-0.01**	-0.01	-0.01**
	(0.002)		(0.003)	(0.01)	(0.004)
$\Delta(n+g+\delta)$	0.004***		0.02***	0.01*	0.02***
	(0.001)		(0.006)	(0.005)	(0.002)
Hausman test (χ^2)				0.01	0.01
				[1.00]	[1.00]
Hansen test (χ^2)	26.31				
	[1.00]				
No. of countries	30	30	30	30	30
No. of observations	1200	1140	1200	1200	1200

Notes: All equations include a constant country-specific term. Values in ( ) and [ ] are standard errors and probability values respectively. For DFE estimates, the standard errors are heteroskedatic consistent. SYS-GMM generates 1124 instruments for 1200 observations. The speed of adjustment for SYS-GMM equals one minus the coefficient on the lagged dependent variable (0.02). Hansen test is the test of overidentifying restrictions (the null hypothesis is that the instruments are valid).***, ** and * indicate significance at the 1%, 5% and 10% levels respectively.

Again, under this specification, we control for HIV/AIDS and the results are reported in Table A4 in the appendix. The results further reiterate the pernicious effects of AIDS on the lives of people in SSA. The results indicate a negative correlation between HIV/AIDS and life expectancy. Further, controlling for AIDS did not significantly alter the effect of economic growth on life expectancy. Thus, against all odds, economic growth is a sine qua non for improving the life span of individuals in SSA. Stated differently, regardless of the incidence and prevalence of AIDS, economic boom increases the ability of individuals to afford better nutrition and have access to health care, which potentially increases the lives of affected persons. Hitherto, such persons would have relinquished life at an earlier date.

4.4 Discussion of Results

Acemoglu and Johnson (2006) pointed out that, although health improvements are a valuable goal within itself, they do not have a significant effect on economic growth. The results in this study corroborate their findings. An important caveat is worth mentioning though, that this study fails to concretely take into account the indirect effect of health on economic growth, though some efforts were made to link that through the impact of education on health. It is expected that, using a single equation to capture the influence of health on economic growth would only capture the labour productivity effect on growth. As a consequence, the impact of health on economic growth would only be underestimated. Furthermore, though the study attempts to establish some linkage between education and health, it only captures the contemporaneous effect of the former on the latter. Conceptually, more educated individuals are more productive (and obtain higher earnings). Also if children with better health and nutrition attain higher level of education and are less likely to play truant and drop out of school early, then improved health in young people would contribute to future productivity. Thus, the health effects in the macroeconomic sense may have long time lags, given that the average worker may have been several years or more which invariably makes the macroeconomic relationship difficult to estimate. This assertion is line with the empirical literature. Moreover, if good health is also linked to longer life, healthier individuals would have more incentive to invest in education and training, as the rate of depreciation of the gains in skills would be lower (Strauss & Thomas, 1998). Apparently therefore, this study had the difficulty in establishing a careful indirect effect of health on economic growth through education.

McDonald and Roberts (2002) also used life expectancy to test the linkage between health capital and growth over a number of samples in a panel data context. The results in this study somewhat corroborate their findings. Their results for 55 Less Developed Countries have the coefficients 0.229, -0.005, 0.106 and -0.308 for investment, education, health and workforce growth respectively. However, they used total years of education as the proxy for education while we use pupil-to-teacher ratio to reflect the quality of education. Though, our results suggest that health capital is seemingly important relative to education capital, the two are quite complementary.

An enchanting feature of this study is its effects to account for the effect of AIDS in order to track down the effect on life expectancy and consequently on economic growth. The coefficient on the variable itself is a manifestation of the iniquitous nature of HIV/AIDS and its effect on the economic development of SSA as well as the life span of affected individuals.

The empirical results also reiterate the importance of removing barriers and fostering trade amongst SSA countries and also internationally. Consequently, the huge income disparities witnessed in the region could be eventually bridged.

4.5 Resource Limitations of the Study

The main limitation of this study is the availability of reliable data that span over a longer period. A number of countries were dropped from the analysis mainly due to data availability. We could use only 30 countries to constitute the sample and even that, we could retrieve data spanning from 1970 on all the variables used in the estimations. Actually, most of the macroeconomic series starts from 1960. Thus using 1970 as a starting point to calculate average growth in SSA is quite dubious. Also, most countries in SSA performed relatively well in the 1970s and then performed worse later. Another major limitation which places a considerable caution in interpreting our results is the quality of data particularly on the GDP per capita and health variables. For instance, a large chunk of the informal sector is unaccounted for in the measurement of GDP. Also, due to lack of reliable data on health, we were forced to use life expectancy as a proxy variable. This might lead to some biases in our results since life expectancy was hampered by some cases of political violence but not necessarily health-related issues.

CHAPTER FIVE

CONCLUSION AND POLICY RECOMMENDATION

In this study, we use the augmented Solow growth model and applied the newly developed panel cointegration estimation strategy to test the relationship between population health and economic growth in SSA for the period 1970 to 2010, alongside some other key determinants of economic growth. Though, this relationship has been empirically tested, the sample usually lumps all countries together even if Less Developed Countries are involved. Also, a more theoretical and exploratory work has been done on the topic, but this study seeks to empirically test it over a number of countries in SSA. Thus, analyzing the role of health capital simultaneously with education on economic performance for a well designated region with similar characteristics is desirable.

Generally, the estimation results of different estimation strategies are quite consistent. Most importantly is the result on the effect of health which does not significantly affect economic growth in SSA. Nevertheless, improvements in the economic performance have the potential of increasing health status and consequently life expectancy in the region. Furthermore, controlling for HIV/AIDS which is ubiquitous in the region produced some interesting results; as the effect of health on economic growth now turned out to be significantly negative. Also, the coefficient on AIDS provides evidence of the lethal nature of the disease and its negative effect on economic performance in the region as well as on the life span of the people. The empirical results also suggest that education is consistently having insignificant effect on economic growth. Indeed, the result is not surprising considering the quality of education in most countries in Africa and the resources channeled into education investment. Another captivating revelation is the complementary effects of both health and education in promoting growth, as the effect of the former is significantly reduced when the latter is omitted from the regression equation. However, the impact of health on economic growth seems greater than education without taking into cognizance their significance levels. The immediate corollary therefore is that, a flotilla of investment in both education and health in SSA is plausibly an option for policymakers to be concerned about. Furthermore, the long-run and short-run results indicate the need for countries in SSA to promote trade since its effects on economic growth has been significantly positive over the sample period.

In view of the intriguing results analysed and considering the economic status of SSA, policymakers should divert their attention and policies to invest more in the public health system as well as providing subsidized and quality education to help the poor greatly. Majority of the population in SSA are considered poor and hence constrained in the expenditures of health care in the event of sudden diseases and ailments. Thus, improving on the efficiency of the public health system as well as providing it at affordable costs would help the poor to access health care, which invariably would improve their health status and consequently life expectancy. This would also have the advantage of reducing the effect of diseases on the cognitive abilities of children who in the long-run can train through school to enhance their human potentialities, and consequently improving per capita income through increased productivity and wages. Also, such policies are expected to help affected individuals who cannot afford the full cost of purchasing antiretroviral drugs and access health care frequently would potentially prolong their life spans and consequently contribute their quota in national development.

References

Acemoglu, D. and Johnson, S. (2006). Disease and development: The effect of life expectancy on economic growth. *NBER Working Paper Series*, No. 12269

Africa's Pulse (2011). *Analysis of Issues Shaping Africa's Economic Future*, Volume 3 available at http://siteresources.worldbank.org/INTAFRICA/Resources/Africas-Pulse-brochure_Vol3.pdf

Arellano, M. and Bond, S. (1991). Some Tests of Specification for Panel Data: Monte Carlo Evidence and an Application to Employment Equations. *Review of Economic Studies*, 58:277-297.

Asteriou, D. and Hall, S. (2007). Applied Econometrics: A Modern Approach (Revised Edition), Palgrave McMillan, Basingstoke.

Baltagi, B. H. (2008). Econometric Analysis of Panel Data (Fourth Edition), John Wiley & Sons, Chichester.

Baltagi, B. H., Griffin, J. M. and Xiong, W. (2000). To pool or not to pool: Homogeneous versus heterogeneous estimators applied to cigarette demand. Review of Economics and Statistics 82: 117-126.

Banerjee, A., Marcellino, M. and Osbat, C. (2005). Testing for PPP: Should We Use Panel Methods?, *Empirical Economics*, 30:77–91.

Barro, R. (1991). Economic growth in a cross-section of countries. *Quarterly Journal of Economics*, 106(2), 403–443.

Barro, R. (1996). Three Models of Health and Economic Growth. *Unpublished manuscript.* Cambridge, MA: Harvard University.

Baum, C. F., Schaffer, M. E., and Stillman, S. (2003). Instrumental variables and GMM: Estimation and testing. Stata Journal 3: 1-31.

Benhabib, J., & Speigel, M. (1994). The role of human capital in economic development: Evidence from aggregate cross-country data. *Journal of Monetary Economics*, *34*(2), 143–173.

Bloom, D. E. and Canning, D. (2000). The Health and Wealth of Nations. *Science* 287: 1207–8.

Bloom, D. E., Canning, D., and Sevilla, J. (2001). The Effect of Health on Economic Growth: Theory and Evidence. *NBER Working Paper* No. 8587.

Bloom, D. E. and Canning, D. (2008). Population Health and Economic Growth. Commission on Growth and Development. Working paper. No. 24.

Bloom, D. E., and Sachs, J. D. (1998). Geography, Demography, and Economic Growth in Africa. *Brookings Papers Econ. Activity*, no. 2: 207–73.

Blundell, R. And Bond, S. (1998). Initial Conditions and Moment Restrictions in Dynamic Panel Data Models. *Journal of Econometrics* 87: 115-143

Breitung, J. (2000). The Local Power of Some Unit Root Tests for Panel Data. In B. Baltagi (ed.), Advances in Econometrics, Vol. 15: Nonstationary Panels, Panel Cointegration, and Dynamic Panels, 161–178, Elsevier Science.

Choi, I. (2001). Unit Root Tests for Panel Data. *Journal of International Money and Finance*, 20(2):249–272.

Cutler, D. M. and Lleras-Muney, A. (2006). Education and Health: Evaluating Theories and Evidence. NBER Working Paper, No. 12352

Elu, J. (2000). Human development in sub-Saharan Africa: Analysis and prospects for the future. Journal of Third World Studies, 17(2) [Association of Third World Studies, Inc.].

Engle, R. and C. Granger (1987). Co-integration and Error-Correction: Representation, Estimation and Testing. *Econometrica*, 55, 251-276.

Falcão, B.L.S; Soares, R. R. (2005). The Demographic Transition and the Sexual Division of Labor. Stanford: Stanford Institute on International Studies, Center on Democracy, Development, and the Rule of Law Working paper, 50.

Fogel, R. W. (2000). *The Fourth Great Awakening and the Future of Egalitarianism*. Chicago and London, University of Chicago Press.

Fogel, R. W. (1991). New sources and new techniques for the study of secular trends in nutritional status, health, mortality and the process of aging. National Bureau of Economic Research, Working Paper Series as Historical Factors and Long Run Growth No. 26.

Fogel, R. W. (1997). New findings on secular trends in nutrition and mortality: some implications for population theory. In: Rosenzweig MR and Stark O, eds. *Handbook of population and family economics*, Vol 1A. Amsterdam, Elsevier, 433–481.

Gallego, J. M. (2000). Aspectos Teóricos sobre la Salud como un Determinante del Crecimiento Económico. *Borradores de Investigación de la Universidad del Rosario* 5.

Gallup, J. L., and Sachs, J. D. (2001). The Economic Burden of Malaria. *American J. Tropical Medicine and Hygiene* 64, suppl. 1 (January): 85–96.

Greene, W. (2003), Econometric Analysis (Fifth Edition), Prentice Hall, New Jersey.

Grossman, M. (1972). On The Concept of Health Capital And The Demand For Health. *Journal of Political Economy* 80: 223-55.

Hadri, K. (2000). Testing for Stationarity in Heterogeneous Panel Data. *Econometrics Journal*, 3:148–161.

Hamoudi, A. A. and Sachs, J. (1999). Economic Consequences of Health Status: A Review of the Evidence. *CID Working Papers Series* No. 30.

Howitt, P. (2005). Health, Human Capital, and Economic Growth: A Schumpeterian perspective. In: López-Casanovas, G.; Rivera, B.; Currais, L. (Eds.). Health and economic growth. Cambridge: The Mit Press.

Im, K., Pesaran, H., and Shin, Y. (2003). Testing for Unit Roots in Heterogeneous Panels. *Journal of Econometrics*, 115:53–74.

Johansen, S. (1988). Statistical Analysis of Cointegration Vectors. *Journal of Economic Dynamics and Control*, 12(2-3), 231-254

Kao, C. and Chiang, M. (2000). On the Estimation and Inference of a Cointegrated Regression in Panel Data. In B. Baltagi (ed.), Advances in Econometrics, Vol. 15: Nonstationary Panels, Panel Cointegration, and Dynamic Panels, 179-222, Elsevier Science.

Kao, C. (1999). Spurious Regression and Residual-Based Tests for Cointegration in Panel Data. *Journal of Econometrics*, 90:1–44.

Knowles, S. and Owen, D. (1995). Health Capital and Cross-Country Variation in Income per Capita in the Mankiw-Romer-Weil model. *Economics Letters*, Vol. 48, pp. 99-106.

Knowles, S. and Owen, D. (1997). Education and Health in an Effective Labour Empirical Growth Model. *Economic Record*, Vol. 73 No. 3, pp. 14-28.

Larsson, R., Lyhagen, J., and Lothgren, M. (2001). Likelihood-Based Cointegration Tests in Heterogeneous Panels. *Econometrics Journal*, 4:109–142.

Levin, A., Lin, C., and Chu, C. (2002). Unit Root Tests in Panel Data: Asymptotic and Finite-Sample Properties. *Journal of Econometrics*, 108:1–24.

Lilliard, L. A. and Weiss, Y. (1997). Uncertain Health and Survival: Effects on End-of-Life consumption. *Journal of Business & Economic Statistics* 15: 254-68.

Lucas, R. E. (1993). Making a Miracle. *Econometrica*, Vol 61, No. 2, March, pp. 251-272

Luft, H. S. (1975). The Impact of Poor Health on Earnings. *Review of Economics and Statistics*, Vol. 57, No. 1, February, pp. 43-57.

Maddala, G. and Wu, S. (1999). A Comparative Study of Unit Root Tests with Panel Data and a New Simple Test. *Oxford Bulletin of Economics and Statistics*, 61:631–52.

Mankiw, N. G., Romer, D. and Weil, D. N. (1992). A Contribution to the Empirics of Economic Growth. *Quaterly Journal of Economics* 107: 407-37.

Martin, P. M. G. (2011). Aid Absorption and Spending in Africa: A Panel Cointegration Approach. *Journal of Development Studies*, ISSN 0022-0388, Vol 47, Issue 12, pp. 1925-1953

Mayer, D., Mora, H., Cermeño, R., and Duryeau, S. (2000). Salud, Crecimiento y Distribución en Latinoamérica y el Caribe: Un Estudio de Determinantes y Comportamiento Regional y Local. *Documentos Técnicos - Investigaciones en Salud Pública*, Panamerican Health Organization.

Mbaku, J. M. (1997). Institutions and Reforms in Africa: The Public Choice Perspective. Westport, CT: Praeger.

McDonald, S. and Roberts, J. (2002). Growth and Multiple Forms of Human Capital in an augmented Solow model: a panel data investigation, Economics Letters, Vol. 74, pp. 271-6.

Mkandawire, T., & Soludo, C. (1999). African Perspectives on Structural Adjustment: Our Continent or Future. New Jersey: Codesria, African World Press.

Pedroni, P. (2004). Panel Cointegration; Asymptotic and Finite Sample Properties of Pooled Time Series Tests with an Application to the PPP Hypothesis. *Econometric Theory*, 20, 597-625.

Pedroni, P. (1999). Critical Values for Cointegration Tests in Heterogeneous Panels with Multiple Regressors. *Oxford Bulletin of Economics and Statistics*, 61:653–70.

Pesaran, H. (2007). A Simple Panel Unit Root Test in the Presence of Cross-Section Dependence. *Journal of Applied Econometrics,* 22(2), 265-312.

Pesaran, H. (2004). General Diagnostic Tests for Cross Section Dependence in Panels. Cambridge Working Papers in Economics 0435, Faculty of Economics, University of Cambridge.

Pesaran, H., Shin, Y., and Smith, R. (1999). Pooled Mean Group Estimation of Dynamic Heterogeneous Panels. *Journal of the American Statistical Association*, 94(446):621–634.

Persyn, D. and Westerlund, J. (2008). Error Correction Based cointegration Tests for Panel Data. Stata Journal 8 (2), 232-241.

Prah R. J., Jamison D. T. & Bloom D. E. (2001). Health and the Economy, in *International Public Health*, edited by Michael H. Merson, Robert E. Black & Anne J. Mills (Sudbury, Massachusetts: Jones e Barlett).

Romer, P. M. (1990). Endogenous technical change. *Journal of Political Economy*, *98*(5), S71–S102.

Roodman, D. (2009). A Note on the Theme of Too Many Instruments. *Oxford Bulletin of Economics and Statistics*, 71(1), 135–158.

Saikkonen, P. (1991). Asymptotically Efficient Estimation of Cointegration Regressions. *Econometric Theory*, 7(1), 1-21.

Sachs, J. D. (2001). Macroeconomics and Health: Investing in Health for Economic Development. Report of the Commission on Macroeconomics and Health. World Health Organization, 2001.

Smith, J. P. (1998). Socioeconomic Status and Health. *American Economic Review* 88: 192-6.

Smith, J. P. (1999). Healthy Bodies and Thick Wallets: The Dual Relation between Health and Socioeconomic Status. *Journal of Economic Perspectives* 13: 145-66.

Stock, J. and M. Watson (1993). A Simple Estimator of Cointegrating Vectors in Higher Order Integrated Systems. *Econometrica*, 61(4), 783-820.

Strauss, J. and Thomas, D. (1995). Health, Nutrition and Economic Development. *Santa Monica, CA, Rand* (*Labour and Population Program Paper Series* 95-23)

United Nations (2007). *The Millennium Development Goals Report* (New York: UN): 4, accessed online at www.un.org/millenniumgoals/pdf/mdg2007.pdf, on May 20, 2012

United Nations (2011) EDStats available at http://web.worldbank.org/WBSITE/EXTERNAL/TOPICS/EXTEDUCATION/EXTDATASTATISTICS/EXTEDSTATS/0,,contentMDK:21528857~menuPK:4324013~pagePK:64168445~piPK:64168309~theSitePK:3232764,00.html?primary

Webber, D.J. (2002). Policies to stimulate growth: should we invest in health or education, Applied Economics, Vol. 34, pp. 1633-43.

Weil, D. N. (2005). Accounting for the Effect of Health on Economic Growth. National Bureau of Economic Research, 11455

Westerlund, J. (2007). Testing for Error Correction in Panel Data. *Oxford Bulletin of Economics and Statistics*, 69(6), 709-748.

World Bank (various years). World Development Report, 2000/2001, 1998–99, 1997, 1995, and 1993 issues. The World Bank, Washington, DC.

World Bank (2011). *African Development Indicators*. Washington DC: World Bank. Available at http://data.worldbank.org/data-catalog/africa-development-indicators

World Bank (2011). *World Development Indicators*. Washington DC: World Bank. Available at http://data.worldbank.org/data-catalog/world-development-indicators

World Health Organisation (2001). *Macroeconomics and Health: Investing in Health for Economic Development*. http://www3.who.int/whosis/cmh.

Appendix

A1: List of Countries in the Sample

Benin, Botswana, Burkina Faso, Burundi, Cameroon, Central African Republic, Chad, Congo Democratic Republic, Congo Republic, Cote d'Ivoire, Gabon, Gambia, Ghana, Guinea Bissau, Kenya, Lesotho, Madagascar, Malawi, Mali, Mauritius, Niger, Rwanda, Senegal, Sierra Leone, South Africa, Swaziland, Togo, Uganda, Zambia, Zimbabwe

Table A1: Summary Statistics

Variable	Obs	Mean	Std. Dev.	Min	Max
GDPPC	1230	754.9061	1121.005	82.67	8594.71
GFCF	1230	19.00695	9.39558	1.93	76.69
LE	1230	50.58369	7.030479	26.82	72.97
PTRATIO	1230	44.5475	13.65549	19.82	100.24
OPEN	1230	67.91698	34.75617	6.32	209.41
$(n+g+\delta)$	1230	2.64674	1.021801	-7.48	9.82

Table A2: Least Squares Regression to Test for Cross-Sectional Dependence

Regressor	Coefficient	Std. Err.	t	P>t
ln*GDPPC*(-1)	-0.0558	0.0094	-5.94	0.000
ln*GFCF*(-1)	0.0291	0.0076	3.82	0.000
ln*LE*(-1)	-0.0473	0.0480	-0.99	0.325
ln*PTRATIO*(-1)	0.0036	0.0140	0.26	0.798
ln*OPEN*(-1)	0.0137	0.0111	1.23	0.218
PGRW(-1)	-0.0025	0.0051	-0.49	0.627
Δln*GDPPC*(-1)	0.1327	0.0368	3.61	0.000
FD ln*GFCF*	0.0104	0.0097	1.07	0.285
Δln*GFCF*(1)	0.0497	0.0099	5.00	0.000
Δln*GFCF*(-1)	-0.0125	0.0097	-1.29	0.199
FDln*LE*	0.4535	1.0390	0.44	0.663
Δln*LE*(1)	0.2010	2.4431	0.08	0.934
Δln*LE*(-1)	-0.6656	1.7864	-0.37	0.710
FDln*PTRATIO*	-0.0045	0.03187	-0.14	0.887
Δln*PTRATIO*(1)	0.0194	0.0324	0.60	0.550
Δln*PTRATIO*(-1)	0.0265	0.0312	0.85	0.395
FD ln*OPEN*	0.0145	0.0146	0.99	0.323
Δln*OPEN*(1)	-0.0055	0.0153	-0.36	0.721
Δln*OPEN*(-1)	0.0128	0.0150	0.86	0.392
FD *PGRW*	0.1605	0.0541	2.97	0.003
Δ*PGRW*(1)	-0.2541	0.0893	-2.85	0.005
Δ*PGRW*(-1)	0.1454	0.0533	2.73	0.007
Year	-0.00004	0.0003	-0.13	0.900
Constant	0.4504	0.5068	0.89	0.374

Dependent Variable: DlnGDPPC FD: First Difference

Breusch-Pagan LM test of independence: Chi2(171) = 214.924, Pr = 0.0128

Table A3: Panel Cointegration Estimation Results for lnRGDPPC controlling for HIV/AIDS

	(1)	(2)	(3)	(4)	(5)
	SYS-GMM	DOLS	PMG	MG	DFE
Dependent variable:	ln*RGDPPC*	ln*RGDPPC*	Δln*RGDPPC*	Δln*RGDPPC*	Δln*RGDPPC*
Convergence coefficients	0.99*** (0.002)		-0.12*** (0.02)	-0.43*** (0.04)	-0.05*** (0.01)
Long-run coefficients					
ln*LE*		3.25*** (0.18)	-0.31** (0.13)	-0.23*** (0.01)	-0.004 (0.48)
ln*GFCF*		0.36*** (0.05)	0.30*** (0.03)	0.11** (0.05)	0.40** (0.16)
ln*PTRATIO*		-0.51*** (0.07)	0.20*** (0.07)	-0.21 (0.17)	0.11 (0.20)
ln*OPEN*		0.31*** (0.06)	-0.01 (0.05)	0.15 (0.11)	0.33* (0.17)
$(n+g+\delta)$		-0.17*** (0.03)	0.01 (0.01)	-0.01 (0.04)	0.08* (0.05)
AIDS		-0.14*** (0.05)	-0.12*** (0.02)	-0.05*** (0.01)	-0.22*** (0.07)
Short-run coefficients					
ΔlnLE	0.04** (0.02)		0.17 (0.66)	0.24 (1.26)	-0.09 (0.14)
ΔlnGFCF	0.03*** (0.01)		0.03** (0.01)	0.01 (0.01)	0.04** (0.02)
ΔlnPTRATIO	-0.01 (0.01)		-0.002 (0.04)	0.04 (0.03)	0.01 (0.02)
ΔlnOPEN	0.004 (0.007)		-0.04** (0.02)	-0.06*** (0.02)	-0.05 (0.04)
$\Delta(n+g+\delta)$	0.002 (0.002)		-0.02 (0.01)	-0.13** (0.06)	0.003 (0.004)
ΔAIDS	-0.01*** (0.003)		-0.004 (0.008)	-0.01 (0.01)	-0.001 (0.008)
Hausman test (χ^2)			18.09*** [0.002]	18.09*** [0.002]	
Hansen test (χ^2)	20.51 [1.00]				
No. of countries	30	30	30	30	30
No. of observations	1200	1140	1200	1200	1200

Notes: All equations include a constant country-specific term. Values in ( ) and [ ] are standard errors and probability values respectively. For DFE estimates, the standard errors are heteroskedatic consistent. SYS-GMM generates 1141 instruments for 1200 observations. The speed of adjustment for SYS-GMM equals one minus the coefficient on the lagged dependent variable (0.01). Hansen test is the test of overidentifying restrictions (the null hypothesis is that the instruments are valid).***, ** and * indicate significance at the 1%, 5% and 10% levels respectively.

Table A4: Panel Cointegration Estimation Results for lnLE controlling for HIV/AIDS

	(1)	(2)	(3)	(4)	(5)
	SYS-GMM	DOLS	PMG	MG	DFE
Dependent variable:	lnLE	lnLE	ΔlnLE	ΔlnLE	ΔlnLE
Convergence	0.97***		-0.04***	-0.03*	-0.04***
coefficients	(0.01)		(0.01)	(0.01)	(0.001)
Long-run coefficients					
ln*RGDPPC*		0.12***	0.17***	2.19***	0.19**
		(0.001)	(0.01)	(0.71)	(0.10)
ln*GFCF*		0.02***	0.05***	0.76	0.13
		(0.008)	(0.01)	(0.75)	(0.08)
ln*PTRATIO*		-0.05***	0.55***	1.52	0.08
		(0.01)	(0.06)	(1.78)	(0.17)
$(n+g+\delta)$		0.03***	0.14***	0.23	0.11***
		(0.003)	(0.004)	(0.14)	(0.02)
AIDS		-0.08***	-0.04***	-0.07***	-0.20***
		(0.002)	(0.01)	(0.02)	(0.04)
Short-run coefficients					
Δln*RGDPPC*	0.0004		-0.004	0.05***	0.01***
	(0.001)		(0.006)	(0.004)	(0.004)
Δln*GFCF*	0.002		0.001	-0.001	-0.001
	(0.002)		(0.002)	(0.002)	(0.002)
Δln*PTRATIO*	-0.003		-0.02**	-0.01	-0.01**
	(0.002)		(0.003)	(0.01)	(0.004)
$\Delta(n+g+\delta)$	0.004***		0.02***	0.01*	0.03***
	(0.001)		(0.006)	(0.005)	(0.002)
ΔAIDS	-0.02		-0.03*	-0.12***	-0.19***
	(0.02)		(0.01)	(0.04)	(0.03)
Hausman test (χ^2)				0.01	0.01
				[1.00]	[1.00]
Hansen test (χ^2)	28.15				
	[1.00]				
No. of countries	30	30	30	30	30
No. of observations	1200	1140	1200	1200	1200

Notes: All equations include a constant country-specific term. Values in ( ) and [ ] are standard errors and probability values respectively. For DFE estimates, the standard errors are heteroskedatic consistent. SYS-GMM generates 1124 instruments for 1200 observations. The speed of adjustment for SYS-GMM equals one minus the coefficient on the lagged dependent variable (0.02). Hansen test is the test of overidentifying restrictions (the null hypothesis is that the instruments are valid).***, ** and * indicate significance at the 1%, 5% and 10% levels respectively.