Essays on Health and Economic Growth

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Abstract of thesis entitled: Essays on Health and Economic growth

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For economists who study the growth theories, the most critical issues they are trying to deal with are the sources of long-term growth and the problem of inequality. Stimulated by the endogenous growth theories, a substantial amount of studies focus on how technology improvement and human capital in education promote economic growth. In this thesis, however, we focus on another important source of growth, the role of health in generating economic growth and generating development traps. We discuss the issues of long-term growth and inequality in the first two essays respectively and examine empirically the relationship between health and economic growth in the last essay.

In the first essay, we analyze the endogenous growth generated by health accumulation. We extend the Barro (1996b) model to consider both the positive and negative effects of health by endogenizing the health depreciation rate. We consider three forms of health depreciation rate: constant health depreciation rate, health depreciation rate determined only by health, and health depreciation rate determined simultaneously by health and education. We also consider the situation when health affects economic growth through entering the utility function directly. By comparing the results from the optimization processes, we find that whether health enters the utility function does not affect long-term growth. What really matters is

the specific form of health depreciation rate.

In the second essay, we analyze the issue of health related development traps. Various mechanisms of health related development traps have been proposed by recent literature. The general characteristics of these mechanisms are that there are stable multiple equilibriums. However, the statistics show that the gap between the rich countries and the poor ones are actually widening from 1960 to 2007. To explain this phenomenon, we develop another mechanism to generate health related development traps, through which the gap between the developed and developing countries is widening. To check the sensitivity of the results to the specific form of health utility function, we also employ a more general form of health utility function.

In the last essay, we complement the first two essays by analyzing empirically the relationship between health expenditure and economic growth. We summarize that there are three main categories of macroeconomic empirical research on the relationship between health and economic growth. Relatively few focuses on how health investment affects economic growth. We analyze this relationship in the last essay by employing both the Mankiw, Romer and Weil (1992) model and the Bassanini and Scarpetta (2001) model. Several econometric methods are used for robustness checking. The statistical results show that health expenditure at least has non-negative effect on economic growth.

論文摘要

在第一篇論文中，我們研究健康所產生的內生增長問題。我們擴展 Barro (1996b) 中的模型，通過內生健康折舊函數來綜合地考慮健康對經濟增長效應。我們考慮了三種健康折舊的情況：第一種是健康折舊是一個固定值，第二種是健康折舊是健康的函數，最後一種是健康折舊是健康和教育的函數。我們也考慮了健康通過影響人們的效用函數從而影響經濟增長的情況。通過比較各種情況的最優化結果，我們發現健康是否進入效用函數並不影響長期經濟增長。真正影響長期經濟增長的是健康折舊的具體形式。

在第二篇論文中，我們研究是的是健康相關的貧困性陷阱問題。最近的研究中，不同的健康相關的貧困性陷阱產生機制被提出。這些機制的共同特點是多重穩定平衡點的存在。可是，統計資料表明，在 1960 到 2007 年間，富裕國家和貧窮國家之間的差距是不斷擴大的。爲了解釋這個現象，我們建立了一個健康相關的貧困性陷阱產生機制，通過這個機制，發達和落後國家之間的差距是不斷擴大的，這個機制更加符合現實生活的實際情況。爲了檢驗我們的研究結果是否對具體的健康效用函數形式敏感，我們也採用另外一種更爲一般的健康效用函數。

在最後一篇論文中，我們通過實證的方法對健康消費和經濟增長之間的關係進行研究，這個是對前兩篇的理論模型的一個補充。我們總結到，宏觀實證分析中，研究健康和經濟增長之間關係的實證研究一共有三大類。本論文則研究這三大類中相對比較少人研究的一類，關於健康投資如何影響經濟增長。我們採用了 Mankiw, Romer and Weil (1992) 模型以及 Bassanini and Scarpetta (2001) 模型來進行回歸分析. 我們採用了幾種常用的計量方法來進行結果的可靠性分析。統計結果表明，健康投資對經濟增長的作用至少是非負的。

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Chapter 1 Introduction

The issue of economic growth is always the hottest topic among economists. The theory of economic growth has a long history dating back to the late 18 century when the analysis of economic growth was at the center of attention of classical economists such as Smith (1776), Malthus (1798) and Ricardo (1817). These studies identified important causes and mechanisms that affect economic growth. The most important result from them is that the accumulation and investment of the production output is the main driving force behind economic growth. The much later works of Ramsey (1928), Young (1928), Schumpeter (1934) and Knight (1944), which emphasize the elements of competition, equilibrium dynamics, diminishing returns, the accumulation of physical and human capital and the monopoly power gained from technology advances, formed a good basis for the neoclassical growth theories and the endogenous growth theories developed after the middle 20 century. The models of Solow (1956) and Swan (1956) use a production function approach where there are constant returns to scale but diminishing return to each input. The equilibrium will exist if certain conditions are satisfied. The growth rate of the economy is determined exclusively by the exogenous technology. In other words, there will be long-term economic growth only if there are continuous new technologies available. One important finding of the neoclassical model is the theory of "conditional convergence" which shows that the growth rate of the economy will be faster the further this economy is below its own equilibrium level. The historical facts show that the positive rate of economic growth persists over a century and there is no trend of decline. The property of diminishing return of the inputs determines that the

neoclassical models explain everything but long-term growth. To overcome this modeling deficiency, researches on endogenous growth such as Romer (1986), Lucas (1988) and Romer (1990), which emphasize the roles technology changes and human capital accumulation in the form of education play, help to generate some important results confirming the important roles of technology changes and education in promoting long-term growth.

For economists who study the growth theories, it is necessary for them to explain the real world situations using economic models and econometric methods and then provide useful suggestions to overcome the obstacles that impede economic growth. The most critical issues which the economists are trying to deal with now are the sources of long-term growth and the problem of inequality. Countries at different development stages, from the developed countries, like the U.S., to the developing countries, like China, are all facing the problem of constructing a sustainable growth model. It is crucial for economists to analyze different sources and mechanisms of economic growth to assist the policymakers to make efficient political decisions. Another important issue is the problem of inequality. Maddison (2001) shows that the ratio of average GDP per capita of the richest group of countries to that of the poorest grew by 90 percent from 1950 to 1998. Mayer-Foulkes (2002) also finds that the same ratio grew by a factor of 2.6 from 1960 to 1995. Furthermore, the statistics shown in Tables 3.1 to 3.3 indicate that the gap between the rich and the poor countries is not only persistent but also widening from 1960 to 2007. It is emergent and challenging for the economists to find remedies to narrow this gap and the analysis of the mechanism behind the generation of this gap becomes

critical. As stated earlier in this introduction, the first wave of endogenous growth models, such as Romer (1986), Lucas (1988), Romer (1990) and other latter studies inspired by these three early studies have emphasized education and technological progress as important sources for long-term economic growth. However, health, as another important form of human capital, has hardly been researched in the theoretical literature. Health, education and income have been regarded as the three pillars of human development in the Human Development Index (HDI) (UNDP, 1990). Health is ranked number one in things men and women desired in life in the huge worldwide survey prepared for the Millennium Report of the Secretary-General of the United Nations. The relatively less coverage of health in theoretical literature has also translated into decisions on resource allocation. The policymakers who make budgetary allocations often think of health only as a good thing but not a kind of investment that can stimulate economic growth. The importance of health only stays as being one asset people value most highly but not a source of growth which results in insufficient allocation of financial resources. However, the characteristics of health in promoting economic growth have been well recognized by historical facts. Fogel (1991, 1997, and 2000) have used historical facts to demonstrate that health is a powerful engine of economic growth. In his study, food supply has been identified as a crucial factor to long-term labor productivity. He argues that "the increase in the amount of calories available for work over the past 200 years must have made a nontrivial contribution to the growth rate of the per capita income of countries such as France and Great Britain." Fogel further explains that the effect of nutrition intake on economic growth is attributable to an increase in the productive labor force

and the provision of sufficient calories to the existing workforce. Each of them contributes 0.11 percent and 0.23 percent to the per capita GDP growth between 1780 and 1980, respectively. In other words, considering that the annual rate of growth in Great Britain during this period is about 1.15 percent, about 30 percent of the British per capita GDP growth is attributable to improvement in nutrition intake. Other researches, such as Barro (1997), Arora (1999), Bloom and Canning (2000) and Hernandez, Fuentes, and Pascual (2001) also show positive effect of other health related variables on economic growth. From the above facts, we know that health has significant impact on economic growth historically. To trace the source of long-term growth and income inequality, among the many causal factors economists have proposed, health stands out as a likely candidate.

The interaction mechanism between health and economic growth is complicated. On one hand, health affects economic growth through channels such as labor productivity, return on education and demographic transition. On the other hand, economic growth would influence health human capital and health investment through various channels such as nutrition intake, housing and medical care services. There is a bilateral causality relationship between health and economic growth. To give an overview of the interaction mechanism between health and economic growth, we use Figure 1.1 to depict the interaction network.

The interaction network presented in Figure 1.1 can be divided into three parts denoted by 'I', 'II', 'III'. In Part I, health affect economic growth through labor productivity. Improvement in health would allow the worker to work more efficiently, increase the amount of effective working hours and lower the probability of being absent from work either by the worker or

his/her family members. Better health status would also increase the life expectancy and thus prolong the working ages which would encourage investment in education because the return on education investment is higher with longer effective working time. All these channels would lead to improvement in labor productivity which results in economic growth. The second main way through which health influences economic growth is health affecting the utility of an individual, which is indicated by part II of Figure 1.1. Grossman (1972) first proposes that health can be viewed as a consumption commodity which enters an individual's utility function directly because sick days are a source of disutility. As being healthy would increase an individual's utility, it would stimulate investment in health which would affect physical capital investment and thus affect economic growth. Most of the existing literature discussing how health affects investment in health through entering the utility function focuses from a microeconomic perspective on the provision of health care services and the demand for health. Examples include Grossman (1972, 1982), Foster (1989), Enrilch and Chuma (1990), Johansson and Lofgren (1995) and Meltzer (1997). There is little research analyzing how health affects economic growth through this mechanism. The few examples include Zon and Muysken (2001, 2003) and Wang, Gong and Li (2008). Part III shows the last main way of the interaction mechanism between health and economic growth. Improvement in economic growth would increase the income level of an individual which would allow the individual to consume more and increase investment in health. Improvement in nutrition intakes and more advanced medical treatments would then lead to improvement in an individual's health level. The most influential researches on this part of

health and growth interaction mechanism are Fogel (1994a, 1994b and 2002). Other studies include the extensive reviews by Strauss and Thomas (1998), Sohn (2000), and Thomas (2001). The three parts of the interaction network illustrated in Figure 1.1 describe the interaction mechanism between health and economic growth from a simple way. In fact, these three parts influence each other. In the existing literature on the relationship between health and economic growth, economists usually follow two or all the three parts of this interaction network.

In this thesis, encouraged by the above historical facts, we first analyze theoretically and systematically the mechanisms of how health, interacting with other factors, such as education and physical capital inputs, affects long-term economic growth. Secondly, we suggest a mechanism that generates health related development traps which explains the vital role of health in determining the widening gap between the rich and poor countries. Lastly, we complement the theoretical analyses with an empirical study analyzing the relationship between health expenditure and economic growth.

There are three essays in this thesis. In the current literature on the effect of health on economic growth, there are two strands of studies. One strand is the analyses on how health determines long-run economic growth and the other strand is the studies on the relationship between health and income inequality. The first two essays focus theoretically on each of these two strands of studies respectively. The last essay examines empirically the relationship between health expenditure and economic growth.

In the first essay, we analyze systematically on how health affects long-term economic growth based on the Barro (1996b) model, which is the

first theoretical framework considering the effect of health on economic growth. In the Barro (1996b) model, health affects economic growth by entering the production function directly, which corresponds to part I of Figure 1.1.

One of the modeling deficiencies of the Barro (1996b) framework is that it is still not able to explain long-term growth. We solve this modeling deficiency by proving that as long as there are constant returns to scale with respect to physical capital and human capital in the forms of both education and health together, there is endogenous growth in the Barro (1996b) model. In the current literature analyzing the effect of health on economic growth, focus is mainly on the positive effect of health on economic growth, such as improving labor productivity, increasing returns to investment in education and longer life expectancy. Few studies take heed of the negative effect of health on economic growth explicitly. Based on the Barro (1996b) framework, inspired by the argument made by Grossman (1972) that health depreciation rate should not be constant, we endogenize the health depreciation rate by considering the following two cases: (1) health depreciation is determined exclusively by health; (2) health depreciation rate is jointly determined by health and education. In these two cases, the negative effect of health on economic growth is reflected explicitly by the endogenous health depreciation rate which is a positive function of health. The optimization results show that when the endogenous health depreciation rate is determined only by health, the negative effect of health on economic growth would be too strong to generate endogenous growth in the long-term. In contrast, if we consider the effect of education on lowering the health depreciation rate simultaneously with the positive effect of health on health

depreciation rate, the effect of education on lowering the health depreciation would be strong enough to counteract the effect of health and we are able to find endogenous growth in the long-term.

Following the idea of Grossman (1972), we also consider the situation that health is regarded as a consumption good which generates 'good health'. For this situation, except for directly entering the production, health also affects economic growth by entering the utility function directly, which corresponds to the second part of Figure 1. The same as the situation that health does not enter the utility function, we consider different forms of endogenous health depreciation rate. The optimization results show that whether health enters the utility function or not does not affect the results of endogenous growth. What really matters is the specific form of health depreciation rate. Because we can view the health depreciation rate as a component of the health accumulation function, it is the exact form of the health accumulation function (or the health production function) playing a determining role in long-run economic growth.

In the second essay, we analyze the problem of income inequality by studying the role health plays in generating development traps. We first illustrate the summary statistics on regional economic performance and health situations. From those statistical results, we find that the income gap between the developed and developing countries is not only persistent but also widening between 1960 and 2007. To explain the mechanism behind this inferior situation in the real world, we develop a model that generates health related development traps. This model we construct is a two period overlapping generations model based on the Arrow-Romer production function. Health enters both the production function and the utility function.

The optimization process shows that health related development traps are generated. Although there are other studies which also generate health related development traps, the one generated in this thesis has a widening gap between the rich and poor countries, which is more consistent with the summary statistics presented in Tables 3.11 to 3.3. Furthermore, we notice that the utility function we use to generate the health related development traps is a logarithm utility function. Although this logarithm utility function is a standard form of utility function used in analyzing the health related development traps, we still want to see whether the specific form of health utility function may affect the resulting health related development traps. We know that the logarithm form of the health utility function is a special form of the utility function with constant absolute risk aversion (CARA) and thus we substitute the logarithm health utility function by the CARA one. In this case, the dynamics of physical capital is jointly determined by its previous value and the value of health. We thus linearize both health and physical capitals around the steady state and get the dynamic equation of physical capital. Although we are not able to prove explicitly whether the equilibrium value is stable or not, we use numerical method to prove that the equilibrium is indeed unstable which indicates that even when we substitute the logarithm health utility function by the corresponding CARA form of utility function, we are still able to generate a widening health related development traps. The health related development traps generated in the second essay provide a possible explanation to the widening gap between the rich and poor countries, which emphasizes the important role of health in generating income inequality.

In the third essay, we complement the two theoretical essays by

conducting an empirical analysis on the relationship between health expenditure and economic growth. There have already been a considerable amount of empirical studies analyzing the relationship between health and economic growth from both microeconomic and macroeconomic perspectives. The macroeconomic literature examines the relationship between health and economic growth through the links among social health spending, social indicators of health and economic growth. Most of these studies focus on the relationship between different social indicators of health and economic growth, which confirm the positive relation between social health indicators and economic growth. There are also some studies analyzing the causality relationship between social spending and social health indicators but the results are mixed. However, relatively little studies gauges the effect of social health expenditure on economic growth directly. In this thesis, we fill in this gap in the existing literature by analyzing the relationship between health expenditure and economic growth. We analyze the effect of health on both per capita output and growth rate while considering simultaneously both health and education as two forms of human capital. To analyze the effect of health expenditure on per capita output, we employ the augmented Solow model while the augmented Bassanini and Scarpetta (2001) model is used to study the relationship between health expenditure and per capita GDP growth. The dataset we employ is a panel dataset consisting of 138 countries at different development stages from all over the world. An East Asia dataset will also be used as a comparison. Because according to Islam (2000), fixed effect estimator (LSDV) has excellent short time series performance compared to instrumental variable estimators and generalized method of moments

(GMM) estimators, we will use LSDV to draw the baseline estimation results. For robustness checking, two stage least square (2SLS), GMM and bias-corrected LSDV estimators will be employed. The statistical results show that health expenditure at least has a non-negative effect on economic growth.

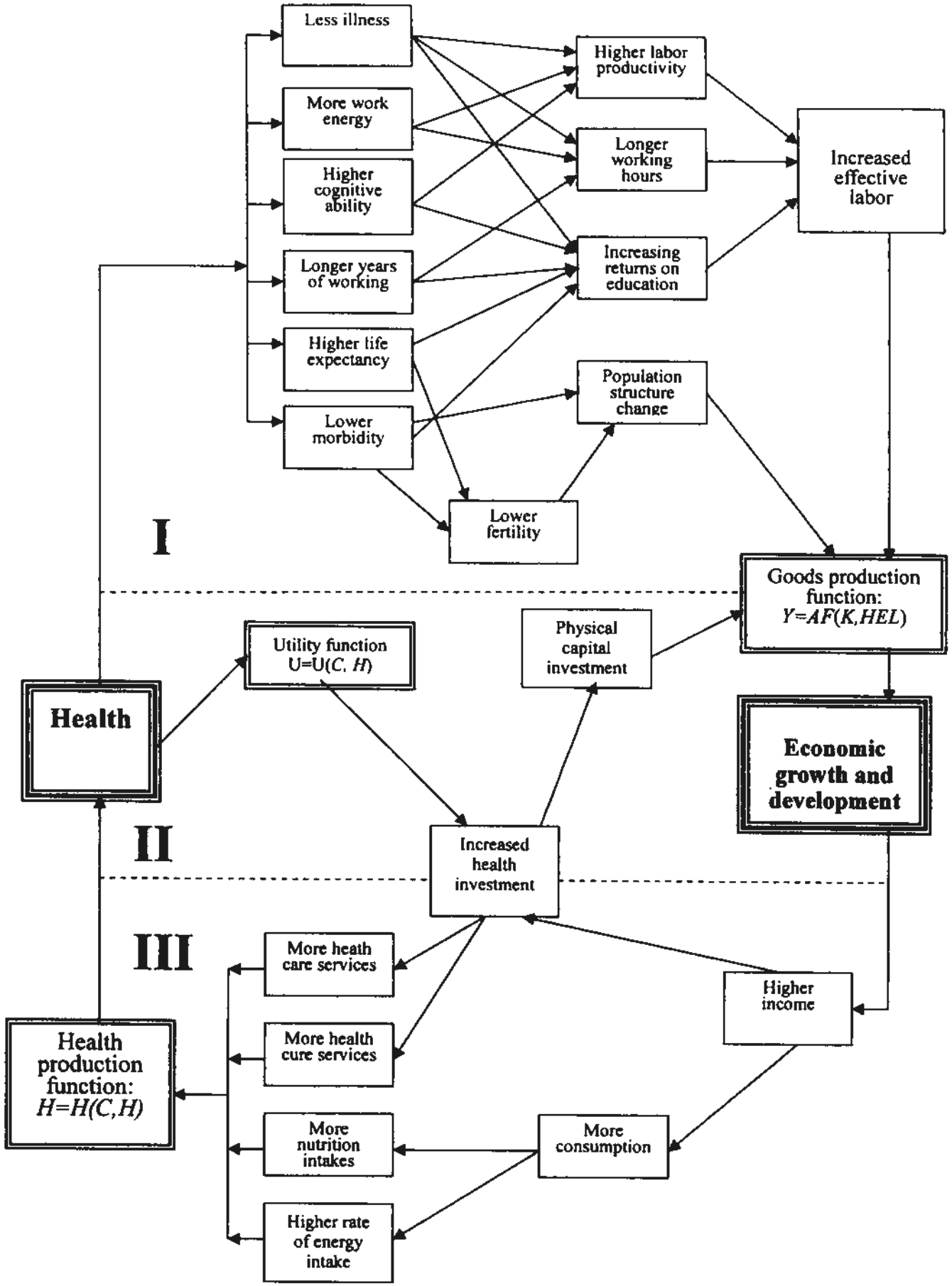


Figure 1.1 The interaction network between health and growth[1]

[1] We thank Professor Wang Dihai for making valuable suggestions on this Figure.

Chapter 2 Endogenous Growth with Health and Human Capital: Does Health Depreciation Rate Matter?

2.1 Introduction

In the current literature on the effect of health on economic growth, there are two strands of studies. One strand is on the analyses of how health determines long-run economic growth and the other strand is on the relationship between health and income inequality. In this essay, our focus is on the first strand of the study. The idea that health can be viewed as another form of human capital has been proposed in early studies such as Mushkin (1962), Becker (1964), Fuchs (1966) and Grossman (1972). However, it is not until Barro (1996b) that the first theoretical framework to analyze the effect of health on economic growth appeared. In his paper, Barro explicitly incorporates health into the production function to consider the effect of health on goods production and derives conclusions similar to those of the neo-classical models. Based on the Lucas (1988) model, Zon and Muysken (2001, 2003) consider the influence of health not only on goods production, but also on an individual's utility. Zon and Muysken (2001, 2003) guess that if the health depreciation rate is an increasing function of health, the endogenous growth derived in their model may disappear. In the current essay, we improve the Barro (1996b) model and Zon and Muysken (2001, 2003) model by systematically analyzing the relationship between health and economic growth based on the Barro (1996b) model. We consider not only the case that health only affects goods production as Barro (1996b) did but also the case that health influences both production and utility as Zon and Muysken (2001, 2003) did. Moreover, our study is an improvement on

Zon and Muysken (2001, 2003) because we prove, not just guess, how health depreciation rate affects economic growth by analyzing different forms of endogenous health depreciation rate. The importance of health depreciation rate was first emphasized by Grossman (1972), which confirms that one central limitation of the demand model of health developed in the paper is that the health depreciation rate is treated constant overtime. This assumption is not appropriate because people with different health status or education levels should have different health depreciation rates. In this essay, we fill in this research gap by proposing an extended Barro (1996b) model which considers different endogenous health depreciation rates. Moreover, following the idea of Grossman (1972), we further analyze the endogenous health depreciation rates in an extended Barro (1996b) model considering health in the utility function. We are interested in analyzing whether endogenous health depreciation rates would affect long-run economic growth and furthermore whether health entering the utility function would change the results. In the remaining part of this introduction, we will provide some background information on the models we construct in the latter sections.

2.1.1 The health depreciation rate

As health depreciation is one component in the health accumulation function, we are interested in endogenizing the health depreciation rate in order to reflect the negative effect of health in promoting economic growth. Our idea of endogenous health depreciation rate is supported by Grossman (1972). In the Grossman (1972) paper, health has been identified as another important form of human capital, which provides a good starting point for

researchers to analyze the relationship between health and economic growth. However, as accepted by Grossman, health depreciation rate should vary over time. To understand why the health depreciation rate should not be constant, we should first understand the definition of health depreciation rate, which is the cost of maintaining the current level of health. There are many examples to show why the health depreciation rate should not be a constant. For example, before a major competition like the Olympic Games, an athlete needs to spend time on training, to eat following the instruction of dietitian and to check his/her body fitness regularly. In order to keep the match fitness, the investment is huge. However, after the competition, he/she no longer needs to keep that high level of match fitness and the expenditure to keep his/her non-match fitness level of health would be lower. Another example is that one of the significant indicators of better health is life expectancy. The increase in life expectancy is attributable to the major advances in medical science which enable the doctors to diagnose and treat illness in a previously impossible way. However, CBO (2008) argues that the nature of the technological advances in medicines and the changes in the clinical practices that followed them tends to raise the health spending, which indicates that to keep a better health, which is indicated by longer life expectancy, it is necessary to spend more on health. By the definition of health depreciation rate, we find that those examples seem to indicate that the health depreciation rate is positively related to health. Therefore, it is interesting to follow the suggestion of Grossman (1972) to endogenize the health depreciation rate and see how different specifications of endogenous health depreciation rate may lead to different results of long-run growth. We will consider three specifications of health depreciation rate. Firstly, because

our analysis is based on the framework of Barro (1996b), we follow the specification in his paper to assume the health depreciation rate as a negative function of health in the short-run but constant in the long-run. Under this assumption, health depreciation rate can be viewed as the mortality rate of an individual. As the health status of an individual improves, the physical condition will decline in a slower rate. Secondly, following the two examples we presented earlier in this sub-section, we consider the situation that the health depreciation rate is an increasing function of health, which is from the standard definition of depreciation rate. [2] The intuition for this specification of health depreciation rate is that as we are healthier and with better physical condition, it needs more investment in health care and health cure services in order to maintain such level of health. CBO (2008) is another example to support the construction of the health depreciation rate as an increasing function of health.[3] Lastly, because more educated people are more efficient producer of health, we also consider the negative effect of education on health depreciation rate by employing one form of health depreciation rate as an increasing function of health but a decreasing function of education.

2.1.2 The traditional models of economic growth

The endogenous model we develop in this essay is based on the

[2] The standard definition of health depreciation rate is that the depreciation rate is the amount needed to be invested in order to keep the current level of health.

[3] Some people may argue that the definition of health depreciation rate as an increasing function of health is not reasonable, because there is no upper limit on the potential level of health. However, we argue that the potential level of health is still uncertain and there may be huge space of improvement for health in the future as more and more researches are conducted. Therefore, we maintain that health depreciation rate is an increasing function of health. Moreover, if we try different specific values of the health depreciation rate, the results are consistent with the results presented later in this essay.

traditional models of economic growth, which refer mainly to the neoclassical models developed between 1950s and 1960s. The Solow-Swan model (Solow 1956 and Swan 1956) provides an easy framework to describe a general equilibrium of an economy. The Cass-Koopmans model (Cass, 1965 and Koopmans, 1965) integrates the consumer optimization conditions of Ramsey (1928) with the neoclassical growth model, which provides the decisions on aggregate economics on a microeconomic basis.

There are two important features that characterize the neoclassical growth models. The first one is conditional convergence. The convergence properties in neoclassical models can be categorized into two cases. The first one is absolute convergence, which received mixed reviews when examined with empirical data.[4] In this case, because all the countries are assumed to have the same intrinsic characteristics[5] except for different initial real GDP per capita levels, the models predict that poor countries will grow faster than the rich ones and eventually all the countries will converge to the same stable level of real GDP per capita. However, in the real world, the empirical evidences tell us that rich countries tend to be richer and healthier as the economies develop and the gap between rich countries and poor countries was widening in the last few decades. To explain this real world scenario, the concept of conditional convergence was proposed. In the settings of conditional convergence, countries are assumed to have different characteristics. The differences in characteristics, such as the propensity to save and invest, the government policies, the leisure time the workers require and the willingness to go to school, lead countries to converge to

[4] See pp 45-47 of Barro and Sala-i-Martin (2005) for a more detailed presentation of the empirical results.

[5] The intrinsic characteristics refer to the saving rates, population growth rates and the depreciation rates, etc.

different steady state positions. Any countries with similar characteristics converge to the same steady state and the difference in growth rates among them depends on the initial per capita GDP level related to the steady-state level. In the context of conditional convergence, a poor country, which the absolute convergent theory predicts that will grow rapidly, may grow in a slow rate because its steady-state position is also low. The conditional convergence theory reconciles the fact of widening gap between the rich countries and poor countries and the convergent property predicted by the neoclassical models. In the framework of neoclassical models, the source behind the convergence, the assumption of diminishing returns in capital, is also important. In the neoclassical growth models, because of diminishing returns, one economy will grow faster if it has less per capita capital in relation to its steady-state level of per capita capital. From the perspective of conditional convergence, this kind of convergence is also affected by characteristics such as the propensity to consume, to have children, the government policies, the openness of the markets and the level of technology the workers are able to access.

Another important feature of neoclassical models is that these models predict that the growth rate of capital per capita is determined entirely by exogenous technology. An economy will cease to grow if there is no change in exogenous technology. In other words, in these kinds of neoclassical models, the steady state per capita growth rate equals the rate of technological progress. Thus, these models are not theoretically satisfactory in explaining the determinants of long-run growth, although they provide interesting and convenient frameworks to analyze the transitional dynamics.

Because of the diminishing returns to capital, the characteristics of the

neoclassical models enable those models to explain everything but the determinants of long-run growth, which is an obvious model deficiency. These models are not helpful for understanding the sources of long-run per capita growth. The endogenous growth models explored in the next subsection overcome this model deficiency of the neoclassical models.

2.1.3 The endogenous growth theory

To solve for the model deficiency of the neoclassical model indicated in the previous subsection, extra sources of growth, typically the inclusion of human components, should be introduced into the neoclassical models so that the diminishing returns does not apply to this broadened class of capital. Following this idea, the endogenous growth models were introduced, which were broadly categorized by the 'engine of growth'. Two groups of endogenous growth models were developed since the mid 1980s. In the first group, growth is generated by positive externality and increasing returns generated from the investment in or the accumulation of physical capital and human capital. In these models, the positive externality from a broadened class of capitals may introduce non-diminishing returns as an economy develops and this externality provides the source for the economy to grow indefinitely. One early example of this line of research is Romer (1986) where he combined the externalities, increasing returns in the production of output, and decreasing returns in the production of new knowledge to derive qualitative conclusions. Xie (1991) provided explicit dynamic solution to the qualitative conclusions of Romer (1986). Lucas (1988) is another early study in the line of research, where investment in schooling is regarded as another channel for endogenous growth. Mulligan and Sala-i-Martin (1991),

Benhabib and Perli (1994) and Xie (1994) provided transitional dynamics for the Lucas (1988) model. Other prototypical examples of these investment-based endogenous growth models, which were based on the building blocks created by Arrow (1962), Uzawa (1965) and Sheshinski (1967), include Barro (1990), Rebelo (1991) and Jones (1995). In the second group, the idea came from the view that technological progress is the only way to escape from diminishing returns in the long run. The 'growth engine' of this group of endogenous growth models is a separate research and development sector which creates new technologies in the form of economic goods and the market rewards the producers who own the new technologies monopoly power over some interval. In the framework of this stream of researches, growth will persist as long as new ideas continue to come out. However, the resulting equilibrium is not Pareto optimum because the competitive framework breaks down. The main examples of this group of technological based researches are Romer (1990), Grossman and Helpman (1991) and Aghion and Howitt (1992).

In this essay, because we concentrate on the relationship between the health human capital and long-run economic growth, we follow the first type of endogenous growth models with a focus on the role of the depreciation rate of health. In the first type of endogenous model, the long run tendency for capital to experience diminishing returns in the neoclassical model is eliminated by broadening the concept of capital to incorporate both physical capital and human capital. The spillover of knowledge of the learning by doing mechanism (Arrow, 1962; Sheshinski, 1967 and Romer, 1986) and the extra returns generated by the incorporation of human capital (see for example, Lucas, 1988) enable the returns to

investment on capital not necessarily diminishing even in the absence of technology progress as an economy develops. The main research interest of this essay is to build on the fundamental ideas of this stream of endogenous growth studies and focus on whether different specifications of health depreciation rate affect the basic macroeconomic relationship between health and long-run economic growth. The concept of human capital in the first wave of the endogenous growth literature mainly confines to education. One example is Lucas (1988), where the model developed by Uzawa (1965) was used to construct a model with endogenous growth generated by human capital accumulation through schooling. However, health, as another important form of human capital, did not receive enough attention until Barro (1996b), in which focus was on the two-way interplay between health and economic growth. Barro (1996b) is the first study to provide a theoretical framework and forms a basis for future researches on the relationship between health and economic growth. However, there are deficiencies in Barro's model: there is no endogenous growth and this model only considered the effect that health affects economic growth through labor productivity but not utility. Extending the Lucas (1988) endogenous growth framework, Zon and Muysken (2001, 2003) analyze the effect of health on economic growth by considering three channels through which health can affect economic growth.[6] In their paper, they followed Grossman (1972) to incorporate health into the utility function and they argued that growth may disappear as demand for health care services was increasing because of an

[6] The three channels Zon and Muysken (2001, 2003) proposed that may affect intertemperoral decision making are: first, health is a prerequisite for the accumulation of human capital and the provision of health services; second, health can generate positive utility of its own; lastly, the provision of health competes with production of goods and accumulation of human capital for labor services.

aging population and this may form a 'crowding out' effect on the healthy labor time that should have been invested in the production of capital goods.

2.1.4 The mechanism behind the generation of endogenous growth of the Barro (1996b)-type models

Although there is no endogenous growth in the Barro (1996b) model, this model provides a good framework for analyzing the effect of health on economic growth. It is interesting to analyze under what conditions endogenous growth can be generated in this Barro (1996b) framework and more importantly, how the endogenous growth generated is affected by the health depreciation rate.

In this essay, with a focus on the effect of health depreciation rate on economic growth, we analyze the mechanism behind the generation of endogenous growth based on Barro (1996b) framework. Firstly, we extend the Barro (1996b) model by assuming constant returns to scale to physical, education and health capitals together while maintaining the same assumptions on the depreciation rates of those three capitals[7] as those in Barro (1996b). Under this scenario, we find that there is endogenous growth in the Barro (1996b) framework model. Secondly, we revise the previous model by assuming constant depreciation rates for physical and education while the depreciation rate for health is assumed to be a function of health only. In this model, however, there is no endogenous growth. Lastly, we maintain the assumption of constant depreciation rates for physical and education but replace the depreciation rate of health by an explicit function

[7] In Barro (1996b), depreciation rate on physical capital is assumed to be constant, while depreciation rates of education and health are assumed to be the same and a decreasing function of health. In the steady state, this health depreciation rate will converge to a constant value.

of both health and education. Endogenous growth exists in this revised model. The results from these different extended Barro (1996b) models show that the definition of depreciation rate of health does matter for endogenous growth of Barro (1996b)-type models. Following the ideas of Grossman (1972) and Zon and Muysken (2001, 2003), we further analyze the effect of different specifications of health depreciation rate on endogenous growth by incorporating health into the utility function. By comparing the results generated from where health enters the utility function with those where only consumption is in the utility function, we find that no matter whether or not health enters the utility function, the endogenous growth in the Barro (1996b)-type models are sensitive to the exact specification of health depreciation rate but not to whether health affects an individual's utility. This result contradicts to what Grossman (1972) and Zon and Muysken (2001, 2003) argue.

The rest of this essay is organized as follows: section 2.2 presents a brief review of the Barro (1996b) model. Based on this framework, we consider in section 2.3 the impact of different specifications of health depreciation rate on the generation of endogenous growth in the scenario of constant returns to scale for physical capital, education and health. In section 2.4, we further analyze the effects of different health depreciation rates on endogenous growth under an extended Barro (1996b) model by incorporating health in the utility function and compare the findings with those obtained in section 2.3 and Zon and Muysken (2001, 2003). Section 2.5 is the concluding remarks.

2.2 The Barro model of health and economic growth

For a long time, economists considered health as a byproduct of economic growth and they seemed to neglect the fact that health is also an important determinant of economic growth. As we stated in the introduction, the endogenous growth models proposed in late 1980s and early 1990s focused overwhelmingly on the effect of human capital in the form of education on economic growth while little attention was paid to the contribution of human capital in the form of health to economic growth.[8] Barro (1996b) proposed a one-sector model which extended the neoclassical model to incorporate the impact of health on economic growth. In his model, health affects economic growth both directly and indirectly. First, health directly enters production function indicating a direct impact of health on productivity. In other words, if other determinants of the production function, such as physical capital, labor and schooling, are all constant, an improvement in an individual's health would increase the productivity. Second, health also determines the depreciation rate of both health and education. Therefore, health contributes to economic growth indirectly through its effect on education.

2.2.1 The Barro growth model revisited

In the Barro model, health is a private good that is financed totally by the individuals themselves. Investments in health include activities such as the purchase of nutrition products, the leisure time spent on sports, the money paid on doctors and medicines, a regular body check, etc.

The economy is a one sector economy. First, total output Y is

[8] Ehrlich and Liu (1991), Meltzer (1995) and Barro (1996a) are the early researches in this direction.

determined in a Cobb-Douglas function by physical capital inputs, K, individual's schooling and other education related factors, S, the health capital of individual, H, and the amount of labor provided, L:

$$Y = AK^{\alpha}S^{\beta}H^{\chi}(L)^{1-\alpha-\beta-\chi} \tag{2.1}$$

where A is the knowledge stock parameter, which represents the exogenously determined technology level. The model assumes that $\alpha > 0, \beta > 0, \chi > 0$, and $\alpha + \beta + \chi < 1$. That is, in this production function, Barro assumes constant returns to scale with respect to the four inputs (physical capital, education, health and labor) but diminishing returns with respect to each of the inputs respectively. This is a key assumption to derive the results of the Barro model. One thing to pay attention to these assumptions is that constant returns to scale with respect to the four inputs imply diminishing returns to scale with respect to the inputs of physical capital, education and health together.

The intensive form of the production function can be obtained by dividing both sides of the Cobb-Douglas function by the effective labor Le^{xt}:

$$\tilde{y} = Y / Le^{xt} = AK^{\alpha}S^{\beta}H^{\chi}(L)^{1-\alpha-\beta-\chi} / Le^{xt} = A\tilde{k}^{\alpha}\tilde{s}^{\beta}\tilde{h}^{\chi} \tag{2.2}$$

where $\tilde{y} = Y / Le^{xt}$, $\tilde{k} = K / Le^{xt}$, $\tilde{s} = S / Le^{xt}$, and $\tilde{h} = H / Le^{xt}$ are quantities of per unit of effective labor. x is the exogenous rate of technology growth and t is a time variable. The representative household in the economy faces a typical intertemporal optimization problem of maximizing the following utility function subjected to the budget constraints:

$$\max_{c_t} \int_0^{+\infty} \frac{c^{1-\gamma}-1}{1-\gamma} e^{-(\rho-n)t} dt$$

$$s.t.: \dot{\tilde{k}} = \tilde{i}_k - (\delta^k + x + n)\tilde{k}$$

$$\dot{\tilde{h}} = \tilde{i}_h - (\delta^h + x + n)\tilde{h}$$

$$\dot{\tilde{s}} = \tilde{i}_s - (\delta^h + x + n)\tilde{s}$$

$$A\tilde{k}^{\alpha}\tilde{s}^{\beta}\tilde{h}^{1-\alpha-\beta-x} = \tilde{i}_k + \tilde{i}_h + \tilde{i}_s + \tilde{c}$$

where Barro assumed the social utility function to trade off future and present consumption possibility with a constant elasticity of substitution. c is consumption per person, $0<\gamma \leq 1$ is the reverse of the intertemporal elasticity of substitution, $\rho > 0$ is the rate of discount, and $n > 0$ is a constant exogenous rate of population growth. $\dot{\tilde{k}}$, $\dot{\tilde{h}}$ and $\dot{\tilde{s}}$ are the evolutions of the three kinds of capital stocks. The depreciation rate of capital stock is assumed to be δ^k. However, different from the standard setting, Barro assumed another key condition here: the depreciation rates for human capital in education and health are equal and depend only on health. The depreciation rate of the human capital in the form of education and health is represented by the function δ^h, where δ^h is a decreasing function of the stock of health capital per capita, h:

$$\delta^h = \delta^h(h) \tag{2.3}$$

By solving the dynamic optimization problem, Barro got the evolution of consumption over time:

$$\frac{\dot{c}}{c} = \frac{1}{\theta}(\alpha A\tilde{k}^{\alpha-1}\tilde{s}^{\beta}\tilde{h}^{x} - \delta^k - \rho) \tag{2.4}$$

where the first two terms inside the parentheses are the net rate of marginal returns on physical capital. The term $\frac{\dot{c}}{c}$, as usual, represents the growth rate of consumption per person c.

From the condition of equal marginal rate of returns to the three kinds

of capitals, the following expression can be obtained:

$$\begin{aligned}
&\alpha A\tilde{k}^{\alpha-1}\tilde{s}^{\beta}\tilde{h}^{\chi}-\delta^{k} \\
&=\beta A\tilde{k}^{\alpha}\tilde{s}^{\beta-1}\tilde{h}^{\chi}-\delta^{h} \\
&=(1-\alpha-\beta)A\tilde{k}^{\alpha}\tilde{s}^{\beta}\tilde{h}^{\chi-1}-\delta^{h}-(s+h)\frac{\partial\delta^{h}}{\partial h}
\end{aligned} \tag{2.5}$$

where s and h are per capita schooling and health. In the long run, the effect of health on health depreciation rate will reach a lower bound and $\frac{\partial\delta^{h}}{\partial h}$ will converge to zero. In other words, the steady state value of health depreciation rate δ^{h} is constant.

From Barro's calculation, much of the dynamics of this model is similar to the neoclassical growth model with single form of capital. The model shows diminishing rate of returns to capital and the growth rate is only determined by exogenous technological changes. There is no endogenous growth existing in this model. A special case Barro mentioned is where the depreciation rates of physical capital and human capital are the same. That is where $\delta^{k}=\delta^{h}$. In this case, equations (2.3) to (2.5) imply that the ratios between the every two of the capitals employed would be constant along the dynamic path. In the steady state, all the capital goods would grow at the same constant rate which we denote as x_1, which is the exogenously determined technology growth rate. Barro also pointed out the negative effect of rising health on human capital depreciation rate and thus increasing the ratios of schooling and health capital to physical.

Although the fundamental results derived from the Barro (1996b) model are similar to those of the neoclassical models, the Barro (1996b) model provides a useful framework for analyzing the interaction between health and economic growth. In the next two sections, we extend the Barro

(1996b) model in two ways to explore how endogenous growth is generated based on the Barro (1996b) framework and more importantly, how different formations of health depreciation rate affect the endogenous growth.

2.3 Barro (1996b) model with different specifications of health depreciation rate and constant returns to scale

We first assume that per capita output is constant returns to scale with respect to physical, education and health capitals together in all the models discussed in this and next section.[9] Because our focus is on how the endogenous health depreciation rate affects economic growth, we assume that depreciation rates of physical capital and education capital are constants. Different specifications of health depreciation rate are then applied to the Barro (1996b) framework. We want to analyze whether endogenous growth in the Barro (1996b)-type models is sensitive to the specifications of health depreciation rate.

2.3.1 Barro (1996b) model with constant health depreciation rate

In the Barro (1996b) model, although the model considers the effect of human capital in the form of both health and education in the production function, the obvious shortcoming of the neoclassical model still exists in this model. The long run per capita growth rate is still determined entirely by the exogenous technological factor which is the rate of technology growth. Thus, we take this established framework and try to elaborate upon this model to get more insights on the conditions that enable endogenous

[9] In the Barro (1996b) model, however, per capita output is assumed to be diminishing returns to scale with respect to physical, education and health capitals together.

long-run economic growth, one characteristic that traditional neoclassical growth model failed to explain.

In this subsection, we assume that $\alpha > 0, \beta > 0, \chi > 0$ which is the same as what is assumed in the basic Barro framework. This condition implies that there are diminishing returns with respect to the three capital inputs respectively. However, we also assume one key condition, $\alpha + \beta + \chi = 1$, which is different from the original Barro (1996b) model. Under this assumption, we assume that the productivity is constant returns to scale in the physical, education and health inputs together. The condition of constant returns to scale will be a default assumption for the models proposed in the rest of this essay.

For the production function, it will not affect the analysis if we continue to assume the technological progress term A to be constant. Thus, the production function of this model is:

$$Y = AK^{\alpha}S^{\beta}H^{1-\alpha-\beta} \tag{2.6}$$

where $1-\alpha-\beta = \chi$. The intensive form of the production function becomes $y = Y/L = AK^{\alpha}S^{\beta}H^{1-\alpha-\beta}/L = Ak^{\alpha}s^{\beta}h^{1-\alpha-\beta}$, where y, k, s, h represent per capita output, physical, education and health capital respectively.

We use one simple form of utility function to analyze the consumer optimization problem presented below:

$$\max_{c_t} \int_0^{\infty} u(c)e^{-(\rho+n)t}dt \tag{2.7}$$

$$s.t.: \dot{k} = I_k - (\delta^k + n)k \tag{2.8}$$

$$\dot{s} = I_s - (\delta^h + n)s \tag{2.9}$$

$$\dot{h} = I_h - (\delta^h + n)h \tag{2.10}$$

where $u(c) = c^r$ [10] and $0<r \leq 1$. The three components I_k, I_h, and I_s represent the gross investment flows per capita.

The Hamiltonian function of this problem[11] is:

$$H = c^r + v_k[I_k - (\delta^k + n)k] + v_s[I_s - (\delta^h + n)s] + v_h[I_h - (\delta^h + n)h] + \lambda[y - I_k - I_h - I_s - c]$$

The first order conditions can be obtained in the usual manner by equating the derivatives of H with respect to c, I_k, I_s, and I_h to 0 and setting the derivatives of H with respect to k, s, h to be:

$$\frac{\partial H}{\partial c} = rc^{r-1} - \lambda = 0 \tag{2.11}$$

$$\frac{\partial H}{\partial I_k} = \frac{\partial H}{\partial I_h} = \frac{\partial H}{\partial I_s} \Rightarrow v_k = v_s = v_h = \lambda \tag{2.12}$$

$$\frac{\partial H}{\partial k} = -\dot{v}_k + (\rho - n)v_k = -v_k(\delta^k + n) + \alpha\lambda k^{\alpha-1}s^{\beta}h^{1-\alpha-\beta} \tag{2.13}$$

$$\frac{\partial H}{\partial s} = -\dot{v}_s + (\rho - n)v_s = -v_s(\delta^h + n) + \beta\lambda k^{\alpha}s^{\beta-1}h^{1-\alpha-\beta} \tag{2.14}$$

$$\frac{\partial H}{\partial h} = -\dot{v}_h + (\rho - n)v_h = -v_h(\delta^h + n) + (1-\alpha-\beta)\lambda k^{\alpha}s^{\beta}h^{-\alpha-\beta} - \frac{\partial \delta^h}{\partial h}(v_h h + v_s s) \tag{2.15}$$

Differentiating equation (2.11) with respect to time and rearranging it together with equations (2.12) and (2.13), we get:

$$\frac{\dot{c}}{c} = \frac{\alpha A(\frac{k}{h})^{\alpha-1}(\frac{s}{h})^{\beta} - \delta - \rho}{1-\gamma} \tag{2.16}$$

Equation (2.16) implies that the growth rate of consumption is

[10] The use of c^r as the utility function is for convenience purpose, which does not affect the key results derived in this essay.

[11] We use a constraint Hamiltonian function in the derivation of the optimal conditions. For technical details of constraint Hamiltonian function, please check Appendix 2.1. The utility function $\frac{c^{1-\gamma}-1}{1-\gamma}e^{-(\rho-n)t}$ is replaced by c^r for convenient purpose as well.

determined by the ratios of physical capital to health and education to health.

Furthermore, in the competitive environment, the marginal products of the three capital inputs are equal, which can also be derived from equations (2.12) to (2.15). Thus, we have the following equalities

$$\begin{aligned} &\alpha A k^{\alpha-1} s^{\beta} h^{1-\alpha-\beta} - \delta^{k} \\ &= \beta A k^{\alpha} s^{\beta-1} h^{1-\alpha-\beta} - \delta^{h} \\ &= (1-\alpha-\beta) A k^{\alpha} s^{\beta} h^{-(\alpha+\beta)} - \delta^{h} - (s+h)\frac{\partial \delta^{h}}{\partial h} \end{aligned} \tag{2.17}$$

In Barro (1996b), the health depreciation rate δ^{h} is defined as a decreasing function of health. To facilitate interpretation, we can view health depreciation rate as mortality rate in the Barro (1996b) model. Barro (1996b) argues that the human capital depreciation rate δ^{h} has a lower bound, which is quite intuitive. The logic behind Barro's argument is that no matter how much you invest in health care measures, for example, doing exercises every day, having a body check regularly, or spending money on nutrition to keep a good health, your health status will depreciate at some level and the mortality rate will rise as your age increases. However, investment in health precautionary measures may improve the health status and decrease the health depreciation rate δ^{h}. In the long run, the model will eventually reach a steady state level where δ^{h} will converge to a lower bound level δ_{0}^{h}. At this level, the impact of health investment on the changes of the depreciation rate of health will be approximately zero. As a result, the constant depreciation rates δ^{k} and δ^{h} appear in the long run. A more detailed argument can be found in Barro (1996b).

In this essay, our research interest is to analyze the long run economic growth. Therefore, we only focus on the steady state situation. In the steady

state, from the second equality of equation (2.17), we have

$$\frac{s}{h} = \frac{\beta}{1-\alpha-\beta} \tag{2.18}$$

Equation (2.18) indicates that in the steady state, the ratio of education to health is constant. In other words, these two human capitals grow at the same rate in the steady state.

Furthermore, from the first equality relation in equation (2.16), we get another equation:

$$\delta^h - \delta^k = \left[\beta A \left(\frac{k}{h}\right)^{\alpha} \left(\frac{s}{h}\right)^{\beta-1} - \alpha A \left(\frac{k}{h}\right)^{\alpha-1} \left(\frac{s}{h}\right)^{\beta} \right] \tag{2.19}$$

In the steady state, we already know that the education to health ratio is constant and the ratio of capital to health can be derived from equation (2.19).

To analyze the balanced growth path, we first assume one special case, where we have equal depreciation rates for the three capital inputs: $\delta^k = \delta^h$. With this assumption, we are able to obtain the ratio of physical capital to health immediately from equations (2.18) and (2.19)

$$\frac{k}{h} = \frac{\alpha}{1-\alpha-\beta} \tag{2.20}$$

Next, we analyze the case where $\delta^k \neq \delta^h$ and assume that $\frac{k}{h} = \omega$. We have the following equation:

$$\delta^h - \delta^k = \left[\beta A \left(\frac{\beta}{1-\alpha-\beta}\right)^{\beta-1} \omega^{\alpha} - \alpha A \left(\frac{\beta}{1-\alpha-\beta}\right)^{\beta} \omega^{\alpha-1} \right] \tag{2.21}$$

Equation (2.21) is a function of ω. If equation (2.21) has positive roots, we are able to prove that for the case where $\delta^h \neq \delta^k$, the ratio of capital to health is also constant, which is the same as the case where $\delta^h = \delta^k$. In Appendix 2.2, we prove that equation (2.21) has at least one

positive root. In other words, the ratio of physical capital to health is also constant in the steady state.

By equations (2.18) and (2.20), we obtain that in the steady state, if the depreciation rates of the three capital inputs are the same, the growth rates of these three capital inputs are also the same.

$$\frac{\dot{k}}{k}=\frac{\dot{s}}{s}=\frac{\dot{h}}{h} \tag{2.22}$$

Based on equations (2.8), (2.9), (2.10), (2.22) and the intensive form of the production function, we have an equation of $\frac{i_k}{k}$ and $\frac{c}{k}$:

$$A(\frac{\beta}{\alpha})^{\beta}\cdot(\frac{1-\alpha-\beta}{\alpha})^{1-\alpha-\beta}=\frac{i_k}{k}\cdot\frac{1}{\alpha}+\frac{c}{k} \tag{2.23}$$

Furthermore, from equation (2.8), we know that in the steady state, $\frac{i_k}{k}$ is constant. Thus, from equation (2.23), we get that the ratio of consumption to capital is also constant in the steady state. In other words, the steady state growth rates of consumption and the three capital inputs are the same.

Differentiating the intensive form of the production function, we find that the growth rate of the output depends on the growth rates of the three capital outputs. Considering that the growth rates of the three capital inputs and the consumption are equal, we conclude that in the steady state, the growth rates of output, the three capital inputs and consumption are the same, which is denoted by r^*:

$$r^*=\frac{\dot{y}}{y}=\frac{\dot{k}}{k}=\frac{\dot{s}}{s}=\frac{\dot{h}}{h}=\frac{\dot{c}}{c} \tag{2.24}$$

Inserting equations (2.18) and (2.20) into equation (2.16), we have the expression for the steady state growth rate r^*:

$$r^*=\frac{\alpha A\left(\frac{\alpha}{1-\alpha-\beta}\right)^{\alpha-1}\left(\frac{\beta}{1-\alpha-\beta}\right)^{\beta}-\delta-\rho}{\theta} \tag{2.25}$$

If we assume $\alpha A\left(\frac{\alpha}{1-\alpha-\beta}\right)^{\alpha-1}\left(\frac{\beta}{1-\alpha-\beta}\right)^{\beta} > \delta+\rho$, the growth rate r^* will be positive and there is an endogenous growth in the steady state. That is, all quantities y, k, s, h, and c will grow at the constant rate r^* shown in equation (2.25).

Substituting equations (2.18) and (2.20) into the intensive form of the production function and rearranging the terms, I have the following expression for the intensive form of output y:

$$y = Ah\cdot\left(\frac{\alpha}{1-\alpha-\beta}\right)^{\alpha}\left(\frac{\beta}{1-\alpha-\beta}\right)^{\beta} \tag{2.26}$$

The relationship between the model derived in this section and the traditional AK model is clear if we regard the term $A\left(\frac{\alpha}{1-\alpha-\beta}\right)^{\alpha}\left(\frac{\beta}{1-\alpha-\beta}\right)^{\beta}$ as the technology progress term in the AK model and health replace physical capital. The technical details of the above results are available in Appendix 2.3.

The above results show that as long as the condition of constant returns to scale with respect to the three capital inputs are satisfied in the original Barro (1996b) model, we are able to derive endogenous growth in the steady state.

Theorem 2.1: In the Barro (1996b) framework, when we broaden the concept of capital to include both education and health, we are able to derive endogenous growth as long as we have constant returns to scale with respect to the three capital inputs together. Health capital plays the same role in generating endogenous growth as education when only education is

considered as human capital. The resulting model is just an AK model.

2.3.2 Barro (1996b) model with health depreciation rate determined only by health

In the previous section, we extend the Barro (1996b) model by assuming constant returns to scale with respect to physical, education and health capitals together. There is endogenous growth in such a model. For that model, we assume as in Barro (1996b) that the depreciation rate of physical capital δ^k is constant. Furthermore, the depreciation rates of education and health capitals, δ^h, are equal, which is assumed to be a decreasing function of health in the short-run but constant in the long-run as in Barro (1996b). However, from the examples we showed in the introduction and the definition of health depreciation rate as the cost to maintain the current level of health, we argue that the depreciation rate of health will be an increasing function of health. In this subsection, we are interested in analyzing whether the result of the existence of endogenous growth in the previous subsection is sensitive to the change of specification of health depreciation rate.

In the model constructed in the remaining part of this subsection, the same as in the previous subsection, we extend the original Barro (1996b) model by assuming constant returns to scale with respect to the three kinds of capitals. However, in this subsection, we assume that the depreciation rate of health is $\delta^h = h^\theta$ where $\theta > 0$, which indicates that the depreciation rate of health can be a positive function of health with either decreasing, constant or increasing marginal rate of returns. For the depreciation rates of physical and education capital, we assume them to

be δ^k and δ^s, which are constants.[12]

The Hamiltonian function for this case is:

$$H = c^{\gamma} + v_k[I_k - (\delta^k + n)k] + v_s[I_s - (\delta^s + n)s] + v_h[I_h - (\delta^h(h) + n)h] + \lambda(k^{\alpha}s^{\beta}h^{1-\alpha-\beta} - I_k - I_s - I_h - c) \quad (2.27)$$

where, as in the previous section, constraint Hamiltonian function is used.

The first order conditions for the Hamiltonian function (2.27) are:

$$\frac{\partial H}{\partial c} = 0 \Rightarrow \gamma c^{\gamma-1} = \lambda \quad (2.28)$$

$$\frac{\partial H}{\partial k} = -\dot{v}_k + (\rho - n)v_k = -v_k(\delta^k + n) + \alpha\lambda k^{\alpha-1}s^{\beta}h^{1-\alpha-\beta} \quad (2.29)$$

$$\frac{\partial H}{\partial s} = -\dot{v}_s + (\rho - n)v_s = -v_s(\delta^s + n) + \beta\lambda k^{\alpha}s^{\beta-1}h^{1-\alpha-\beta} \quad (2.30)$$

$$\frac{\partial H}{\partial h} = -\dot{v}_h + (\rho - n)v_h = -v_h(\delta^h + n) + (1-\alpha-\beta)\lambda k^{\alpha}s^{\beta}h^{-\alpha-\beta} - v_h\delta_h^h(h)h \quad (2.31)$$

$$\frac{\partial H}{\partial I_k} = \frac{\partial H}{\partial I_s} = \frac{\partial H}{\partial I_h} = 0 \Rightarrow v_k = v_s = v_h = \lambda \quad (2.32)$$

where $\delta_h^h = \frac{\partial \delta^h}{\partial h} = \theta h^{\theta-1}$.

From equations (2.28) to (2.32), we have the equations for consumption growth rate:

$$-(r-1)\frac{\dot{c}}{c} = -(\rho + \delta^k) + \alpha k^{\alpha-1}s^{\beta}h^{1-\alpha-\beta} \quad (2.33)$$

$$-(r-1)\frac{\dot{c}}{c} = -(\rho + \delta^s) + \beta k^{\alpha}s^{\beta-1}h^{1-\alpha-\beta} \quad (2.34)$$

$$-(r-1)\frac{\dot{c}}{c} = -(\rho + h^{\theta}) + (1-\alpha-\beta)k^{\alpha}s^{\beta}h^{-\alpha-\beta} - \theta h^{\theta} \quad (2.35)$$

If we assume that $\frac{\dot{k}}{k} = \frac{\dot{s}}{s} = \frac{\dot{h}}{h} = \frac{\dot{c}}{c} = 0$ on the balanced growth path, we have the following equations from equations (2.33) to (2.35):

$$\alpha k^{\alpha-1}s^{\beta}h^{1-\alpha-\beta} = \rho + \delta^k \quad (2.36)$$

[12] Different from Barro (1996b), we assume that the education depreciation rate δ^s is not a function of health, because an individual's health status will not affect the knowledge this individual has already obtained. As long as this individual is still alive, the knowledge he/she has learnt will not disappear. However, we may extend this model from this direction in future extension.

$$\beta k^{\alpha} s^{\beta-1} h^{1-\alpha-\beta} = \rho + \delta^{s} \tag{2.37}$$

$$(1-\alpha-\beta) k^{\alpha} s^{\beta} h^{-\alpha-\beta} = (1+\theta) h^{\theta} + \rho \tag{2.38}$$

From equations (2.36) and (2.38), we have an equation of k as a function of h and other parameters:

$$k = \frac{\alpha h\left[(1+\theta)h^{\theta} + \rho\right]}{(1-\alpha-\beta)\left(\rho + \delta^{k}\right)} \tag{2.39}$$

From equations (2.37) and (2.38), we have an equation of s as a function of h and other parameters:

$$s = \frac{\beta h\left[(1+\theta)h^{\theta} + \rho\right]}{(1-\alpha-\beta)(\rho + \delta^{s})} \tag{2.40}$$

Inserting equation (2.39) and (2.40) into equation (2.36), we are able to obtain the equilibrium value of h, which we define as h^{*}:

$$h^{*} = \left[\frac{(1-\alpha-\beta)\left(\rho+\delta^{k}\right)^{\frac{\alpha}{\alpha+\beta-1}} (\rho+\delta^{s})^{\frac{\beta}{\alpha+\beta-1}}}{(1+\theta)\alpha^{\frac{\alpha}{\alpha+\beta-1}} \beta^{\frac{\beta}{\alpha+\beta-1}}} - \frac{\rho}{1+\theta} \right]^{\frac{1}{\theta}} \tag{2.41}$$

Assume that $\dfrac{(1-\alpha-\beta)\left(\rho+\delta^{k}\right)^{\frac{\alpha}{\alpha+\beta-1}} (\rho+\delta^{s})^{\frac{\beta}{\alpha+\beta-1}}}{(1+\theta)\alpha^{\frac{\alpha}{\alpha+\beta-1}} \beta^{\frac{\beta}{\alpha+\beta-1}}} = \kappa$, then the equilibrium value of health is:

$$h^{*} = \left(\kappa - \frac{\rho}{1+\theta} \right)^{\frac{1}{\theta}} \tag{2.42}$$

By putting the equilibrium value h^{*} into equations (2.39) and (2.40), we have the equilibrium values of k and s, which we define as k^{*} and s^{*}:

$$k^{*} = \frac{(1+\theta)\kappa\alpha\left(\kappa - \dfrac{\rho}{1+\theta} \right)^{\frac{1}{\theta}}}{(1-\alpha-\beta)\left(\rho+\delta^{k}\right)} \tag{2.43}$$

$$s^* = \frac{(1+\theta)\kappa\beta\left(\kappa - \frac{\rho}{1+\theta}\right)^{\frac{1}{\theta}}}{(1-\alpha-\beta)(\rho+\delta^s)} \tag{2.44}$$

In the steady state, the growth rates $\dot{k} = \dot{s} = \dot{h} = \dot{c} = 0$ and therefore the budget constraint is:

$$k^{*\alpha} s^{*\beta} h^{*1-\alpha-\beta} - (\delta^k + n)k^* - (\delta^s + n)s^* - (\delta^h(h) + n)h^* - c^* = 0 \tag{2.45}$$

The steady state value of consumption c can be derived from equations (2.42) to (2.45):

$$\begin{aligned} c^* = & \left[\frac{(1+\theta)\kappa\alpha\left(\kappa - \frac{\rho}{1+\theta}\right)^{\frac{1}{\theta}}}{(1-\alpha-\beta)\left(\rho+\delta^k\right)}\right]^{\alpha}\left[\frac{(1+\theta)\kappa\beta\left(\kappa - \frac{\rho}{1+\theta}\right)^{\frac{1}{\theta}}}{(1-\alpha-\beta)(\rho+\delta^s)}\right]^{\beta}\left(\kappa - \frac{\rho}{2}\right)^{1-\alpha-\beta} \\ & - (\delta^k + n)\frac{(1+\theta)\kappa\alpha\left(\kappa - \frac{\rho}{1+\theta}\right)^{\frac{1}{\theta}}}{(1-\alpha-\beta)\left(\rho+\delta^k\right)} - (\delta^s + n)\frac{(1+\theta)\kappa\beta\left(\kappa - \frac{\rho}{1+\theta}\right)^{\frac{1}{\theta}}}{(1-\alpha-\beta)(\rho+\delta^s)} \\ & - \left(\kappa - \frac{\rho}{1+\theta} + n\right)\left(\kappa - \frac{\rho}{1+\theta}\right)^{\frac{1}{\theta}} \end{aligned} \tag{2.46}$$

It is clear from the above derivations that if we assume that the health depreciation rate is an endogenous function of only health while both the depreciation rates of physical and education capitals are constants, there will be constant steady state levels of k, s, h, and c. In other words, we are not able to find endogenous growth in the Barro (1996b) model when we assume that the health depreciation rate is an increasing function of health. The intuition behind this result is that in the real world, to maintain a better health status, an individual need to invest more time and goods that could have been used for other purposes, like the production of goods. The endogenous health depreciation rate as an increasing function of health accounts for this negative effect of increasing health on economic growth.

We find in this subsection that if we consider this negative effect of health, which affects economic growth through the increasing cost of health accumulation, endogenous growth would disappear. It seems that the endogenous growth obtained from the previous section is sensitive to the definition of the depreciation rate of health. For the technical details of the model presented in this subsection, please refer to Appendix 2.4.

2.3.3 Barro (1996b) model with health depreciation rate determined simultaneously by health and education

From the two Barro (1996b)-type models we constructed in the previous two subsections, we know that endogenous growth may be sensitive to the specification of health depreciation rate. In subsection 2.3.2, we assumed that health depreciation rate is determined only by health itself and is a positive function of health. However, one may argue that education can also affect health depreciation rate. The effects of education on health depreciation rate are mixed. An individual with higher education may have better chance to get a job with higher pay. Nevertheless, such kinds of high-paid jobs, for example, financial analysts, lawyers, and auditors, are usually associated with extremely high pressure and unhealthy working hours. The cost of maintaining a healthy body is also higher. This does not mean that higher education has only negative effect on decreasing health depreciation rate. If an individual is well-educated, he/she may obtain more useful information on how to keep a healthy body in a more efficient and effective way, which can lower the cost of maintaining the same level of health. In this essay, however, we assume that the positive effect of education on decreasing health depreciation rate dominate the negative

effect so that an individual with better education may decrease the cost of maintaining the current level of health.

In this subsection, we consider health depreciation rate as a function of both health and education:

$$\delta^h(h,s) = h^\theta s^{-\theta} \tag{2.47}$$

where $\theta > 0$. Equation (2.47) implies that the higher the health status of an individual, the greater the cost of maintaining this level of health. In contrast, the higher the education level of an individual, the lower the cost of keeping the same level of health.

The corresponding Hamiltonian function for the case in this subsection is:

$$\begin{aligned} H = {} & c^\gamma + v_k[I_k - (\delta^k + n)k] + v_s[I_s - (\delta^s + n)s] \\ & + v_h[I_h - (\delta^h(h,s) + n)h] + \lambda(k^\alpha s^\beta h^{1-\alpha-\beta} - I_k - I_s - I_h - c) \end{aligned} \tag{2.48}$$

The first order conditions with respect to the control and state variables for equation (2.48) are:

$$\frac{\partial H}{\partial c} = 0 \Rightarrow \gamma c^{\gamma-1} = \lambda \tag{2.49}$$

$$\frac{\partial H}{\partial k} = -\dot{v}_k + (\rho - n)v_k = -v_k(\delta^k + n) + \alpha\lambda k^{\alpha-1} s^\beta h^{1-\alpha-\beta} \tag{2.50}$$

$$\frac{\partial H}{\partial s} = -\dot{v}_s + (\rho - n)v_s = -v_s(\delta^s + n) + \beta\lambda k^\alpha s^{\beta-1} h^{1-\alpha-\beta} - v_h\delta_s^h(h,s)h \tag{2.51}$$

$$\frac{\partial H}{\partial h} = -\dot{v}_h + (\rho - n)v_h = -v_h(\delta^h + n) + (1-\alpha-\beta)\lambda k^\alpha s^\beta h^{-\alpha-\beta} - v_h\delta_h^h(h,s)h \tag{2.52}$$

$$\frac{\partial H}{\partial I_k} = \frac{\partial H}{\partial I_s} = \frac{\partial H}{\partial I_h} = 0 \Rightarrow v_k = v_s = v_h = \lambda \tag{2.53}$$

where $\delta_h^h = \dfrac{\partial \delta^h}{\partial h} = \theta h^{\theta-1} s^{-\theta}$ and $\delta_s^h = \dfrac{\partial \delta^h}{\partial s} = -\theta h^\theta s^{-\theta-1}$.

The equations for the growth rate of consumption can be derived from equations (2.49) to (2.53):

$$-(r-1)\frac{\dot{c}}{c} = -(\rho+\delta^k) + \alpha k^{\alpha-1} s^{\beta} h^{1-\alpha-\beta} \tag{2.54}$$

$$-(r-1)\frac{\dot{c}}{c} = -(\rho+\delta^s) + \beta k^{\alpha} s^{\beta-1} h^{1-\alpha-\beta} + \theta h^{1+\theta} s^{-1-\theta} \tag{2.55}$$

$$-(r-1)\frac{\dot{c}}{c} = -(\rho+h^{\theta}s^{-\theta}) + (1-\alpha-\beta)k^{\alpha}s^{\beta}h^{-\alpha-\beta} - \theta h^{\theta}s^{-\theta} \tag{2.56}$$

Assume that $\frac{k}{h} = x$ and $\frac{s}{h} = y$, then equivalent expressions for equations (2.54) to (2.56) are:

$$-(r-1)\frac{\dot{c}}{c} = -(\rho+\delta^k) + \alpha x^{\alpha-1} y^{\beta} \tag{2.57}$$

$$-(r-1)\frac{\dot{c}}{c} = -(\rho+\delta^s) + \beta x^{\alpha} y^{\beta-1} + \theta y^{-1-\theta} \tag{2.58}$$

$$-(r-1)\frac{\dot{c}}{c} = -(\rho+y^{-\theta}) + (1-\alpha-\beta)x^{\alpha}y^{\beta} - \theta y^{-\theta} \tag{2.59}$$

From equations (2.57) to (2.59), we have:

$$\delta^s - \delta^k + \alpha x^{\alpha-1} y^{\beta} - \theta y^{-1-\theta} = \beta x^{\alpha} y^{\beta-1} \tag{2.60}$$

$$\alpha x^{\alpha-1} y^{\beta} + (1+\theta) y^{-\theta} - \delta^k = (1-\alpha-\beta) x^{\alpha} y^{\beta} \tag{2.61}$$

Because it is too complicated to derive the equilibrium values for x and y from the above equations, we check for the case when $\delta^k = \delta^s = 0$. If $\delta^k = \delta^s$, equations (2.60) and (2.61) become:

$$x^{\alpha-1} y^{\beta-1} (\alpha y - \beta x) = \theta y^{-1-\theta} \tag{2.62}$$

$$x^{\alpha-1} y^{\beta-1} [\alpha y - (1-\alpha-\beta)xy] = -(1+\theta) y^{-\theta} \tag{2.63}$$

After some algebra manipulation, we are able to derive equations of y as functions of x from equations (2.62) and (2.63)

$$y = -\frac{\theta}{1+\theta} + \frac{\beta+\theta(1-\alpha)}{(1+\theta)\alpha} x \tag{2.64}$$

$$y^{-(\theta+\beta)} = \frac{(1-\alpha-\beta)}{(1+\theta)} x^{\alpha} - \frac{\alpha}{1+\theta} x^{\alpha-1} \tag{2.65}$$

Equations (2.64) and (2.65) are linear and nonlinear functions of x, respectively. For equation (2.65), we know that $y^{-(\theta+\beta)} \geq 0$, which indicates

that $x \geq \frac{\alpha}{1-\alpha-\beta}$.

By total differentiation of equation (2.65), we have:

$$\frac{dy}{dx} = \frac{\alpha}{(1+\theta)} x^{\alpha-2}\left[\frac{(1-\alpha-\beta)x-(\alpha-1)}{-(\theta+\beta)y^{-(\theta+\beta+1)}}\right] = 0 \Leftrightarrow x = -\frac{1-\alpha}{1-\alpha-\beta} \in (-\infty,-1) \quad (2.66)$$

Because $x \geq \frac{\alpha}{1-\alpha-\beta}$, we know that:

$$\frac{dy}{dx} < 0 \quad (2.67)$$

Furthermore, we can obtain the limit of y from the limit of $y^{-(\theta+\beta)}$:

$$\lim_{x\to+\infty} y^{-(\theta+\beta)} = \lim_{x\to+\infty}\left[\frac{(1-\alpha-\beta)}{(1+\theta)}x^{\alpha} - \frac{\alpha}{1+\theta}x^{\alpha-1}\right] = +\infty \Leftrightarrow \lim_{x\to+\infty} y = 0 \quad (2.68)$$

$$\begin{aligned}&\lim_{x\to\frac{\alpha}{1-\alpha-\beta}} y^{-(\theta+\beta)} = \lim_{x\to\frac{\alpha}{1-\alpha-\beta}}\left[\frac{(1-\alpha-\beta)}{(1+\theta)}x^{\alpha} - \frac{\alpha}{1+\theta}x^{\alpha-1}\right] = 0 \\ &\Leftrightarrow \lim_{x\to\frac{\alpha}{1-\alpha-\beta}} y = +\infty\end{aligned} \quad (2.69)$$

From equations (2.67) to (2.69), we know that equation (2.65) is a decreasing function of x while we already know that equation (2.64) is a linear function of x with positive slope. We draw the graph of equations (2.64) and (2.65) in Figure 2.1, from which we know that there should be a pair of positive roots x^* and y^*.

We defined in this subsection that x is the ratio of capital to health and y is the ratio of education to health. The existence of steady state values of x and y implies that the growth rates of physical, education and health capitals are constants in the steady state. In other words, there is endogenous growth in the Barro (1996b)-type model proposed in this subsection.

We have also analyze the case when $\delta^s \neq \delta^k$ and there are also steady state values of x and y existing in this framework. The results of this subsection indicate that if we consider the effects of both health and

education on the health depreciation rate, the negative effect of education on lowering the health depreciation rate dominates the positive effect created by health, which results in the existence of endogenous growth. More technical details can be found in Appendix 2.5.

From subsections 2.3.1 to 2.3.3, we know that the endogenous growth derived from the Barro (1996b)-type models is sensitive to different specifications of the endogenous health depreciation rate. If we follow Barro (1996b) to define health depreciation rate as a decreasing function of health in the short-run but constant in the long-run, we are able to find endogenous growth as long as there are constant returns to scale with respect to physical, education and health capitals together. However, as we know, the definition of health depreciation rate is the cost of maintaining the current level of health. From the examples shown in the introduction, this health depreciation rate should be a positive function of health itself. Thus, we keep the constant returns to scale assumption of section 2.3.1 but assume that the health depreciation rate is a positive function of only health in section 2.3.2. The maximization results show that there is no endogenous growth in this case. Nevertheless, if we assume the health depreciation rate as a function of health and education, where health depreciation rate is an increasing function and a decreasing function of health and education respectively, we are able to find endogenous growth in this model. It is obvious that the specifications of health depreciation rate determine the existence of endogenous growth in the Barro (1996b)-type models even if we assume constant returns to scale of output with respect to the three kinds of capitals together.

2.4 Barro (1996b) Model with Health Entering Utility Function

To obtain further insights on how the endogenous health depreciation rate affect the long-run economic growth, we analyze a more complex problem: the case where health enters the utility function. The influential Grossman (1972) paper argues that health could generate output in the form of "healthy time" and using health service can be viewed as another form of consumption. Built on the idea, Zon and Muysken (2001, 2003) construct a model considering the utility function with both consumption and health. They find that the preference for health may lead to a slowdown in growth because people may choose for investment in health instead of producing goods as health is now another way to obtain utility. Although the Barro (1996b) paper considers the impact of health on economic growth by incorporating health into the production function, the impact of health on economic growth through utility is not considered in that model. Therefore, based on the Barro (1996b) framework, we introduce health into both the utility function and production function and study whether health entering the utility function will affect the results of long-run steady state growth derived in the previous section. In the previous section, we find that when the condition of constant returns to scale with respect to the three kinds of capitals is satisfied, the Barro (1996b) model will have endogenous growth. However, by further investigation, we also find that the endogenous growth derived from the Barro (1996b) model in subsection 2.3.1 is sensitive to different specifications of health depreciation rate.

In this section, we intend to study the situation when we also consider health affect economic growth through entering the utility function directly, whether the conclusion we drew in section 2.3 that endogenous growth in

Barro (1996b)-type models are sensitive to the specification of health depreciation rate is still correct. Following the procedures of section 2.3, all the models analyzed in this section assume constant returns to scale with respect to the three kinds of capitals together. We first analyze the Barro (1996b) model with health entering the utility function. Second, based on the first case, we replace the health depreciation rate of Barro (1996b) by the one determined exclusively by health itself. Last, we consider the case when the health depreciation rate is a function of both health and education. By comparing the results from different specifications of health depreciation rate derived in this section with those from section 2.3, we are able to find that whether health entering into the utility function would affect the effect of endogenous health depreciation rate on long-run economic growth.

Different from Zon and Muysken (2001, 2003), which consider the effect of health on economic growth through the impact of health on knowledge accumulation in the tradition of Lucas (1988), we follow the Barro (1996b) framework and consider three channels through which health affects economic growth. First, the health status of the population in the aggregate level may determine the total 'healthy time' available and if people feel healthier, they may work more productively. This channel of health effect on economic growth in the aggregate level is reflected by the incorporation of health explicitly into the production function. Second, as a special form of human capital, the accumulation of health competes with the accumulation of education and the production of goods for the 'healthy time'. This property was also recognized by Bloom and Canning (2000). In this essay, this channel of health affecting economic growth is through the health depreciation rate. Last, following Grossman (1972) and Zon and

Muysken (2001, 2003), we consider the generation of utility by health. As stated in Grossman (1972), health can be viewed as a special form of durable goods which could produce the commodity 'healthy time'. People gain utility from the consumption of 'healthy time'. As a result, health may also affect economic growth through entering the utility function directly. The first channel seems having positive effect on economic growth while the last two channels may have negative impact.

2.4.1 Barro (1996b) model with constant health depreciation rate and health entering the utility function

Throughout this section, we assume as in section 2.3 that there are constant returns to scale with respect to the three kinds of capitals. For the utility function, we choose the same form of utility function as the one used in section 2.3, which is $u(c,h)=c^{\gamma}h^{1-\gamma}$. The three components i_k, i_s and i_h represent the gross investment flows in the three capitals in per capita terms.

Considering the direct utility effect from health, the household maximization problem is modified as follow:

$$\max_{c,h} \int_0^{+\infty} u(c,h)e^{-(\rho+n)t}dt \tag{2.70}$$

$$s.t.: \dot{k} = i_k - (\delta^k + n)k \tag{2.71}$$

$$\dot{s} = i_s - (\delta^s + n)s \tag{2.72}$$

$$\dot{h} = i_h - (\delta^h + n)h \tag{2.73}$$

$$k^{\alpha}s^{\beta}h^{1-\alpha-\beta} = i_k + i_h + i_s + c \tag{2.74}$$

where we assume the same as in Barro (1996b) that the depreciation rate of physical capital δ^k is constant and the depreciation rates of education equals that of health, which is a decreasing function of health. In the steady state, the health depreciation rate will converge to a constant value δ^h. In other words, we analyze the steady state where $\delta^s = \delta^h = \text{constant}$.

The constraint Hamiltonian function for this problem is represented as the following equation:

$$\begin{aligned} H = {} & c^{\gamma} h^{1-\gamma} + v_k[I_k - (\delta^k + n)k] + v_s[I_s - (\delta^s + n)s] \\ & + v_h[I_h - (\delta^h + n)h] + \lambda(k^{\alpha} s^{\beta} h^{1-\alpha-\beta} - I_k - I_s - I_h - c) \end{aligned} \tag{2.75}$$

The first order conditions with respect to the control variables and state variables are:

$$\frac{\partial H}{\partial c} = 0 \Rightarrow \gamma \left(\frac{c}{h}\right)^{\gamma-1} = \lambda \tag{2.76}$$

$$\frac{\partial H}{\partial k} = -\dot{v}_k + (\rho - n)v_k = -v_k(\delta^k + n) + \alpha\lambda k^{\alpha-1} s^{\beta} h^{1-\alpha-\beta} \tag{2.77}$$

$$\frac{\partial H}{\partial s} = -\dot{v}_s + (\rho - n)v_s = -v_s(\delta^s + n) + \beta\lambda k^{\alpha} s^{\beta-1} h^{1-\alpha-\beta} \tag{2.78}$$

$$\begin{aligned} \frac{\partial H}{\partial h} &= -\dot{v}_h + (\rho - n)v_h \\ &= \frac{(1-\gamma)}{\gamma}\left(\frac{c}{h}\right)\lambda - v_h(\delta^h + n) + (1-\alpha-\beta)\lambda k^{\alpha} s^{\beta} h^{-\alpha-\beta} \end{aligned} \tag{2.79}$$

$$\frac{\partial H}{\partial I_k} = \frac{\partial H}{\partial I_s} = \frac{\partial H}{\partial I_h} = 0 \Rightarrow v_k = v_s = v_h = \lambda \tag{2.80}$$

We assume that $x = \frac{k}{h}$, $y = \frac{s}{h}$ and $z = \frac{c}{h}$. Together with the condition implied by equation (2.80), equations (2.78) to (2.80) then become:

$$-\dot{\lambda} + (\rho - n)\lambda = -\lambda(\delta^k + n) + \alpha\lambda x^{\alpha-1} y^{\beta} \tag{2.81}$$

$$-\dot{\lambda} + (\rho - n)\lambda = -\lambda(\delta^s + n) + \beta\lambda x^{\alpha} y^{\beta-1} \tag{2.82}$$

$$-\dot{\lambda}+(\rho-n)\lambda=\frac{(1-r)}{r}z\lambda-\lambda(\delta^h+n)+(1-\alpha-\beta)\lambda x^{\alpha}y^{\beta} \tag{2.83}$$

From equation (2.76) and $z=\frac{c}{h}$, we are able to derive the following two equations:

$$\gamma z^{\gamma-1}=\lambda \tag{2.84}$$

$$(\gamma-1)\frac{\dot{z}}{z}=\frac{\dot{\lambda}}{\lambda} \tag{2.85}$$

From equations (2.82) to (2.85), we have:

$$\alpha x^{\alpha-1}y^{\beta}-(\rho+\delta^k)=\beta x^{\alpha}y^{\beta-1}-(\rho+\delta^s) \tag{2.86}$$

$$\beta x^{\alpha}y^{\beta-1}-(\rho+\delta^s)=\frac{1-\gamma}{\gamma}z+(1-\alpha-\beta)x^{\alpha}y^{\beta}-(\rho+\delta^h) \tag{2.87}$$

On the balance growth path, we have:

$$0=(1-\gamma)\frac{\dot{z}}{z}=\alpha x^{\alpha-1}y^{\beta}-(\rho+\delta^k) \tag{2.88}$$

$$=\beta x^{\alpha}y^{\beta-1}-(\rho+\delta^s) \tag{2.89}$$

$$=\frac{1}{\gamma}z+(1-\alpha-\beta)x^{\alpha}y^{\beta}-(\rho+\delta^h) \tag{2.90}$$

Dividing equation (2.88) by equation (2.89) and inserting the resulting equation back into equation (2.88), we have the steady state level of x:

$$x^*=\left[\frac{(\rho+\delta^k)^{1-\beta}(\rho+\delta^s)^{\beta}}{\alpha^{1-\beta}\beta^{\beta}}\right]^{\frac{1}{\alpha+\beta-1}} \tag{2.91}$$

By putting the steady state value of x into equation (2.88), we get the steady state value of y:

$$y^*=\left[\frac{(\rho+\delta^k)^{\alpha}}{\alpha^{\alpha}\beta^{1-\alpha}(\rho+\delta^s)^{\alpha-1}}\right]^{\frac{1}{\alpha+\beta-1}} \tag{2.92}$$

Replacing x and y in equation (2.87) by x^* and y^* we obtained from equations (2.91) and (2.92), we have the steady state value of z:

$$z^* = \frac{\gamma}{1-\gamma}\left\{(\rho+\delta^h)-(1-\alpha-\beta)\left[\frac{(\rho+\delta^k)^\alpha(\rho+\delta^s)^\beta}{\alpha^\alpha\beta^\beta}\right]^{\frac{1}{\alpha+\beta-1}}\right\} \tag{2.93}$$

Equations (2.91) to (2.93) implies that the ratio of physical capital to health capital, education capital to health capital and consumption to health capital are all constants in the steady state, which indicates that physical capital, education, health, and consumption grow at constant rate at the steady state. We assume the constant growth rate as $\frac{\dot{k}}{k}=\frac{\dot{s}}{s}=\frac{\dot{h}}{h}=\frac{\dot{c}}{c}=g$. Moreover, from the resource constraint condition[13], we have:

$$x^\alpha y^\beta-(\delta^k+n)x-(\delta^s+n)y-(\delta^h+n)-z=(x+y+1)g$$

$$\Leftrightarrow g=\frac{x^{*\alpha}y^{*\beta}-(\delta^k+n)x^*-(\delta^s+n)y^*-(\delta^h+n)-z^*}{x^*+y^*+1} \tag{2.94}$$

Equations (2.91) to (2.94) indicate that there is endogenous growth in the Barro (1996b) model as long as we assume constant returns to scale with respect to the three kinds of capitals even when we assume health entering the utility function. The technical details are available in Appendix 2.6.

2.4.2 Barro (1996b) model with health depreciation rate determined by health and health entering the utility function

Just like subsection 2.3.2, we follow the examples on the relationship between health depreciation rate and the health status presented in the introduction and consider the definition of the health depreciation rate as the cost of maintaining the current level of health. We thus first consider the health depreciation rate as a function of health itself:

$$\delta^h=h^\theta \tag{2.95}$$

[13] The resource constraint equation is:

$$k^\alpha s^\beta h^{1-\alpha-\beta}-\dot{k}-(\delta^k+n)k-\dot{s}-(\delta^s+n)s-\dot{h}-(\delta^h+n)h-c=0$$

where $\theta > 0$, which means that the marginal effect of health can be either a decreasing, constant, or increasing function of health.

Different from the model we constructed in subsection 2.3.2, we assume that both health and consumption enter the utility function. For this case, we assume that health enter the utility function in the form of a multiplier of consumption c. The constraint Hamiltonian function for this case is represented as follows:

$$\begin{aligned} H &= c^{\gamma}h^{1-\gamma} + v_k[I_k - (\delta^k + n)k] + v_s[I_s - (\delta^s + n)s] \\ &\quad + v_h[I_h - (\delta^h(h) + n)h] + \lambda(k^{\alpha}s^{\beta}h^{1-\alpha-\beta} - I_k - I_s - I_h - c) \end{aligned} \tag{2.96}$$

The corresponding first order conditions for the Hamiltonian function presented above are:

$$\frac{\partial H}{\partial c} = 0 \Rightarrow \gamma\left(\frac{c}{h}\right)^{\gamma-1} = \lambda \tag{2.97}$$

$$\frac{\partial H}{\partial k} = -\dot{v}_k + (\rho - n)v_k = -v_k(\delta^k + n) + \alpha\lambda k^{\alpha-1}s^{\beta}h^{1-\alpha-\beta} \tag{2.98}$$

$$\frac{\partial H}{\partial s} = -\dot{v}_s + (\rho - n)v_s = -v_s(\delta^s + n) + \beta\lambda k^{\alpha}s^{\beta-1}h^{1-\alpha-\beta} \tag{2.99}$$

$$\begin{aligned} \frac{\partial H}{\partial h} &= -\dot{v}_h + (\rho - n)v_h \\ &= \frac{(1-\gamma)}{\gamma}\left(\frac{c}{h}\right)\lambda - v_h(\delta^h(h) + n) + (1-\alpha-\beta)\lambda k^{\alpha}s^{\beta}h^{-\alpha-\beta} - v_h\delta_h^h h \end{aligned} \tag{2.100}$$

$$\frac{\partial H}{\partial I_k} = \frac{\partial H}{\partial I_s} = \frac{\partial H}{\partial I_h} = 0 \Rightarrow v_k = v_s = v_h = \lambda \tag{2.101}$$

From equation (2.97), we assume a dynamic equation of λ as a function of c and h:

$$(\gamma - 1)\left(\frac{\dot{c}}{c} - \frac{\dot{h}}{h}\right) = \frac{\dot{\lambda}}{\lambda} \tag{2.102}$$

On the balanced growth path, we have $\frac{\dot{k}}{k} = \frac{\dot{s}}{s} = \frac{\dot{h}}{h} = \frac{\dot{c}}{c} = 0$, which

indicates that $\frac{\dot{\lambda}}{\lambda} = 0$.

From equations (2.98) to (2.101), we are able to derive equations of $\frac{\dot{\lambda}}{\lambda}$:

$$\frac{-\dot{\lambda}}{\lambda} = -(\delta_k + \rho) + \alpha k^{\alpha-1} s^{\beta} h^{1-\alpha-\beta} = 0 \tag{2.103}$$

$$\frac{-\dot{\lambda}}{\lambda} = -(\delta_s + \rho) + \beta k^{\alpha} s^{\beta-1} h^{1-\alpha-\beta} = 0 \tag{2.104}$$

$$\frac{-\dot{\lambda}}{\lambda} = (1+\theta)h^{1+\theta} + \rho h - \frac{(1-\gamma)}{\gamma} c = (1-\alpha-\beta)k^{\alpha} s^{\beta} h^{1-\alpha-\beta} \tag{2.105}$$

To simplify our calculation, we assume here the depreciation rates of physical capital and education capital are equal,[14] i.e., $\delta^k = \delta^s = \delta$. From equations (2.103) and (2.104), we have:

$$s = \frac{\beta}{\alpha} k \tag{2.106}$$

Replacing s in equation (2.103) by equation (2.106), we then have equations of k and s as a function of h respectively:

$$k = \left(\frac{\delta + \rho}{\alpha^{1-\beta} \beta^{\beta}} \right)^{\frac{1}{\alpha+\beta-1}} h = \omega_1 h \tag{2.107}$$

$$s = \frac{\beta}{\alpha} \omega_1 h \tag{2.108}$$

From the constraint equation, we can have an expression for c:

$$c = k^{\alpha} s^{\beta} h^{1-\alpha-\beta} - (\delta + n)k - (\delta + n)s - (h^{\theta} + n)h \tag{2.109}$$

Replacing consumption c in equation (2.105) by equation (2.109) and substituting k and s by equations (2.108) and (2.109), we then have:

[14] We have also calculated the results generated from where $\delta^k \neq \delta^s$ and the results are the same as the one we present in this subsection, although they are much more complicated.

$$(1+\theta)h^{\theta}+\rho-\frac{(1-\gamma)}{\gamma}\left\{\left[\omega_1^{\alpha+\beta}\left(\frac{\beta}{\alpha}\right)^{\beta}-(\delta+n)(1+\frac{\beta}{\alpha})\omega_1\right]-(h^{\theta}+n)\right\} \quad (2.110)$$
$$=(1-\alpha-\beta)\left(\frac{\beta}{\alpha}\right)^{\beta}\omega_1^{\alpha+\beta}$$

The equilibrium value of health h can be derived from equation (2.110):

$$h^{*}=\left\{\frac{(1-\alpha-\beta)\left(\frac{\beta}{\alpha}\right)^{\beta}\omega_1^{\alpha+\beta}+\frac{(1-r)}{r}\left[\omega_1^{\alpha+\beta}\left(\frac{\beta}{\alpha}\right)^{\beta}-(\delta+n)(1+\frac{\beta}{\alpha})\omega_1-n\right]-\rho}{(1+\theta)+\frac{(1-\gamma)}{\gamma}}\right\}^{\frac{1}{\theta}}$$

The corresponding steady state values of k, s and c, are:

$$k^{*}=\omega_1 h^{*}$$

$$s=\frac{\beta}{\alpha}k^{*}$$

$$c=k^{*\alpha}s^{*\beta}h^{*1-\alpha-\beta}-(\delta+n)k^{*}-(\delta+n)s^{*}-(h^{\theta}+n)h^{*}$$

Consistent with the results we obtained in subsection 2.3.2, there is no endogenous growth if we assume that health depreciation rate is a function of only health and this function is a non-decreasing function. Technical details of this subsection are available in Appendix 2.7.

2.4.3 Barro (1996b) model with health depreciation rate determined simultaneously by both health and education and health entering the utility function

In this subsection, we consider the situation where health depreciation rate is determined simultaneously by health and education. We follow section 2.3.3 to assume that both the depreciation rates of physical capital and education capital are constant while the health depreciation rate is

assumed to be an increasing function of health and a decreasing function of education. However, we consider health entering the utility function in this subsection. The exact form of the health depreciation rate for this subsection is:

$$\delta^h(h,s) = h^{\theta} s^{-\theta} \tag{2.111}$$

where $\theta > 0$.

In this case, the utility function is the product of a consumption exponential function and a health exponential function. The constraint utility function for this case is:

$$\begin{aligned} H = {} & c^{\gamma} h^{1-\gamma} + v_k[I_k - (\delta^k + n)k] + v_s[I_s - (\delta^s + n)s] \\ & + v_h[I_h - (\delta^h(h,s) + n)h] + \lambda(k^{\alpha} s^{\beta} h^{1-\alpha-\beta} - I_k - I_s - I_h - c) \end{aligned} \tag{2.112}$$

The first order conditions for the control variables and state variables in equation (2.112) are:

$$\frac{\partial H}{\partial c} = 0 \Rightarrow \gamma \left(\frac{c}{h}\right)^{\gamma-1} = \lambda \tag{2.113}$$

$$\frac{\partial H}{\partial k} = -\dot{v}_k + (\rho - n)v_k = -v_k(\delta^k + n) + \alpha\lambda k^{\alpha-1} s^{\beta} h^{1-\alpha-\beta} \tag{2.114}$$

$$\frac{\partial H}{\partial s} = -\dot{v}_s + (\rho - n)v_s = -v_s(\delta^s + n) + \beta\lambda k^{\alpha} s^{\beta-1} h^{1-\alpha-\beta} - v_h \delta_s^h h \tag{2.115}$$

$$\begin{aligned} \frac{\partial H}{\partial h} &= -\dot{v}_h + (\rho - n)v_h \\ &= \frac{(1-\gamma)}{\gamma}\left(\frac{c}{h}\right)\lambda - v_h(\delta^h + n) + (1-\alpha-\beta)\lambda k^{\alpha} s^{\beta} h^{-\alpha-\beta} - v_h \delta_h^h h \end{aligned} \tag{2.116}$$

$$\frac{\partial H}{\partial I_k} = \frac{\partial H}{\partial I_s} = \frac{\partial H}{\partial I_h} = 0 \Rightarrow v_k = v_s = v_h = \lambda \tag{2.117}$$

We assume the ratios of physical capital, education and consumption to health respectively as $x = \frac{k}{h}$, $y = \frac{s}{h}$ and $z = \frac{c}{h}$. In the steady state, we assume that these ratios are constants.

From equations (2.114) to (2.117), we have equations of $\frac{\dot{\lambda}}{\lambda}$:

$$-\frac{\dot{\lambda}}{\lambda} = -(\rho + \delta^k) + \alpha x^{\alpha-1} y^{\beta} \tag{2.118}$$

$$-\frac{\dot{\lambda}}{\lambda} = -(\rho + \delta^s) + \beta x^{\alpha} y^{\beta-1} + \theta y^{-\theta-1} \tag{2.119}$$

$$-\frac{\dot{\lambda}}{\lambda} = -(\rho + y^{-\theta}) + (1-\alpha-\beta) x^{\alpha} y^{\beta} + \frac{(1-\gamma)}{\gamma} z - \theta y^{-\theta} \tag{2.120}$$

On the balanced growth path, from equation (2.113), we have:

$$\gamma \left(\frac{c}{h}\right)^{\gamma-1} = \lambda \Rightarrow (\gamma-1)\frac{\dot{z}}{z} = \frac{\dot{\lambda}}{\lambda} = 0 \tag{2.121}$$

From equations (2.118) to (2.121), we have the following three equations:

$$-(\rho + \delta^k) + \alpha x^{\alpha-1} y^{\beta} = 0 \tag{2.122}$$

$$-(\rho + \delta^s) + \beta x^{\alpha} y^{\beta-1} + \theta y^{-\theta-1} = 0 \tag{2.123}$$

$$-(\rho + y^{-\theta}) + (1-\alpha-\beta) x^{\alpha} y^{\beta} + \frac{(1-\gamma)}{\gamma} z - \theta y^{-\theta} = 0 \tag{2.124}$$

Using equations (2.122) to (2.124), we are able to get another equation of y and z:

$$\begin{aligned} \beta\frac{(1-\gamma)}{\gamma}(z) = {} & \beta\rho + \beta(1+\theta)\left(\frac{1}{y}\right)^{\theta} \\ & -(1-\alpha-\beta)\beta\left\{\frac{\alpha y\left[(\rho+\delta^s) - \theta y^{-\theta-1}\right]}{\beta\left(\rho+\delta^k\right)}\right\}^{\alpha} \end{aligned} \tag{2.125}$$

Dividing equation (2.123) by equation (2.124) and rearranging the terms, we have an equation of y and z:

$$\beta\frac{(1-\gamma)}{\gamma} z = \beta\rho + \beta(1+\theta)\left(\frac{1}{y}\right)^{\theta} - (1-\alpha-\beta)\left[\left(\rho+\delta^s\right)y - \theta\left(\frac{1}{y}\right)^{\theta}\right] \tag{2.126}$$

From equations (2.125) and (2.126), we have an equation of y only, from which the equilibrium value y^* can be generated:

$$\left(\rho+\delta^{x}\right)y^{1+\theta}-\beta\left(\frac{\alpha}{\rho+\delta^{k}}\right)^{\frac{\alpha}{1-\alpha}}y^{\theta}-\theta=0 \qquad (2.127)$$

Using method similar to Appendix 2.2, we are able to find at least one positive root for equation (2.127), which indicates that there is equilibrium value of y. The equilibrium value of x and z can be derived from equations (2.122) and (2.125) respectively.

From the above calculations, we are able to find that there is endogenous growth generated by the model we constructed in this subsection. Technical details can be found in Appendix H.

Theorem 2.2: When health enters both the production function and utility function of the Barro (1996b) framework, endogenous growth still exists and it is sensitive to the definition of the health depreciation rate. Whether health enters the utility function or not does not change the effect of health depreciation rate on economic growth in the long-run.

In summary, as long as there are constant returns to scale for the three capital inputs together, there would be endogenous growth in the Barro (1996b) model and the inclusion of health into the utility function does not affect the existence of endogenous growth. However, by comparing the results for the case when health capital only enter the production function (section 2.3) and the case when health capital enters both the production function and the utility function (section 2.4), we find that the results derived from these two sections are very consistent. The resulting endogenous growth is sensitive to the specifications of the health depreciation rate. If we consider health depreciation rate as an increasing

function of health capital, which reflects the higher cost to maintain the better health status, the resources occupied by the allocation to produce 'good health' would affect the production of goods to a level that there would be no endogenous growth in the long-run. However, if we also consider more efficient investment in health as a result of people being equipped with better knowledge, which is reflected by the inclusion of both health and education in the function of health depreciation rate, the negative effect of education on health depreciation rate would dominate the positive effect from health. Endogenous growth would exist in this case.

As we stated in the previous sections, there are several channels through which health may affect economic growth. From the results of this section, it seems that when health is viewed as a kind of utility, the positive channels, for example, the increase in health status can generate more "healthy time" an agent is able to provide, an increase in health leads to an agent to work more productively, may dominate the negative channels, for example, a preference for health care measures when the income of an agent increase, which results in less time spent on production.

The results derived in this section is an improvement to what was found in Zon and Muysken (2001, 2003) where, based on the Lucas (1988) framework, they also consider the situation that health enters the utility function. In Zon and Muysken (2001, 2003), they are not able to derive endogenous growth for health as they argue that when health enter the utility function, the negative effect from the preference for health may dominate the positive effects which affect the economic growth through the production function. However, based on the Barro (1996b) framework, we arrive at the conclusion that there is endogenous growth even when health

enters the utility function, although this endogenous growth is subjected to the exact form of health depreciation rate. Zon and Muysken (2001, 2003) assumed that increase in longevity leads to higher demand for health care services and the provision of health care services crowds out the scare resources that are supposed to be used in capital good production. In our model, however, we find that what determines the long-run effect of health on economic growth is not whether health enters the utility function but the specification of the endogenous health depreciation rate. If the health depreciation rate is determined only by health itself, the cost of maintaining the current level of health will increase as people's preference for health increases when they become healthier and have longer lives. However, if the health depreciation rate is determined not only by health but also by education, we find that there is endogenous growth in the model, which implies that the effect of education on decreasing the health depreciation rate may dominate the effect generated by health.

2.5 Concluding Remarks

For the analysis of the relationship between health and economic growth, there are two main streams of researches. One stream is on how health affects economic growth and the other stream is on the relationship between health and inequality. In this essay, we focus on the first line of research and analyze the fundamental macroeconomic relationship between health and long-run economic growth with an emphasis on the effect of health depreciation rate. The model we construct here is based on the Barro (1996b) framework, which is the first theoretical framework that describes the interplay between physical capital and human capital in the form of both

education and health. Although the Barro (1996b) model successfully introduced health into the production function, this model has the same model deficiency as those of the neoclassical models. In this model, economic growth is only determined by the exogenous technological factor. Thus, this model is still not able to explain the determinants of long run growth.

The first contribution of this essay is to solve the model deficiency in Barro (1996b) and find the condition under which there is endogenous growth. From the derivation of the original Barro (1996b) model, we find that the condition of diminishing returns with respect to the three capital inputs, $\alpha + \beta + \chi < 1$, may be an important factor behind the result of no endogenous growth of the original model. Thus, we adjust this condition to allow for constant returns to scale with respect to these three capital inputs and check whether endogenous growth may be determined by whether the condition of constant returns to scale for the three capital inputs together is satisfied.

The second contribution of this essay, which is also the main contribution of this essay, is that we endogenize the health depreciation rate. In most of the health literature, focus of the researchers is on the positive effect of health on economic growth by increasing, for example, the labor productivity, improving the efficiency of learning, and achieving longer life expectancy. However, health also has negative effect on economic growth. For example, investment in health requires goods and 'healthy time' that could have been used for goods production or knowledge accumulation. From the influential Grossman (1972) paper, we know that health depreciation rate should not be constant. The examples shown in the

introduction also indicate that health depreciation rate should be an increasing function of health. By definition, the health depreciation rate is the cost of maintaining the current level of health. The health depreciation rate as an increasing function of health reflects the negative effect of health on economic growth by demanding more resources to maintain a higher level of health. Zon and Muysken (2001, 2003) also guess that health depreciation rate, as an increasing function of health, may lead to no endogenous growth in the long-run. Therefore, based on the results generated by the extended Barro (1996b) model, we introduce endogenous health depreciation rate and analyze whether different specifications of health depreciation rate may alter the result of endogenous growth derived in the extended Barro (1996b) model.

The last contribution of our model is that we further extend the Barro (1996b) model by introducing health into the utility function following the idea proposed by Grossman (1972). In Grossman (1972), the author argues that health could generate a special kind of goods called "healthy time" and people can benefit from the consumption of health services which leads to health being viewed as another form of consumption. Thus, we extend the Barro (1996b) model from another direction by introducing health into both the utility function and production function, just as what Zon and Mushken (2001, 2003) did, which is another channel through which health affects economic growth. Like the procedures of analyzing the extended Barro (1996b) model without health entering the utility function, we also examine whether the result of endogenous growth is sensitive to the different specifications of health depreciation rate. By comparing the results with and without health entering the utility function, we are able to determine

whether health entering the utility function or not would affect long-run economic growth.

The first extension of the Barro (1996b) framework indicates that when the condition of constant returns to scale with respect to the three capital inputs (physical capital, education and health) together is satisfied, output and consumption would grow at the same constant rate in the steady state. In other words, there is endogenous growth in the original Barro (1996b) model as long as the constant returns to scale condition is satisfied. However, from our sensitivity analysis with respect to the health depreciation rate, we find that the endogenous growth in the extended Barro (1996b) model depends on the exact form of health depreciation rate. If the health depreciation rate is an increasing function of h, there is no endogenous growth in the Barro (1996b) model. In contrast, if we consider that education can also affect the health depreciation rate and thus the health depreciation rate is a function of both health and education, we are able to derive endogenous growth in the Barro (1996b) model. These results indicate that when we only consider the increasing cost of better health status, the negative effect of health would lead to no endogenous growth in the long-run. However, if we also consider the fact that people would invest more efficiently on health if they manage more knowledge, which is reflected by the inclusion of education in the function of health depreciation rate, there would be endogenous growth in the long-run growth. It is obvious that endogenous health depreciation rate has a significant effect on long-run economic growth.

The idea for the second extension is from the results derived from Zon and Muysken (2001, 2003), where they extended the Lucas (1988) model by

introducing health into both the production function and utility function. One important result from Zon and Muysken (2001, 2003) is that the preference for health may undermine economic growth. In this essay, we follow the idea of Grossman (1972) and Zon and Muysken (2001, 2003) and extend the Barro (1996b) model by considering health in both the production function and the utility function. The results from this line of extension are consistent with what we have derived in section 2.3. We are able to find endogenous growth in the steady state if there is constant health depreciation rate in the long-run or when the health depreciation rate is determined simultaneously by health and education while there is no endogenous growth if the health depreciation rate is determined only by a non-decreasing function of health.

The findings from the extensions to the Barro (1996b) framework in sections 2.3 and 2.4 of this essay show that health is another form of human capital that affects long run economic growth. In the Barro (1996b) framework, as long as there are constant returns with respect to the three capital inputs as a whole, there would be endogenous growth in the long-run economic growth. Health capital plays the same role in generating endogenous growth as education when only education is considered as human capital. However, the existence of the endogenous growth in the Barro (1996b) model is sensitive the specification of the endogenous health depreciation rate. When the health depreciation rate is an increasing function of health, there would be no endogenous growth. Nevertheless, the inclusion of education simultaneously with health into the function of health depreciation rate would dominate the effect of health on the health depreciation rate and endogenous growth exists again. The comparison of

the results from sections 2.3 and 2.4 show that whether health enters the utility function or not does not play a central role to the generation of endogenous growth because the results from sections 2.3 and 2.4 are very consistent. In contrast, the specification of the endogenous function of health depreciation rate does affect the existence of endogenous growth. The results derived from this essay indicate that there are several channels through which health affects long-run economic growth. However, the negative effect of health on economic growth, which is reflected by the health depreciation rate as an increasing function of health capital, may be so significant that leads to no endogenous growth in the long-run. Nevertheless, the role of education on increasing the efficiency of investment on health, which is reflected by the inclusion of education simultaneous with health as the determinants of the endogenous health depreciation rate, is so important that it counteracts the negative effect of health on long-run growth.

The importance of the endogenous health depreciation rate and the insignificant effect of health affecting an individual's utility on economic growth indicate that the preference for health is not important in determining long-run economic growth, which is contradict to what emphasized in Grossman (1972) and Zon and Muysken (2001, 2003). In contrast, this study proves that it is the exact form of the health accumulation function (or the health production function), which contains an endogenous health depreciation rate that plays a determinant role in long-run economic growth.

In this paper, the limitation to our models is that we only prove the existence of endogenous growth in the Barro (1996b) model and the

important role of the exact specification of the endogenous health depreciation on the existence of endogenous growth. However, it is also interesting to study the transitional dynamics and check the stability conditions. In the future studies, we may apply the results obtained in this essay to analyze the transitional dynamics of these models.

Appendix 2.1: The constraint Hamiltonian function

Suppose we want to maximize $\int_0^T F(x,u)e^{-\beta t}dt$, $s.t.: \dot{x} = f(x,u)$, where x is a state variable and u is a control variable, then the Hamiltonian function for this maximization problem is:

$$H = F(x,u)e^{-\beta t} + \lambda f(x,u) \tag{2.1.1}$$

The first order conditions with respect to the control variables and state variables are:

$$\frac{\partial H}{\partial u} = 0 \Rightarrow F_u e^{-\beta t} + \lambda f_u = 0 \tag{2.1.2}$$

$$\frac{\partial H}{\partial x} = -\dot{\lambda} \Rightarrow F_x e^{-\beta t} + \lambda f_x = -\dot{\lambda} \tag{2.1.3}$$

$$\frac{\partial H}{\partial \lambda} = \dot{x} \Rightarrow \dot{x} = f(x,u) \tag{2.1.4}$$

The transversality condition is $\lim_{t\to\infty} x\lambda = 0$.

Next, we assume $\lambda = \zeta e^{-\beta t}$ and $\dot{\lambda} = \dot{\zeta} e^{-\beta t} - \beta\zeta e^{-\beta t}$. We then insert λ and $\dot{\lambda}$ into equations (2.1.2) and (2.1.3):

$$F_u e^{-\beta t} + \zeta e^{-\beta t} f_u = 0 \Rightarrow F_u + \zeta f_u = 0 \tag{2.1.5}$$

$$\begin{aligned} &F_x e^{-\beta t} + \zeta e^{-\beta t} f_x = -\left(\dot{\zeta} e^{-\beta t} - \beta\zeta e^{-\beta t}\right) \\ &\Rightarrow F_x + \zeta f_x = -\dot{\zeta} + \beta\zeta \end{aligned} \tag{2.1.6}$$

Substituting $\lambda = \zeta e^{-\beta t}$ into equation (2.1.1), the Hamiltonian function changes to:

$$\begin{aligned} H &= F(x,u)e^{-\beta t} + \zeta e^{-\beta t} f(x,u) \\ &= \left[F(x,u) + \zeta f(x,u)\right]e^{-\beta t} \\ &= \bar{H}e^{-\beta t} \end{aligned}$$

where $\bar{H}$ is the constraint Hamiltonian function. The first order conditions for the maximization are:

$$\frac{\partial \bar{H}}{\partial u} = 0 \Rightarrow F_u + \zeta f_u = 0 \tag{2.1.7}$$

$$\frac{\partial \bar{H}}{\partial x} = -\dot{\zeta} + \beta\zeta \Rightarrow F_x + \zeta f_x = -\dot{\zeta} + \beta\zeta \qquad (2.1.8)$$

where the transversality condition for this constraint Hamiltonian function is $\lim_{t\to\infty} x\zeta e^{-\beta t} = 0$.

Comparing equations (2.1.5) and (2.1.6) and equations (2.1.7) and (2.1.8), we find that the first order conditions for the original Hamiltonian function and the constraint Hamiltonian function are the same. We can use the constraint Hamiltonian function to solve the original optimization problem.

Appendix 2.2: Proof of a positive root for equation (2.21)

Equation (2.21) is a function of the capital to health ratio:

$$\delta^h - \delta^k = \left[\beta A \left(\frac{\beta}{1-\alpha-\beta} \right)^{\beta-1} (\omega)^{\alpha} - \alpha A \left(\frac{\beta}{1-\alpha-\beta} \right)^{\beta} (\omega)^{\alpha-1} \right] \qquad (2.2.1)$$

Assume that $\beta A \left(\frac{\beta}{1-\alpha-\beta} \right)^{\beta-1} = c_1$, $\alpha A \left(\frac{\beta}{1-\alpha-\beta} \right)^{\beta} = c_2$ and $\delta^k - \delta^h = c_3$, equation (2.2.1) can be rewritten as a function of ω:

$$f(\omega) = c_1 \omega^{\alpha} - c_2 \omega^{\alpha-1} + c_3 = 0 \qquad (2.2.2)$$

where $\omega \in (0, +\infty)$. We have already discussed the special case where $c_3 = 0$. In the analysis below, we only consider the case where $c_3 \neq 0$. If the steady state ratio of capital to health ratio is positive, $f(\omega)$ should have a positive root.

We know that for the two extreme values 0 and $+\infty$, $f(0) = -\infty$ and $f(+\infty) = c_1 \omega^{\alpha} + c_3 > 0$. This result implies that the curve of $f(\omega)$ must intersect the positive part of x-axis at least once. In other words, there exists at least one positive root for the ratio of physical capital to health. As a result, when the depreciation rates for physical capital and human capital are not equal, we are still able to derive a constant ratio for physical capital to health.

Appendix 2.3: The derivation of the balanced growth path

The dynamic equilibrium conditions are available in subsection 2.3.2. From equation (2.11), we obtain:

$$\frac{\dot{c}}{c} = \frac{-\frac{\dot{v}_k}{v_k} - \rho + n}{1-\gamma} \tag{2.3.1}$$

Rearrange equation (2.13) to get the expression for $\frac{\dot{v}_1}{v_1}$ and insert it into equation (2.3.1), we get the familiar equation for the growth rate of consumption, which is corresponding to equation (3.11):

$$\frac{\dot{c}}{c} = \frac{\alpha A(k)^{\alpha-1}(s)^{\beta}(h)^{1-\alpha-\beta} - \delta^k - \rho}{1-\gamma} = \frac{\alpha A(\frac{k}{h})^{\alpha-1}(\frac{s}{h})^{\beta} - \delta^k - \rho}{1-\gamma} \tag{2.3.2}$$

Furthermore, from the condition that the net marginal products of the three capital inputs are equal, which can also be obtained from equation (2.13) to (2.15), we have the following equalities, which corresponding to equation (2.17):

$$\begin{aligned} &\alpha A k^{\alpha-1} s^{\beta} h^{1-\alpha-\beta} - \delta^k \\ &= \beta A k^{\alpha} s^{\beta-1} h^{1-\alpha-\beta} - \delta^h \\ &= (1-\alpha-\beta) A k^{\alpha} s^{\beta} h^{-(\alpha+\beta)} - \delta^h - (s+h)\frac{\partial \delta^h}{\partial h} \end{aligned} \tag{2.3.3}$$

Using the same argument as Barro (1996b), we assume that the human capital depreciation rate δ^h will gradually converge to a lower bound level δ_0^h in the steady state.

Assuming that there is a steady state, then from the second equality relation in equation (2.3.3), we derive the steady state ratio of education to health

$$\frac{s}{h} = \frac{\beta}{1-\alpha-\beta} \tag{2.3.4}$$

Furthermore, by manipulating the first equality relation in equation

(2.3.3), we get another equation:

$$\delta^h - \delta^k = \left[\beta A \left(\frac{k}{h}\right)^{\alpha} \left(\frac{s}{h}\right)^{\beta-1} - \alpha A \left(\frac{k}{h}\right)^{\alpha-1} \left(\frac{s}{h}\right)^{\beta} \right] \tag{2.3.5}$$

From equation (2.3.5), we notice that the growth rate of consumption per capita is determined by the ratios of physical capital to health capital $\frac{k}{h}$ and education capital to health capital $\frac{s}{h}$. From equation (2.3.4), we already found that the ratio $\frac{s}{h}$ is constant in the steady state. To determine the consumption growth rate, we need to find out the dynamics of the ratio $\frac{k}{h}$.

Equation (2.3.5) implies that because $\frac{s}{h}$ is constant in the steady state, this equation can be viewed as a function of the ratio of physical to health capital $\frac{k}{h}$. We can analyze the balanced growth path by considering whether the depreciation rates for physical capital and human capital are equal.

Consider the special case where the depreciation rates of physical capital δ and human capital δ^h are the same. In this case, $\delta^h - \delta^k = 0$ and from equation (2.3.5), we have:

$$\left[\beta A \left(\frac{k}{h}\right)^{\alpha} \left(\frac{s}{h}\right)^{\beta-1} - \alpha A \left(\frac{k}{h}\right)^{\alpha-1} \left(\frac{s}{h}\right)^{\beta} \right] = \delta^h - \delta^k = 0 \Rightarrow \frac{k}{s} = \frac{\alpha}{\beta} \tag{2.3.6}$$

Multiplying equation (2.3.4) by equation (2.3.6), we have an expression for health capital h in terms of physical capital k:

$$\frac{s}{h} \cdot \frac{k}{s} = \frac{k}{h} = \frac{\beta}{1-\alpha-\beta} \cdot \frac{\alpha}{\beta} \Rightarrow h = \frac{1-\alpha-\beta}{\alpha} \cdot k \tag{2.3.7}$$

By the equality relations of equations (2.3.6) and (2.3.7), we know that

when the depreciation rates of physical and human capital are the same, the three capitals are growing at the same rate. That is:

$$\frac{\dot{k}}{k} = \frac{\dot{s}}{s} = \frac{\dot{h}}{h} \tag{2.3.8}$$

Equation (2.3.8), together with equations (2.8) to (2.10) and the condition that the depreciation rates of physical and human capital are the same, we are able to get the following relations:

$$\frac{i_k}{k} = \frac{i_s}{s} = \frac{i_h}{h} \Rightarrow$$

$$i_s = i_k \cdot \frac{s}{k} \tag{2.3.9}$$

$$i_h = i_k \cdot \frac{h}{k} \tag{2.3.10}$$

The household's budget constraint is:

$$y = Ak^{\alpha} s^{\beta} h^{1-\alpha-\beta} = i_k + i_s + i_h + c \tag{2.3.11}$$

Inserting equation (2.3.6), (2.3.7), (2.3.9), and (2.3.10) into the budget constraint equation (2.3.11), we get the following equation:

$$\begin{aligned} & Ak^{\alpha}(\frac{\beta}{\alpha})^{\beta} k^{\beta} (\frac{1-\alpha-\beta}{\alpha})^{1-\alpha-\beta} k^{1-\alpha-\beta} \\ & = i_k + i_k \cdot \frac{s}{k} + i_k \cdot \frac{h}{k} + c \\ & = i_k + i_k \cdot \frac{\beta}{\alpha} + i_k \cdot \frac{1-\alpha-\beta}{\alpha} + c \end{aligned} \tag{2.3.12}$$

Dividing both sides of equation (2.3.12) by k, we get the following equation:

$$A(\frac{\beta}{\alpha})^{\beta} \cdot (\frac{1-\alpha-\beta}{\alpha})^{1-\alpha-\beta} = \frac{i_k}{k} \cdot \frac{1}{\alpha} + \frac{c}{k} \tag{2.3.13}$$

Dividing both sides of equation (2.8) by k, we have:

$$\frac{\dot{k}}{k} = \frac{i_k}{k} - (\delta + n) \tag{2.3.14}$$

In the steady state, $\frac{\dot{k}}{k}$, δ and n are all constant. Therefore, from

equation (2.3.14) we know that $\frac{i_k}{k}$ should be constant in the steady state. From equation (2.3.13), we further know that $\frac{c}{k}$ is also constant, which means that the growth rates of consumption and physical capital are the same in the steady state.

Differentiating the intensive form of the production function with respect to time, we have:

$$\frac{\dot{y}}{y} = \alpha\frac{\dot{k}}{k} + \beta\frac{\dot{s}}{s} + (1-\alpha-\beta)\frac{\dot{h}}{h} \tag{2.3.15}$$

From equation (2.21), we know that the growth rate of k, s, and h are the same in the steady state, so the growth rate of y should also be the same as the growth rate of the three capitals. As a result, the steady state growth rate of y, k, s, h and c are all the same in the steady state, which is denoted by r^*:

$$r^* = \frac{\dot{y}}{y} = \frac{\dot{k}}{k} = \frac{\dot{s}}{s} = \frac{\dot{h}}{h} = \frac{\dot{c}}{c} \tag{2.3.16}$$

The expression for this steady state growth rate r^* is:

$$\begin{aligned} r_1^* &= \frac{\dot{c}}{c} = \frac{\alpha A(\frac{k}{h})^{\alpha-1}(\frac{s}{h})^{\beta} - \delta - \rho}{1-r} \\ &= \frac{\alpha A(\frac{\alpha}{1-\alpha-\beta})^{\alpha-1}(\frac{\beta}{1-\alpha-\beta})^{\beta} - \delta - \rho}{1-r} \end{aligned} \tag{2.3.17}$$

Inserting the ratios of the capitals into the intensive form of the production function and rearranging the terms, we have:

$$y = Ak^{\alpha}s^{\beta}h^{1-\alpha-\beta} = Ah\cdot(\frac{k}{h})^{\alpha}(\frac{s}{h})^{\beta} = Ah\cdot(\frac{\alpha}{1-\alpha-\beta})^{\alpha}(\frac{\beta}{1-\alpha-\beta})^{\beta} \tag{2.3.18}$$

Appendix 2.4: Barro (1996b)-type model with health depreciation rate determined only by health

The constraint Hamiltonian function for this model is:

$$H = c^{\gamma} + v_k[I_k - (\delta^k + n)k] + v_s[I_s - (\delta^s + n)s] + v_h[I_h - (\delta^h(h) + n)h] + \lambda(k^{\alpha}s^{\beta}h^{1-\alpha-\beta} - I_k - I_s - I_h - c) \tag{2.4.1}$$

The corresponding first order conditions with respect to the control and state variables are:

$$\frac{\partial H}{\partial c} = 0 \Rightarrow rc^{\gamma-1} = \lambda \tag{2.4.2}$$

$$\frac{\partial H}{\partial k} = -\dot{v}_k + (\rho - n)v_k = -v_k(\delta^k + n) + \alpha\lambda k^{\alpha-1}s^{\beta}h^{1-\alpha-\beta} \tag{2.4.3}$$

$$\frac{\partial H}{\partial s} = -\dot{v}_s + (\rho - n)v_s = -v_s(\delta^s + n) + \beta\lambda k^{\alpha}s^{\beta-1}h^{1-\alpha-\beta} \tag{2.4.4}$$

$$\frac{\partial H}{\partial h} = -\dot{v}_h + (\rho - n)v_h = -v_h\left(\delta^h(h) + n\right) + (1-\alpha-\beta)\lambda k^{\alpha}s^{\beta}h^{-\alpha-\beta} - v_h\delta_h^h(h)h \tag{2.4.5}$$

$$\frac{\partial H}{\partial I_k} = \frac{\partial H}{\partial I_s} = \frac{\partial H}{\partial I_h} = 0 \Rightarrow v_k = v_s = v_h = \lambda \tag{2.4.6}$$

The equation of budget constraint is:

$$k^{\alpha}s^{\beta}h^{1-\alpha-\beta} - \dot{k} - (\delta^k + n)k - \dot{s} - (\delta^s + n)s - \dot{h} - (\delta^h(h) + n)h - c = 0 \tag{2.4.7}$$

From equation (2.4.2), we obtain the equation of $\dot{\lambda}$:

$$\gamma c^{\gamma-1} = \lambda \Leftrightarrow \gamma(\gamma-1)c^{\gamma-2}\dot{c} = \dot{\lambda} \tag{2.4.8}$$

Substituting λ and $\dot{\lambda}$ by $\gamma c^{\gamma-1}$ and $\gamma(\gamma-1)c^{\gamma-2}\dot{c}$ respectively into equations (2.4.3) to (2.4.5) and consider equation (2.4.6), we have:

$$-\gamma(\gamma-1)c^{\gamma-2}\dot{c} + (\rho-n)\gamma c^{\gamma-1} = -\gamma c^{\gamma-1}(\delta^k + n) + \alpha\gamma c^{\gamma-1}k^{\alpha-1}s^{\beta}h^{1-\alpha-\beta} \tag{2.4.9}$$

$$-\gamma(r-1)c^{\gamma-2}\dot{c} + (\rho-n)\gamma c^{\gamma-1} = -\gamma c^{\gamma-1}(\delta^s + n) + \beta\gamma c^{\gamma-1}k^{\alpha}s^{\beta-1}h^{1-\alpha-\beta} \tag{2.4.10}$$

$$-\gamma(\gamma-1)c^{\gamma-2}\dot{c} + (\rho-n)\gamma c^{\gamma-1} = -\gamma c^{\gamma-1}\left(\delta^h(h) + n\right) + (1-\alpha-\beta)\gamma c^{\gamma-1}k^{\alpha}s^{\beta}h^{-\alpha-\beta} - \gamma c^{\gamma-1}\delta_h^h(h)h \tag{2.4.11}$$

Assuming that $\delta^h = h^\theta$ and δ^k and δ^s are constants, we are able to derive the equations of consumption growth rate from equations (2.4.9) to (2.4.11):

$$-(\gamma-1)\frac{\dot{c}}{c} = -(\rho+\delta^k) + \alpha k^{\alpha-1}s^{\beta}h^{1-\alpha-\beta} \tag{2.4.12}$$

$$-(\gamma-1)\frac{\dot{c}}{c} = -(\rho+\delta^s) + \beta k^{\alpha}s^{\beta-1}h^{1-\alpha-\beta} \tag{2.4.13}$$

$$-(\gamma-1)\frac{\dot{c}}{c} = -(\rho+h^\theta) + (1-\alpha-\beta)k^{\alpha}s^{\beta}h^{-\alpha-\beta} - \theta h^\theta \tag{2.4.14}$$

We assume that $\frac{\dot{k}}{k} = \frac{\dot{s}}{s} = \frac{\dot{h}}{h} = \frac{\dot{c}}{c} = 0$ and then we have:

$$-(\gamma-1)\frac{\dot{c}}{c} = -(\rho+\delta^k) + \alpha k^{\alpha-1}s^{\beta}h^{1-\alpha-\beta} = 0 \Leftrightarrow \alpha k^{\alpha-1}s^{\beta}h^{1-\alpha-\beta} = \rho+\delta^k \tag{2.4.15}$$

$$-(\gamma-1)\frac{\dot{c}}{c} = -(\rho+\delta^s) + \beta k^{\alpha}s^{\beta-1}h^{1-\alpha-\beta} = 0 \Leftrightarrow \beta k^{\alpha}s^{\beta-1}h^{1-\alpha-\beta} = \rho+\delta^s \tag{2.4.16}$$

$$\begin{aligned} &-(\gamma-1)\frac{\dot{c}}{c} = -(\rho+h^\theta) + (1-\alpha-\beta)k^{\alpha}s^{\beta}h^{-\alpha-\beta} - \theta h^\theta = 0 \\ &\Leftrightarrow (1-\alpha-\beta)k^{\alpha}s^{\beta}h^{-\alpha-\beta} = (1+\theta)h^\theta + \rho \end{aligned} \tag{2.4.17}$$

From equations (2.4.15) and (2.4.16), we have:

$$\frac{\alpha k^{\alpha-1}s^{\beta}h^{1-\alpha-\beta}}{\beta k^{\alpha}s^{\beta-1}h^{1-\alpha-\beta}} = \frac{\rho+\delta^k}{\rho+\delta^s} \Leftrightarrow \frac{s}{k} = \left(\frac{\rho+\delta^k}{\rho+\delta^s}\right)\frac{\beta}{\alpha} \tag{2.4.18}$$

From equations (2.4.15) and (2.4.17), we have:

$$\frac{\alpha k^{\alpha-1}s^{\beta}h^{1-\alpha-\beta}}{(1-\alpha-\beta)k^{\alpha}s^{\beta}h^{-\alpha-\beta}} = \frac{\rho+\delta^k}{(1+\theta)h^\theta+\rho} \Leftrightarrow \frac{\alpha h}{(1-\alpha-\beta)k} = \frac{\rho+\delta^k}{(1+\theta)h^\theta+\rho}$$

$$\Leftrightarrow k = \frac{\alpha h\left[(1+\theta)h^\theta+\rho\right]}{(1-\alpha-\beta)\left(\rho+\delta^k\right)} \tag{2.4.19}$$

From equations (2.4.16) and (2.4.17), we also have:

$$\frac{\beta k^{\alpha}s^{\beta-1}h^{1-\alpha-\beta}}{(1-\alpha-\beta)k^{\alpha}s^{\beta}h^{-\alpha-\beta}} = \frac{\rho+\delta^s}{(1+\theta)h^\theta+\rho} \Leftrightarrow \frac{\beta h}{(1-\alpha-\beta)s} = \frac{\rho+\delta^s}{(1+\theta)h^\theta+\rho}$$

$$\Leftrightarrow s = \frac{\beta h\left[(1+\theta)h^\theta+\rho\right]}{(1-\alpha-\beta)(\rho+\delta^s)} \tag{2.4.20}$$

Insert equations (2.4.19) and (2.4.20) into equation (2.4.15), we then

have:

$$\alpha\left\{\frac{\alpha h\left[(1+\theta)h^{\theta}+\rho\right]}{(1-\alpha-\beta)\left(\rho+\delta^{k}\right)}\right\}^{\alpha-1}\left\{\frac{\beta h\left[(1+\theta)h^{\theta}+\rho\right]}{(1-\alpha-\beta)(\rho+\delta^{s})}\right\}^{\beta}h^{1-\alpha-\beta}=\rho+\delta^{k}$$

$$\Leftrightarrow\left[(1+\theta)h^{\theta}+\rho\right]^{\alpha+\beta-1}$$

$$=\frac{\left[(1-\alpha-\beta)\left(\rho+\delta^{k}\right)\right]^{\alpha-1}\left[(1-\alpha-\beta)(\rho+\delta^{s})\right]^{\beta}\left(\rho+\delta^{k}\right)}{\alpha^{\alpha}\beta^{\beta}}$$

$$=\frac{(1-\alpha-\beta)^{\alpha+\beta-1}\left(\rho+\delta^{k}\right)^{\alpha}(\rho+\delta^{s})^{\beta}}{\alpha^{\alpha}\beta^{\beta}}$$

$$\Leftrightarrow h^{*}=\left[\frac{(1-\alpha-\beta)\left(\rho+\delta^{k}\right)^{\alpha}{}^{\alpha+\beta-1}(\rho+\delta^{s})^{\beta}{}^{\alpha+\beta-1}}{(1+\theta)\alpha^{\alpha+\beta-1}\beta^{\alpha+\beta-1}}-\frac{\rho}{1+\theta}\right]^{\frac{1}{\theta}} \qquad (2.4.21)$$

Assuming that $\frac{(1-\alpha-\beta)\left(\rho+\delta^{k}\right)^{\alpha}{}^{\alpha+\beta-1}(\rho+\delta^{s})^{\beta}{}^{\alpha+\beta-1}}{(1+\theta)\alpha^{\alpha+\beta-1}\beta^{\alpha+\beta-1}}=\kappa$, then we have:

$$h^{*}=\left(\kappa-\frac{\rho}{1+\theta}\right)^{\frac{1}{\theta}}$$

From equations (2.4.19) and (2.4.21), we have:

$$k^{*}=\frac{\alpha\left(\kappa-\frac{\rho}{1+\theta}\right)^{\frac{1}{\theta}}\left[(1+\theta)\left(\kappa-\frac{\rho}{1+\theta}\right)+\rho\right]}{(1-\alpha-\beta)\left(\rho+\delta^{k}\right)}=\frac{(1+\theta)\kappa\alpha\left(\kappa-\frac{\rho}{1+\theta}\right)^{\frac{1}{\theta}}}{(1-\alpha-\beta)\left(\rho+\delta^{k}\right)} \qquad (2.4.22)$$

From equations (2.4.20) and (2.4.21), we have:

$$s^{*}=\frac{\beta\left(\kappa-\frac{\rho}{1+\theta}\right)^{\frac{1}{\theta}}\left[(1+\theta)\left(\kappa-\frac{\rho}{1+\theta}\right)+\rho\right]}{(1-\alpha-\beta)(\rho+\delta^{s})}$$

$$=\frac{(1+\theta)\kappa\beta\left(\kappa-\frac{\rho}{1+\theta}\right)^{\frac{1}{\theta}}}{(1-\alpha-\beta)(\rho+\delta^{s})} \qquad (2.4.23)$$

Insert equations (2.4.21) to (2.4.23) into equation (2.4.7), we have the equilibrium value of consumption c:

$$
\begin{aligned}
c^{*} &= k^{\alpha} s^{\beta} h^{1-\alpha-\beta} - (\delta^{k} + n)k - (\delta^{'} + n)s - (h^{\theta} + n)h \\
&= \left[\frac{(1+\theta)\kappa\alpha\left(\kappa - \frac{\rho}{1+\theta}\right)^{\frac{1}{\theta}}}{(1-\alpha-\beta)\left(\rho+\delta^{k}\right)}\right]^{\alpha}\left[\frac{(1+\theta)\kappa\beta\left(\kappa - \frac{\rho}{1+\theta}\right)^{\frac{1}{\theta}}}{(1-\alpha-\beta)(\rho+\delta^{'})}\right]^{\beta}\left(\kappa - \frac{\rho}{2}\right)^{1-\alpha-\beta} \\
&\quad -(\delta^{k} + n)\frac{(1+\theta)\kappa\alpha\left(\kappa - \frac{\rho}{1+\theta}\right)^{\frac{1}{\theta}}}{(1-\alpha-\beta)\left(\rho+\delta^{k}\right)} \\
&\quad -(\delta^{'} + n)\frac{(1+\theta)\kappa\beta\left(\kappa - \frac{\rho}{1+\theta}\right)^{\frac{1}{\theta}}}{(1-\alpha-\beta)(\rho+\delta^{'})} \\
&\quad -(\kappa - \frac{\rho}{1+\theta} + n)\left(\kappa - \frac{\rho}{1+\theta}\right)^{\frac{1}{\theta}}
\end{aligned}
\tag{2.4.24}
$$

Equations (2.4.21) to (2.4.24) indicate that there are equilibrium values k^{*}, s^{*}, h^{*} and c^{*} and there will be no endogenous growth when we assume the health depreciation rate as $\delta^{h}(h) = h^{\theta}$.

Appendix 2.5: Barro (1996b)-type model with health depreciation rate determined simultaneously by both health and education

The constraint Hamiltonian function for this model is:

$$H = c^{\gamma} + v_k[I_k - (\delta^k + n)k] + v_s[I_s - (\delta^s + n)s] + v_h[I_h - (\delta^h(h,s) + n)h] + \lambda(k^{\alpha}s^{\beta}h^{1-\alpha-\beta} - I_k - I_s - I_h - c) \quad (2.5.1)$$

The corresponding first order conditions with respect to the control and state variables are:

$$\frac{\partial H}{\partial c} = 0 \Rightarrow \gamma c^{\gamma-1} = \lambda \quad (2.5.2)$$

$$\frac{\partial H}{\partial k} = -\dot{v}_k + (\rho - n)v_k = -v_k(\delta^k + n) + \alpha\lambda k^{\alpha-1}s^{\beta}h^{1-\alpha-\beta} \quad (2.5.3)$$

$$\frac{\partial H}{\partial s} = -\dot{v}_s + (\rho - n)v_s = -v_s(\delta^s + n) + \beta\lambda k^{\alpha}s^{\beta-1}h^{1-\alpha-\beta} - v_h\delta_s^h(h,s)h \quad (2.5.4)$$

$$\frac{\partial H}{\partial h} = -\dot{v}_h + (\rho - n)v_h = -v_h(\delta^h + n) + (1-\alpha-\beta)\lambda k^{\alpha}s^{\beta}h^{-\alpha-\beta} - v_h\delta_h^h(h,s)h \quad (2.5.5)$$

$$\frac{\partial H}{\partial I_k} = \frac{\partial H}{\partial I_s} = \frac{\partial H}{\partial I_h} = 0 \Rightarrow v_k = v_s = v_h = \lambda \quad (2.5.6)$$

The equation of budget constraint is:

$$k^{\alpha}s^{\beta}h^{1-\alpha-\beta} - \dot{k} - (\delta^k + n)k - \dot{s} - (\delta^s + n)s - \dot{h} - (\delta^h(h) + n)h - c = 0 \quad (2.5.7)$$

From equation (2.5.2), we obtain the equation of $\dot{\lambda}$:

$$\gamma c^{\gamma-1} = \lambda \Leftrightarrow \gamma(\gamma-1)c^{\gamma-2}\dot{c} = \dot{\lambda} \quad (2.5.8)$$

Substituting λ and $\dot{\lambda}$ by $rc^{\gamma-1}$ and $\gamma(\gamma-1)c^{\gamma-2}\dot{c}$ respectively into equations (2.5.3) to (2.5.5) and consider equation (2.5.6), we have:

$$-\gamma(\gamma-1)c^{\gamma-2}\dot{c} + (\rho-n)\gamma c^{\gamma-1} = -\gamma c^{\gamma-1}(\delta^k + n) + \alpha\gamma c^{\gamma-1}k^{\alpha-1}s^{\beta}h^{1-\alpha-\beta} \quad (2.5.9)$$

$$-\gamma(\gamma-1)c^{\gamma-2}\dot{c} + (\rho-n)\gamma c^{\gamma-1} = -\gamma c^{\gamma-1}(\delta^s + n) + \beta\gamma c^{\gamma-1}k^{\alpha}s^{\beta-1}h^{1-\alpha-\beta} - \gamma c^{\gamma-1}\delta_s^h(h,s)h \quad (2.5.10)$$

$$-\gamma(\gamma-1)c^{\gamma-2}\dot{c} + (\rho-n)\gamma c^{\gamma-1} = -\gamma c^{\gamma-1}(\delta^h(s,h) + n) + (1-\alpha-\beta)\gamma c^{\gamma-1}k^{\alpha}s^{\beta}h^{-\alpha-\beta} - \gamma c^{\gamma-1}\delta_h^h(s,h)h$$

(2.5.11)

Assume $\delta^h(h,s) = h^\theta s^{-\theta}$ and let $\frac{\dot{k}}{k}, \frac{\dot{s}}{s}, \frac{\dot{h}}{h}$, and $\frac{\dot{c}}{c}$ being constants, then we have the following equations of consumption growth:

$$-(\gamma-1)\frac{\dot{c}}{c} = -(\rho+\delta^k) + \alpha k^{\alpha-1}s^\beta h^{1-\alpha-\beta} \tag{2.5.12}$$

$$-(\gamma-1)\frac{\dot{c}}{c} = -(\rho+\delta^s) + \beta k^\alpha s^{\beta-1}h^{1-\alpha-\beta} + \theta h^{1+\theta}s^{-1-\theta} \tag{2.5.13}$$

$$-(\gamma-1)\frac{\dot{c}}{c} = -(\rho+h^\theta s^{-\theta}) + (1-\alpha-\beta)k^\alpha s^\beta h^{-\alpha-\beta} - \theta h^\theta s^{-\theta} \tag{2.5.14}$$

Assuming that $\frac{k}{h} = x$ and $\frac{s}{h} = y$, then we have:

$$-(\gamma-1)\frac{\dot{c}}{c} = -(\rho+\delta^k) + \alpha x^{\alpha-1}y^\beta \tag{2.5.15}$$

$$-(\gamma-1)\frac{\dot{c}}{c} = -(\rho+\delta^s) + \beta x^\alpha y^{\beta-1} + \theta y^{-1-\theta} \tag{2.5.16}$$

$$-(\gamma-1)\frac{\dot{c}}{c} = -(\rho+y^{-\theta}) + (1-\alpha-\beta)x^\alpha y^\beta - \theta y^{-\theta} \tag{2.5.17}$$

From equations (2.5.15) to (2.5.17), we have:

$$\delta^s - \delta^k + \alpha x^{\alpha-1}y^\beta - \theta y^{-1-\theta} = \beta x^\alpha y^{\beta-1} \tag{2.5.18}$$

$$\alpha x^{\alpha-1}y^\beta + (1+\theta)y^{-\theta} - \delta^k = (1-\alpha-\beta)x^\alpha y^\beta \tag{2.5.19}$$

We assume that $\delta^s = \delta^k = 0$. Then equations (2.5.18) and (2.5.19) change to the following two equations:

$$x^{\alpha-1}y^{\beta-1}(\alpha y - \beta x) = \theta y^{-1-\theta} \tag{2.5.20}$$

$$x^{\alpha-1}y^{\beta-1}[\alpha y - (1-\alpha-\beta)xy] = -(1+\theta)y^{-\theta} \tag{2.5.21}$$

Dividing equation (2.5.20) by equation (2.5.21), we have:

$$\frac{x^{\alpha-1}y^{\beta-1}(\alpha y - \beta x)}{x^{\alpha-1}y^{\beta-1}[\alpha y - (1-\alpha-\beta)xy]} = \frac{\theta y^{-1-\theta}}{-(1+\theta)y^{-\theta}}$$

$$\Leftrightarrow (1+\theta)\alpha y - (1+\theta)\beta x + \theta\alpha - \theta(1-\alpha-\beta)x = 0$$

$$\Leftrightarrow y = -\frac{\theta}{1+\theta} + \frac{\beta+\theta(1-\alpha)}{(1+\theta)\alpha}x \tag{2.5.22}$$

From equations (2.5.20) and (2.5.21), we are able to derive a nonlinear equation of y as a function of x:

$$y^{-(\theta+\beta)} = \frac{(1-\alpha-\beta)}{(1+\theta)} x^{\alpha} - \frac{\alpha}{1+\theta} x^{\alpha-1} \tag{2.5.23}$$

From equation (2.5.23), we have:

$$y^{-(\theta+\beta)} = \frac{(1-\alpha-\beta)}{(1+\theta)} x^{\alpha} - \frac{\alpha}{1+\theta} x^{\alpha-1} \geq 0$$

$$\Leftrightarrow \frac{(1-\alpha-\beta)}{(1+\theta)} x \geq \frac{\alpha}{1+\theta}$$

$$\Leftrightarrow x \geq \frac{\alpha}{1-\alpha-\beta}$$

By total differentiation of equation (2.5.23), we are able to find the marginal effect of x on y:

$$y^{-(\theta+\beta)} = \frac{(1-\alpha-\beta)}{(1+\theta)} x^{\alpha} - \frac{\alpha}{1+\theta} x^{\alpha-1}$$

$$\Leftrightarrow -(\theta+\beta) y^{-(\theta+\beta+1)} dy = \left(\frac{\alpha(1-\alpha-\beta)}{(1+\theta)} x^{\alpha-1} - \frac{\alpha(\alpha-1)}{1+\theta} x^{\alpha-2} \right) dx$$

$$\Leftrightarrow \frac{dy}{dx} = \frac{\alpha}{(1+\theta)} x^{\alpha-2} \left(\frac{(1-\alpha-\beta)x - (\alpha-1)}{-(\theta+\beta) y^{-(\theta+\beta+1)}} \right) = 0$$

$$\Leftrightarrow x = -\frac{1-\alpha}{1-\alpha-\beta} \in (-\infty, -1)$$

It is obvious that if $x > -\frac{1-\alpha}{1-\alpha-\beta}$, then the marginal effect of x on y is negative:

$$\frac{dy}{dx} = \frac{\alpha}{(1+\theta)} x^{\alpha-2} \left(\frac{(1-\alpha-\beta)x + (1-\alpha)}{-(\theta+\beta) y^{-(\theta+\beta+1)}} \right) < 0 \tag{2.5.24}$$

We can also derive the limit of $y^{-(\theta+\beta)}$ by the following two equations:

$$\lim_{x \to +\infty} y^{-(\theta+\beta)} = \lim_{x \to +\infty} \left[\frac{(1-\alpha-\beta)}{(1+\theta)} x^{\alpha} - \frac{\alpha}{1+\theta} x^{\alpha-1} \right] = +\infty \tag{2.5.25}$$

$$\Leftrightarrow \lim_{x \to +\infty} y = 0$$

$$\lim_{x \to \left(1-\frac{\alpha}{\alpha+\beta}\right)^{+}} y^{(\theta+\beta)} = \lim_{x \to \left(1-\frac{\alpha}{\alpha+\beta}\right)^{+}} \left[\frac{(1-\alpha-\beta)}{(1+\theta)} x^{\alpha} - \frac{\alpha}{1+\theta} x^{\alpha-1} \right] = 0$$
$$\Leftrightarrow \lim_{x \to (1-\alpha)^{+}} y = +\infty \qquad (2.5.26)$$

Equations (2.5.24) to (2.5.26) indicate that equation (2.5.23) is a decreasing function of x and with a lower bound $y_0>0$. By putting the curves of equation (2.5.22) and (2.5.23) into one graph (Figure 2.1), we are able to find a pair of equilibrium values x^* and y^*, which, by definition, indicates that the growth rates of physical, education and health capitals are the same in the long-run. In other words, there is endogenous growth in this Barro (1996b)-type model.

Appendix 2.6: Barro (1996b)-type model with constant health depreciation rate and health entering the utility function: $u(c,h) = c^{\gamma} h^{1-\gamma}$

The constraint Hamiltonian function for this case is:

$$\begin{aligned} H = {} & c^{\gamma} h^{1-\gamma} + v_k[I_k - (\delta^k + n)k] + v_s[I_s - (\delta^s + n)s] \\ & + v_h[I_h - (\delta^h + n)h] + \lambda(k^{\alpha} s^{\beta} h^{1-\alpha-\beta} - I_k - I_s - I_h - c) \end{aligned} \tag{2.6.1}$$

The first order conditions for the control variables and state variables of the above equation are:

$$\frac{\partial H}{\partial c} = 0 \Rightarrow \gamma \left(\frac{c}{h}\right)^{\gamma - 1} = \lambda \tag{2.6.2}$$

$$\frac{\partial H}{\partial k} = -\dot{v}_k + (\rho - n)v_k = -v_k(\delta^k + n) + \alpha\lambda k^{\alpha-1} s^{\beta} h^{1-\alpha-\beta} \tag{2.6.3}$$

$$\frac{\partial H}{\partial s} = -\dot{v}_s + (\rho - n)v_s = -v_s(\delta^s + n) + \beta\lambda k^{\alpha} s^{\beta-1} h^{1-\alpha-\beta} \tag{2.6.4}$$

$$\begin{aligned} \frac{\partial H}{\partial h} = {} & -\dot{v}_h + (\rho - n)v_h = \frac{(1-r)}{r}\left(\frac{c}{h}\right)\lambda \\ & - v_h(\delta^h + n) + (1-\alpha-\beta)\lambda k^{\alpha} s^{\beta} h^{-\alpha-\beta} - \frac{\partial \delta^h}{\partial h}\left(v_h h + v_s s\right) \end{aligned} \tag{2.6.5}$$

$$\frac{\partial H}{\partial I_k} = \frac{\partial H}{\partial I_s} = \frac{\partial H}{\partial I_h} = 0 \Rightarrow v_k = v_s = v_h = \lambda \tag{2.6.6}$$

We assume that $x = \frac{k}{h}$, $y = \frac{s}{h}$ and $z = \frac{c}{h}$. Together with the condition implied by equation (2.6.6), equations (2.6.3) to (2.6.5) then become:

$$-\dot{\lambda} + (\rho - n)\lambda = -\lambda(\delta^k + n) + \alpha\lambda x^{\alpha-1} y^{\beta} \tag{2.6.7}$$

$$-\dot{\lambda} + (\rho - n)\lambda = -\lambda(\delta^s + n) + \beta\lambda x^{\alpha} y^{\beta-1} \tag{2.6.8}$$

$$-\dot{\lambda} + (\rho - n)\lambda = \frac{(1-r)}{r} z\lambda - \lambda(\delta^h + n) + (1-\alpha-\beta)\lambda x^{\alpha} y^{\beta} \tag{2.6.9}$$

Assume that $\frac{\dot{k}}{k} = \frac{\dot{s}}{s} = \frac{\dot{h}}{h} = \frac{\dot{c}}{c} = \text{constant}$, then on the balanced growth

path, we can get $\frac{\dot{\lambda}}{\lambda} = (r-1)\frac{\dot{z}}{z} = 0$ from the condition $\gamma z^{\gamma-1} = \lambda$. Using this condition, on the balanced growth path, equations (2.6.7) to (2.6.9) becomes:

$$0 = (1-\gamma)\frac{\dot{z}}{z} = -\frac{\dot{\lambda}}{\lambda} = \alpha x^{\alpha-1} y^{\beta} - (\rho + \delta^k) \tag{2.6.10}$$

$$= \beta x^{\alpha} y^{\beta-1} - (\rho + \delta^s) \tag{2.6.11}$$

$$= \frac{1-\gamma}{\gamma} z + (1-\alpha-\beta) x^{\alpha} y^{\beta} - (\rho + \delta^h) \tag{2.6.12}$$

From equations (2.6.10) and (2.6.11), we have an equation y as a function of x:

$$\frac{\alpha}{\beta}\frac{y}{x} = \frac{\rho+\delta^k}{\rho+\delta^s} \Leftrightarrow y = \left(\frac{\rho+\delta^k}{\rho+\delta^s}\right)\left(\frac{\beta}{\alpha}\right)x \tag{2.6.13}$$

Inserting equation (2.6.13) into equation (2.6.10), we have:

$$\alpha x^{\alpha-1}\left[\left(\frac{\rho+\delta^k}{\rho+\delta^s}\right)\left(\frac{\beta}{\alpha}\right)x\right]^{\beta} - (\rho+\delta^k) = 0$$

$$\Rightarrow x^* = \left[\frac{(\rho+\delta^k)}{\alpha\left(\frac{\rho+\delta^k}{\rho+\delta^s}\right)^{\beta}\left(\frac{\beta}{\alpha}\right)^{\beta}}\right]^{\frac{1}{\alpha+\beta-1}} = \left[\frac{(\rho+\delta^k)^{1-\beta}(\rho+\delta^s)^{\beta}}{\alpha^{1-\beta}\beta^{\beta}}\right]^{\frac{1}{\alpha+\beta-1}} \tag{2.6.14}$$

Putting equation (2.6.14) into equation (2.6.13), we have the steady state value of y:

$$y^* = \left(\frac{\rho+\delta^k}{\rho+\delta^s}\right)\left(\frac{\beta}{\alpha}\right)\left\{\frac{(\rho+\delta^k)}{\alpha\left[\left(\frac{\rho+\delta^k}{\rho+\delta^s}\right)\left(\frac{\beta}{\alpha}\right)\right]^{\beta}}\right\}^{\frac{1}{\alpha+\beta-1}} \tag{2.6.15}$$

$$= \left[\frac{(\rho+\delta^k)^{\alpha}}{\alpha^{\alpha}\beta^{1-\alpha}(\rho+\delta^s)^{\alpha-1}}\right]^{\frac{1}{\alpha+\beta-1}}$$

By equations (2.6.12), (2.6.14) and (2.6.15), we have the steady state

value of z:

$$z = \frac{\gamma}{1-\gamma}\left\{(\rho+\delta^h)-(1-\alpha-\beta)\left[\frac{(\rho+\delta^k)^{1-\beta}(\rho+\delta')^{\beta}}{\alpha^{1-\beta}\beta^{\beta}}\right]^{\frac{\alpha+\beta}{\alpha+\beta-1}}\left(\frac{\rho+\delta^k}{\rho+\delta'}\right)^{\beta}\left(\frac{\beta}{\alpha}\right)^{\beta}\right\}$$

$$\Rightarrow z^* = \frac{\gamma}{1-\gamma}\left\{(\rho+\delta^h)-(1-\alpha-\beta)\left[\frac{(\rho+\delta^k)^{\alpha}(\rho+\delta')^{\beta}}{\alpha^{\alpha}\beta^{\beta}}\right]^{\frac{1}{\alpha+\beta-1}}\right\} \qquad (2.6.16)$$

We assume the constant growth rate as $\frac{\dot{k}}{k} = \frac{\dot{s}}{s} = \frac{\dot{h}}{h} = \frac{\dot{c}}{c} = g$. Moreover, from the resource constraint condition, we have:

$$k^{\alpha}s^{\beta}h^{\alpha+\beta} - \left(\frac{k}{h}\right)\left(\frac{\dot{k}}{k}\right) - (\delta^k+n)\left(\frac{k}{h}\right) - \left(\frac{s}{h}\right)\left(\frac{\dot{s}}{s}\right)$$

$$-(\delta'+n)\left(\frac{s}{h}\right) - \frac{\dot{h}}{h} - (\delta^h+n) - \frac{c}{h} = 0$$

$$x^{\alpha}y^{\beta} - (\delta^k+n)x - (\delta'+n)y - (\delta^h+n) - z = (x+y+1)g$$

$$\Leftrightarrow g = \frac{x^{*\alpha}y^{*\beta} - (\delta^k+n)x^* - (\delta'+n)y^* - (\delta^h+n) - z^*}{x^*+y^*+1} \qquad (2.6.17)$$

Appendix 2.7: Barro (1996b)-type model with health depreciation rate determined by health and health entering the utility function: $u(c,h) = c^{\gamma} h^{1-\gamma}$

The constraint Hamiltonian function for this case is:

$$H = c^{\gamma} h^{1-\gamma} + v_k[I_k - (\delta^k + n)k] + v_s[I_s - (\delta^s + n)s] + v_h[I_h - (\delta^h(h) + n)h] + \lambda(k^{\alpha} s^{\beta} h^{1-\alpha-\beta} - I_k - I_s - I_h - c)$$

The corresponding first order conditions for the control variables and state variables are:

$$\frac{\partial H}{\partial c} = 0 \Rightarrow \gamma\left(\frac{c}{h}\right)^{\gamma-1} = \lambda \tag{2.7.1}$$

$$\frac{\partial H}{\partial k} = -\dot{v}_k + (\rho - n)v_k = -v_k(\delta^k + n) + \alpha\lambda k^{\alpha-1} s^{\beta} h^{1-\alpha-\beta} \tag{2.7.2}$$

$$\frac{\partial H}{\partial s} = -\dot{v}_s + (\rho - n)v_s = -v_s(\delta^s + n) + \beta\lambda k^{\alpha} s^{\beta-1} h^{1-\alpha-\beta} \tag{2.7.3}$$

$$\frac{\partial H}{\partial h} = -\dot{v}_h + (\rho - n)v_h = \frac{(1-\gamma)}{\gamma}\left(\frac{c}{h}\right)\lambda - v_h(\delta^h(h) + n) + (1-\alpha-\beta)\lambda k^{\alpha} s^{\beta} h^{-\alpha-\beta} - v_h\delta_h^h h \tag{2.7.4}$$

$$\frac{\partial H}{\partial I_k} = \frac{\partial H}{\partial I_s} = \frac{\partial H}{\partial I_h} = 0 \Rightarrow v_k = v_s = v_h = \lambda \tag{2.7.5}$$

From equation (2.7.1), we have a dynamic equation of λ as a function of c and h:

$$(\gamma - 1)\left(\frac{\dot{c}}{c} - \frac{\dot{h}}{h}\right) = \frac{\dot{\lambda}}{\lambda} \tag{2.7.6}$$

On the balanced growth path, we assume $\frac{\dot{k}}{k} = \frac{\dot{s}}{s} = \frac{\dot{h}}{h} = \frac{\dot{c}}{c} = 0$, which, together with equation (2.119), indicates that $\frac{\dot{\lambda}}{\lambda} = 0$ on the balanced growth path.

As a result, equations (2.7.2) to (2.7.4) become:

$$\frac{-\dot{\lambda}}{\lambda} = -(\delta^k + \rho) + \alpha k^{\alpha-1} s^{\beta} h^{1-\alpha-\beta} = 0 \tag{2.7.7}$$

$$\frac{-\dot{\lambda}}{\lambda} = -(\delta^s + \rho) + \beta k^{\alpha} s^{\beta-1} h^{1-\alpha-\beta} = 0 \tag{2.7.8}$$

$$\frac{-\dot{\lambda}}{\lambda} = \frac{(1-\gamma)}{\gamma}\left(\frac{c}{h}\right) - (h^{\theta} + \rho) + (1-\alpha-\beta)k^{\alpha} s^{\beta} h^{-\alpha-\beta} - \theta h^{\theta} = 0$$

$$\Rightarrow (1+\theta)h^{1+\theta} + \rho h - \frac{(1-\gamma)}{\gamma} c = (1-\alpha-\beta)k^{\alpha} s^{\beta} h^{1-\alpha-\beta} \tag{2.7.9}$$

For simplicity reason, without loss of generalarity, we assume that $\delta^k = \delta^s = \delta$. With this assumption, we have the relationship between s and k from equations (2.7.7) and (2.7.8):

$$s = \frac{\beta}{\alpha} k \tag{2.7.10}$$

Replacing s in equation (2.7.7) by equation (2.7.10), we have an equation of k as a function of h:

$$\alpha k^{\alpha-1}\left(\frac{\beta}{\alpha} k\right)^{\beta} h^{1-\alpha-\beta} = \delta^k + \rho$$

$$\Rightarrow k = \left(\frac{\delta_k + \rho}{\alpha^{1-\beta}\beta^{\beta} h^{1-\alpha-\beta}}\right)^{\frac{1}{\alpha+\beta-1}} = \left(\frac{\delta_k + \rho}{\alpha^{1-\beta}\beta^{\beta}}\right)^{\frac{1}{\alpha+\beta-1}} h = \omega_1 h \tag{2.7.11}$$

$$\Rightarrow s = \frac{\beta}{\alpha} k = \frac{\beta}{\alpha} \omega_1 h \tag{2.7.12}$$

On the balanced growth path, the equation of budget constraint turns to:

$$k^{\alpha} s^{\beta} h^{1-\alpha-\beta} - \dot{k} - (\delta^k + n)k - \dot{s} - (\delta^s + n)s - \dot{h} - (\delta^h(h,s) + n)h - c = 0$$

$$\Leftrightarrow c = k^{\alpha} s^{\beta} h^{1-\alpha-\beta} - (\delta^k + n)k - (\delta^s + n)s - (h^{\theta} + n)h \tag{2.7.13}$$

From equations (2.7.9) and (2.7.13), we are able to get an equation of only k, s and h. Replacing k and s by equations (2.7.11) and (2.7.12), we have:

$$(1+\theta)h^{\theta} + \rho - \frac{(1-r)}{r}\left\{\left[\omega^{\alpha+\beta}\left(\frac{\beta}{\alpha}\right)^{\beta} - (\delta+n)(1+\frac{\beta}{\alpha})\omega\right] - (h^{\theta}+n)\right\}$$

$$= (1-\alpha-\beta)\left(\frac{\beta}{\alpha}\right)^{\beta}\omega^{\alpha+\beta}$$

$$\Rightarrow \left[(1+\theta)+\frac{(1-r)}{r}\right]h^{\theta}$$

$$= (1-\alpha-\beta)\left(\frac{\beta}{\alpha}\right)^{\beta}\omega^{\alpha+\beta} + \frac{(1-r)}{r}\left[\omega^{\alpha+\beta}\left(\frac{\beta}{\alpha}\right)^{\beta} - (\delta+n)(1+\frac{\beta}{\alpha})\omega - n\right] - \rho$$

$$h^{*} = \left\{\frac{(1-\alpha-\beta)\left(\frac{\beta}{\alpha}\right)^{\beta}\omega^{\alpha+\beta} + \frac{(1-r)}{r}\left[\omega^{\alpha+\beta}\left(\frac{\beta}{\alpha}\right)^{\beta} - (\delta+n)(1+\frac{\beta}{\alpha})\omega - n\right] - \rho}{(1+\theta)+\frac{(1-r)}{r}}\right\}^{\frac{1}{\theta}}$$

The corresponding steady state values of k, s and c, are:

$$k^{*} = \omega_1 h^{*}$$

$$s = \frac{\beta}{\alpha}k^{*}$$

$$c = k^{*\alpha}s^{*\beta}h^{*1-\alpha-\beta} - (\delta+n)k^{*} - (\delta+n)s^{*} - (h^{\theta}+n)h^{*}$$

Appendix 2.8: Barro (1996b)-type model with health depreciation rate determined simultaneously by both health and education and health entering the utility function: $u(c,h)=c^{\gamma}h^{1-\gamma}$

The constraint Hamiltonian function for this case is:

$$\begin{aligned}H &= c^{\gamma}h^{1-\gamma}+v_k[I_k-(\delta^k+n)k]+v_s[I_s-(\delta^s+n)s]\\ &\quad +v_h[I_h-(\delta^h(h,s)+n)h]+\lambda(k^{\alpha}s^{\beta}h^{1-\alpha-\beta}-I_k-I_s-I_h-c)\end{aligned} \tag{2.8.1}$$

The corresponding first order conditions for the control variables and state variables are:

$$\frac{\partial H}{\partial c}=0\Rightarrow\gamma\left(\frac{c}{h}\right)^{\gamma-1}=\lambda \tag{2.8.2}$$

$$\frac{\partial H}{\partial k}=-\dot{v}_k+(\rho-n)v_k=-v_k(\delta^k+n)+\alpha\lambda k^{\alpha-1}s^{\beta}h^{1-\alpha-\beta} \tag{2.8.3}$$

$$\frac{\partial H}{\partial s}=-\dot{v}_s+(\rho-n)v_s=-v_s(\delta^s+n)+\beta\lambda k^{\alpha}s^{\beta-1}h^{1-\alpha-\beta}-v_h\delta_s^h h \tag{2.8.4}$$

$$\begin{aligned}\frac{\partial H}{\partial h} &= -\dot{v}_h+(\rho-n)v_h\\ &=\frac{(1-r)}{r}\left(\frac{c}{h}\right)\lambda-v_h(\delta^h+n)+(1-\alpha-\beta)\lambda k^{\alpha}s^{\beta}h^{-\alpha-\beta}-v_h\delta_h^h h\end{aligned} \tag{2.8.5}$$

$$\frac{\partial H}{\partial I_k}=\frac{\partial H}{\partial I_s}=\frac{\partial H}{\partial I_h}=0\Rightarrow v_k=v_s=v_h=\lambda \tag{2.8.6}$$

We assume the ratios of physical capital, education and consumption to health respectively as $x=\frac{k}{h}$, $y=\frac{s}{h}$ and $z=\frac{c}{h}$. In the steady state, we assume that these ratios are constants.

From equations (2.8.7) to (2.8.8), we have equations of $\frac{\dot{\lambda}}{\lambda}$:

$$-\frac{\dot{\lambda}}{\lambda}=-(\rho+\delta^k)+\alpha x^{\alpha-1}y^{\beta} \tag{2.8.9}$$

$$-\frac{\dot{\lambda}}{\lambda}=-(\rho+\delta^s)+\beta x^{\alpha}y^{\beta-1}+\theta y^{-\theta-1} \tag{2.8.10}$$

$$-\frac{\dot{\lambda}}{\lambda}=-(\rho+y^{-\theta})+(1-\alpha-\beta)x^{\alpha}y^{\beta}+\frac{(1-r)}{r}(z)-\theta y^{-\theta} \tag{2.8.11}$$

On the balanced growth path, from equation (2.8.12), we have:

$$\gamma\left(\frac{c}{h}\right)^{\gamma-1} = \lambda \Rightarrow (\gamma-1)\frac{\dot{z}}{z} = \frac{\dot{\lambda}}{\lambda} = 0 \tag{2.8.12}$$

From equations (2.8.9) to (2.8.12), we have the following three equations:

$$-(\rho+\delta^k) + \alpha x^{\alpha-1} y^{\beta} = 0 \tag{2.8.13}$$

$$-(\rho+\delta^s) + \beta x^{\alpha} y^{\beta-1} + \theta y^{-\theta-1} = 0 \tag{2.8.14}$$

$$-(\rho+y^{-\theta}) + (1-\alpha-\beta)x^{\alpha} y^{\beta} + \frac{(1-\gamma)}{\gamma}(z) - \theta y^{-\theta} = 0 \tag{2.8.15}$$

From equations (2.8.13) and (2.8.14), we obtain an equation of x as a function of y:

$$\frac{\alpha x^{\alpha-1} y^{\beta}}{\beta x^{\alpha} y^{\beta-1}} = \frac{\rho+\delta^k}{\rho+\delta^s-\theta y^{-\theta-1}}$$

$$\Leftrightarrow x = \frac{\alpha y\left[(\rho+\delta^s)-\theta y^{-\theta-1}\right]}{\beta\left(\rho+\delta^k\right)} \tag{2.8.16}$$

Dividing equation (2.8.14) by equation (2.8.15) and rearrange the terms, we have an equation of y and z:

$$\frac{\beta}{(1-\alpha-\beta)y} = \frac{\rho+\delta^s-\theta y^{-\theta-1}}{-\frac{(1-\gamma)}{\gamma}(z)+\rho+(1+\theta)y^{-\theta}}$$

$$\Rightarrow \beta\frac{(1-r)}{r}z = \beta\rho + \beta(1+\theta)\left(\frac{1}{y}\right)^{\theta} - (1-\alpha-\beta)\left[\left(\rho+\delta^s\right)y - \theta\left(\frac{1}{y}\right)^{\theta}\right] \tag{2.8.17}$$

Using equations (2.8.13) to (2.8.15), we are able to get another equation of y and z:

$$-(\rho+y^{-\theta}) + (1-\alpha-\beta)\left\{\frac{\alpha y\left[(\rho+\delta^s)-\theta y^{-\theta-1}\right]}{\beta\left(\rho+\delta^k\right)}\right\}^{\alpha} y^{\beta} + \frac{(1-r)}{r}z - \theta y^{-\theta} = 0$$

$$\Rightarrow \beta\frac{(1-r)}{r}z = \beta\rho + \beta(1+\theta)\left(\frac{1}{y}\right)^{\theta} - (1-\alpha-\beta)\beta\left\{\frac{\alpha y\left[(\rho+\delta^{s})-\theta y^{-\theta-1}\right]}{\beta\left(\rho+\delta^{k}\right)}\right\}^{\alpha} \tag{2.8.18}$$

From equations (2.8.17) and (2.8.18), we have an equation of y only, from which the equilibrium value y^{*} can be generated:

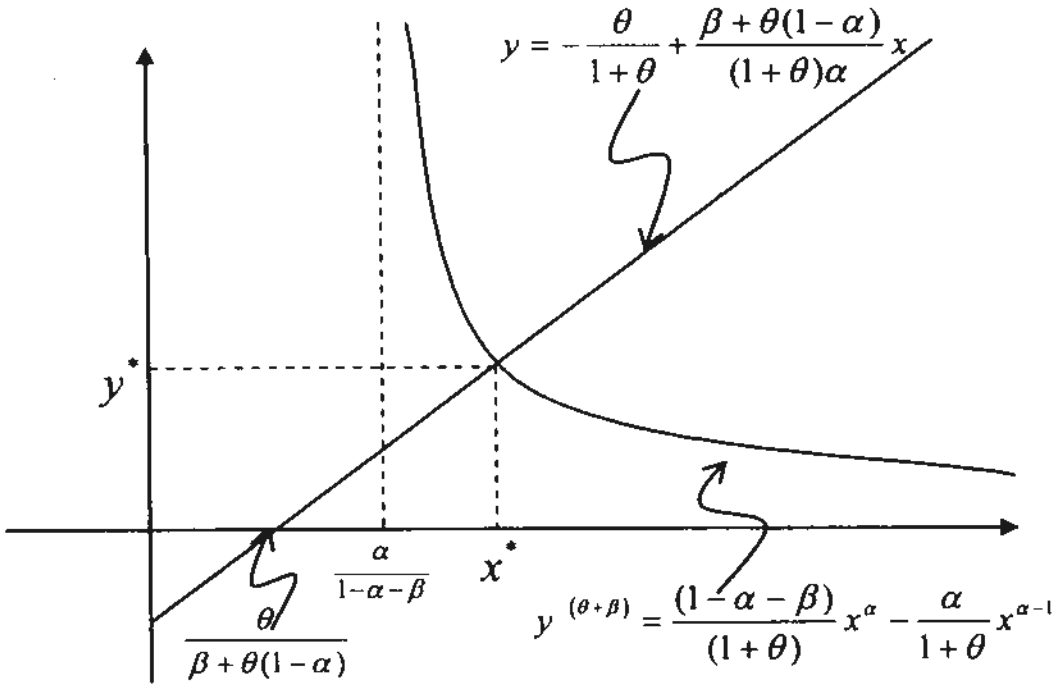
$$\left(\rho+\delta^{s}\right)y-\theta y^{-\theta} = \beta\left\{\frac{\alpha\left[(\rho+\delta^{s})y-\theta y^{-\theta}\right]}{\beta\left(\rho+\delta^{k}\right)}\right\}^{\alpha}$$

$$\Rightarrow \left(\rho+\delta^{s}\right)y^{1+\theta} - \beta\left(\frac{\alpha}{\rho+\delta^{k}}\right)^{\frac{\alpha}{1-\alpha}} y^{\theta} - \theta = 0 \tag{2.8.19}$$

Using the method similar to Appendix 2.2, we are able to find at least one positive root for equation (2.8.19), which indicates that there is equilibrium value of y. The equilibrium value of x and z can be derived from equations (2.8.13) and (2.8.16) respectively.

From the above calculations, we are able to find that there is endogenous growth generated by the model we constructed in this subsection.

Figure 2.1 Dynamics of Barro (1996b)-type model in section 2.3.3



Chapter 3 Health Investment, Development Traps and Economic Growth

3.1 Introduction

In the first essay, we study the effect of health in stimulating economic growth together with education human capital in an endogenous growth framework. From that essay, we are able to identify the role health plays in generating endogenous growth by the following ways: (1) affecting the effective labor and thus the output productivity, (2) entering the utility function, (3) being a core component of health depreciation rate. In the current essay, however, in order to explain the real-world situation that rich countries usually end up with higher capital, better health and higher consumption than the poor ones, we study another important issue related to health, the health related development traps (poverty traps).

Except for the analysis on the determinants of long run economy growth, another stream of research on economic growth is on income distribution. One of the methods to explain the issue of income inequality is the theory of development traps, which are receiving more and more attention after 2000. The issue of health related development traps did not receive much attention until the appearance of a series of findings on this issue from the recent literature. Arcand (2001) argues that health in terms of nutrition can have significant effect directly or indirectly through life expectancy or education on income distribution. Mayer-Foulkes (2002) shows that countries of different development levels can be categorized into two convergent clusters, one with high income and health achievement, another with low income and poor health. The study of health related development traps provides a new direction for the study of economic effect

of health, not just constrained to the growth promoting and poverty relieving effect of health.

The disparity in per capita output between different economies has been documented and explained by a number of papers, such as Alesina (1997), Jones (1997), McGrattan and Schmitz (1998) and Parente and Prescott (2000). In this essay, we start with the presentation of macroeconomic development data from 1960 to 2007. The summary statistics of regional economic performance data are shown in Table 3.1. The most significant fact derived from the data is that except for those countries in East Asia and Pacific areas, the developing countries are not catching up with their developed counterparts. For example, the ratio of income of high income OECD countries to that of the world increased from 3.68 in 1960 to 5.00 in 2007 while the ratio of income of sub-Saharan Africa area to that of the world dropped from 0.18 in 1960 to 0.1 in 2007. In fact, the income gap between the less developed countries (LDCs) and the advanced capitalist nations was widening in the past 50 years. This persistent and widening disparity in observed income levels in different economies can be better illustrated by Table 3.2, where countries are grouped by different income levels. A case in point is the ratios of income relative to world for different income groups which show that the difference between the indices of low income group and high income group widened from 3.49 in 1960 to 4.75 in 2007. Table 3.3 presents annual growth rates of GDP per capita for different income level groups. The statistics in Table 3 indicate that the growth rates of low income group are slower and much more unstable than those of the high income group.

The above facts suggest that consistent with the previous empirical

studies, there exists persistent income disparity among different income groups. Furthermore, our data implies that from 1960 to 2007, this income gap is not only persistent but also widening.[15] These statistical evidences contradict with the traditional convergence theory of economic growth (for example, Solow, 1956; Swan, 1956) which indicates the catching up between low income and high income groups. It is also not consistent with the multiple steady-states mechanism which was used to explain the development traps in recent economic growth literature (Chakraborty, 2004; Bunzel and Qiao, 2005; Hemimi, Tabata and Futagami, 2007). Therefore, it is worthy of constructing one mechanism which can reflect the widening gaps between the rich and the poor. There are multiple sources for the global divergence observed in the statistics in Tables 3.1 to 3.4, such as research and development (R&D), physical capital accumulation, education, and health. Based on these sources, a series of models can be developed using the idea of multiple steady states in income dynamics which generate development traps to explain the phenomena.

Development traps are unfavorable situation, in which an economy ends up with an inferior equilibrium while there may exist better equilibriums. Azariadis (1996, 2001) surveys a class of theoretical explanations for the development traps, where development traps are attributable to several main factors such as misleading governments, incomplete markets, non-convexities and capital mobility. Early studies on multiple steady states focus mainly on development traps generated by physical capital accumulation (Becker and Barro, 1989; Galor and Weil,

[15] Similar findings can be found in Pritchett (1997) and Quah (1993, 1996, 1997), which use shorter time period.

1996), human capital in the form of education (Azariadis and Drazen, 1990; Benabou, 1996; Durlauf, 1993, 1996; Galor and Zeria, 1993; Galor and Tsiddon, 1997), and R&D (Acemoglu, Aghion and Zilibotti, 2002; Howitt and Mayer-Foulkes, 2002). However, as Galor and Mayer-Foulkes (2004) argue that the basic needs of investment in health should be satisfied in order for the investment in education and physical capital to be effective, health is also an important factor in determining the income distribution. Furthermore, the empirical studies of Fogel (1991, 1994a, 1994b), Fogel and Wimmer (1992), and Arora (2001) also corroborate the critical role of health in economic growth. Thus, focus has been turned to the role of health in generating development traps in recent studies as health itself not only has significant impact on economic growth but also influence economic growth through other channels like physical capital accumulation and education.

In this essay, we develop another mechanism to generate development traps. From Table 3.4, we find that health in terms of life expectancy and morbidity is positively related to income per capita. The comparison between income per capita and health indicators for two periods 1960 to 1970 and 1997 to 2007 also indicates that a rise in average per capita income is associated with better health status no matter measured in which health indicator or in which development category. Furthermore, Table 3.4 also implies that wealthier countries are usually associated with healthier people. We focus on the dynamic interaction between health and economic growth in a model of health human capital investment. The health-related development traps give rise to two classes of families, one poorer, less healthy, and unskilled and the other richer, healthier and skillful. By using a

two-period overlapping generations model and the extended Arrow-Romer (Arrow, 1962; Romer, 1986) production function, we analyze the accumulation and interaction of physical capital and health capital and their effect on determining long-run growth. We show that development traps result in twin-peaked distribution, which is consistent with the recent literature on health related poverty traps (e.g. Blackburn and Cipriani, 2002; Chakraborty, 2004; Haaparanta and Puhakka, 2004 and Chakraborty and Das, 2005). Moreover, our study shows that not only the development traps may persist but also the gap between the two classes of countries may even widen despite the assumption of the standard convex technology and preferences.

There also exists a body of literature using overlapping generation models to study the development traps generated by health in terms of mortality, longevity and fertility (Ehrilch and Lui, 1991; Jones, 2001; Kalemli-Ozcan, 2002; Morand, 2004; Lagerlof, 2003; Tamura, 2006). Health is regarded as a prerequisite for sustained economic growth. In a country with low life expectancy and high mortality rate, savings and investment will be lower than what traditional theories predict. Blackburn and Cipriani (2002) construct a three period overlapping generations model where economic and demographic outcomes are determined jointly by a model of longevity, fertility and growth. Chakraborty (2004) uses a two-period overlapping generations model with endogenous mortality to generate development trap. His results suggest that improving longevity and lower mortality risks are beneficial to economic growth. Countries with different levels of human capital will diverge in living standard and mortality may result in development trap. Similar to Chakraborty (2004),

Haaparanta and Puhakka (2004) study development traps by introducing endogenous time preference into an overlapping generations model. Contrary to the Chakraborty (2004) model which links current capital stock to time preference, Haaparanta and Puhakka (2004) determine time preference by optimum savings and investments in patients. Chakraborty and Das (2005) propose a mechanism linking the high intergenerational correlation of economic status and persistent health disparity in a two period overlapping generations model. Hemmi, Tabata and Futagami (2007) analyze the interaction between decisions about financing after-retirement health stocks and precautionary saving motives. The wealth status of an individual determines different saving decisions and this may give rise to multiple steady-state and thus development traps. Osang and Sarkar (2008) consider the lifetime uncertainty in an economy with human capital-led endogenous growth by using an overlapping generations framework. They argue that lifetime uncertainty may affect private decisions on both physical and human capital accumulation and this may be another source to cause income divergence.

In the above papers, the development traps are explained by the mechanism of stable multiple steady-states. However, from the statistical evidence presented in Tables 3.1 to 3.3, the income gap between LDCs and the advanced countries was widening in the sample period. The stable multiple steady-states mechanism may not accord strongly with the empirical evidences, because it is only able to reflect the fact that the countries may be divided into two classes while the widening divergence between the two classes is not able to be reflected in those studies. In this paper, however, we propose another dynamic general equilibrium model of

health human capital, physical capital and growth, in which development traps are generated by the instability of steady states which results in widening difference in economic status of different countries. This mechanism fits the summary statistics of the historical evidence we present in Tables 3.1 to 3.3 and explains the source behind the widening income inequality. It is a good complement to the existing literature on explaining the mechanism of the generation of development traps by health.

This essay is organized as follows. First, a basic theoretical framework is presented in section 3.2. We present a two period overlapping generations model, where consumption and health enter the utility function. An extended Arrow-Romer production function with health investment is also proposed. Section 3.3 shows the competitive equilibrium for the model constructed in section 3.2. The analysis of the aggregate dynamics which shows the widening development traps is given in section 3.4. Concluding remarks appear in section 3.5.

3.2 Theoretical benchmark model

3.2.1 The utility function

The relationship between health and economic growth can be understood in a two-direction way. Firstly, health investment can affect economic growth[16]. Higher level of health is usually associated with higher productivity and this will lead to higher economic growth (Barro, 1996a, 1996b; Strauss and Thomas, 1998 and Bloom et.al, 2004). For example, health can affect economic growth through mortality, fertility, longevity and

[16] Zon and Muysken (2001, 2003) discussed the macroeconomic effect of health capital derived from investment in health on economic growth. It argued that there are generally four channels that health influence economic growth: 1) better health is beneficial to the accumulation of education human capital; 2) the consumption of health service increases an agent's utility; 3) improvement in health level increases the labor productivity; 4) higher health status is accompanied with increment in longevity and an ageing population.

many other channels (Kalemli-Ozcan, Ryder and Weil, 2000; Kalemli-Ozcan, 2002, 2003 and Morand, 2004). Health can also promote economic growth through another form of human capital, education human capital. In return, however, economic growth is also able to encourage the growth of health. Grossman (1972) argues that higher rate of economic growth results in higher wage rate and an agent with higher salary is able to invest more in medical care and cure measures. Furthermore, agents with higher income are also associated with higher consumptions and nutrition intakes, which is another channel to improve health that is widely recognized by the existing literature (Strauss and Thomas, 1998; Thomas, 2001; Fogel, 1994a, 1994b, and 2002). In this essay, we concentrate on both the direct and indirect benefits that health provides to individual consumers and then affects economic growth. On one hand, health is widely regarded by the health literature as one form of utility besides consumption. One pioneer work is Grossman (1972) which argues that health can be viewed as a kind of durable capital stock that can generate output in the form of "healthy time" and using health services is also another form of consumption. Health capital is different from other kind of human capital, like education, because education only affects an agent's ability to produce final consumption goods or to earn money. In contrast, the stock of health determines the amount of "healthy time" an agent is able to provide to produce the final goods or to earn the money. Health investment can improve the health status of an agent and thus improve the agent's utility, which is the direct effect of health investment. On the other hand, if an agent has better health, this agent is able to work for a longer time and more productive, which will improve the efficient labor productivity. When the

agent can provide more effective labor, this agent will normally receive higher salary and then spend more. More consumption will improve one's utility. This is the indirect channel that health affects an agent's utility, which is achieved by improving an agent's effective labor and then consumption.

We assume that an agent lives for two periods, young and old and the agent only works when young. An agent makes consumption in both young and old periods and the consumption in these two periods affects the agent's utility. In general, there are two kinds of health investments. One is health care measures and the other one is health cure measures. We stress in this essay the important role of investment in health care measures plays in affecting the health utility while the role health cure measures plays in the determination of an agent's utility is reflected through the indirect effect of health. Therefore, for health capital stock, we assume that only old age health enters the utility function, because the direct utility of better health is not obvious when an agent is young. For example, even if a young agent is ill, this agent can recover much quicker than an old one and the direct utility from better health is not that obvious. What really matter for a young agent is the indirect effect of health, such as the amount of effective labor he/she is able to provide. The healthy hours that an agent is able to offer would directly determine the agent's income. In contrast, the direct effect of health on an agent's utility is more obvious to an old agent. When an agent is old, this agent is retired from work. This agent uses the saving from the young period to support the old age consumption. There is no indirect effect of health from effective labor for this old agent. When an old agent is ill, the agent usually needs much more time to recover and the chance of a

particular illness to threat an agent's life is also much higher. As a result, an old agent is more likely to invest in health care (or health precautionary) measures. Investment in health precautionary measures can lower the probability of getting ill and thus improve an agent's life expectancy. Therefore, the agents are interested in the investment in precautionary measures. The consumption of health services is related mainly to health precautionary measures that can generate direct benefits and thus affect an old agent's utility.[17]

For simplicity, we also assume logarithm felicity utility functions. Based on the two channels that health affects one's utility, we have the following utility function for a generation-t agent's expected life-time utility:

$$U_t = \ln c_{t,t} + \beta\left(\ln c_{t,t+1} + \ln h_{t,t+1}\right) \tag{3.1}$$

where the parameter β represents the survival probability from young to old; c_t denotes the consumption of an generation-t agent and h_t denotes a generation-t agent's health capital.

In what follows, we omit the generation subscripts to avoid clustered notations. Let us denote, for example, $\ln c_t = \ln c_{t,t}$, $\ln h_t = \ln h_{t,t}$, $\ln c_{t+1} = \ln c_{t,t+1}$, and $\ln h_{t+1} = \ln h_{t,t+1}$.

An agent offers a unit of labor and obtains both labor income and capital income. Since the better an agent's health status is, the more effective labor the agent can offer and the higher wage an agent is able to

[17] Of course, one may argue that investment in health cure measures can save an agent's life when the agent gets a serious illness and this can also generate direct utility. What we want to stress in this essay is that investment in health precautionary measures is what an agent is more likely to encounter. An investment in health precautionary measures is what the agent can control and the utility generated from investment in these measures is more stable and obvious.

obtain, we assume that both the effective labor and the wage rate are functions of health h. In other words, an agent with health h offers $f(h)$ effective labor and the agent's wage rate is $w(h)$. On the other hand, in order to achieve a health level of h, an agent needs to invest $T(h)$. After making the previous assumptions, we arrive at the following equation of a particular agent's budget constraints:

$$c_t + T(h_t) + s_t = w(h_t) \tag{3.2}$$

$$c_{t+1} = (1 + r_t)s_t \tag{3.3}$$

Where s_t denotes a generation-t agent's saving when young and r_t is the interest rate in time t.

An agent's optimal problem is as given below:

$$\begin{aligned} &\max_{c,h} U_t(c_t, c_{t+1}, h_{t+1}) \\ &s.t.: c_t + T(h_t) + s_t = w(h_t) \\ &\qquad c_{t+1} = (1 + r_{t+1})s_t \end{aligned} \tag{3.4}$$

3.2.2 Production function

We assume an Arrow-Romer production function as given below:

$$Y_t = A(K_t)^{\alpha}(E_t L_t)^{1-\alpha} \tag{3.5}$$

where Y denotes the total output and K denotes the total physical capital input.

E denotes the efficient labor parameter, which is proportional to the per capita capital:

$$E_t = \frac{K_t}{N_t} = \bar{k} \tag{3.6}$$

where N_t denotes the amount of total population. For a more detailed description of the model and justification of this assumption, see Barro and

Sala-i-Martin (2005).

L_t denotes the total efficient labor input and it is proportional to an agent's health investment:

$$L_t = f(h)N_t \tag{3.7}$$

where $f(.)$ is the efficient labor production function of health as mentioned before and the total population is N_t. To simplify our analysis, we assume that N_t is constant in each period and there is no population growth. For further simplification, we assume that there is no population growth and N can be standardized to one. The multiplication of E and L stands for the total effective Labor.

From equations (3.5) to (3.7), we arrive at the intensive form of the production function:

$$y_t = Y_t/N = A(f(h_t)\bar{k})^{1-\alpha}(k_t)^{\alpha} \tag{3.8}$$

In the competitive equilibrium, all the agents will have the same per capita capital. Furthermore, the assumption of profit maximizing firms and the factor market clearing condition indicate that both the rate of returns of physical capital and wage rates are equal to their respective marginal productivities:

$$r_t = \alpha A\phi(h_t) \tag{3.9}$$

$$w_t = (1-\alpha)\phi(h_t)Ak_t \tag{3.10}$$

where $\phi(h_t) = (f(h_t))^{1-\alpha}$ and we assume that $\phi'(h_t) > 0$, $\phi''(h_t) < 0$, $\lim_{h_t \to 0}\phi'(h_t) = +\infty$, $\lim_{h_t \to \infty}\phi'(h_t) = 0$, $\phi(0) = 0$ and $\lim_{h_t \to \infty}\phi(h_t) = \bar{\phi} > 0$.

3.3 Competitive equilibrium

The representative agent's optimization problem can be written in the

following indirect form by substituting the constraints into the utility function:

$$L = \ln(w(h_t) - T(h_t) - s_t) + \beta\{\ln[(1 + r_{t+1})s_t] + \ln h_{t+1}\} \tag{3.11}$$

Solving the above Hamiltonian function, we have the following two first order conditions with respect to saving and health, respectively:

$$\frac{1}{w(h_t) - T(h_t) - s_t} = \frac{\beta}{s_t} \tag{3.12}$$

$$\frac{w'(h_t) - T'(h_t)}{w(h_t) - T(h_t) - s_t} = -\frac{\beta}{h_t} \tag{3.13}$$

From equations (3.12) and (3.13), we can get the following two equations:

$$s_t = \beta\left(w(h_t) - T(h_t) - s_t\right) \tag{3.12'}$$

$$((\beta + 1)(T'(h_t)h_t - w'(h_t)h_t) = \beta(w(h_t) - T(h_t)) \tag{3.13'}$$

We assume that the elasticity of health cost $T(h)$ with respect to health is ε_T and the elasticity of wage rate with respect to health is ε_ϕ. That is:

$$\varepsilon_T = \frac{T'(h_t)h_t}{T(h_t)} \tag{3.14}$$

$$\varepsilon_\phi = \frac{w'(h_t)h_t}{w(h_t)} \tag{3.15}$$

Then, by equations (3.12'), (3.13'), (3.14) and (3.15), we have the following two equilibrium levels of s and $T(h)$ as a function of h and k:

$$s^* = \frac{\beta(\varepsilon_T - \varepsilon_\phi)}{(\beta + 1)\varepsilon_T + \beta} w(h^*) \tag{3.16}$$

$$T(h^*) = (1 - \alpha)\phi(h^*)Ak^* \frac{(\beta + 1)\varepsilon_\phi + \beta}{(\beta + 1)\varepsilon_T + \beta} \tag{3.17}$$

As shown in equation (3.16), the saving rate of an agent depends on the two health elasticities (ε_T and ε_ϕ), the wage rate (w) and the survival probability from young to old (β). Firstly, the patience of an agent is positively related to the survival probability. Thus, the higher the probability of surviving from young to old, the higher amount of income an agent is

willing to save for old. Secondly, if we hold other factors constant, the larger the differences between the health cost and the health production elasticities, the higher the saving rate. The intuition behind this relationship can be interpreted in this way: the larger the differences between the cost of increasing the health level of an agent by one unit and the benefits from the increment of one unit of effective labor, the more willingness the young agent is to save more money for the old period, because it is less attractive to invest in health. Lastly, if the two elasticities are constant, the higher the wage rate, the higher the saving rate. This implies that the better the health status of an agent, the higher wage rate and the higher chance he/she prefers to save than consume in his/her young age.

Equation (3.17) is the equilibrium level of health expenditure. From this equation, we find that if the probability of surviving from young to old is increased, the amount of health expenditure to maintain the current level of health will decrease. In contrast, the higher the amount of effective labor per unit increment of health, which is indicated by $\phi(h)$, the higher cost of maintaining the current level of health. Similarly, the higher the elasticity of output to effective labor, the higher the health expenditure, as indicated by the parameter $(1-\alpha)$. The intuition behind these causality relationships is that higher $\phi(h)$ would lead to higher labor productivity and thus higher wage rate, which results in the increment of cost to maintain the current level of health, because those jobs with higher payment are usually associated with higher pressure, more competition, and longer working hours, which require more investment in health in order to keep a health body. The same logic applies to the rise in the elasticity of output to effective labor.

From equations (3.16) and (3.17), we derive the equilibrium ratio of total cost to health cost:

$$\frac{T^*(h)+c^*}{T^*(h)} = 1 + \frac{\varepsilon_T - \varepsilon_\phi}{\beta + \varepsilon_\phi + \beta\varepsilon_\phi} \tag{3.18}$$

Because the two elasticity ε_T and ε_ϕ are positive, Equation (3.18) implies that the difference between the two health elasticities $(\varepsilon_T - \varepsilon_\phi)$ is also positive. That is, $\varepsilon_T > \varepsilon_\phi$.

Equation (3.18) implies that the higher the survival probability, the higher the share of health expenditure in total expenditure. We already know that health expenditure is negatively related to survival probability. The positive relationship between survival probability and the ratio of health expenditure to total expenditure indicates that when a young agent has a higher probability to survive to old age, this agent would be more patient and incline to consume less to save for future investment. The decline in consumption is more than that of health expenditure. Furthermore, the larger the difference between the two health elasticities, the less willingness is for agents to spend on health. The technical details of the derivation of the equations (3.16) to (3.18) are available in Appendix 3.1.

Moreover, from equations (3.8) and (3.17), we obtain the equilibrium ratio of health expenditure to total output:

$$\frac{T^*(h)}{y^*} = (1-\alpha)\frac{(\beta+1)\varepsilon_\phi + \beta}{(\beta+1)\varepsilon_T + \beta} \tag{3.19}$$

Equation (3.19) shows that the higher the survival probability, the lower the health expenditure to output. Considering that we obtain in equation (3.18) that the higher the survival probability, the less spending on

health expenditure and consumption, we know that in equilibrium as the survival probability increases, an agent intends to save more for old age but spend less on health and consumption. Moreover, the larger the difference between the health cost elasticity and health benefit elasticity, the less possibility for an agent to invest in health, which is indicated by the lower ratio of health expenditure to output.

Theorem 3.1: a) Holding other factors constant, an increase in ε_T leads to a decrease in the ratio of health expenditure to total expenditure and the ratio of health expenditure to total output while an increase in ε_ϕ will increase the ratio of health expenditure to total expenditure and the ratio of health expenditure to total output.

b) Holding other factors constant, an increase in the difference between the two health elasticities or in the survival probability leads to increment of the ratio of health expenditure to total expenditure but decrease in the ratio of health expenditure to output.

c) If either $\frac{d\varepsilon_T}{dh} < 0$ or $\frac{d\varepsilon_\phi}{dh} > 0$ or $\frac{d\left(\varepsilon_T - \varepsilon_\phi\right)}{dh} < 0$, then with the health level increase, the ratio of health expend to total expenditure will increase. In contrast, if either $\frac{d\varepsilon_T}{dh} > 0$, $\frac{d\varepsilon_\phi}{dh} < 0$ or $\frac{d\left(\varepsilon_T - \varepsilon_\phi\right)}{dh} > 0$, then with the health level increase, the ratio of health expend to total expenditure will decrease.

d) If either $\frac{d\varepsilon_T}{dh} < 0$ or $\frac{d\varepsilon_\phi}{dh} > 0$, the ratio of health expenditure to total output will increase as the health level increase. In contrast, if either $\frac{d\varepsilon_T}{dh} > 0$ and $\frac{d\varepsilon_\phi}{dh} < 0$, the ratio of health expenditure to total output will

decrease as the health level increase.

We can regard ε_T and ε_ϕ as the health cost elasticity and health production elasticity, respectively. Theorem 3.1.a indicates that higher health cost elasticity and lower health production elasticity induce lower health expenditure to total expenditure and lower health expenditure to total output, vice versa. In the real world, the less developed countries usually do not have well established public medical care systems and social welfare protection schemes. The marginal cost of increasing the health status of an agent is very high. Most of the people in those countries work to earn a salary that is just able to satisfy their basic needs and only a very low portion of their salary can be used in health. Moreover, the healthy time they are able to provide does not have high marginal return because what they are able to do is usually labor intensive, low technology work usually with low wage rate. As a result, in those less developed countries, because of the relatively higher health cost elasticity and lower health benefit elasticity, most of the income they receive has gone to the survival needs and these people tend to invest less in health. By contrast, there are well organized social health protection systems in the developed countries financed mainly by the government subsidies. Therefore, the marginal cost of becoming healthier is lower. Moreover, there are much more job opportunities in those developed countries. The benefits from being able to work for a longer time and more efficiently are obvious and this determines that people in the developed countries, which are associated with lower health cost elasticity but higher health productivity elasticity, tend to invest more in health.

Consequently, the ratio of health expenditure to output is higher in the developed countries than in the developing countries. This relationship is corroborated by the statistics we calculated from World Development Indicators 2008, in which we find that the average ratio of public health expenditure to GDP is 8.88 percent from 1971 to 2005 for North America compared to 4.92 percent of South Asia for the same period.

Theorem 3.1b has been explained in the previous part of this section and we turn our focus on the last two sub-Theorems. Theorems 3.1c and 3.1d are very intuitive. They show that if higher health leads to lower health cost elasticity but higher health benefit elasticity, people incline to invest more in health. Consistent with Theorem 1a, Theorems 3.1c and 3.1d show that the divergence in health status between the less developed countries and the developed countries may attribute to the unfavorable situation in the less developed countries and the favorable situation in the developed countries. For example, in the developed countries, when people are healthier, more effective labor is available, which induces higher productivity and in turn more investment in medical related researches. The development in medical area will lower the health cost elasticity and it will also increase the health benefit elasticity because more healthy time is available to other production area. In the less developed countries, however, one may need to walk several hours in order to see a doctor while there are district clinics near your home in the developed countries. To be healthier, the health cost elasticity of people in the less developed areas is much higher while being healthier may not significantly increases their income due to their low education level, which gives less impetus to invest in health. The vicious health investment cycle in the less developed countries and the relatively

favorable situation in the developed countries generate a channel through which the health related development traps may appear.

3.4. Dynamics of the aggregate economy

3.4.1 Analysis of the dynamics of capital per capita

In section 3.3, we have obtained the equilibrium levels of the key variables from the optimization process, which enable us to derive the possible causality relationships that may cause the health related development traps. In this section, based on the optimization results derived in section 3.3, we prove the existence of such a health related development traps. In order to derive the dynamic equations of the per capita physical capital, we have to define the specific forms of health cost and health production functions. In general, we have to invest more in order to maintain a higher level of health and better health status would result in more effective labor. Therefore, we have the following two assumptions:

$$T(h_t) = h_t^{\theta} \tag{3.20}$$

$$\phi(h_t) = h_t^{\sigma} \tag{3.21}$$

where $\theta > 0$ and $\sigma > 0$. Inserting these two specific forms of health cost function and effective labor production function into equations (3.14) and (3.15), we show that the two elasticities are determined only by θ or σ:

$$\varepsilon_T = \theta \tag{3.22}$$

$$\varepsilon_\phi = \sigma \tag{3.23}$$

Moreover, from equation (3.18), we know that $\varepsilon_T > \varepsilon_\phi$, which implies that $\theta > \sigma$.

Inserting equations (3.20) to (3.23) into equations (3.10) and (3.17), we

are able to derive the equilibrium level of health level h:

$$h_t^{\theta-\sigma} = \frac{T(h_t)}{\phi(h_t)} = (1-\alpha)Ak_t \frac{(\beta+1)\varepsilon_\phi + \beta}{(\beta+1)\varepsilon_T + \beta}$$

$$\Leftrightarrow h^* = \left[(1-\alpha)Ak^* \frac{(\beta+1)\theta+\beta}{(\beta+1)\sigma+\beta}\right]^{\frac{1}{\theta-\sigma}} \tag{3.24}$$

Equation (3.24) is the equilibrium level of health human capital. Because $\theta > \sigma$, we know that h^* is positively related to technology parameter A, per capita physical capital k, the output elasticity of health $(1-\alpha)$ and the probability of survival from young to old β, all of which are consistent with our expectation. According to equation (3.24), an economy with higher development level, which is indicated by its advanced technology, higher level of capital accumulation and longer life expectancy, is associated with higher equilibrium level of health.

From equations (3.8) and (3.24), we obtain the equilibrium ratio of health capital to total output:

$$\begin{aligned}\frac{h^*}{y^*} &= \frac{\left[(1-\alpha)\frac{(\beta+1)\theta+\beta}{(\beta+1)\sigma+\beta}\right]^{\frac{1}{\theta-\sigma}}}{Ak^*h^{*\sigma}} \\ &= \left[(1-\alpha)\frac{(\beta+1)\theta+\beta}{(\beta+1)\sigma+\beta}\right]^{\frac{1-\sigma}{\theta-\sigma}} (Ak^*)^{\frac{1-\sigma}{\theta-\sigma}}\end{aligned} \tag{3.25}$$

Equation (3.25) implies that the higher the technological parameter A, the higher the ratio of health capital to total output. Inadequate social infrastructure and inefficient policies, which reduce A, could reduce the health-output ratio. Equation (3.25) also indicates that the higher the capital per capita, the higher the ratio of health capital to total output. Moreover, the higher the elasticity of output with respect to health capital[18], the higher the

[18] In this case, this elasticity $\varepsilon_{yh} = (1-\alpha)\theta$.

ratio of health capital to total output. The developed countries usually have more advanced technologies, higher capital per capita and elasticities, thus equation (3.25) implies that as the economy develops, health accounts for a more and more important role.

Since $K_t = s_{t-1}L_{t-1}$ and we assume that there is no population growth, we have the following dynamic equation of per capita physical capital:

$$k_t = s_{t-1} = \frac{\beta(\varepsilon_T - \varepsilon_\phi)}{(\beta+1)\varepsilon_T + \beta} w(h_{t-1})$$

$$= \frac{\beta(\varepsilon_T - \varepsilon_\phi)}{(\beta+1)\varepsilon_T + \beta}(1-\alpha)\phi(h_{t-1})Ak_{t-1}$$

$$= \frac{\beta(\varepsilon_T - \varepsilon_\phi)}{(\beta+1)\varepsilon_T + \beta}(1-\alpha)Ak_{t-1}\left[(1-\alpha)Ak_{t-1}\frac{(\beta+1)\varepsilon_\phi + \beta}{(\beta+1)\varepsilon_T + \beta}\right]^{\frac{\sigma}{\theta-\sigma}}$$

$$= \left[\frac{(1-\alpha)A}{(\beta+1)\theta+\beta}\right]^{\frac{\theta}{\theta-\sigma}} [(\beta+1)\sigma+\beta]^{\frac{\sigma}{\theta-\sigma}} \beta(\theta-\sigma)k_{t-1}^{\frac{\theta}{\theta-\sigma}} \qquad (3.26)$$

The dynamic equation of per capita physical capital (equation (3.26)) indicates a non-stable growth path, which is shown in Figure 3.1. If an economy starts out with a high enough capital stock above the instable equilibrium steady state k^*, it will grow persistently. By contrast, if an economy starts out below the instable equilibrium it would not be able to escape the vicious cycle of poverty and poor health because the negative growth rate of the economy will lead to persistent low income and the economy would end up with an inferior equilibrium. Due to pervasive poverty, neither can this economy afford to invest in the much needed public health system, nor can an agent in this kind of economy save and invest in other areas required for the development of this economy. Low investment rate causes consistently low income and poorer health. The low income and poor health interact with each other, which contributes to the even wider

gaps between the developed countries and the developing countries. On one hand, this result is consistent with the recent literature (Blackburn and Cipriani 2002; Chakraborty 2004; Haaparanta and Puhakka 2004 and Chakraborty and Das 2005) that there may be development traps existing when the development of an economy is determined by health. On the other hand, although our model is another mechanism to generate health related development traps, the development traps generated by our model is different from those of the recent literature. In the models of the recent literature, the development traps are derived from the existence of multiple steady states. Thus, countries with different initial capital per capita may end up with different steady states and the gap between any two steady states is constant. For the mechanism constructed in this essay, the most obvious characteristic of our model is that our model indicates that the positive steady state is unstable and an economy that starts out above the equilibrium level k^* will grow at a positive rate while an economic with an initial level of capital stock lower than k^* will grow at a negative rate (See Theorem 3.2 below). Under this scenario, the rich countries tend to be richer and healthier and the poor countries suffer from the development traps and be poorer and with lower health level. The widening gap between the poor and rich countries is what the implication of our model different from those recent literatures. The scenario proposed in our model seems more consistent with what is happening in the real world, since in the real world, countries with different initial level of capital per capita will grow in different rates and the country differences is widening, which is also evidenced by the facts presented in the introduction.

Equation (3.26) also indicates that the an economy will be more likely

to fall into the development trap if the technology level A in this economy is lower; the elasticity of output with respect to health $(1-\alpha)$ is lower, and the lower the probability of surviving from young to old (i.e., β).

Theorem 3.2: Under the mechanism constructed in this essay, the economy has an unstable equilibrium steady state. When the per capita capital is greater than the equilibrium level, the economy will grow persistently. In contrast, if per capita capital is less than the equilibrium level, the economy can retrogress persistently.

(Proof: See appendix 3.2)

The existence of the health related development traps has important policy implications. Our model shows that as long as an economy starts with an initial value of per capita capital lower than the unstable equilibrium, this economy would converge to the inferior equilibrium, which is a situation of the development traps. To explain from an individual level, if the wage of an unskilled agent is too low for his/ her family to satisfy the basic needs of his/ her children's health and education development and the public provision is also inadequate, his/ her children would stay in poverty and this vicious cycle would continue. The confirmation of the existence of health related development traps is consistent with early studies such as Chakraborty (2004), Haaparanta and Puhakka (2004), Charkraborty and Das (2005) and Mayer-Foulkes (2008). However, our paper not only stresses the crucial role of health in generating development traps, but also suggests that if the developing countries trapped in the inferior equilibrium are not able to break this cycle of poverty, the gap between them and the developed countries

would keep widening. To assist those entrapped developing countries to go out of the vicious cycle, we suggest that the policymakers in those less developed countries should provide sufficient support for public health system. To eliminate poverty traps, governments in the developing countries should provide free or at least affordable public health services to the poor, which would at least help to satisfy the basic health requirement of the children in poor families. Galor and Mayer-Foulkes (2004) argue that early child development is essential to education performance. The provision of sufficient public health services would also be conductive to counteract the inadequate investment in health due to low salary or credit constraints. Furthermore, the health status of people in developing countries could be improved if more public health expenditure is invested in projects providing better sanitation, hygiene, more hospitals, higher ratios of doctors to patients and beds to patients. Last but not least, the efficiency of the public health system is also critical to the elimination of poverty traps. World Bank (1993) finds that public health expenditure is often spent on the relatively more expensive disease treatments that mainly benefit the wealthy. To increase the efficiency of the public health system, government policymakers should first consider the satisfaction of the basic needs of health rather than the quality of life. One possible source of low health status in the developing countries is the inadequate information on the benefits of better health and the measures to effectively improve personal health. Therefore, it is beneficial to set up more schools, especially primary schools, to provide fundamental education to the children of the poor and equip them with basic knowledge of health. Students with better knowledge could have higher chances of getting a job with higher salary, which would induce an agent to

be more patient to invest in health. Better health would then be beneficial to education.

3.4.2 A general form of health utility function

Although the logarithm utility function is the standard form of utility function in the OLG models analyzing the development traps[19], we still want to see whether the specific form of health utility function may affect the results derived in the previous sections. We know that the logarithm form of the health utility function is a special form of the constant absolute rise aversion (CARA) utility function and we can loose the logarithm form of the utility function of health to a general form $g(h)$, where $g'(h) > 0$ and $g''(h) < 0$. In the following, we will specify a specific CARA health utility function to check the robustness of the development traps derived in the previous sections to this general form utility function. Therefore, the agent's maximization problem is as follow:

$$\max_{c,h} U = \ln c_t + \beta(\ln c_{t+1} + g(h_{t+1})) \tag{3.27}$$

$$s.t.: c_t + T(h_t) + s_t = w(h_t) \tag{3.28}$$

$$c_t = (1 + r_{t+1})s_t \tag{3.29}$$

Using similar procedures when we assume the logarithm utility function, we get the corresponding expressions for s_t and $T(h_t)$:

$$s_t = \frac{\beta w(h_t)(\varepsilon_T - \varepsilon_\phi)}{(1+\beta)\varepsilon_T + \beta g'(h_t)h_t} \tag{3.16'}$$

$$T(h_t) = \frac{[(1+\beta)\varepsilon_\phi + \beta g'(h_t)h_t]}{[(1+\beta)\varepsilon_T + \beta g'(h_t)h_t]} w(h_t) \tag{3.17'}$$

From equations (3.27) to (3.29), we have:

[19] See the recent papers analyzing health related development traps like Blackburn and Cipriani (2002), Chakraborty (2004), Bunzel and Qiao (2005) and Hemimi, Tabata and Futagami (2007).

$$\frac{T(h_t)+c_t}{T(h_t)} = 1+\frac{\varepsilon_T-\varepsilon_\phi}{(1+\beta)\varepsilon_\phi+\beta g'(h_t)h_t} \tag{3.18'}$$

$$\frac{T(h_t)}{y_t} = (1-\alpha)\frac{(\beta+1)\varepsilon_\phi+\beta g'(h_t)h_t}{(\beta+1)\varepsilon_T+\beta g'(h_t)h_t} \tag{3.19'}$$

Theorem 3.3: a) If ε_T and ε_ϕ are constants, and assuming that $u(c,h) = \ln c_{t,t} + \beta\left(\ln c_{t,t+1} + g(h_{t+1})\right)$, then when the health level increase, the ratio of health expend to total expend will increase if $\left|\frac{\partial g'(h_t)}{\partial h_t}\right| < 1$.

b) The threshold level of capital per capita will decrease when the health level increase if $\left|\frac{\partial g'(h_t)}{\partial h_t}\right| > 1$.

(Proof: See appendix 3.3)

Next, we turn to the dynamics of the aggregate economy. Assuming that the health cost function and health production function follow the same specific forms defined in section 3.4.1, then the corresponding health to output ratio is:

$$\frac{h_t}{y_t} = \left[(1-\alpha)\frac{(\beta+1)\theta+\beta g'(h_t)h_t}{(\beta+1)\sigma+\beta g'(h_t)h_t}\right]^{\frac{1-\sigma}{\theta-\sigma}} (Ak_t)^{\frac{1-\sigma}{\theta-\sigma}} \tag{3.25'}$$

The generalization of health utility function does not change the main findings presented in section 3.2.4.

The dynamic equation of per capita capital for the case of general health utility function is:

$$k_t = s_{t-1} = \frac{\beta(\varepsilon_T-\varepsilon_\phi)}{(\beta+1)\varepsilon_T+\beta g'(h)h} w(h_{t-1})$$

$$= \frac{\beta(\varepsilon_T - \varepsilon_\phi)}{(\beta+1)\varepsilon_T + \beta g'(h)h}(1-\alpha)Ak_{t-1}\left[(1-\alpha)Ak_{t-1}\frac{(\beta+1)\varepsilon_\phi + \beta g'(h)h}{(\beta+1)\varepsilon_T + \beta g'(h)h}\right]^{\frac{\sigma}{\theta-\sigma}}$$

$$= \left[\frac{(1-\alpha)A}{(\beta+1)\theta + \beta g'(h)h}\right]^{\frac{\theta}{\theta-\sigma}}\left[(\beta+1)\sigma + \beta g'(h)h\right]^{\frac{\sigma}{\theta-\sigma}}\beta(\theta-\sigma)k_{t-1}^{\frac{\theta}{\theta-\sigma}} \quad (3.26')$$

Different from the situation when we assume that $g(h) = \ln h$, the development traps can not be derived from equation (3.26') because the coefficient of k_{t-1} now is affected by the dynamics of health and we are not able to judge whether there exists development traps. To solve this problem, we have to consider simultaneously the dynamic of both k_t and h_t and linearize the two dynamic equations around the steady point to check the stability of the equilibriums. We assume the health utility function to be the following CARA form utility function:

$$g(h_t) = \frac{h_t^\gamma}{\gamma} \quad (3.30)$$

where $0 < \gamma < 1$ and if $\gamma = 0$, the utility function turns to the logarithm form like the one we use in the previous two sections. Substituting equation (3.30) into equation (3.27) and based on similar maximization process, we obtain two dynamic equations of k_t and h_t:

$$h_t^{\theta-\sigma}\left(\theta + bh_t^\gamma\right) = (1-\alpha)k_t\left(\sigma + bh_t^\gamma\right) \quad (3.31)$$

$$k_{t+1} = b\left[(1-\alpha)k_t h_t^\sigma - h_t^\theta\right] \quad (3.32)$$

In the equilibrium, we have $k_{t+1} = k_t = k^*$ and $h_{t+1} = h_t = h^*$. We have proved in Appendix 3.3 that in equilibrium there are two equilibrium points. In order to check the robustness of the widening development traps we generated in the previous sections, we need to check the stability of the equilibrium points. We linearize equations (3.31) and (3.32) around the

equilibrium point (k^*, h^*):

$$\begin{aligned}&\left[\theta(\theta-\sigma)h_t^{\theta-\sigma-1}+b(\theta+\gamma-\sigma)h_t^{\theta+\gamma-\sigma-1}-b(1-\alpha)k_t\gamma h_t^{\gamma-1}\right](h_t-h^*)\\&=\left[(1-\alpha)\sigma+b(1-\alpha)h_t^{\gamma}\right](k_t-k^*)\end{aligned} \tag{3.33}$$

$$k_{t+1}-k^*=\left[b(1-\alpha)k_t\sigma h_t^{\sigma-1}-b\theta h_t^{\theta-1}\right](h_t-h^*)+b(1-\alpha)h_t^{\sigma}(k_t-k^*) \tag{3.34}$$

From equations (3.33) and (3.34), we get the dynamic equation of k_t:

$$k_{t+1}-k^*=\Omega(k_t-k^*) \tag{3.35}$$

where

$$\Omega=b(1-\alpha)\left\{\frac{\left[(1-\alpha)k^*\sigma h^{*\sigma}-\theta h^{*\theta}\right]\left(\sigma+bh^{*\gamma}\right)}{\theta(\theta-\sigma)h^{*\theta-\sigma}+b(\theta+\gamma-\sigma)h^{*\theta+\gamma-\sigma}-b(1-\alpha)k^*\gamma h^{*\gamma}}+h^{*\sigma}\right\}$$

The stability of k_t around the equilibrium can be checked from equation (3.35). If $\Omega>1$, the equilibrium point is unstable, which is consistent with that of the special case with logarithm utility function. Because it is not able to prove explicitly $\Omega>1$, we have to use numerical method to prove it. We follow the assumptions of this essay[20] such that $\theta=1.25$, $\sigma=0.75$, $\gamma=0.25$, $b=$ 0.25 and $\alpha=\frac{1}{3}$.[21] Firstly we use Mathlab program to solve equations (3.31) and (3.32) for the equilibrium value of k and h. Secondly, we insert the equilibrium values of k and h together with the assumed values of the parameters stated above into Ω and we prove that $\Omega>1$. Thus, we prove that we are able to generate the same development traps as the one in section 3.4.1. In other words, the

[20] In this paper, we derive that $\theta>1>\sigma>0$. Furthermore, we know from the definitions that $0<\gamma<1$ and $0<b<0.5$. We follow Mankiw, Romer and Weil (1992) to assume that $\alpha=\frac{1}{3}$.

[21] We have also tried other values of the parameters and still able to prove the unstable equilibrium point.

development traps we generate in section 3.4.1 are not sensitive to the specific form of health utility function. Detailed proof of the unstable equilibrium point of this subsection can be found in Appendix 3.4.

3.5. Concluding Remarks

The persistence of international income differences has long been attracting interest from researchers. Graham and Temple (2003) argue that development traps is one of the significant mechanisms offered to explain the persistent differences in income. The sources of development traps are multiple. There are some empirical and theoretical studies analyzing the mechanism of the generation of development traps from different sources. In this essay, we develop another mechanism to generate development traps by constructing an overlapping generations model to analyze the health related development traps. The economic effects of health on economic growth have been well recognized in the historical studies, empirical analyses as well as in the first essay of this thesis while the role health plays in generating development traps has not been studied in depth in empirical and theoretical literature. There is no theoretical study analyzing this issue until after 2000 and most of the mechanisms proposed in those recent studies generate multiple stable steady states where poor countries usually end up with an inferior equilibrium although better equilibrium is available. The health related development traps generated in this essay is similar but different from those of the early studies: although there are also multiple steady states generated in our model, only one of the equilibriums in our models is stable and this equilibrium is an inferior equilibrium. In contrast, countries with initial capital level higher than the unstable equilibrium

would growth persistently. This mechanism reflects that the gap between the rich and poor countries are widening, which is consistent with the historical facts presented in Tables 3.1 to 3.4.

Agents in our model live for two periods: young and old but they only work when young. The health status of the young agents determines the amount of healthy working hours they are able to offer which will affect the labor productivity directly. Furthermore, we notice that young agents can recover from illness in much less time than their old counterparts so the direct utility for being healthy is not obvious for young agents. Therefore, the model we constructed in this essay is the one with health entering both the utility function and the production function. In particular, we consider only the old age utility of health while the utility from improvement of the health status of young agents is reflected indirectly from the increment in labor productivity and thus higher wages which lead to higher consumption.

The confirmation of the existence of the health related poverty traps in our model highlights the important role of health in generating development traps. To avoid this health related poverty traps, government policymakers should pay attention to the following factors about market failures, which are possible sources of development traps: 1) excessive impatience due to poverty and the inability to satisfy the basic needs, which would discourage health investment (see for example Haaparanta and Puhakka 2004); 2) credit market imperfection which prevents the acquisition of nutrition and necessary health services for early child development (see for example Banerjee and Newman, 1993; Galor and Zeira, 1993); 3) inadequate information on the benefits from early child development, health and education. These market failures can only be effectively overcome by

implementing more public investment measures in both health and education by the government planners in those developing countries. To avoid the poverty traps, we propose that policymakers in the developing countries should invest in public health system which provides free or at least cheap public health services to the poor. The policymakers should also pay attention to improve the efficiency of the public health system because, for example, empirical evidence from World Bank (1993) shows that a significant portion of the public health expenditure is spent in the relatively more expensive diseases which usually benefits more the wealthy. We also suggest the investment in setting up more schools to teach children of the poor the benefits of being healthy and the measures of improving health, which would not only enable a young agent equipped with more knowledge on keeping a healthy body but also increase the chance of obtaining a job with higher salary which would encourage the young agent to invest more in health. These measures should be beneficial to the overcome the market failures and thus development traps.

For the developing countries trapped in the development traps to raise productive forces to pull themselves out of this inferior situation, it is essential for the government policymakers to implement the following two general procedures: firstly, they should adopt the right measures to ensure that the market functions normally, which provide capacity to hire the human capital generated by investment in health and education basic needs; secondly, effective and integrated government policies should also be employed to overcome the market failures that are presented above, which would help to provide the much needed young agents who are equipped with adequate nutrition and quality education.

Appendix 3.1: The derivation of the two ratios

From the two first order conditions (3.12) and (3.13), we get the following two equations:

$$s_t = \beta\left(w(h_t) - T(h_t) - s\right) \Leftrightarrow (\beta+1)s_t = \beta\left(w(h_t) - T(h_t)\right) \tag{3.1.1}$$

$$T'(h_t)h_t - w'(h_t)h_t = s_t \tag{3.1.2}$$

From equations (3.1.1) and (3.1.2), we get:

$$((\beta+1)(T'(h_t)h_t - w'(h_t)h_t) = \beta(w(h_t) - T(h_t))$$

$$\Leftrightarrow (\beta+1)\varepsilon_T T(h_t) + \beta T(h_t) = (\beta+1)\varepsilon_\phi w(h_t) + \beta w(h_t) \tag{3.1.3}$$

where $\varepsilon_T = \dfrac{T'(h_t)h_t}{T(h_t)}$, $\varepsilon_\phi = \dfrac{w'(h_t)h_t}{w(h_t)}$

The expression for $T(h)$ can be arrived from equation (3.1.3):

$$T(h_t) = \frac{(\beta+1)\varepsilon_\phi + \beta}{(\beta+1)\varepsilon_T + \beta} w(h_t) = (1-\alpha)\phi(h_t)Ak_t \frac{(\beta+1)\varepsilon_\phi + \beta}{(\beta+1)\varepsilon_T + \beta} \tag{3.1.4}$$

Based on equations (3.1.1), (3.1.2) and (3.1.4), we obtain the equation for the optimum saving rate:

$$\begin{aligned} s &= T'(h_t)h_t - w'(h_t)h_t \\ &= \frac{\beta\left[w(h_t) - T(h_t)\right]}{(\beta+1)} \\ &= \frac{\beta w(h_t) - \beta\dfrac{(\beta+1)\varepsilon_\phi + \beta}{(\beta+1)\varepsilon_T + \beta} w(h_t)}{(\beta+1)} \\ &= \frac{\beta(\varepsilon_T - \varepsilon_\phi)}{(\beta+1)\varepsilon_T + \beta} w(h_t) \end{aligned} \tag{3.1.5}$$

The ratio of health expenditure to total expenditure can be derived easily from the above equations

$$\begin{aligned} \frac{T(h_t) + c_t}{T(h_t)} = \frac{w(h_t) - s_t}{T(h_t)} &= \frac{w(h_t) - \dfrac{\beta(\varepsilon_T - \varepsilon_\phi)}{(\beta+1)\varepsilon_T + \beta} w(h_t)}{\dfrac{(\beta+1)\varepsilon_\phi + \beta}{(\beta+1)\varepsilon_T + \beta} w(h_t)} \\ &= \frac{\beta + \varepsilon_T + \beta\varepsilon_\phi}{\beta + \varepsilon_\phi + \beta\varepsilon_\phi} = 1 + \frac{\varepsilon_T - \varepsilon_\phi}{\beta + \varepsilon_\phi + \beta\varepsilon_\phi} \end{aligned} \tag{3.1.6}$$

$$\frac{T(h_t)}{y_t} = \frac{(1-\alpha)\phi(h_t)Ak_t \dfrac{(\beta+1)\varepsilon_\phi + \beta}{(\beta+1)\varepsilon_T + \beta}}{\phi(h_t)Ak_t} = (1-\alpha)\frac{(\beta+1)\varepsilon_\phi + \beta}{(\beta+1)\varepsilon_T + \beta} \tag{3.1.7}$$

Appendix 3.2: Proof of Theorem 3.2

Equation (3.26) is the dynamic equation for physical capital per capita. Based on this equation, we analyze the dynamic behavior of the economy. First, we establish the following lemma.

Lemma 3.2.1. Define the right hand side of equation (3.26) as $R(k)$:

$$R(k_{t-1}) = \left[\frac{(1-\alpha)A}{(\beta+1)\theta+\beta}\right]^{\frac{\theta}{\theta-\sigma}} \left[(\beta+1)\sigma+\beta\right]^{\frac{\sigma}{\theta-\sigma}} \beta(\theta-\sigma)k_{t-1}^{\frac{\theta}{\theta-\sigma}} \tag{3.2.1}$$

This $R(k)$ satisfies the following properties:

a) $R(0)=0$;

b) $R'(k^*)>1$, where k^* is the equilibrium level of per capita physical capital

c) $R''(k)\geq 0$

d) $\lim_{k\to\infty}\frac{R(k)}{k}>1$

e) $\lim_{k\to 0}R'(k)\to 0$

Proof. The first property is easy to check. To proof property b), we first need to find the equilibrium level of per capita physical capital. In equilibrium, the level of per capita physical capital is stable. That is $k_t = k_{t-1} = k^*$ when k^* is at steady state. Thus, by setting the k_t and k_{t-1} at both sides of equation (3.26) to k^*, we are able to derive the equilibrium level of k:

$$k^* = \frac{\left[\frac{(\beta+1)\theta+\beta}{(1-\alpha)A}\right]^{\frac{\theta}{\sigma}}}{\left[(\beta+1)\sigma+\beta\right]\left[\beta(\theta-\sigma)\right]^{\frac{\theta-\sigma}{\sigma}}} \tag{3.2.2}$$

The first order condition of $R(k)$ is:

$$R'(k)=\left[\frac{(1-\alpha)A}{(\beta+1)\theta+\beta}\right]^{\frac{\theta}{\theta-\sigma}}\left[(\beta+1)\sigma+\beta\right]^{\frac{\sigma}{\theta-\sigma}}\beta\theta k_{t-1}^{\frac{\sigma}{\theta-\sigma}} \tag{3.2.3}$$

When the economy is at equilibrium, replace k^* for k_{t-1} in equation (3.2.3) and we have the first order condition at the equilibrium level:

$$R'(k^*)=\left[\frac{(1-\alpha)A}{(\beta+1)\theta+\beta}\right]^{\frac{\theta}{\theta-\sigma}}\left[(\beta+1)\sigma+\beta\right]^{\frac{\sigma}{\theta-\sigma}}\beta\theta\left\{\frac{\left[\frac{(\beta+1)\theta+\beta}{(1-\alpha)A}\right]^{\frac{\theta}{\sigma}}}{\left[(\beta+1)\sigma+\beta\right]\left[\beta(\theta-\sigma)\right]^{\frac{\theta-\sigma}{\sigma}}}\right\}^{\frac{\sigma}{\theta-\sigma}} \tag{3.2.4}$$

$$=\frac{\theta}{\theta-\sigma}>1$$

The second order condition of $R(k)$ is straightforward:

$$R''(k)=\left[\frac{(1-\alpha)A}{(\beta+1)\theta+\beta}\right]^{\frac{\theta}{\theta-\sigma}}\left[(\beta+1)\sigma+\beta\right]^{\frac{\sigma}{\theta-\sigma}}\beta\theta\sigma k_{t-1}^{\frac{2\sigma-\theta}{\theta-\sigma}} \tag{3.2.5}$$

The terms before k_{t-1} at the right hand side of equation (3.2.5) is positive and $k_{t-1}^{\frac{2\sigma-\theta}{\theta-\sigma}}\geq 0$, which indicates that $R''(k)\geq 0$.

To proof $\lim_{k\to\infty}\frac{R(k)}{k}>1$, we have:

$$\lim_{k\to\infty}\frac{R(k)}{k}=\left[\frac{(1-\alpha)A}{(\beta+1)\theta+\beta}\right]^{\frac{\theta}{\theta-\sigma}}\left[(\beta+1)\sigma+\beta\right]^{\frac{\sigma}{\theta-\sigma}}\beta(\theta-\sigma)k_{t-1}^{\frac{\sigma}{\theta-\sigma}}$$

$$=\frac{\left[\frac{(1-\alpha)A}{(\beta+1)\theta+\beta}\right]^{\frac{\theta}{\theta-\sigma}}\left[(\beta+1)\sigma+\beta\right]^{\frac{\sigma}{\theta-\sigma}}\beta(\theta-\sigma)}{k_{t-1}^{\frac{\sigma}{\theta-\sigma}}}$$

$$=\left[\frac{(1-\alpha)A}{(\beta+1)\theta+\beta}\right]^{\frac{\theta}{\theta-\sigma}}\left[(\beta+1)\sigma+\beta\right]^{\frac{\sigma}{\theta-\sigma}}\beta(\theta-\sigma)\lim_{k\to\infty}\frac{1}{k_{t-1}^{\frac{\sigma}{\theta-\sigma}}}$$

The term $\lim_{k\to\infty}\frac{1}{k_{t-1}^{\frac{\sigma}{\theta-\sigma}}}>1$ and the terms before this limit are constant.

Therefore, $\lim_{k\to\infty}\frac{R(k)}{k}>1$.

To determine the value of $\lim_{k\to 0} R'(k)$, we need to discuss the following two cases:

Firstly, when $\frac{\sigma}{\theta-\sigma}<1$ or $\theta>2\sigma$, then $\lim_{k\to 0} R'(k)\to\infty$. However, in this case the curve of $R(k)$ crosses the 45-degree line from above, which contradicts to lemma 3.2.1.b we just proved.

Secondly, when $\frac{\sigma}{\theta-\sigma}>1$ and $\theta>\sigma$ or $\sigma<\theta<2\sigma$, then $\lim_{k\to 0} R'(k)\to 0$, which is consistent with the properties we just proofed.

Theorem 3.2 is proved from the above results. By a) above, zero is always a steady state of the dynamic equation (3.26). By b) to e) above, we know another equilibrium exists and the phase map is monotonically increasing and crosses the 45-degree line from below and stay above the 45-degree line in the long run. Therefore, there are two equilibria for the dynamic equation (3.26). One is zero, which is stable. The other is the one when the phase map crosses the 45-degree line from below, which is an unstable equilibrium.

Appendix 3.3: Proof of Theorem 3.3

In a general setting, if we assume the utility function of health as $g(h)$, then the agent's maximization problem is as given below:

$$U = \ln c_t + \beta(\ln c_{t+1} + g(h_{t+1}))$$

$$s.t.: c_t + T(h_t) + s_t = w(h_t)$$

$$c_{t+1} = (1 + r_{t+1})s_t$$

The two first order conditions are as follow:

$$\frac{\partial U}{\partial h_t} = \frac{w'(h_t) - T'(h_t)}{w(h_t) - T(h_t) - s_t} = -\beta g'(h_t) \tag{3.3.1}$$

$$\frac{\partial U}{\partial s_t} = \frac{1}{w(h_t) - T(h_t) - s_t} = \frac{\beta}{s_t} \tag{3.3.2}$$

From (3.3.1), we get:

$$s_t = \beta(w(h_t) - T(h_t) - s_t) \Leftrightarrow (1+\beta)s_t = \beta(w(h_t) - T(h_t)) \tag{3.3.3}$$

From (C.2), we get:

$$\beta g'(h_t)(w(h_t) - T(h_t) - s_t) = T'(h_t) - w'(h_t) \tag{3.3.4}$$

From (3.3.1) and (3.3.2) together, we get:

$$s_t = \frac{T'(h_t) - w'(h_t)}{g'(h_t)} \tag{3.3.5}$$

Insert (3.3.5) into (3.3.3), we have:

$$(1+\beta)\frac{T'(h_t) - w'(h_t)}{g'(h_t)} = \beta(w(h_t) - T(h_t))$$

$$\Leftrightarrow (1+\beta)T'(h_t) + \beta T(h_t)g'(h_t) = (1+\beta)w'(h_t) + \beta w(h_t)g'(h_t)$$

$$\Leftrightarrow \left[(1+\beta)\frac{\varepsilon_T}{h_t} + \beta g'(h_t)\right]T(h_t) = \left[(1+\beta)\frac{\varepsilon_\phi}{h_t} + \beta g'(h_t)\right]w(h_t)$$

$$\Leftrightarrow T(h_t) = \frac{(1+\beta)\varepsilon_\phi + \beta g'(h_t)h_t}{(1+\beta)\varepsilon_T + \beta g'(h_t)h_t}w(h_t) = \eta(h_t)w(h_t) \tag{3.3.6}$$

$$\Leftrightarrow s_t = \frac{\beta w(h_t)\left[1 - \dfrac{(1+\beta)\varepsilon_\phi + \beta g'(h_t)h_t}{(1+\beta)\varepsilon_T + \beta g'(h_t)h_t}\right]}{1+\beta} = \frac{\beta w(h_t)(\varepsilon_T - \varepsilon_\phi)}{(1+\beta)\varepsilon_T + \beta g'(h_t)h_t} \tag{3.3.7}$$

$$\Leftrightarrow \frac{T(h_t)+c_t}{T(h_t)} = \frac{w(h_t)-s_t}{T(h_t)} = 1+\frac{1-\eta(h_t)}{\eta(h_t)(1+\beta)} \tag{3.3.8}$$

where ε_ϕ and ε_T are the two elasticity defined in the previous sections

and $\eta(h_t) = \dfrac{(1+\beta)\varepsilon_\phi + \beta g'(h_t)h_t}{(1+\beta)\varepsilon_T + \beta g'(h_t)h_t}$ (3.3.9)

Further simplify equation (3.3.8), we have:

$$\begin{aligned}\frac{T(h_t)+c_t}{T(h_t)} &= 1+\frac{(1+\beta)\dfrac{\varepsilon_T}{h_t}+\beta g'(h_t)-(1+\beta)\dfrac{\varepsilon_\phi}{h}-\beta g'(h)}{\left[(1+\beta)\dfrac{\varepsilon_\phi}{h_t}+\beta g'(h_t)\right](1+\beta)} \\ &= 1+\frac{\varepsilon_T-\varepsilon_\phi}{(1+\beta)\varepsilon_\phi+\beta g'(h_t)h_t}\end{aligned} \tag{3.3.10}$$

We know that $g(h)$ is a concave function and an increase in health level h_t will lead to a decrease in $g'(h_t)$. As a result, when ε_T and ε_ϕ are constant, an increase in an agent's health level will increase the health expenditure to total expenditure if $\left|\dfrac{\partial g'(h_t)}{\partial h_t}\right| < 1$. Meanwhile, as the survival probability increases, the ratio of health expenditure to total expenditure will increase. The intuition behind this relationship is that the survival probability is usually a positive function of the economy's development level. As the survival probability increases, an agent in the more developed society will take more precautionary measures for their old period health status.

Appendix 3.4: Proof of the unstable stability equilibrium under the general health utility function

We assume a CARA health utility function $g(h_t)=\frac{h_t^{\gamma}}{\gamma}$ $(0<\gamma<1)$. Following the definition in section 3.4.1, we assume that the health cost function is $T(h_t)=h_t^{\theta}$ and the effective labor generation function is $\phi(h_t)=h_t^{\sigma}$ where $\theta>\sigma$. Then the utility function is:

$$U=\ln c_t+\beta(\ln c_{t+1}+g(h_{t+1}))$$

$$s.t.: c_t+T(h_t)+s_t=w(h_t) \tag{3.4.1}$$

$$c_{t+1}=(1+r_{t+1})s_t \tag{3.4.2}$$

The per capita output is:

$$y_t=\frac{Y_t}{N}=(f(h_t)\bar{k})^{1-\alpha}(k_t)^{\alpha} \tag{3.4.3}$$

In the competitive equilibrium, we have the following two conditions regarding the rate of returns of physical capital and wage rate:

$$r_t=\alpha A\phi(h)=\alpha h_t^{\sigma} \tag{3.4.4}$$

$$w_t=(1-\alpha)kh_t^{\sigma} \tag{3.4.5}$$

The representative agent's optimization problem can be written in the following indirect form by substituting the constraints into the utility function:

$$L=\ln(w(h_t)-T(h_t)-s_t)+\beta\{\ln[(1+r_{t+1})s_t]+g(h_{t+1})\} \tag{3.4.6}$$

Differentiate equation (3.4.6) with respect to s and h, we have:

$$\frac{\beta}{s_t}=\frac{1}{w(h_t)-T(h_t)-s_t}=\frac{1}{(1-\alpha)k_th_t^{\sigma}-h_t^{\theta}-s_t}$$

$$\Leftrightarrow (1-\alpha)k_th_t^{\sigma}-h_t^{\theta}=\frac{(\beta+1)}{\beta}s_t \tag{3.4.7}$$

$$\frac{w'(h_t)-T'(h_t)}{w(h_t)-T(h_t)-s}=-\beta g'(h_t) \tag{3.4.8}$$

Assume that $b=\frac{\beta}{\beta+1}$ and insert $g(h_t)=\frac{h_t^{\gamma}}{\gamma}$, $T(h_t)=h_t^{\theta}$, and $\phi(h_t)=h_t^{\sigma}$ into equations (3.4.7) and (3.4.8), we have:

$$k_{t+1}=s_t=\frac{\beta}{\beta+1}\left[(1-\alpha)k_t h_t^{\sigma}-h_t^{\theta}\right]=b\left[(1-\alpha)k_t h_t^{\sigma}-h_t^{\theta}\right] \tag{3.4.9}$$

From equations (3.4.7) and (3.4.8), we obtain an equation of s_t

$$s_t=-\frac{w'(h_t)-T'(h_t)}{g'(h_t)}=\frac{\theta h_t^{\theta-1}-(1-\alpha)\sigma k_t h_t^{\sigma-1}}{g'(h_t)} \tag{3.4.10}$$

From equations (3.4.9) and (3.4.10), we derive another dynamic equation of k_t and h_t

$$\frac{\theta h_t^{\theta-1}-(1-\alpha)\sigma k_t h_t^{\sigma-1}}{g'(h_t)}=b\left[(1-\alpha)k_t h_t^{\sigma}-h_t^{\theta}\right]$$

$$\Leftrightarrow \frac{\theta h_t^{\theta-1}-(1-\alpha)\sigma k_t h_t^{\sigma-1}}{(1-\alpha)k_t h_t^{\sigma}-h_t^{\theta}}=bg'(h_t)=bh_t^{\gamma-1}$$

$$\Leftrightarrow \theta h_t^{\theta-1}-(1-\alpha)\sigma k_t h_t^{\sigma-1}=bh_t^{\gamma-1}\left[(1-\alpha)k_t h_t^{\sigma}-h_t^{\theta}\right]$$

$$\Leftrightarrow \theta h_t^{\theta-1}+bh_t^{\theta+\gamma-1}=b(1-\alpha)k_t h_t^{\sigma+\gamma-1}+(1-\alpha)\sigma k_t h_t^{\sigma-1}$$

$$\Leftrightarrow h_t^{\theta-1}\left(\theta+bh_t^{\gamma}\right)=(1-\alpha)k_t h_t^{\sigma-1}\left(\sigma+bh_t^{\gamma}\right)$$

$$\Leftrightarrow h_t^{\theta-\sigma}\left(\theta+bh_t^{\gamma}\right)=(1-\alpha)k_t\left(\sigma+bh_t^{\gamma}\right) \tag{3.4.11}$$

$$k_{t+1}=b\left[(1-\alpha)k_t h_t^{\sigma}-h_t^{\theta}\right] \tag{3.4.12}$$

In the equilibrium $k_{t+1}=k_t=k^*$ and $h_{t+1}=h_t=h^*$, thus we have:

$$(1-\alpha)k^*=h^{*\theta-\sigma}\frac{\theta+bh^{*\gamma}}{\sigma+bh^{*\gamma}} \tag{3.4.11'}$$

$$k^*=b\left[(1-\alpha)k^*h^{*\sigma}-h^{*\theta}\right]\Leftrightarrow bh^{*\theta}=\left[b(1-\alpha)h^{*\sigma}-1\right]k^* \tag{3.4.12'}$$

From equations (3.4.11) and (3.4.12), we have:

$$bh_t^{\theta} = \left[b(1-\alpha)h_t^{\sigma} - 1\right] h_t^{\theta-\sigma} \frac{\left(\theta + bh_t^{\gamma}\right)}{(1-\alpha)\left(\sigma + bh_t^{\gamma}\right)}$$

$$\Leftrightarrow \frac{b(1-\alpha)h_t^{\sigma}}{b(1-\alpha)h_t^{\sigma} - 1} = \frac{\left(\theta + bh_t^{\gamma}\right)}{\left(\sigma + bh_t^{\gamma}\right)}$$

$$\Leftrightarrow \frac{\theta - \sigma}{\theta + bh_t^{\gamma}} = \frac{1}{b(1-\alpha)h_t^{\sigma}}$$

$$\Leftrightarrow (\theta - \sigma)(1-\alpha)h_t^{\sigma} = \theta/b + h_t^{\gamma}$$

$$\Leftrightarrow \begin{cases} (\theta-\sigma)(1-\alpha)h_t^{\sigma-\gamma} = \dfrac{\theta}{b}h_t^{-\gamma} + 1 \text{ (if } \sigma > \gamma) \\ (\theta-\sigma)(1-\alpha) = \dfrac{\theta}{b}h_t^{-\sigma} + h_t^{\gamma-\sigma} \quad \text{(if } \sigma < \gamma) \end{cases}$$

We know the in the case when $\sigma > \gamma$, there are equilibrium values of (k^*, h^*). To analyze whether the equilibrium is stable, we need to linearize equations (3.4.11) and (3.4.12):

$$\begin{aligned} &\left[\theta(\theta-\sigma)h_t^{\theta-\sigma-1} + b(\theta+\gamma-\sigma)h_t^{\theta+\gamma-\sigma-1}\right](h_t - h^*) \\ &= b(1-\alpha)k_t\gamma h_t^{\gamma-1}(h_t - h^*) + \left[(1-\alpha)\sigma + b(1-\alpha)h_t^{\gamma}\right](k_t - k^*) \end{aligned}$$

$$\begin{aligned} &\Leftrightarrow \left[\theta(\theta-\sigma)h_t^{\theta-\sigma-1} + b(\theta+\gamma-\sigma)h_t^{\theta+\gamma-\sigma-1} - b(1-\alpha)k_t\gamma h_t^{\gamma-1}\right](h_t - h^*) \\ &= \left[(1-\alpha)\sigma + b(1-\alpha)h_t^{\gamma}\right](k_t - k^*) \end{aligned} \tag{3.4.13}$$

$$k_{t+1} - k^* = \left[b(1-\alpha)k_t\sigma h_t^{\sigma-1} - b\theta h_t^{\theta-1}\right](h_t - h^*) + b(1-\alpha)h_t^{\sigma}(k_t - k^*) \tag{3.4.14}$$

Inserting equation (3.4.13) into equation (3.4.14), we have the dynamic equation of k_t:

$$k_{t+1} - k^* = \Omega(k_t - k^*) \tag{3.4.15}$$

where

$$\Omega = b(1-\alpha)\left\{\frac{\left[(1-\alpha)k^*\sigma h^{*\sigma} - \theta h^{*\theta}\right]\left(\sigma + bh^{*\gamma}\right)}{\theta(\theta-\sigma)h^{*\theta-\sigma} + b(\theta+\gamma-\sigma)h^{*\theta+\gamma-\sigma} - b(1-\alpha)k^*\gamma h^{*\gamma}} + h^{*\sigma}\right\}$$

To prove whether the equilibrium value of (k^*, h^*) is stable, we only need to check whether $\Omega > 1$. Because it is not able to solve explicitly whether $\Omega > 1$, we have to use the numerical method. We assume that $\theta = 1.25$, $\sigma = 0.75$, $\gamma = 0.25$, $b = 0.25$ and $\alpha = \frac{1}{3}$, then we have $h^* = 245.33$ and $k^* = 26.01$. By inserting the equilibrium values of k^* and

h^* together with the parameter values stated above into Ω, we have $\Omega = 1.8 > 1$. Therefore, we prove that the equilibrium is unstable, which is consistent with what we have found when we assume the logarithm utility function. In other words, the health related development traps, which generate widening gaps between the developing and developed economies, are robust to the use of general form of health utility function.

Appendix 3.5: Tables and Figure

Table 3.1 Regional indices of economic performance, 1960-2007

Development Category	Income relative to world				Income related to Rich			
	1960	1980	2000	2007	1960	1980	2000	2007
World	1.00	1.00	1.00	1.00	0.27	0.23	0.20	0.20
East Asia & Pacific	0.06	0.07	0.18	0.27	0.02	0.02	0.04	0.05
Sub-Saharan Africa	0.18	0.15	0.10	0.10	0.05	0.03	0.02	0.02
Latin America & Caribbean	0.86	0.91	0.75	0.76	0.23	0.21	0.15	0.15
Euro Area	2.64	3.43	3.88	3.72	0.72	0.79	0.76	0.74
High income: OECD	3.68	4.33	5.09	5.00	1.00	1.00	1.00	1.00

Source: The statistics are compiled and calculated by the author based on data from World Development Indicators 2008.

Table 3.2 Income group indices of economic performance, 1960-2007

Development Category	Income relative to world				Income related to Rich			
	1960	1980	2000	2007	1960	1980	2000	2007
World	1.00	1.00	1.00	1.00	0.28	0.24	0.20	0.21
LDC	0.09	0.07	0.05	0.06	0.02	0.02	0.01	0.01
Low income	0.10	0.08	0.06	0.07	0.03	0.02	0.01	0.01
Middle income	0.22	0.25	0.27	0.33	0.06	0.06	0.06	0.07
High income	3.59	4.22	4.91	4.82	1.00	1.00	1.00	1.00

Source: The statistics are compiled and calculated by the author based on data from World Development Indicators 2008.

Table 3.3 Annual per capita GDP growth rates, 1960-2007 (Std. deviations in parentheses)

Development Category	1960-1979	1980-1999	2000-2007
World	2.72	1.24	1.94
	(1.4)	(1.07)	(1.05)
Low income	1.3	0.49	1.96
	(2.71)	(1.4)	(1.44)
Middle income	3.12	1.8	4.86
	(1.25)	(1.32)	(1.2)
High income	3.57	2.01	1.77
	(1.28)	(1.18)	(0.91)

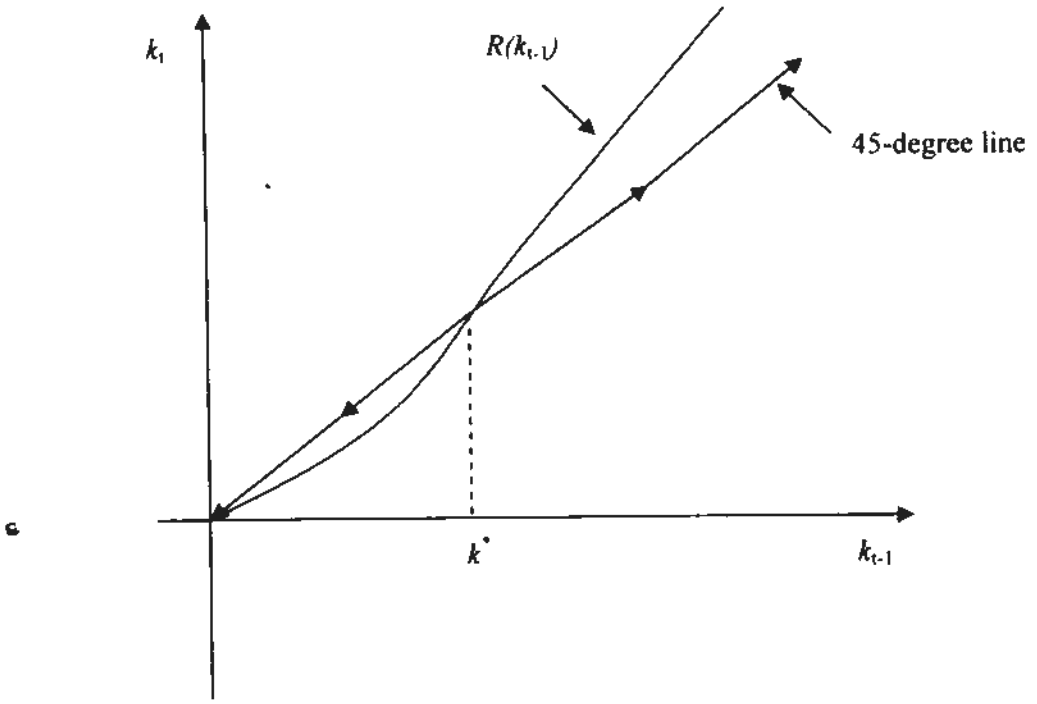
Source: The statistics are compiled and calculated by the author based on data from World Development Indicators 2008.

Table 3.4 Average per capita income and health indicators, 1960-1970 and 1997-2007

Development Category	World		Low income		Middle income		High income	
Time	1960-1970	1997-2007	1960-1970	1997-2007	1960-1970	1997-2007	1960-1970	1997-2007
Income per capita (constant 2000 US$)	2853.34	5390.97	253.28	355.28	608.08	1566.66	10702.57	26305.13
Life expectancy at birth, total (years)	55.53	67.4	44.16	55.98	52.76	67.94	69.87	78.26
Mortality rate, under-5 (per 1,000)	166.6	76.54	235.47	139.16	147.87	52.44	30.84	6.87
Mortality rate, infant (per 1,000 live births)	109.31	51.81	142.84	87.43	101.02	40.19	25.72	5.76
Fertility rate, total (births per woman)	4.88	2.65	6.62	4.56	5.37	2.36	2.75	1.7

Source: The statistics are compiled and calculated by the author based on data from World Development Indicators 2008.

Figure 3.1 Dynamics of the growth path of per capita capital



Chapter 4 Health Expenditure and Economic Growth: An Empirical Analysis

4.1 Introduction

In the previous two essays, we build theoretical models to analyze two important issues on the basic relationship between health and economic growth. In the first essay, we extend the Barro (1996b) model to analyze the effect of health on generating long-run economic growth with an emphasis on how the negative effect of health, reflected by the endogenous health depreciation rate, affects the long run economic growth. We find that health would generate endogenous growth if the health depreciation rate is constant in the long-run or the endogenous health depreciation rate is determined simultaneously by health and education. We then study in the second essay another important issue of health, the issue of "development traps", by providing a new mechanism on how the development traps can be generated by health. One result generated from the model developed in the second essay is the positive ratio of health expenditure to output. From these two essays, we identify theoretically the core role of health in economic development. In this essay, we complement the previous two theoretical essays by providing empirical evidence on the role of health in promoting economic growth.

There have already been some existing literatures examining the health and growth relationship by analyzing how the population health status may influence economic growth. In this essay, we examine empirically on another aspect of health by investigating how health expenditure, proxied by public health expenditure, may affect economic growth and production output. Except for the previous two essays, the role of human capital in the

forms of health and education has also been well recognized by a series of papers as not only the key determinant of economic growth but also one of the ultimate goals to expand human freedom more generally (Ravallion and Chen, 1997; Schultz, 1999; Sen, 1999; and Squire, 1993). The vital roles of health and education have also been reflected by their significant roles in almost all of the important development plans proposed by the government policymakers. For example, of the eight time-bounded goals of the revised version of Millennium Development Goals (MDGs)[22] released on January 2008, tow of them are related to health and two of them are related to education. The important roles of health and education in MDGs call on rich and poor countries to boost their efforts to achieve the goals in some crucial health and education indicators. Another example is the 4 trillion RMB economic stimulus package announced by the Chinese State Council on October 2008, where increasing spending in health and education services is also among the key measures.

In most of the countries, the provision of health and education services necessary for the accumulation of these two forms of human capital are supported by public spending. Therefore, it is of great interest for us to analyze empirically the relationship between public expenditure on health and education and economic growth. Because our main research interest is in health, most of the analysis followed will focus on health, although education will also be considered simultaneously. From Figure 1.1 shown in the introduction of this thesis, we know that on one hand increase in public health expenditure may improve the average health level of the population

[22] The Millennium Development Goals, translated from the Millennium Declaration which was endorsed by leaders of 189 countries at the United Nations on September 2000, is a roadmap, set out eight time-bound and measurable goals to be reached by 2015.

and thus increase the return on education investment and provide more effective labor, which promote economic growth. Nevertheless, on the other hand, increment in health investment may occupy resources that may have been used for physical capital production and increase in life expectancy may introduce more health care expenditure. This "crowding out" effect created by increasing health expenditure may induce the slowdown of economic growth. The econometric analysis conducted in this essay may provide some empirical evidence on which effect of health expenditure may play a dominant role.

This essay intends to fill the gap in the existing literature by providing an empirical study assessing the relationship between health expenditure and economic growth. We analyze the effect of health on both per capita output and growth rate while considering simultaneously both health and education as two forms of human capital. The two growth models we employ for analyzing the relationship between health expenditure and growth are the augmented Bassanini and Scarpetta (2001) model and the augmented Solow model. We first study the health and growth relationship by incorporating a panel dataset covering 138 countries in different development levels from all over the world. Next, as a comparison and robustness checking, we focus on countries from East Asia and compare the results derived from this sub-sample with those from the whole sample. The time span of these two datasets is from 1971 to 2005. Because according to Islam (2000), fixed effect estimator (LSDV) has excellent short time series performance compared to instrumental variable estimators and generalized method of moments (GMM) estimators, we will use LSDV to draw the baseline estimation results. For robustness checking, two stage least squares

(2SLS), GMM and bias-corrected LSDV estimators will also be employed.

The rest of the paper is organized as follows. Section 4.2 discusses the empirical literature on the relationship among health, education and economic growth. Section 4.3 outlines the modeling details, econometric issues and discusses the data used in the empirical analysis. Section 4.4 presents the estimation results of the growth regressions and discusses the main findings. Finally, Section 4.5 offers concluding remarks.

4.2 Literature review

Health and education have been well recognized as the core dimensions of human capital in theoretical literature. The recognition of these two aspects of human capital as key determinants of long run economic growth was encouraged by the significant advances in endogenous growth theory in the late 1980s. There is considerable theoretical support for education in fostering economic growth.[23] However, relatively less attention was given to health as another aspect of human capital to affect long run economic growth. Although early studies, such as Arrow (1962), Ehrlich and Liu (1991), Mushkin (1962), and Schultz (1961), have identified health as another important form of human capital, only theoretical links between health and growth were proposed. Becker (1962) is another example where a decline in the working age death rate would increase earning because more effective working hours could be provided. The first theoretical model considering the impact of both health and education did not appear until Barro (1996b). Van Zon and Muysken (2001,

[23] The examples of theoretical studies regarding the role of education are Arrow (1962), Becker, Murphy and Tamura (1990), Lucas (1988), Rebelo (1991), Mulligan and Sala-i-Martin (1992), Romer (1986, 1990) and Schulz (1961).

2003) extend the Lucas (1988) model and follow the idea of Grossman (1972)[24] by introducing health into both the production function and utility function. They argue that health influences economic growth through three channels[25] and the demand for health care services may occupy resources which could have been used for goods production.

Similar situation appears in empirical studies. Most early empirical studies identified human capital narrowly as education. Accumulation of human capital through education has featured dominantly in empirical work examining the determinants of long run economic growth (Barro, 1991, 1996a, 1996b; Barro and Sala-i-Martin, 1992, 2005; Coulombe et.al, 2004; Krueger and Lindahl, 2001; Levine and Renelt, 1992; Mankiw, Romer and Weil, 1992; Romer, 1990; and Sala-i-Martin, 1997).

Compared with the substantial empirical work focusing on education as human capital, empirical analyses on the effect of health capital on growth are relatively thin. The empirical studies on the relationship between health and economic growth are available from both microeconomic and macroeconomic perspectives. The microeconomic literature examines the health and growth relationship through the links from health inputs to health status and then to labor productivity and wages, which is shown in Figure 4.1. The health inputs in Figure 4.1 are generally represented by nutrition intakes, while examples of health status variables include life expectancy, mortality, healthy working hours, cognitive functioning, and reasoning

[24] Grossman (1972) regards health as a kind of durable good. Investment in health human capital would result in more healthy time and thus higher marginal return, which implies an increase in labor productivity and therefore promote economic growth.

[25] The three channels Zon and Muysken (2001, 2003) propose that may affect intertemperoral decision making are: first, health is a prerequisite for the accumulation of human capital and the provision of health services; second, health can generate positive utility of its own; lastly, the provision of health competes with production of goods and accumulation of human capital for labor services.

ability. An individual with better health status is often with more strength and higher stamina, which result in higher productivity and lower possibility to be absent from work and therefore more likely to demand higher wage. Strauss and Thomas (1998) present an excellent review of microeconomic evidence on the variation in wage explained by indicators of health status. Some recent studies also examine the link between health inputs and labor productivity and wages. Positive effects on labor productivity and wage through health inputs are found in Alderman, Hoddinott, and Kinsey (2006), Behrman et al. (2003), Bleakley (2007), Maccini and Yang (2005), Miguel and Kremer (2005), Thomas et al. (2002, 2003). Some studies[26] even examined more than one link in Figure 4.1. All these microeconomic works show that nutrition or health status indicators are ideal predictors of economic status.

The macroeconomic literature examine the relationship between health and economic growth through the links among social health spending, social indicators of health and economic growth, which is shown in Figure 4.2. Examples of the social indicators of health output are life expectancy at birth, mobility rate and mortality rate. Different from the microeconomic literature, the studies in macroeconomic level do not focus on the channels through which health affect economic growth. Cross country macroeconomic data is used to analyze whether there is positive relationship between health and economic growth from an aggregate level. The empirical studies of health on economic growth are usually conducted by considering education simultaneously. Knowles and Owen (1995, 1997) find

[26] Thomas and Frankenberg (2002) provide an extensive review of examples for the studies analyzing more than one link in Figure 1.

that there is a significant statistical relationship between health and growth with education having a modest role. In contrast, Webber (2002) reaches different conclusions and argues that growth oriented policies should focus on investments in education over health. However, in another study, McDonald and Roberts (2002) conduct a panel data analysis of 77 countries and their findings seem to be consistent with those of Knowles and Owen (1995). Studies of the relationship between health and economic growth have also been conducted to analyze an individual country. Li and Huang (2009) examine the relationship among education, health and economic growth based on China's provincial data from 1978 to 2005 and the results show that both health and education have positive effect on economic growth and there is a trade-off between these two forms of human capital investment. Other recent studies, for example, Bloom and Canning (2001, 2003), Bloom, Canning, and Sevilla (2004), Gyimah-Brempong, and Wilson (2004), Mayer et al. (2001) and Weil (2007) also find a positive link between health output indicators and economic growth. In contrast, studies of the link between social spending and health have generated mixed results. Positive impact from social spending on health indicators are recognized in Baldacci et al. (2003), Gupta et al. (2003) and Hojman (1996). In contrast, Filmer and Pritchett (1997, 1998), Musgrove (1996) and Pritchett (1996) are not able to find a significant positive impact on health indicators from social spending.

The macroeconomic literature on the health and economic growth relationship concentrates mainly on the effect of aggregate health indicators, such as life expectancy and mortality rate, on economic growth. Comparatively little attention has been given to the impact of social

spending on economic growth. In this essay, we analyze the relationship between social health expenditure and economic growth. We seek to assess the statistical effects of social health expenditure on both the growth rate of GDP and the level of per capita GDP.

4.3 Data and model description

4.3.1 Model description

Since the significant development of the new growth theories in mid-1980s, there has been a considerable amount of empirical papers estimating economic growth, in which cross-sectional data are generally used. However, although encouraged by the findings of the theoretical development in endogenous growth theory, those empirical works are still generally based on the neo-classical models[27] which were developed in the 1950s and 1960s. Barro (2002) argues that the endogenous growth models help to account for the long-run economic growth for developed countries, although there is diminishing return to the accumulation of physical capital and human capital. In contrast, the neo-classical models are useful for explaining the growth difference between different countries. The endogenous growth models play a complementary rather than a competing role to the neoclassical growth models. One prototypical empirical study is Mankiw, Romer and Weil (1992) which extend the Solow (1956) neoclassical growth model to include human capital in the form of education. Knowles and Owen (1995) and Islam (1995) further extend the Mankiw, Romer and Weil (1992) model by incorporating health human capital into

[27] The main examples of the neoclassical models include Solow (1956), Swan (1956), Cass (1965) and Koopmans (1965).

the production function. The Bassanini and Scarpetta (2001) consider both the short-run and long-run effect of human capital in the form of education on economic growth.

The empirical models we employ here are based on Bassanini and Scarpetta (2001) and Knowles and Owen (1995). Firstly, we follow the procedures of Knowles and Owen (1995) to include both health and education as two aspects of human capital into the Mankiw, Romer and Weil (1992) model. The resulting baseline equation is shown in equation (4.1), with further transformations of the baseline equation shown in equations (4.1.21') to (4.1.23') in Appendix 4.1, which transform the baseline equation according to whether we are interested in analyzing the levels or the growth rates of the two dimensions of human capital.

$$\ln y_{it_2} = \alpha_{10} + \alpha_{11} \ln y_{it_1} + \alpha_{12} \ln s_{ki} + \alpha_{13} \ln s_{si} + \alpha_{14} \ln s_{hi} - \xi_1 \ln p_{it} + \varepsilon_{1i} \qquad (4.1)$$

where y_{it} is per capita output; p_{it} is "workforce growth"; s_{ki}, s_{si}, s_{hi} are investments in physical, education and health capitals, respectively.

Secondly, we extend the Bassanini and Scarpetta (2001) model by including health human capital into an equation with growth rate as dependent variable. In Bassanini and Scarpetta (2001), the output growth rate, rather then the logarithm of output level is used as dependent variable. Furthermore, both the levels and growth rates of human capital or human capital investment are incorporated into the regression equations (see equations (4.1.20), (4.1.26'), (4.1.27') and (4.1.28') in Appendix 4.1). These growth equations are similar to the "error-correction" models which are widely used in time-series research. The advantage of using these error-correction models is that they consider both the short-run and long-run

effects of human capitals on economic growth. The baseline equation for the extended Bassanini and Scarpetta (2001) model is shown in equation (4.2).

$$\Delta \ln y_{it} = a_{10} + \varphi \ln y_{it_1} + a_{11} \ln s_{kt} + a_{12} \ln s_{st} + a_{13} \ln s_{ht} + \psi_1 \ln p_{it} + b_{11} \Delta \ln s_{st} + b_{12} \Delta \ln s_{ht} + \varepsilon_{1t} \quad (4.2)$$

The technical details for both the extended Knowles and Owen (1995) model and the Bassanini and Scarpetta (2001) model are presented in Appendix 4.1.

As we discussed in section 4.2, the empirical macroeconomic literature on the relationship between health and economic growth focuses mainly on the effect of health indicators, such as life expectancy and mortality rate, on economic growth. However, we are interested in analyzing the statistical relationship between health expenditure and economic growth. Therefore, for the different transformations of the baseline equations (4.1) and (4.2) which are shown in Appendix 4.1, we employ the equations with health investment entering the regression equations (equations (4.1.18), (4.1.20), (4.1.21') and (4.1.26') in Appendix 4.1). For the equations with growth rate as the depending variable (equations (4.1.20) and (4.1.26') in Appendix 4.1), both the level and changes in the two forms of human capitals are included as independent variables. The theoretical reasons for the inclusion of both the levels and changes of the human capital variables are that the stock of human capital drives growth (Romer 1986, 1990) and changes in human capital would also stimulate economic growth (Lucas 1988). The derivation of the growth equations in Bloom, Canning, and Sevilla (2004) and Gyimah-Brempong, and Wilson (2004) also support the inclusion of both levels and changes of human capital in the growth equation.

Next, we discuss the definitions of the structural variables. The core

variables included in all the regressions of our analysis are the real per capita GDP (RGDP), investment saving ratios (INV) and the summary of population growth rate, depreciation rate and technological progress $(n+g+\delta)$ [28], which sometimes is called "workforce growth". The proxy variable for investment saving ratios is the share of investment spending in GDP. For the sum of depreciation rate and technological progress ($g+\delta$), we follow Mankiw, Romer, and Weil (1992)'s assumption to assume it to be 0.05 (5 percent) and the same for all the countries in all years.[29] To select proper proxy variables for health and education, we base on the following rules: 1) the proxy variables must be comparable across different economies; 2) the proxy variable of health must address the characteristics of health expenditure and the proxy variable for education should reflect the aspects of education status and be comparable to the health expenditure proxy variable; 3) they should be estimable and data are available. For the health investment proxy variable, the absolute government spending figures are not comparable because of the data quality and the problem of exchange rate. We thus employ the variable public health expenditure as a percentage of GDP (HEXP) as a proxy variable for health expenditure. The selection of the proxy variable for education is always a controversial topic in applied economic growth research. Mankiw, Romer and Weil (1992) use the average schooling years of the total population over 15 years old while Barro and Lee (1993) have improved the work of Mankiw, Romer and Weil (1992) by extending the dataset to give the average number of schooling years of people with age over 25. In some of

[28] In the growth equation, according to the derivation of the growth equation, only population growth is included in the regression.

[29] Changing this ratio does not seem to alter the estimation results significantly. Thus, we use this number throughout the estimation.

the recent researches, besides the two commonly used measures mentioned before, the enrolment ratios for primary, secondary, and tertiary education are also used. In this paper, however, because the focus is on the statistical effect of health expenditure on economic growth, we employ the public spending on education (PSE) as one of the proxy variables for education, which would enable the convenient comparison of the effects of health and education expenditure on economic growth. Furthermore, we use student to teacher ratio for primary schools (PTRATIO) as another proxy variable to education. PTRATIO is regarded as the education variable which reflects the fundamental quality of education or the education level of a particular country. We employ the student to teacher ratio for primary schools but not for secondary schools because the countries in our sample are at different stages of development in education. The adoption of student to teacher ratio for primary schools enables us to include more observations into our dataset.

4.3.2 Estimation methodology

There are two datasets we employ in this empirical study. One is a world dataset composed of 138 countries and the other one is an East Asia dataset comprising of 10 countries/regions. Both of these two datasets consist of countries/regions at different development stages. It is not appropriate to assume homogeneity in characteristics such as technology, environmental issues, and social atmosphere. Therefore, panel data models are preferred over cross-section models which were used in Mankiw, Romer and Weil (1992). Baltagi (2001) also supports using panel data estimation by arguing that panel data estimation is the most suitable choice for growth

regression because it allows variation of technology across countries and captures the dynamic effect which is not reflected in cross-section estimation. The estimation models of equations (4.1) and (4.2) are actually examples of the dynamic panel data model of the following form:

$$y_{it} = \gamma y_{i,t-1} + \beta x_{i,t} + \mu_i + \varepsilon_{it} \quad i=1,\ldots,N,\ t=1,\ldots,T \tag{4.3}$$

where N and T stand for total number of countries/regions and total number of time, respectively. γ is a scalar and β is $1\times(K-1)$, $x_{i,t}$ is $(K-1)\times 1$ of control variables. μ_i is the fixed effect variable and ε_{it} follows independent identical distribution (i.i.d) $N(0,\sigma_\varepsilon^2)$.

In panel data estimation, we usually encounter five problems: small sample bias, endogenity, heterogeneity, measurement error and omitting variable bias. There are a number of panel estimators, each of which may have certain advantage in overcoming one or several of the above problems. In the remaining part of this subsection, we will discuss the selection of the appropriate panel estimators for this empirical study.

First of all, we would not use ordinary least squares (OLS) in this study because OLS ignores the individual effects which cause omitting variable bias. Furthermore, OLS estimation may attribute predicting power of the fixed effect to the lagged term, which induces serious up-ward bias. The other commonly used methods, such as the fixed effect estimator (least squares dummy variable estimator, LSDV), random effect estimation (generalized least squares estimation, GLS), two stage least squares (2SLS), and generalized method of moments (GMM) can solve the problems appearing in OLS. Secondly, one of the key characteristics of growth-convergence equations we analyze in this empirical study is that the

exogenous variables are correlated with the country/region specific effect. Thus, the random effect estimations which rely on the assumption of random effect are not appropriate. Similar argument is also available in Islam (1995).

The exclusion of OLS and random effect estimators results in LSDV, 2SLS, and GMM for our selection. For the LSDV, 2SLS and GMM panel estimators, we would use LSDV as our baseline method. 2SLS and GMM would be used as complementary methods and for robustness checking. The LSDV estimation is performed on equation (4.4), which is a within group transformation of equation (4.3).

$$y_{it} - y_{i.} = \gamma(y_{i,t-1} - y_{i.,-1}) + \beta(x_{i,t-1} - x_{i.}) + (\varepsilon_{it} - \varepsilon_{i.}) \qquad (4.4)$$

The reason for us to use LSDV as our baseline method is that the datasets we use are with short time series but relatively large number of countries/regions. Although Nickel (1981) argues that LSDV estimator for dynamic panel data model, just like the one used in our study, is not consistent for large N and finite T dataset, we should emphasize that this theoretical property is only asymptotic. Monte Carlo simulations should be done to provide evidence on small sample properties. What is well known to us is that Monte Carlo simulations are more useful if the exercise is customized to the model whose estimation is in question. Islam (2000) performs Monte Carlo study on a number of commonly use panel estimators for the Growth-Convergence model. Judged by both small sample bias and root mean squared errors (RMSE), LSDV displays excellent small sample performance and performs better to GMM and instrumental variable (IV) estimators. One possible reason offered by Islam (2000) for the theoretically

better estimators like GMM and IV not performing well in small sample is that their theoretical properties depend on the optimal weighting matrix which should be estimated in practice and may contain noises in the data. Therefore, LSDV given below would be used as our baseline panel estimator.

However, LSDV estimator can not solve the problem of endogenity. In our model, there may be bilateral causality relationship between the growth and the two forms of human capital, which may generate problem of endogenity. To check the robustness of our estimation results to endogenity, we perform 2SLS estimation. Lagged values of the two dimensions of human capital are used as instrumental variables, which are natural and standard choice of instruments in this kind of analysis. Another kind of IV estimator can also solve the problem of endogenity. Anderson and Hsiao (1981) suggest one kind of IV estimator using lagged difference and levels as potential instruments to the first difference of the dynamic model which is shown in equation (4.5).

$$y_{it} - y_{i,t-1} = \gamma(y_{i,t-1} - y_{i,t-2}) + \beta(x_{i,t-1} - x_{i,t-2}) + (\varepsilon_{it} - \varepsilon_{i,t-1}) \qquad (4.5)$$

Theoretically, the Anderson and Hsiao (1981) IV estimator is a consistent estimator and longer lags can be used to improve the model efficiency. However, longer lags used in the Anderson and Hsiao (1981) IV estimator means losing more degree of freedom and there is a trade-off between consistency and efficiency. Holtz-Eakin, Neway, and Rosen (1988) suggest that one way to get around this trade-off is to use GMM estimator which was later formalized by Arellano and Bond (1991) to use the orthogonality conditions between lagged values of y_{it} and the error terms to

obtain additional "GMM-style" instruments. As a comparison, the standard "IV-style" instruments used in the 2SLS and the "GMM-style" instruments are presented in equations (4.6) and (4.7), respectively.

$$Z_i = \begin{bmatrix} \cdot \\ y_{i1} \\ \vdots \\ y_{i,T-2} \end{bmatrix} \tag{4.6}$$

$$Z'_i = \begin{bmatrix} [y_{i1}] & 0 & \cdots & 0 \\ 0 & [y_{i1}, y_{i2}] & \cdots & 0 \\ \vdots & \vdots & \cdots & \vdots \\ 0 & 0 & \cdots & [y_{i1}, y_{i2}, \ldots, y_{i,T-2}] \end{bmatrix} \tag{4.7}$$

We see that in the Arellano and Bond (1991) GMM estimation, all the possible lags are used as instruments and the missing values are substituted by zeros which results in a more efficient estimation over the Anderson and Hsiao (1981) IV estimator. In this essay, we would employ both the 2SLS and GMM estimators to overcome the endogenity problem.

For the problem of heterogeneity, we use a robust estimator of the covariance matrix of the parameters to be estimated in the GMM estimation. This covariance matrix is consistent in the presence of any pattern of heteroskedasticity and autocorrelation within panels. For the problem of measurement errors, there is no particular panel estimator to deal with this problem. We would use the five year average data to alleviate this problem.

Furthermore, One obvious advantage of the use of the set of estimation methods (LSDV, 2SLS, one-step GMM, two-step GMM) is that the comparison of estimation results from different estimation methods enable us to check whether the results derived are sensitive to the problem of

nonstationarity in some of the variables, such as RGDP and the workforce growth. The extensive survey of Baltagi and Kao (2000) on the nonstationary panels states that the GMM method is recommended for the nonstationary panels. The comparison of the results from various methods tells us the extend of the problem of nonstationary variables.

For technical details of the LSDV, 2SLS and GMM estimators, please see Appendix 4.2.

4.3.3 Data description

The dataset we use in this study is a panel dataset with 138 countries from 1971 to 2005. Of the 138 countries, 16 countries are from East Asia and Pacific (EAP), 29 countries from Europe (EU), 29 countries from Latin America and Caribbean (LA), 16 countries from Middle East and North Africa (ME), 2 countries from North America (NA), 40 countries from Sub-Saharan African (SSA) and 6 from South Asia (SAS). The list of the countries in our sample is available in Appendix 4.4. Five-year averages are used to smooth out short-turn fluctuations and minimize measurement errors. Furthermore, to make comparison to the statistical results derived from this world dataset, we also collect an annual dataset of East Asia[30] with the same time span. The education variables are from the World Development Indicators (2008) and the World Bank's EdStats database while the data of the ratio of health expenditure to GDP is from the World Development Indicators (2008), the World Health Organization's WHO Statistical Information System (WHOSIS) and World Development Report

[30] The countries/regions in the East Asia dataset include China, Hong Kong, Indonesia, Korea, Japan, Malaysia, Philippines, Singapore, Taiwan and Thailand. The data of this East Asia dataset is from World Development Indicators and the statistical yearbooks of the respectively countries.

(2008). All the other macroeconomic and population variables are from World Development Indicators (2008).

The total number of observations in each of the regressions may vary significantly because of the data availability for different combinations of variables. The coverage for the macroeconomic and population data, such as physical capital investment, GDP per capita, population growth rate are more complete and a large number of countries have coverage of these variables for the whole sample period from 1971 to 2005. However, for the proxy variables of the two dimensions of human capital, the observations are much less. Observations may vary, depending on different human capital proxy variables used. Furthermore, the inclusion of the IV variables to check the robustness of the results derived in this study may also lead to further difference in sample size.

The summary statistics of the key variables analyzed in our study Table 4.1 presents real GDP per capita (RGDP), per capita GDP growth, investment, population, and health and education indicators for countries with data of at least two periods. First, we check the summary statistics of health expenditure. One significant fact is that government planners from all the regions have invested similar percentage of GDP in public health expenditure (between 5 to 6 percent) except for North America, which invested an average of 8.88 percent of GDP. Similar situation happens in public spending on education. But in this case, public spending on education has two extreme cases. One is the average spending of 6.36 percent for North America and the other one is 2.63 percent for South Asia while the corresponding values for other regions are around the average value of the world, which is 4.41 percent. The means of RGDP per capita range from

413.14 (constant 2000 US$, thereafter) of South Asia to 23189 of North America and only Europe and North America have the means of RGDP per capita above 10000. For the average growth rates of GDP per capita, East Asia and Pacific region is the top performer, with an average growth rate of 4.57 percent, more than double of the average growth rate of the world (2.17 percent) while the growth rates of other regions are in the range of 1.3 percent to 3.2 percent. For population growth rates, we notice that Europe has the lowest average population growth of 0.48 percent which is much lower then the world average level of 1.84 percent.

4.4 Estimation results

As already discussed in section 4.3, although panel data models, which are the most suitable choice of accounting for country differences, have obvious advantages over cross-sectional models, there are problems such as small sample bias, endogenity, measurement error and heterogeneity. Different estimation methods have their own advantages in overcoming certain shortcomings of the panel models. Following Islam (1995, 2000), we choose fixed-effect panel estimation (LSDV) as our baseline estimation because from the results of the Monte Carlo study on the Growth-Convergence model, it performs well in short time series like the one we use in this study. To check whether our baseline LSDV results are sensitive to the problems that LSDV is not able to overcome, we do robustness checking by applying other panel estimators on our world dataset. As stated in Knowles and Owen (1995), one possible source of estimation bias is the simultaneity of the explanatory variables, which causes the problem of endogenity. To fit into our study, the possible source of

estimation bias is that investment in human capital is likely to stimulate economic growth but reversely, better living standards may induce demand for education and healthier life. To account for the possible endogenity, we also estimate 2SLS with the lagged values of health and education indicators as instruments (see also Baldacci *et al.*, (2004) and Bloom *et al.* (2004) for examples using lagged values of endogenous variables as instrumental variables). Furthermore, to test the sensitivity of our estimation results from LSDV, we also use both the one-step and two-step GMM estimators to overcome the problem of endogenity and heterogeneity. Lastly, for the augmented Mankiw, Romer and Weil (1992) model, we perform the bias-corrected LSDV (LSDVC) estimator proposed by Bruno (2005).[31]

4.4.1 The baseline estimation results using LSDV

To understand the statistical relationship between health expenditure and economic growth thoroughly, we first use the world dataset to estimate the augmented Bassanini and Scarpetta (2001) growth equation with per capita GDP growth rate as dependent variable and both the level and changes of the two dimensions of human capital included as independent variable. Secondly, we also analyze the augmented Mankiw, Romer and Weil (1992) model using the world data. For the augmented Bassanini and Scarpetta (2001) growth equation, we have run 12 regressions. The three baseline variables (lagged value of RGDP, physical capital investment and the sum of population growth rate, depreciation rate and technology

[31] We do not run the LSDVC estimation for the augmented Bassanini and Scarpetta (2001) model because we are only able to find the program solving the dynamic equation with the lagged term of the dependent variable included in the regression while the augmented Bassanini and Scarpetta (2001) do not have the lagged term of the dependent variable as independent variable. We would leave this problem for future research.

progress) are included in all the 12 regression equations. The first regression is the regression on these three baseline variables. For regressions (4.2) to (4.4), we add one of the three proxy variables for health or education into the regression equation (health expenditure as a percentage of GDP, student to teacher ratio for primary schools and public spending on education), one for each time. For equations (4.5) and (4.6), we add HEXP and one of the education proxy variables into the regression equations simultaneously. For equations (4.7) to (4.12), we add different combinations of stocks of human capital and changes in human capital into the regression equations. The comparison of these 12 regression equations offers us answer to the question that whether the effect of health expenditure on per capita GDP growth rate is robust to the inclusion of levels or changes of health or/and education indicators. For the augmented Mankiw, Romer and Weil (1992) model, the first regression is the baseline regression (equation (4.1)), which has the same independent variables as those of the augmented Bassanini and Scarpetta (2001) growth equation. The three human capital indicators are added into the baseline regression sequentially, as shown in equations (4.2) to (4.4). The last two equations are regressions with health expenditure and one of the two education proxy variables. Furthermore, to make comparison, we run the above two regression models on an East Asia dataset.

Firstly, we discuss the results of the augmented Bassanini and Scarpetta (2001) model. The regression results are shown in Table 4.2. Equation (1) is the baseline regression and all the three baseline variables are statistically significant with expected signs. Moreover, by comparing the 12 equations of Table 4.2, we find that the statistical results of the first two baseline variables (lagged value of real GDP and physical capital investment) are

very stable. Both of them are with expected signs and most of them are significant. These results are consistent of those of McDonald and Roberts (2002). The negative and significant signs on the lagged value of per capita *RGDP* indicate the convergence of per capita growth rates for different countries in our sample while the significant positive coefficients of fixed capital investment reflect the positive effect of physical capital investment on per capita GDP growth. The coefficients on population growth rates, however, are consistently negative although some of them are not significant. Turning to the human capital proxy variables, we find that when the health and education proxy variables (public health expenditure as a percentage of GDP, student to teacher ratio for primary schools, public spending on education as a percentage of GDP) are added into the baseline regression individually (see equations (2) to (4) in Table 4.2), only health expenditure is statistically significant, although all of them are with the expected signs. Equations (5) to (6) in Table 4.2 show the regression results with health expenditure and one of the education proxy variables added into the baseline regression. The signs of the three human capital proxy variables are consistent with our expectation but the inclusion of different education proxy variables has different effects on the significant level of health expenditure. Although the inclusion of student to teacher ratio lowers the significant level of health expenditure, the effect of health expenditure on per capita GDP growth is still statistically significant. In contrast, if we consider the public spending on health and education simultaneously, the significance level of health expenditure would drop sharply while the coefficient on public spending on education would be significant. This result indicates that when we consider the public spending of health and education

simultaneously, education spending seems to have a more significant effect on per capita GDP growth. Furthermore, we also consider the short-run effect of these human capital proxy variables on per capita GDP growth. The results are presented in equations (7) to (12) in Table 4.2. The coefficients of health expenditures are consistently positive and significant except equation (10), where both the level and changes of public spending on education are included together with health expenditure into the baseline regression. However, if we consider both the levels and changes of health and education expenditures simultaneously, we find that both health and education expenditure are statistically significant and the effect of health expenditure is statistically more significant than that of education expenditure. For the changes of health expenditure on per capita GDP growth, we find that there are correction effects to the long term growth trend, which is indicated by the negative signs on the coefficients of the health expenditure changes. We further find that both the changes of health and education expenditure correct the growth rate to the long term path, although some of the effects are not statistically significant. The 12 regressions on the world data show that the effect of health expenditure on economic growth is at least non-negative and most of the regressions even show statistically significant effects of health expenditure on economic growth even when we also consider the short-run changes on health and education proxy variables. Furthermore, not only the levels of health spending have non-negative effect on economic growth, the changes on health expenditure may also help to correct the short run fluctuation of per capita GDP back to the long term growth trend. The comparison of health and education expenditure shows that if we consider both the levels and

changes of these two kinds of expenditure, both of them have statistically significant positive effect on economic growth while if we do not consider the short-run changes of these two forms of human capital expenditure simultaneously, expenditure on education would have a more significant positive effect on per capita GDP growth.

To make comparison, we also analyze an East Asia dataset. The statistical results are shown in Table 4.3. Firstly, we check the three core variables in the baseline regression. We find that the coefficients of both the lagged per capita GDP and physical capital investment are consistently with the expected signs while the signs on the coefficients of population growth rate are mixed and consistently insignificant. The significance levels of lagged per capita GDP are similar to those obtained from world data. Although the signs of the coefficients on physical capital investment are positive, most of them are not significant. The effects of the two core variables, physical capital investment and population growth rate, on per capita GDP growth are not as significant as those in the world data. However, the performance of the three human capital proxy variables is much better than those we get from the regression on world dataset. Comparing the 12 regressions, we notice that the coefficients of health expenditure are consistently positive and significant. Expect for the consistent significant coefficients, we find that one unit change in health expenditure will cause more than 0.12 unit change in per capita GDP growth in the East Asia dataset. By contrast, on average only 0.05 unit of change in per capita GDP growth will occur for one unit change in health expenditure for the world dataset. When we add these three health and education proxy variables into the baseline regression individually (see equations (2) to (4) in

Table 4.3), all of them are statistically significant with expected signs. In contrast, only health expenditure has statistically significant effect on per capital GDP growth when we use the world dataset (see equations (2) to (4) in Table 4.2). If we include both the health and education proxy variables into the baseline equation (see equations (5) and (6) in Table 4.3), the effects of both health expenditure and the two education proxy variables are statistically significant. The inclusion of the level of public spending on education into the baseline regression with health expenditure does not alleviate the positive effect of health expenditure on per capita GDP growth to an insignificant level, although the inclusion of the level of public spending on education does lower the effect of health expenditure. Equations (7) to (12) of Tables 4.3 presents the regression results of the case when short-run changes in the three kinds of human capital are also considered in the baseline regression. We find that the inclusion of these terms of changes in the human capital does not affect the effect of health expenditure on economic growth. Consistent with the results obtained from the world dataset, short run changes in health expenditure would correct the fluctuation of per capita GDP growth back to the long run trend. For the two education proxy variables, the effects of student to teacher ratio for schools on per capita GDP growth are more significant in this East Asia dataset than in the world dataset while the effects of public spending on education on per capita GDP growth are a little 'fragile', depending on the inclusion of the level and change of the health expenditure variable. The statistical results for this East Asia dataset show that the effect of health expenditure on economic growth is statistically more significant in East Asia than in the world, with both the level and change of health expenditure having

consistent significant statistical effect on per capita GDP growth. The effects of the two education proxy variables on per capita GDP are also more significant when we use the East Asia dataset, although the effect of public spending on education on per capita GDP growth still depends on the inclusion of level or change of health expenditure.

In addition to the above analysis on the augmented Bassanini and Scarpetta (2001) model, we also analyze the effect of health expenditure on economic growth using the augmented Mankiw, Romer and Weil (1992) model. We follow the procedures of analyzing the augmented Bassanini and Scarpetta (2001) model by first studying the world dataset and then comparing the statistical results derived from regressions on the world dataset with those from the East Asia dataset. Firstly, we analyze the regression results of the world dataset, which are presented in Table 4.4. There are statistical results of 6 regressions in Table 4.4. Like the analysis of the augmented Bassanini and Scarpetta (2001) model, we first regress the level of *RGDP* on the three core variables. The regression results of this baseline regression show that all the three core variables are statistically significant and with the correct signs. Comparing the coefficients and significance levels of these three proxy variables, we find that the signs of the coefficients of these variables are consistent in all the 6 regressions. Except for the one in regression (2) for the 'workforce variable', all the other variables are statistically significant. Secondly, we add each of the three human capital proxy variables into the baseline regression, one for each time (see equations (2) to (4) of Table 4.4). The results are in accord with those from the augmented Bassanini and Scarpetta (2001) model with only the coefficient on health expenditure statistically significant. The

coefficients on student to teacher ratio for primary schools and public spending on education are both with the correct signs but not significant. Lastly, we include health expenditure and one of the two education proxy variables into the baseline equations (see equations (5) and (6) of Table 4.4). The two education proxy variables are both significant and with expected signs. We obtain the coefficients of -0.04 and 0.02 for PTRATIO and PE, respectively, from the regressions with no health expenditure. We also can get another set of coefficients -0.09 and 0.04 on PTRATIO and PE, respectively, from the regressions with health expenditure included in the regression. The comparison of these two pairs of coefficients on PTRATIO and PE indicates that the inclusion of health expenditure simultaneously with the two education proxy variables would increase their effects on per capita GDP. Nevertheless, for health expenditure, if we consider it together with education proxy variable at the same time, its effect on per capita GDP will decrease. Similar to what we find from the regression results of the augmented Bassanini and Scarpetta (2001) model, if we include both health and education expenditure into the baseline equation, the effect of health expenditure on per capita GDP will turn insignificant and the effect of public spending on education would be more significant than that of health expenditure.

We follow the procedure of analyzing the augmented Bassanini and Scarpetta (2001) model by considering the case of East Asia. The regression results are shown in Tables 4.5. The comparison of the 6 regressions of Table 4.5 shows that the three core variables in the baseline regression are all statistically significant and with correct signs, which are consistent with the results in Table 4.4. Like the results we derive from the augmented

Bassanini and Scarpetta (2001) model, the performance of the three human capital proxy variables are a lot better in the East Asia case. We include each of the three health and education proxy variables into the baseline regression respectively (see equations (2) to (4) in Table 4.5). All the three variables are with the correct signs and statistically significant. If we consider health expenditure and the education proxy variable at the same time, the results in equations (5) and (6) of Table 4.5 indicate that the significance level of health expenditure on per capita GDP will not be affected. For the two education proxy variables, the inclusion of health expenditure would affect public spending on education but not student to teacher ratio for primary schools. The coefficients on health expenditure when public spending on education is included and not included are 0.48 and 0.49, respectively, which are much higher than those of public spending on education. situation is the same for the significant levels of these coefficients. It seems that public spending on health is favorable to education in East Asia. However, as argued by Li and Huang (2009), who analyze one of the key members of East Asia countries, China, this statistically more favorable position of health may be the result of the higher investment in health than education. We examine the summary statistics of Table 4.1 and find that the average percentage of public health expenditure in GDP is higher than that of education in all the regions. There is perhaps a trade-off between the effectiveness of investment and its related cost.

4.4.2 Robustness check of the LSDV results

As stated in section 4.3.2, although LSDV has relatively better performance on Monte Carlo simulations, this method can not solve the

problem of endogenity, which is one of the common problems in panel data estimation. The previous estimation done by LSDV could be biased if endogenity appears. It is necessary to do a robustness check on those baseline estimation results. In this subsection, we will perform 2SLS, one-step and two-steps GMM, and LSDVC estimation on the world dataset to do the robustness check. The most important issue is to check whether the effect of health expenditure on economic growth and production output is sensitive to different panel estimation methods. We first re-examine the above statistical results by employing the 2SLS, where we choose the lagged values of the health and education proxy variables as instrumental variables. The regression results show great similarity between the fixed effect estimations and those of the 2SLS estimation in terms of the signs of the human capital proxy variables. We find that all these three human capital proxy variables are with the expected signs, although some of them are statistically insignificant due to significant drop in number of observations. This finding is consistent with Knowles and Owen (1995), Webber (2002) and Li and Huang (2009). The statistical results of the two-stage fixed effect estimations for world are shown in Tables 4.6 and 4.7.

As stated in Nickel (1981) and in section 3 of this essay, the LSDV estimator is theoretically inconsistent in dynamic panel data models such as the one used in this essay. One theoretically consistent estimator with higher efficiency level is the GMM estimator from Arellano and Bond (1991), although this asymptotically better estimator does not perform as well as the LSDV estimator in the small sample Growth-Convergence model Monte Carlo simulations of Islam (2000). With the adoption of a robust estimator of the covariance matrix of the parameters to be estimated in the GMM

estimation, GMM estimators could solve the problem of heteroskedasticity and autocorrelation within panels. Therefore, we also perform GMM estimation on the two models we study in this essay. As a comparison, LSDVC estimation is also included together with the GMM results for the augmented Mankiw, Romer and Weil (1992) model. The estimation results are presented in Tables 4.8 and 4.9. Table 4.8 presents the results of the augmented Bassanini and Scarpetta (2001) model. The three baseline variables together with health expenditure are included in all the 8 equations in Table 4.8. Various combinations of levels and differences of the three human capitals are included together with the baseline variables to check the sensitivity of the effect of health expenditure on economic growth. Equations (1) and (3) are the one-step GMM estimation of the equation with only one form of the education proxy included respectively. Equations (2) and (4) are the corresponding two-steps GMM estimations. Equations (5) to (8) are the corresponding one-step and two-steps GMM estimations with the difference terms of human capital included in the regression. From the regression results of Table 4.8, we find that all the three human capital proxy variables have expected signs. The coefficients on health expenditure are consistently positive, although they are not significant in equations (1) and (2). The difference terms of health expenditure are consistently negative, which indicate that the short term fluctuations will be corrected back to the long-term trend and the long-run relationship between health expenditure and economic growth would be stable. The two education proxy variables are with expected signs, but their significance level is a little low. Table 4.9 shows the statistical results of augmented Mankiw, Romer and Weil (1992) model. The first three equations are respectively the one-step GMM,

two-steps GMM and LSDVC estimations for the regression with PTRATIO as proxy for education while the remaining three regressions are the corresponding regressions with *PE* as proxy for education. The coefficients of health expenditure are consistently positive but some of them are not significant. Similar to health expenditure, the two education proxy variables are with correct signs but not consistently significant. Especially, the significance levels of the LSDVC estimations (equations (3) and (6) of Table 4.9) are lower than their GMM counterparts. By comparing the GMM and LSDVC estimation results with those of LSDV, we find that they are consistent in signs but lower in significance levels for the GMM and LSDVC estimations. The reasons may be the loss of observations caused by the use of lagged terms as instruments, the bias correction process and the difference method used in GMM. However, after these robustness and sensitivity checking using 2SLS, GMM and LSDVC estimators, we still can draw the conclusion that health expenditure at least have non-negative effect on economic growth.

4.5 Concluding Remarks

The previous two essays analyze theoretically the relationship between health and long run economic growth and the mechanism of how health generates the "development traps". Both these two essays confirm the role of investment in health in promoting economic growth. Inspired by the prominent development of the endogenous growth theory in mid 1980s, there is a considerable amount of empirical studies on economic growth. However, most of them focus on education as human capital. One prototypical example of these empirical studies on the relationship between

education human capital and economic growth is Mankiw, Romer and Weil (1992), which is augmented by Knowles and Owen (1995) to consider the role of health as another important form of human capital. The macroeconomic empirical literature on the relationship between health and economic growth focus on using aggregate data to analyze whether there is statistical relationship of health on economic growth. They generally analyze the nexus among social health expenditure, social health indicators and economic outcome. For the current empirical studies on the links among health expenditure, social health indicators and economic outcome, most of the studies concentrate on how investment in health care or health cure services can improve the social health status, which is reflected by the commonly used indicators like life expectancy and infant mortality rate. There are also substantial amount of studies investigating the link between those social health indicators and economic growth. However, relatively little studies gauges the effect of social health expenditure on economic growth directly.

In this paper, we contribute to the current macroeconomic empirical literature by assessing the statistical relationship between social health expenditure on economic growth. We employ both the augmented Bassanini and Scarpetta (2001) model and the augmented Mankiw, Romer and Weil (1992) model to evaluate the health expenditure and growth relationship. The model proposed in Bassanini and Scarpetta (2001) is a model with per capita GDP growth rate as dependent variable and both the levels and changes of the human capital variables are included as independent variables. This model is one form of the "error-correction" model widely used in time series literature. We extend the Bassanini and Scarpetta (2001)

model by incorporating both health and education as human capital. To make an in depth analysis on the health expenditure and growth relationship, we also analyze the augmented Mankiw, Romer and Weil (1992) model following the procedures proposed by Knowles and Owen (1995). There are two dataset studied in this essay: one is a world dataset consisting of 138 countries with a time span from 1975 to 2007. The other dataset used for comparison is an East Asia dataset composed of 10 countries with the same time coverage. The estimation method we use is fixed effect panel estimation which minimizes the problem of homogeneity and provides satisfactory small sample properties. To check the robustness of our baseline estimation results from LSDV, we also employ the 2SLS, GMM and LSDVC estimators to check the problem of endogenity, heterogeneity and potential serial correlation in error terms.

The statistical results from the extended Bassanini and Scarpetta (2001) model show that the statistical effect of health expenditure on per capita GDP growth are consistently significant except when we consider the levels of public health expenditure and public spending on education simultaneously. In this case, the inclusion of public spending on education would lead to insignificant positive effect of health expenditure on economic growth. However, if we also consider the short-run effects of these two human capital expenditures, the effect of health expenditure is more significant than that of education expenditure. In contrast, the statistical results using the East Asia dataset show that the effect of health expenditure on economic growth are not affected by the simultaneously inclusion of public spending on health and education into the baseline regression. Furthermore, the effects of the three health and education proxy

variables derived from the East Asia dataset are higher and statistically more significant than those obtained from the regressions on the world dataset. Moreover, the statistical results also indicate that the short-run changes in health expenditure correct the fluctuation of per capita GDP growth rate back to the long term trend.

We also consider the augmented Mankiw, Romer and Weil (1992) model. Similar to what we find from the regression results of the augmented Bassanini and Scarpetta (2001) model, if we include both health and education expenditure into the baseline equation, the effect of health expenditure on per capita GDP will turn insignificant and the effect of public spending on education would be more significant than that of health expenditure. The analysis of the East Asia dataset shows that the performance of the three human capital proxy variables are a lot better in the East Asia case, which is consistent with the findings in the augmented Bassanini and Scarpetta (2001) model. The coefficients and significance levels of health expenditure when both health and education public expenditures are included in the baseline regression are higher than those of the public education expenditure in the East Asia case. It seems that public spending on health is superior to education in promoting economic growth in East Asia. However, in a similar study analyzing the case of a group of 10 East Asia countries, Li and Huang (2010) argue that this statistically more favorable position of health in East Asia may attribute to the higher investment in health than education. This is the case as shown in the summary statistics in Table 4.1.

The robustness and sensitivity check by employing the 2SLS, GMM and LSDVC estimators confirm the robustness of the effects of health

expenditure on both economic growth and production output. Thus, together with the baseline statistical results from the augmented Bassanini and Scarpetta (2001) model and the augmented Mankiw, Romer and Weil (1992) model, we can safely draw the conclusion that the effect of health expenditure on economic growth is at least non-negative in the world and positive in East Asia. Indeed, if we consider both the short-run and long-run effect of the two forms of human capital expenditures, health expenditure would be statistically more significant than education expenditure. The statistical effect of health and education on economic growth is much higher in East Asia than in the world. The consideration of public education expenditure would affect the effect of health expenditure on economic growth for the case of the world, while the positive effect of health expenditure on economic growth will not be affected by the inclusion of education proxy variables for the case of East Asia. The statistical favorable position of public health expenditure over public spending on education in East Asia may be attributable to the higher percentage of public health expenditure to GDP than that of education.

The results from the first two theoretical essays indicate that health is an important factor that affects economic growth. The overall effect of health on economic growth depends on the exact form of the health accumulation function, which is a function of health and health depreciation rate. Different specifications of the health accumulation function may lead to different results of endogenous growth. In this empirical essay, the statistical results show that health at least has non-negative effect on long run economic growth, which is consistent with cases 3 and 6 of essay 1, which state that when health and education enter into the function of health

depreciation rate, there would be endogenous growth. The suggestions to the policymakers are that public health expenditure is an effective measure to stimulate economic growth. To achieve the Millennium Development Goals released by the United Nations, there are only five years remained for countries to implement more efficient policies and strengthen the macroeconomic environment to stimulate development in public health services. The current difficult time of post-financial tsunami also requires policymakers to spend the money more wisely and efficiently in providing sufficient health service to the public, which is extremely important to the developing countries.

In the future study, improvement on this essay can be made if higher quality data and extra observations on health expenditure are available. Moreover, we may try more advanced estimation methods which may have better performance on the nonstationary data.

Appendix 4.1 Theoretical Model

The theoretical framework of this paper follows the procedures of Knowles and Owen's (1995), Islam (1995), and Bassanini and Scarpetta (2001). We consider the extended Solow model to include human capital in two forms – health and education - in a panel data case. The model assumes an aggregate production function with constant returns to scale. The Cobb-Douglas production function with labor-augmenting technological progress, for country i and time period t, is:

$$Y_{it} = K_{it}^{\alpha} S_{it}^{\beta} H_{it}^{\gamma} (A_{it} L_{it})^{1-\alpha-\beta-\gamma}, \qquad 0 < \alpha, \beta, \gamma < 1 \text{ and } \alpha + \beta + \gamma < 1 \quad (4.1.1)$$

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

$$L_{it} = L_{i0} e^{n_{it} t} \quad (4.1.2)$$

$$A_{it} = A_{i0} e^{g_{it} t} \quad (4.1.3)$$

Further, Knowles and Owen assume that the growth rates n_{it} and g_{it} are exogenously given, i.e., $n_{it} = n_i$ (assumed to be the same over time for country i), and $g_{it} = g$ (same for all countries and over time). The growth rate of the number of effective unit of labor, $A_{it} L_{it}$, is therefore $n_i + g$. The rates of savings, population growth and technological progress are constant and are exogenously given.

Define s_{ki}, s_{si}, and s_{hi} as the constant fractions of output that is invested in physical capital, education, and health, respectively, for country *i*. Define $\tilde{k}$, $\tilde{s}$, and $\tilde{h}$ as the stocks of physical capital, education, and health per effective unit of labor, respectively, i.e., $\tilde{k} = K/(AL)$, $\tilde{s} = S/(AL)$, $\tilde{h} = H/(AL)$. Similarly, define $\tilde{y}$ as the level of output per effective unit of labor, i.e., $\tilde{y} = Y/(AL)$. Therefore the output *y* can be written as

$$\tilde{y}_{it} = \tilde{k}_{it}^{\alpha}\tilde{s}_{it}^{\beta}\tilde{h}_{it}^{\gamma} \tag{4.1.4}$$

The dynamics of $\tilde{k}$, $\tilde{s}$, and $\tilde{h}$ are given as the following:

$$\dot{\tilde{k}}_{it} = s_{ki}\tilde{y}_{it} - (n_i + g + \delta)\tilde{k}_{it} = s_{ki}\tilde{k}_{it}^{\alpha}\tilde{s}_{it}^{\beta}\tilde{h}_{it}^{\gamma} - (n_i + g + \delta)\tilde{k}_{it} \tag{4.1.5}$$

$$\dot{\tilde{s}}_{it} = s_{si}\tilde{y}_{it} - (n_i + g + \delta)\tilde{s}_{it} = s_{si}\tilde{k}_{it}^{\alpha}\tilde{s}_{it}^{\beta}\tilde{h}_{it}^{\gamma} - (n_i + g + \delta)\tilde{s}_{it} \tag{4.1.6}$$

$$\dot{\tilde{h}}_{it} = s_{hi}\tilde{y}_{it} - (n_i + g + \delta)\tilde{h}_{it} = s_{hi}\tilde{k}_{it}^{\alpha}\tilde{s}_{it}^{\beta}\tilde{h}_{it}^{\gamma} - (n_i + g + \delta)\tilde{h}_{it} \tag{4.1.7}$$

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

$$\tilde{k}_i^* = \left(\frac{s_{ki}^{1-\beta-\gamma} s_{si}^{\beta} s_{hi}^{\gamma}}{n_i + g + \delta} \right)^{1/\theta} \tag{4.1.8}$$

$$\tilde{s}_i^* = \left(\frac{s_{ki}^{\alpha} s_{si}^{1-\alpha-\gamma} s_{hi}^{\gamma}}{n_i + g + \delta} \right)^{1/\theta} \tag{4.1.9}$$

$$\tilde{h}_i^* = \left(\frac{s_{ki}^{\alpha} s_{si}^{\beta} s_{hi}^{1-\alpha-\beta}}{n_i + g + \delta} \right)^{1/\theta} \tag{4.1.10}$$

with $\theta = 1 - \alpha - \beta - \gamma$. Define $p_i = n_i + g + \delta$. Substituting equations (4.1.3), (4.1.8), (4.1.9), and (4.1.10) into the production function (4.1.4) and taking logs, we obtain the implied steady-state income per capita:

$$\ln y_i^* = \ln A_{i0} + gt + \frac{\alpha}{\theta}\ln s_{ki} + \frac{\beta}{\theta}\ln s_{ni} + \frac{\gamma}{\theta}\ln s_{hi} - \frac{1-\theta}{\theta}\ln p_{ni} \qquad (4.1.11)$$

where $y_i^* = Y_i^*/L_i^*$ is the steady state per capita output for country *i* at time *t*.

The equation derived is about what determines the level of income per capita. However what we are interested in is the determinants of economic growth. As a result, we follow the ideas of linearization from Bassanini and Scarpetta (2001), Mankiw et al. (1992) and Romer (2002) to convert the level equation (A.11) to get the growth equation. We first define $\tilde{y}_i^*$ as the steady level of income per effective unit of labor, and $\tilde{y}_{ni}$ as its value at any time *t* for country *i* . The rate of convergence is given as:

$$\frac{d\ln \tilde{y}_{ni}}{dt} = \lambda_i[\ln \tilde{y}_i^* - \ln \tilde{y}_{ni}] \qquad (4.1.12)$$

where $\lambda_i = (n_i + g + \delta)(1 - \alpha - \beta - \gamma) = (n_i + g + \delta)\theta$.[3] Equation (4.1.12) implies:

$$\ln \tilde{y}_{ni_2} = (1 - e^{-\lambda_i \tau})\ln \tilde{y}_i^* + e^{-\lambda_i \tau}\ln \tilde{y}_{ni_1} \qquad (4.1.13)$$

where $\tau = t_2 - t_1$. We therefore can easily get:

$$\ln \tilde{y}_{ni_2} - \ln \tilde{y}_{ni_1} = (1 - e^{-\lambda_i \tau})(\ln \tilde{y}_i^* - \ln \tilde{y}_{ni_1}) \qquad (4.1.14)$$

[3] See Barro & Sala-i-Martin (1995, pp. 87-8) for derivation.

Substituting equation (4.1.11) into equation (4.1.14) yields

$$\ln \tilde{y}_{it_2} - \ln \tilde{y}_{it_1} = (1-e^{-\lambda t})\left(\frac{\alpha}{\theta}\ln s_{kit} + \frac{\beta}{\theta}\ln s_{sit} + \frac{\gamma}{\theta}\ln s_{hit} - \frac{1-\theta}{\theta}\ln p_{it} - \ln \tilde{y}_{it_1}\right) \quad (4.1.15)$$

Since $\tilde{y}_{it}$ is the income per effective labor and we are interested in income per capita, we can substitute $\ln \tilde{y}_{it} = \ln y_{it} - \ln A_{0i} - gt$ into equation (4.1.15) and get the following growth equation:

$$\begin{aligned}\ln y_{it_2} = {} & e^{-\lambda t}\ln y_{it_1} + (1-e^{-\lambda t})\frac{\alpha}{\theta}\ln s_{kit} + (1-e^{-\lambda t})\frac{\beta}{\theta}\ln s_{sit} \\ & + (1-e^{-\lambda t})\frac{\gamma}{\theta}\ln s_{hit} - (1-e^{-\lambda t})\frac{1-\theta}{\theta}\ln p_{it} \\ & + (1-e^{-\lambda t})\ln A_{0i} + g(t_2 - e^{-\lambda t}t_1)\end{aligned} \quad (4.1.16)$$

By some further algebra manipulations, we obtain a growth equation with the economic growth rate as the dependent variable:

$$\begin{aligned}\Delta \ln y_{it} = {} & -(1-e^{-\lambda t})\ln y_{it_1} + (1-e^{-\lambda t})\frac{\alpha}{\theta}\ln s_{kit} + (1-e^{-\lambda t})\frac{\beta}{\theta}\ln s_{sit} \\ & + (1-e^{-\lambda t})\frac{\gamma}{\theta}\ln s_{hit} - (1-e^{-\lambda t})\frac{1-\theta}{\theta}\ln p_{it} + (1-e^{-\lambda t})\ln A_{0i} \\ & + g(t_2 - e^{-\lambda t}t_1)\end{aligned} \quad (4.1.17)$$

Following the argument of Bassanini and Scarpetta (2001) that the parameter g cannot be distinguished from constant term empirically, equations (4.1.16) and (4.1.17) could be expressed as follows:

$$\ln y_{it_2} = \alpha_{10} + \alpha_{11}\ln y_{it_1} + \alpha_{12}\ln s_{kit} + \alpha_{13}\ln s_{sit} + \alpha_{14}\ln s_{hit} - \xi_1 \ln p_{it} + \varepsilon_{1it} \quad (4.1.18)$$

$$\Delta \ln y_{it} = a_{10} - \varphi_1 \ln y_{it_1} + a_{11}\ln s_{kit} + a_{12}\ln s_{sit} + a_{13}\ln s_{hit} - \psi_1 \ln p_{it} + \varepsilon_{1it} \quad (4.1.19)$$

To account for the possible short-run effect from the two dimensions of human capital in the data, we add short-run regressors into equation (4.1.19),

which is an error-correction model:

$$\Delta \ln y_{it} = a_{10} - \varphi \ln y_{it_1} + a_{11} \ln s_{kt} + a_{12} \ln s_{st} + a_{13} \ln s_{ht} - \psi_1 \ln p_{it} + b_{11} \Delta \ln s_{st} + b_{12} \Delta \ln s_{ht} + \varepsilon_{1t} \tag{4.1.20}$$

Equations (4.1.18) and (4.1.19) investigate the effects of investment in human capital on economic growth. However, if we are interested in the level of human capital stock on economic growth, we can convert equations (4.1.9) and (4.1.10) to express s_{st} and s_{ht} in terms of $\tilde{s}_t^*$ and $\tilde{h}_t^*$ respectively and substitute those converted expressions into equation (4.1.16). The resulting equations are:

$$\begin{aligned} \ln y_{it_2} = {} & e^{-\lambda_1 \tau} \ln y_{it_1} + (1 - e^{-\lambda_1 \tau}) \frac{\alpha}{1-\alpha-\gamma} \ln s_{kt} + (1 - e^{-\lambda_1 \tau}) \frac{\beta}{1-\alpha-\gamma} \ln \tilde{s}_t^* \\ & + (1 - e^{-\lambda_1 \tau}) \frac{\gamma}{1-\alpha-\gamma} \ln s_{ht} - (1 - e^{-\lambda_1 \tau}) \frac{(\alpha+\gamma)}{1-\alpha-\gamma} \ln p_{it} \\ & + (1 - e^{-\lambda_1 \tau}) \ln A_{0t} + g(t_2 - e^{-\lambda_1 \tau} t_1) \end{aligned} \tag{4.1.21}$$

$$\begin{aligned} \ln y_{it_2} = {} & e^{-\lambda \tau} \ln y_{it_1} + (1 - e^{-\lambda_1 \tau}) \frac{\alpha}{1-\alpha-\beta} \ln s_{kt} + (1 - e^{-\lambda_1 \tau}) \frac{\beta}{1-\alpha-\beta} \ln s_{st} \\ & + (1 - e^{-\lambda_1 \tau}) \frac{\gamma}{1-\alpha-\beta} \ln \tilde{h}_t^* - (1 - e^{-\lambda_1 \tau}) \frac{(\alpha+\beta)}{1-\alpha-\beta} \ln p_{it} \\ & + (1 - e^{-\lambda_1 \tau}) \ln A_{0t} + g(t_2 - e^{-\lambda \tau} t_1) \end{aligned} \tag{4.1.22}$$

$$\begin{aligned} \ln y_{it_2} = {} & e^{-\lambda \tau} \ln y_{it_1} - (1 - e^{-\lambda_1 \tau}) \frac{\alpha}{1-\alpha} [\ln s_{kt} - \ln p_{it}] \\ & + (1 - e^{-\lambda_1 \tau}) \frac{\beta}{1-\alpha} \ln \tilde{s}_t^* + (1 - e^{-\lambda_1 \tau}) \frac{\gamma}{1-\alpha} \ln \tilde{h}_t^* \\ & + (1 - e^{-\lambda \tau}) \ln A_{0t} + g(t_2 - e^{-\lambda \tau} t_1) \end{aligned} \tag{4.1.23}$$

From equations (4.1.6), (4.1.7), (4.1.9) and (4.1.10), we have expressions for $\ln s^*$ and $\ln h^*$:

$$\ln \tilde{s}_{it_2} = (1 - \varphi) \ln \tilde{s}_t^* + \varphi \ln \tilde{s}_{it_1} \Leftrightarrow \ln \tilde{s}_t^* = \ln \tilde{s}_{it_2} + \frac{1-\varphi}{\varphi} \Delta \ln (\tilde{s}_{it}) \tag{4.1.24}$$

$$\ln \tilde{h}_{it_2} = (1-\varphi)\ln \tilde{h}_i^* + \varphi \ln \tilde{h}_{it_1} \Leftrightarrow \ln \tilde{h}_i^* = \ln \tilde{h}_{it_2} + \frac{1-\varphi}{\varphi}\Delta \ln\left(\tilde{h}_{it}\right) \qquad (4.1.25)$$

Inserting equations (4.1.24) and (4.1.25) into equations (4.1.21) to (4.1.23) and the corresponding equations with growth rate as dependent variable are:

$$\begin{aligned}\Delta \ln y_{it} = {} & (1-e^{-\lambda t})\ln y_{it_1} + (1-e^{-\lambda t})\frac{\alpha}{1-\alpha-\gamma}\ln s_{kit} \\ & + (1-e^{-\lambda t})\frac{\beta}{1-\alpha-\gamma}\ln \tilde{s}_{it_2} \\ & + (1-e^{-\lambda t})\frac{\gamma}{1-\alpha-\gamma}\ln s_{hit} - (1-e^{-\lambda t}) \\ & + (1-e^{-\lambda t})\frac{1-\varphi}{\varphi}\frac{\beta}{1-\alpha-\gamma}\Delta \ln\left(\tilde{s}_{it}\right) + \frac{(\alpha+\gamma)}{1-\alpha-\gamma}\ln p_{it} \\ & + (1-e^{-\lambda t})\ln A_{0i} + g(t_2 - e^{-\lambda t}t_1)\end{aligned} \qquad (4.1.26)$$

$$\begin{aligned}\Delta \ln y_{it} = {} & (1-e^{-\lambda t})\ln y_{it_1} + (1-e^{-\lambda t})\frac{\alpha}{1-\alpha-\beta}\ln s_{kit} \\ & + (1-e^{-\lambda t})\frac{\beta}{1-\alpha-\beta}\ln s_{eit} + (1-e^{-\lambda t})\frac{\gamma}{1-\alpha-\beta}\ln \tilde{h}_{it_2} \\ & + (1-e^{-\lambda t})\frac{\gamma}{1-\alpha-\beta}\frac{1-\varphi}{\varphi}\Delta \ln\left(\tilde{h}_{it}\right) - (1-e^{-\lambda t})\frac{(\alpha+\beta)}{1-\alpha-\beta}\ln p_{it} \\ & + (1-e^{-\lambda t})\ln A_{0i} + g(t_2 - e^{-\lambda t}t_1)\end{aligned} \qquad (4.1.27)$$

$$\begin{aligned}\Delta \ln y_{it} = {} & (1-e^{-\lambda t})\ln y_{it_1} - (1-e^{-\lambda t})\frac{\alpha}{1-\alpha}[\ln s_{kit} - \ln p_{it}] \\ & + (1-e^{-\lambda t})\frac{\beta}{1-\alpha}\ln \tilde{s}_{it_2} + (1-e^{-\lambda t})\frac{\gamma}{1-\alpha}\ln \tilde{h}_{it_2} \\ & + (1-e^{-\lambda t})\frac{\gamma}{1-\alpha}\frac{1-\varphi}{\varphi}\Delta \ln\left(\tilde{h}_{it}\right) \\ & + (1-e^{-\lambda t})\frac{\beta}{1-\alpha}\frac{1-\varphi}{\varphi}\Delta \ln\left(\tilde{s}_{it}\right)(1-e^{-\lambda t})\ln A_{0i} \\ & + g(t_2 - e^{-\lambda t}t_1)\end{aligned} \qquad (4.1.28)$$

Using the same argument as for deriving equations (4.1.18) and (4.1.20), we have the corresponding equations for equations (4.1.21) to (4.1.23) and (4.1.26) to (4.1.28):

$$\ln y_{it_2} = \alpha_{20} + \alpha_{21} \ln y_{it_1} + \alpha_{22} \ln s_{ki} + \alpha_{23} \ln s_i^* + \alpha_{24} \ln s_{hi} - \xi_2 \ln p_{it} + \varepsilon_{2i} \quad (4.1.21')$$

$$\ln y_{it_2} = \alpha_{30} + \alpha_{31} \ln y_{it_1} + \alpha_{32} \ln s_{ki} + \alpha_{33} \ln s_{ei} + \alpha_{34} \ln h_i^* - \xi_3 \ln p_{it} + \varepsilon_{3i} \quad (4.1.22')$$

$$\ln y_{it_2} = \alpha_{40} + \alpha_{41} \ln y_{it_1} + \alpha_{42} \ln s_{ki} + \alpha_{43} \ln s_i^* + \alpha_{44} \ln h_i^* - \xi_4 \ln p_{it} + \varepsilon_{4i} \quad (4.1.23')$$

$$\Delta \ln y_{it} = a_{20} - \varphi_2 \ln y_{it_1} + a_{21} \ln s_{ki} + a_{22} \ln s_i^* + a_{23} \ln s_{hi} - \psi_2 \ln p_{it} + b_{21} \Delta \ln s_{ei} + b_{22} \Delta \ln s_{hi} + \varepsilon_{2i} \quad (4.1.26')$$

$$\Delta \ln y_{it} = a_{30} - \varphi_3 \ln y_{it_1} + a_{31} \ln s_{ki} + a_{32} \ln s_{ei} + a_{13} \ln h_i^* - \psi_3 \ln p_{it} + b_{31} \Delta \ln s_{ei} + b_{32} \Delta \ln s_{hi} + \varepsilon_{3i} \quad (4.1.27')$$

$$\Delta \ln y_{it} = a_{40} - \varphi_4 \ln y_{it_1} + a_{41} \ln s_{ki} + a_{42} \ln s_i^* + a_{43} \ln h_i^* - \psi_4 \ln p_{it} + b_{41} \Delta \ln s_{ei} + b_{42} \Delta \ln s_{hi} + \varepsilon_{4i} \quad (4.1.28')$$

The employment of the specific regression equations depends on whether the level or change of the variables is used.

Appendix 4.2 Technical details on econometric methods

Fixed effect estimator

Fixed effect estimator or LSDV estimator is one of the most commonly used panel estimators. For growth regressions, because the control variables are usually correlated with the unobserved individual effects, LSDV is preferable to random effects models. The Monte Carlo simulations results on the growth-convergence model performed by Islam show that LSDV is the preferred method in sample with small time observations but large number of countries. However, since we use panel data to capture the omitted individual effects, which are ignored by cross-section models, these individual effects are likely to correlate with the regressors. To see this, we see the following dynamic fixed effect model:

$$y_{it} = \gamma y_{i,t-1} + \beta x_{i,t} + \mu_i + \varepsilon_{it} \quad i = 1,\ldots,N;\ t = 1,\ldots,T \tag{4.2.1}$$

where N and T stand for total number of countries/regions and total number of time, respectively. γ is a scalar and β is $1\times(K-1)$, $x_{i,t}$ is $(K-1)\times 1$ of control variables. μ_i is fixed effect and ε_{it} follows independent identical distribution (i.i.d) $N(0,\sigma_\varepsilon^2)$.

Furthermore, we assume that:

$$E(\varepsilon_{it},\varepsilon_{js}) = 0 \quad \text{for} \quad i \neq j \quad \text{and} \quad t \neq s \tag{4.2.2}$$

$$E(x_{it}\varepsilon_{it}) = 0 \forall i,j,t,s \tag{4.2.3}$$

Subtracting equation (4.2.1) by its time mean, we get:

$$y_{it} - y_{i.} = \gamma(y_{i,t-1} - y_{i,t-1}) + \beta(x_{it-1} - x_{i.}) + (\varepsilon_{it} - \varepsilon_{i.}) \qquad (4.2.4)$$

where for any variable k_{it}, $k_{i.} = \frac{1}{T}\sum_{t=1}^{T} k_{it}$ is the time mean.

LSDV estimates the parameters γ and β by performing OLS on equation (4.2.4). Because both $y_{i,t-1}$ and $\varepsilon_{i.}$ contain ε_{it-1}, LSDV estimation is biased. Nickel (1981) estimates that the bias is of O(1/T).

GMM estimator

According to Hansen (1982), theoretically, one consistent estimator for dynamic panel data model is GMM estimator, which uses the orthogonality conditions between lagged values of y_{it} and the error terms to obtain additional "GMM-style" instruments. We first discuss the derivation of the GMM estimator and then apply the idea of GMM to the case of dynamic panel data estimation. Some of the results derived here were developed by Hansen (1982) and Hamilton (1994).

The GMM estimation is developed and generalized from the classical method of moments estimator. The general idea of the classical method of moments is as follows.

Given a vector of $(b \times 1)$ unknown parameters θ that characterize the distribution of an observed variable y_t. Assume that there are b distinct population moments of the random variable y_t can be characterized by the unknown parameters θ:

$$E(y_t^i) = f(\theta) \text{ for } i = i_1, i_2, \ldots, i_b \qquad (4.2.5)$$

The classical method of moments estimate of θ is to equate the

population moments of the random variable y_t to the corresponding observed sample moments:

$$f(\theta) = \frac{1}{T}\sum_{t=1}^{T} y_t^i \quad \text{for } i = i_1,\ i_2,\ \ldots, i_h \tag{4.2.6}$$

We take the normal method of moments as an example. Suppose x_1, x_2,..., x_T follow i.i.d. $N(\delta, \sigma^2)$ distribution. There are two unknown parameters: $\theta_1 = \delta$ and $\theta_2 = \sigma^2$. The first and second population moments are: $\mu_1 = \delta$ and $\mu_2 = \delta^2 + \sigma^2$. The corresponding sample first moment and second moment are: $\mu_1' = \bar{X} = \frac{1}{T}\sum_{t=1}^{T} x_t$ and $\mu_2' = \frac{1}{T}\sum_{t=1}^{T} x_t^2$. The classical method of moments is to solve the following two equations:

$$\delta = \frac{1}{T}\sum_{t=1}^{T} x_t \tag{4.2.7}$$

$$\delta^2 + \sigma^2 = \frac{1}{T}\sum_{t=1}^{T} x_t^2 \tag{4.2.8}$$

The resulting estimations are:

$$\delta_1 = \bar{x} \tag{4.2.9}$$

$$\sigma_1 = \frac{1}{T}\sum_{t=1}^{T} (x_t - \bar{x})^2 \tag{4.2.10}$$

In the classical method of moments estimation, b population parameters require b simple moment conditions. If we have only one population parameter but two simple moment conditions, we are usually not able to choose a parameter estimate so as to satisfy two moment conditions. The idea to solve this problem is to choose a parameter estimate which is as close as possible to the two moment conditions by minimizing the following criterion function:

$$Q = \vartheta' W \vartheta \tag{4.2.11}$$

where ϑ is a (2×1) moment vector and W is a (2×2) weighting matrix.

This idea of using the minimizing criterion equation (4.2.11) to estimate the unknown parameters was called "minimum chi-square" by Cramer (1946), Ferguson (1958) and Rothenberg (1973) and "minimum distance estimator" by Malinvaud (1970). But the formal description of this idea was made by Hansen (1982) who called this method "generalized method of moments" (GMM) and derived the asymptotic properties for this method.

The description of the GMM estimation by Hansen (1982) is as follows. Let a_t be a $(j \times 1)$ vector of variables that are observed at time t and let δ denotes an unknown $(b \times 1)$ vector of parameters, and let $h(\delta, a_t)$ be an $(r \times 1)$ vector-value function: $h:(R^b \times R^j) \to R^r$. The r rows of the vector-value function are usually called orthogonality conditions. Let δ^* be the true value of δ which is characterized by the property that:

$$E\{h(\delta^*, a_t)\} = 0 \tag{4.2.12}$$

Let $\phi_T \equiv (a_1', a_2', ..., a_T')$ be a $(Tj \times 1)$ vector containing all the sample observations and denote the $(r \times 1)$ vector-value function $\vartheta(\delta, \phi_T)$ as the sample average of $h(\delta, a_t)$:

$$\vartheta(\delta, \phi_T) = \frac{1}{T}\sum_{t=1}^{T} h(\delta, a_t) \tag{4.2.13}$$

The idea of the GMM estimation is to choose δ so as to make the sample moment $\vartheta(\delta, \phi_T)$ as close as to the population moment of zero. In other words, the GMM estimator $\tilde{\delta}$ is the one that minimizes the scalar:

$$Q(\delta, \phi_T) = \left[\vartheta(\delta, \phi_T)\right]' W_T \left[\vartheta(\delta, \phi_T)\right] \tag{4.2.14}$$

where W_T is a $r \times r$ weighting matrix.

The classical method of moment is a special case of the GMM estimation. For the example of normal distribution we showed earlier, we

have $r = b = 2$, $a_t = x_t$, $W_t = 1$

$$h(\delta, a_t) = \begin{bmatrix} x_t - \theta \\ x_t^2 - (\theta^2 + \sigma^2) \end{bmatrix} \tag{4.2.15}$$

and the corresponding function of $\vartheta(\delta, \phi_t)$ is:

$$\vartheta(\delta, \phi_T) = \begin{bmatrix} \frac{1}{T}\sum_{t=1}^{T} x_t - \theta \\ \frac{1}{T}\sum_{t=1}^{T} x_t^2 - (\theta^2 + \sigma^2) \end{bmatrix} \tag{4.2.16}$$

According to Hansen (1982), if the number of parameters to be estimated (b) equals to the number of orthogonality conditions (r), then the objective function $Q(\delta, \phi_t)$ would be minimized by setting:

$$\vartheta(\tilde{\delta}, \phi_T) = 0 \tag{4.2.17}$$

If the number of orthogonality conditions is greater than that of parameters to be estimated, the minimization would be determined by the optimal weighting matrix W_T.

Assume that the function $h(\delta, a_t)$ valued at the true parameter values, the process $\{h(\delta, a_t)\}_{t=-\infty}^{\infty}$ is a strictly stationary process with mean zero and autocovariance given by

$$\rho_d = E\left\{\left[h(\delta, a_t)\right]\left[h(\delta, a_{t-d})\right]'\right\} \tag{4.2.18}$$

Assume that the autocovariances are absolutely summable and we can define:

$$S = \sum_{d=-\infty}^{\infty} \rho_d \tag{4.2.19}$$

where from Hamilton (1994) we know that S is the asymptotic variance of the sample mean of $h(\delta, a_t)$:

$$S = \lim_{t \to \infty} T \cdot E\left\{\left[h(\delta, a_t)\right]\left[h(\delta, a_{t-d})\right]'\right\}$$

The optimal weighting matrix W_T for equation (4.2.14) is S^{-1} which minimizes equation (4.2.14) when $\bar{\delta}$ is chosen:

$$Q(\delta,\phi_T)=\left[\vartheta(\delta,\phi_t)\right]' S^{-1}\left[\vartheta(\delta,\phi_t)\right] \tag{4.2.20}$$

If the vector process $\{h(\delta,a_t)\}_{t=-\infty}^{\infty}$ is serially uncorrelated, S could be consistently estimated by

$$S^{*}=\frac{1}{T}\sum_{t=1}^{T}\left[h(\delta,a_t)\right]\left[h(\delta,a_{t-d})\right] \tag{4.2.21}$$

If the vector process $\{h(\delta,a_t)\}_{t=-\infty}^{\infty}$ is serially correlated, S could be estimated following Newey-West (1987):

$$S^{*}=\rho_{0,T}+\sum_{d=1}^{q}\left\{1-\left[\frac{d}{q+1}\right]\right\}\left(\rho_{v,T}+\rho_{v,T}'\right)$$

where $\rho_{v,T}=\frac{1}{T}\sum_{t=d+1}^{T}\left[h(\delta,a_t)\right]\left[h(\delta,a_{t-d})\right]'$.

The most important property of the GMM estimator is the property of consistency. We present the proof of the asymptotic distribution property of the GMM estimator, which was developed by Hansen (1982) as follows:

Firstly, we assume $\bar{\delta}$ as the value of parameters that minimize equation (B.20). Thus, this GMM estimator $\tilde{\delta}$ should be a solution to the following system of nonlinear equations:

$$\left\{\left.\frac{\partial\vartheta(\delta,a_T)}{\partial\delta'}\right|_{\delta=\tilde{\delta}_T}\right\}'\times\tilde{S}_T^{-1}\times\left[\vartheta\left(\tilde{\delta},a_T\right)\right]=0 \tag{4.2.22}$$

where $\left.\frac{\partial\vartheta(\delta,a_T)}{\partial\delta'}\right|_{\delta=\tilde{\delta}_T}$ is a $(r\times a)$ matrix of derivatives of the function of $\vartheta(\delta,a_T)$ with respect to δ where $r\geq a$, $\tilde{S}$ is a $(r\times r)$ weighting matrix with the property that $\tilde{S}\rightarrow S$ and $\vartheta\left(\tilde{\delta},a_T\right)$ is a $(r\times 1)$ vector

evaluated at $\tilde{\delta}$.

Hansen (1982) further assumes that:

$$\bar{\delta}_t \xrightarrow{p} \delta^* \tag{4.2.23}$$

$$\sqrt{T}\cdot\vartheta\left(\delta^*, a_T\right) \xrightarrow{d} N(0,S) \tag{4.2.24}$$

$$\text{plim}\left\{\left.\frac{\partial\vartheta(\delta,a_T)}{\partial\delta'}\right|_{\delta\ a_T}\right\} = \text{plim}\left\{\left.\frac{\partial\vartheta(\delta,a_T)}{\partial\delta'}\right|_{\delta=\delta_t^*}\right\} = \varphi' \tag{4.2.25}$$

for any sequence $\left\{\delta_t\right\}_{t=1}^{\infty}$ satisfying the condition that $\bar{\delta}_t \xrightarrow{p} \delta^*$.

Let $\vartheta_i(\delta, a_T)$ be the ith element of $\vartheta(\delta, a_T)$ and by the first order Taylor expansion, we have:

$$\vartheta_i\left(\tilde{\delta}, a_T\right) = \vartheta_i\left(\delta^*, a_T\right) + \left[\left.\frac{\partial\vartheta_i(\delta,a_T)}{\partial\delta}\right|_{\delta=\hat{\delta}_{i,T}}\right]'\left(\tilde{\delta}-\delta^*\right) \tag{4.2.26}$$

for some $\hat{\delta}_{i,T}$ between δ^* and $\tilde{\delta}$. We also define that:

$$D_T' = \begin{bmatrix} \left[\left.\frac{\partial\vartheta_1(\delta,a_T)}{\partial\delta}\right|_{\delta=\hat{\delta}_{1,T}}\right]' \\ \left[\left.\frac{\partial\vartheta_2(\delta,a_T)}{\partial\delta}\right|_{\delta\ \hat{\delta}_{2,T}}\right]' \\ \vdots \\ \left[\left.\frac{\partial\vartheta_r(\delta,a_T)}{\partial\delta}\right|_{\delta=\hat{\delta}_{r,T}}\right]' \end{bmatrix} \tag{4.2.27}$$

Stacking the r elements, we have:

$$\vartheta_t\left(\tilde{\delta}, a_T\right) = \vartheta_t\left(\delta^*, a_T\right) + D_T'\left(\tilde{\delta}-\delta^*\right) \tag{4.2.28}$$

Multiplying both sides of equation (B.28) by the following matrix

$$\left[\left.\frac{\partial\vartheta_t(\delta,a_T)}{\partial\delta'}\right|_{\delta=\tilde{\delta}}\right]' \times \tilde{S}_T^{-1}$$

we then have:

$$\left[\left.\frac{\partial \vartheta_t(\delta, a_T)}{\partial \delta'}\right|_{\delta=\tilde{\delta}}\right]' \times \bar{S}_T^{-1} \times \vartheta_t\left(\tilde{\delta}, a_T\right)$$

$$= \left[\left.\frac{\partial \vartheta_t(\delta, a_T)}{\partial \delta'}\right|_{\delta=\tilde{\delta}}\right]' \times \bar{S}_T^{-1} \times \vartheta_t\left(\delta^*, a_T\right) \tag{4.2.29}$$

$$+ \left[\left.\frac{\partial \vartheta_t(\delta, a_T)}{\partial \delta'}\right|_{\delta=\tilde{\delta}}\right]' \times \bar{S}_T^{-1} \times D_T\left(\tilde{\delta} - \delta^*\right)$$

From equation (4.2.22), we know that the left hand side of equation (4.2.29) is zero, so we have:

$$\left(\tilde{\delta} - \delta^*\right) = -\left\{\left[\left.\frac{\partial \vartheta_t(\delta, a_T)}{\partial \delta'}\right|_{\delta=\tilde{\delta}}\right]' \times \bar{S}_T^{-1} \times D_T\right\}^{-1} \tag{4.2.30}$$

$$\times \left[\left.\frac{\partial \vartheta_t(\delta, a_T)}{\partial \delta'}\right|_{\delta=\tilde{\delta}}\right]' \times \bar{S}_T^{-1} \times \vartheta_t\left(\delta^*, a_T\right)$$

Equations (4.2.25) and (4.2.29) imply that:

$$\sqrt{T}(\tilde{\delta} - \delta^*) \xrightarrow{l'} \kappa\sqrt{T} \cdot \vartheta_t\left(\delta^*, a_T\right) \tag{4.2.31}$$

where $\kappa = -\left(DS^{-1}D\right)^{-1} \times DS^{-1}$.

Equations (4.2.24) and (4.2.31) indicate that:

$$\sqrt{T}(\tilde{\delta} - \delta^*) \xrightarrow{l} N(0, \upsilon) \tag{4.2.32}$$

where $\upsilon = \kappa S \kappa' = \left(DS^{-1}D'\right)^{-1}$.

In other words, we have proved that the GMM estimator is a consistent estimator.

For the case of the dynamic panel estimation, we take difference for equation (4.2.1) to eliminate the individual effects and obtain:

$$y_{it} - y_{i,t-1} = \gamma(y_{i,t-1} - y_{i,t-2}) + \beta(x_{i,t-1} - x_{i,t-2}) + (\varepsilon_{it} - \varepsilon_{i,t-1}) \tag{4.2.33}$$

For $t = 3$, y_{i1} is a valid instrument. If $t = 4$, y_{i2} and y_{i1} are both valid

instruments. Following this fashion, we can add additional instrument for each time moving forward. Therefore, for time T, the set of instruments is $(y_{i1}, y_{i2}, \ldots, y_{T-2})$.

We stack the instrument sets and get

$$Z_i' = \begin{bmatrix} [y_{i1}] & 0 & \cdots & 0 \\ 0 & [y_{i1}, y_{i2}] & \cdots & 0 \\ \vdots & \vdots & \cdots & \vdots \\ 0 & 0 & \cdots & [y_{i1}, y_{i2}, \ldots, y_{i,T-2}] \end{bmatrix} \tag{4.2.34}$$

We define $Q = [\varepsilon_{i3} - \varepsilon_{i2}, \ldots, \varepsilon_{iT} - \varepsilon_{iT-1}]$ and the GMM estimator estimates the parameters in equation (4.2.33) by minimize a generalized metric, based on a positive semi-definite quadratic form. The metric is:

$$\left\| E_N\left[z\left(\varepsilon_{it} - \varepsilon_{it-1}\right)\right] \right\| = \left\| \frac{1}{N} Z'Q \right\| = \frac{1}{N} Q'Z\bar{W}Z'Q \tag{4.2.35}$$

We assume that $\delta = [\gamma, \beta]$. The minimization process is:

$$\frac{d}{d\delta}\|Z'Q\| = \frac{d}{dQ}\|Z'Q\|\frac{dQ}{d\delta} = \frac{d}{dQ}\left(\frac{1}{N} Q'(Z\bar{W}Z')Q \right)\frac{d\left(\Delta Y - \Delta X\delta\right)}{d\delta}$$

$$= \frac{2}{N} Q'Z\bar{W}Z'(-\Delta X)$$

$$\Leftrightarrow 0 = Q'Z\bar{W}Z'\Delta X = \left(\Delta Y - \Delta X\delta\right)' Z\bar{W}Z'\Delta X$$

$$\Leftrightarrow \delta = \left(\Delta X'Z\bar{W}Z'\Delta X\right)^{-1} \Delta X'Z\bar{W}Z'\Delta Y \tag{4.2.36}$$

where W is a weighting matrix to be estimated; $\Delta X = x_{i,t} - x_{i,t-1}$ and $\Delta Y = y_{i,t} - y_{i,t-1}$. Hansen (1982) states that the GMM estimators derived in equation (4.2.36) are consistent. However, because the instruments used in the GMM estimation are to some degree correlated to the regressors in finite

sample estimation, the GMM estimators are also biased, which is the same as 2SLS.

To make the GMM estimation in equation (4.2.36) feasible, we have to estimate the optimal weighting matrix W. One possible candidate for W is of the form $\sigma^2 I$, in which the errors are *i.i.d*, then the GMM estimator becomes:

$$\delta_{2SLS} = \left(\Delta X' Z \left(Z'Z\right)^{-1} Z'\Delta X\right)^{-1} \Delta X' Z \left(Z'Z\right)^{-1} Z'\Delta Y \qquad (4.2.37)$$

which is the 2SLS estimator. In other words, if the errors are *i.i.d*, the feasible efficient GMM estimator becomes 2SLS estimator.

For the more complicated form of the weighting matrix W, we can follow the usual practice from Arellano and Bond (1991) and Baltagi (2002) to assume:

$$\bar{W} = (Z'HZ)^{-1} \qquad (4.2.38)$$

where

$$H = I_N \otimes G$$

$$G = \begin{pmatrix} 2 & -1 & & \\ -1 & 2 & -1 & \\ & -1 & 2 & \ddots \\ & & \ddots & \ddots \end{pmatrix}$$

The resulting GMM estimator based on H is the one-step GMM estimator:

$$\delta_3 = \left(\Delta X' Z \left(Z'(I_N \otimes G)Z\right)^{-1} Z'\Delta X\right)^{-1} \Delta X Z \left(Z'(I_N \otimes G)Z\right)^{-1} Z'\Delta Y \quad (4.2.39)$$

Based on the one-step GMM estimation, we are able to use the residual from the one-step GMM to estimate the optimal weighting matrix W. The advantage of this estimation of the weighting matrix is that we do not need to consider the initial condition or distribution of the residual ε_i. The resulting two-step GMM estimator is:

$$\delta_4 = \left(\Delta X' Z \varpi^{-1} Z' \Delta X\right)^{-1} \Delta X' Z \varpi^{-1} Z' \Delta Y \quad (4.2.40)$$

where $\varpi = \sum_{i=1}^{N} Z_i'(\Delta\varepsilon_i)(\Delta\varepsilon_i)' Z_i$

Appendix 4.3 Tables and Figures

Table 4.1 Summary statistics by main variables.

	Variable	NOB	Mean	Standard Deviation	Maximum	Minimum
World	RGDP	1163	6126.37	8761.51	53986.05	84.35
	GRW	1142	2.15	3.72	32.23	-12.11
	INV	1053	21.70	7.51	66.43	2.63
	POP	1239	1.84	1.52	16.62	-8.88
	HEXP	729	5.61	1.68	15.30	1.50
	PTRATIO	994	30.42	13.72	84.79	6.57
	PSE	842	4.41	2.65	41.78	0.59
EU	RGDP	239	14471.10	10422.76	53986.05	480.97
	GRW	231	2.86	2.86	17.61	-8.58
	INV	220	22.77	4.90	44.35	3.96
	POP	261	0.48	0.98	2.54	-8.50
	HEXP	215	5.96	1.48	11.30	2.50
	PTRATIO	197	17.99	5.96	37.80	6.57
	PSE	186	4.90	1.50	8.68	1.43
ME	RGDP	131	7104.07	9130.30	49513.96	445.72
	GRW	127	1.71	4.38	14.89	-12.10
	INV	129	22.82	6.30	41.68	8.95
	POP	144	3.06	2.50	16.62	-8.88
	HEXP	69	5.38	1.70	9.90	2.00
	PTRATIO	116	23.75	7.91	47.47	9.40
	PSE	104	4.93	1.80	9.86	0.88
NA	RGDP	18	23188.77	7134.66	38064.84	12985.68
	GRW	18	1.77	0.96	3.17	-0.94
	INV	16	19.80	1.76	22.94	16.81
	POP	18	2.01	3.88	0.86	1.69
	HEXP	18	8.88	3.94	15.30	4.74
	PTRATIO	10	16.25	2.78	23.37	13.81
	PSE	14	6.36	1.10	8.49	4.78

Table 4.1 Summary statistics by main variables. **(Continued)**

	Variable	NOB	Mean	Standard Deviation	Maximum	Minimum
EA	RGDP	135	8448.08	9954.14	40328.20	122.29
	GRW	135	4.57	3.40	16.15	-6.00
	INV	129	24.91	7.36	45.83	2.63
	POP	144	1.67	0.93	4.62	-1.00
	HEXP	81	5.13	1.56	8.44	1.80
	PTRATIO	120	25.55	7.46	56.87	9.07
	PSE	102	3.81	1.45	7.79	0.99
LA	RGDP	252	3441.48	2943.37	16242.04	408.39
	GRW	251	1.85	3.27	14.84	-8.46
	INV	210	20.81	6.30	52.29	20.80
	POP	261	1.64	0.97	3.44	-1.80
	HEXP	130	5.86	1.46	10.10	2.80
	PTRATIO	209	28.83	8.34	61.52	12.25
	PSE	175	3.86	1.62	9.02	1.07
SSA	RGDP	337	859.13	1358.56	7838.52	84.38
	GRW	330	1.36	4.35	32.23	-10.35
	INV	301	19.61	9.35	66.43	3.18
	POP	358	2.55	1.06	7.41	-5.35
	HEXP	187	5.18	1.51	12.30	1.50
	PTRATIO	301	43.62	12.93	84.79	13.72
	PSE	221	4.66	4.30	41.78	0.68
SAS	RGDP	51	413.14	246.10	1181.62	138.57
	GRW	50	3.17	2.63	12.02	-4.51
	INV	48	22.78	9.46	57.73	12.93
	POP	53	2.11	0.80	3.68	-1.53
	HEXP	29	4.92	1.42	9.00	2.80
	PTRATIO	41	38.58	11.37	63.59	13.81
	PSE	40	2.63	1.16	6.97	0.59

* The unit for RGDP is constant 2000 US $ while the units for GRW, INV, POP, HEXP, PSE are percentage.

* Of the 138 countries, 16 countries are from East Asia and Pacific (EAP), 29 countries from Europe (EU), 29 countries from Latin America and Caribbean (LA), 16 countries from Middle East and North Africa (ME), 2 countries from North America (NA), 40 countries from Sub-Saharan African (SSA) and 6 from South Asia (SAS).

Table 4.2 Fixed-effect panel data estimation results for the world for the augmented Bassanini and Scarpetta (2001) model

Independent Var.	1	2	3	4	5	6	7	8	9	10	11	12
ln(RGDP1)	-0.09	-0.09	-0.11	-0.11	-0.12	-0.07	-0.11	-0.13	-0.08	-0.07	-0.11	-0.08
	(-6.41)	(-4.72)	(-6.71)	(-6.33)	(-5.06)	(-3.00)	(-3.53)	(-5.12)	(-3.49)	(-3.13)	(-3.48)	(-3.53)
ln(INV)	0.17	0.16	0.16	0.17	0.16	0.16	0.17	0.15	0.16	0.16	0.18	0.16
	(11.38)	(8.14)	(10.66)	(9.35)	(8.01)	(6.81)	(5.72)	(8.08)	(6.68)	(6.89)	(6.27)	(6.71)
POP	-0.01	-0.01	-0.02	-0.02	-0.02	-0.02	-0.01	-0.02	-0.02	-0.02	-0.01	-0.02
	(-3.26)	(-1.58)	(-3.47)	(-3.87)	(-2.58)	(-2.71)	(-0.31)	(-3.79)	(-2.36)	(-2.71)	(-0.78)	(-2.37)
ln(HEXP)		0.05			0.04	0.02	0.08	0.05	0.06	0.02	0.08	0.06
		(2.83)			(2.04)	(1.06)	(2.23)	(2.76)	(2.46)	(1.12)	(2.17)	(2.39)
ln(PTRATIO)			-0.04		-0.09		-0.07	-0.09			-0.08	
			(-1.63)		(-2.89)		(-1.78)	(-2.55)			(-2.07)	
ln(PE)				0.02		0.04			0.03	0.05		0.04
				(1.02)		(2.01)			(1.84)	(2.34)		(1.95)
Dln(HEXP)							-0.06		-0.06		-0.05	-0.06
							(-2.51)		(-2.77)		(-1.92)	(-2.55)
Dln(PTRATIO)								0.03			0.06	
								(1.09)			(1.53)	
Dln(PE)										-0.03		-0.02
										(-1.25)		(-0.67)
CONSTANT	0.26	0.24	0.54	0.39	0.79	0.09	0.53	0.84	0.14	0.1	0.58	0.15
	(2.27)	(1.49)	(3.17)	(2.81)	(3.11)	(0.45)	(1.63)	(3.11)	(0.71)	(0.47)	(1.73)	(0.71)
NOB	943	618	761	692	478	435	353	422	435	435	309	427
Adjust R^2	0.89	0.91	0.89	0.88	0.92	0.89	0.92	0.93	0.89	0.88	0.93	0.86
F-value	58.86	27.05	41.52	37.72	21.17	16.68	10.19	19.85	15.49	14.19	9.14	13.32

Table 4.3 Fixed-effect panel data estimation results for East Asia for the augmented Bassanini and Scarpetta (2001) model

Independent Var.	1	2	3	4	5	6	7	8	9	10	11	12
ln(RGDP1)	-0.03	-0.11	-0.06	-0.05	-0.13	-0.11	-0.14	-0.13	-0.13	-0.11	-0.14	-0.13
	(-3.09)	(-4.85)	(-4.32)	(-4.15)	(-5.46)	(-4.93)	(-5.88)	(-5.44)	(-5.44)	(-5.18)	(-5.83)	(-5.58)
ln(INV)	0.05	0.04	0.05	0.07	0.06	0.06	0.07	0.06	0.06	0.08	0.07	0.08
	(1.98)	(1.19)	(2.21)	(2.65)	(1.66)	(1.56)	(1.91)	(1.62)	(1.71)	(2.12)	(1.93)	(2.19)
POP	-0.01	0.01	-0.01	-0.01	0.01	-0.01	0.01	0.01	0.01	-0.01	0.01	0.01
	(-0.55)	-0.61	(-0.02)	(-0.93)	-1.05	(-0.13)	-1.23	-1.06	-0.32	(-0.32)	-1.21	-0.08
ln(HEXP)		0.19			0.13	0.16	0.20	0.13	0.23	0.16	0.20	0.22
		(3.13)			(2.01)	(2.49)	(2.76)	(1.99)	(3.31)	(2.55)	(2.76)	(3.27)
ln(PTRATIO)			-0.10		-0.22		-0.19	-0.22			-0.19	
			(-2.98)		(-2.57)		(-2.12)	(-2.55)			(-2.06)	
ln(PE)				0.06		0.08			0.06	0.11		0.09
				(3.04)		(1.93)			(1.49)	(2.71)		(2.15)
Dln(HEXP)							-0.34		-0.34		-0.34	-0.30
							(-3.13)		(-3.07)		(-3.13)	(-2.74)
Dln(PTRATIO)								0.02			-0.05	
								(0.09)			(-0.25)	
Dln(PE)										-0.24		-0.21
										(-2.74)		(-2.29)
CONSTANT	0.14	0.57	0.66	0.13	1.45	0.49	1.37	1.45	0.55	0.41	1.35	0.47
	(1.46)	(3.12)	(3.33)	(1.34)	(3.76)	(2.58)	(3.42)	(3.69)	(2.81)	(2.17)	(3.31)	(2.38)
NOB	342	223	342	342	223	223	214	223	214	223	214	214
	0.89	0.91	0.9	0.92	0.92	0.93	0.93	0.88	0.89	0.91	0.91	0.92
F-value	4.26	6.6	5.49	5.59	6.74	6.09	7.45	5.59	7.01	6.49	6.36	6.87

Table 4.4 Fixed-effect panel data estimation for the world for the augmented Mankiw, Romer and Weil (1992) model

Independent Var.	1	2	3	4	5	6
ln(RGDP1)	0.91	0.91	0.90	0.90	0.89	0.93
	(67.33)	(47.68)	(56.46)	(53.25)	(36.91)	(39.22)
ln(INV)	0.17	0.15	0.16	0.16	0.15	0.16
	(11.22)	(7.85)	(10.20)	(8.95)	(7.55)	(6.60)
COM	-0.06	0.06	-0.02	-0.05	0.05	-0.07
	(-2.32)	(1.05)	(-0.34)	(-1.10)	(0.84)	(-1.07)
ln(HEXP)		0.06			0.05	0.02
		(3.32)			(2.47)	(1.21)
ln(PTRATIO)			-0.05		-0.09	
			(-1.87)		(-2.91)	
ln(PE)				0.02		0.04
				(1.06)		(1.97)
CONSTANT	0.36	0.10	0.55	0.45	0.62	0.18
	(2.86)	(0.51)	(2.91)	(2.64)	(2.20)	(0.73)
NOB	943	617	760	692	477	435
Adjust R^2	0.85	0.84	0.86	0.85	0.86	0.85
F-value	1549	605	993	776	415	351

Table 4.5 Fixed effect panel data estimation for East Asia for the augmented Mankiw, Romer and Weil (1992) model

Independent Var.	1	2	3	4	5	6
ln(RGDP1)	0.86	0.63	0.79	0.84	0.61	0.63
	(49.65)	(18.47)	(36.83)	(46.71)	(17.41)	(18.42)
ln(INV)	0.18	0.16	0.19	0.20	0.20	0.16
	(4.08)	(2.45)	(4.40)	(4.53)	(3.01)	(2.45)
COM	-0.29	-0.16	-0.20	-0.26	-0.16	-0.16
	(-3.12)	(-1.28)	(-2.19)	(-2.81)	(-1.32)	(-1.29)
ln(HEXP)		0.49			0.32	0.48
		(4.56)			(2.71)	(4.41)
ln(PTRATIO)			-0.31		-0.46	
			(-5.23)		(-3.05)	
ln(PE)				0.10		0.01
				(2.64)		(0.19)
CONSTANT	1.13	2.24	2.53	1.02	4.07	2.23
	(4.01)	(5.43)	(6.63)	(3.53)	(5.63)	(5.29)
NOB	354	227	354	354	227	227
Adjust R^2	0.92	0.81	0.93	0.92	0.82	0.81
F-value	1345	231	1093	1028	193	184

Table 4.6 Two-stage fixed effect panel data estimation for the world for the augmented Bassanini and Scarpetta (2001) model

Independent Var.	1	2	3	4	5	6	7	8	9	10	11	12
ln(RGDP1)	-0.09	-0.18	-0.12	-0.11	-0.23	0.01	-0.14	-0.19	0.01	-0.01	-0.11	0.01
	(-6.41)	(-4.46)	(-5.93)	(-5.24)	(-3.01)	(0.03)	(-3.49)	(-3.03)	(0.41)	(-0.07)	(-3.48)	(0.43)
ln(INV)	0.17	0.14	0.17	0.17	0.16	0.27	0.18	0.16	0.27	0.29	0.18	0.28
	(11.38)	(4.01)	(10.42)	(7.53)	(4.39)	(6.61)	(6.15)	(4.51)	(6.74)	(6.81)	(6.27)	(7.02)
POP	-0.01	-0.01	-0.02	-0.02	-0.02	-0.02	-0.01	-0.02	-0.01	-0.02	-0.01	-0.01
	(-3.26)	(-0.47)	(-3.39)	(-3.37)	(-1.41)	(-1.13)	(-0.66)	(-1.40)	(-0.75)	(-1.06)	(-0.78)	(-0.66)
ln(HEXP)		0.48			0.36	0.06	0.08	0.34	0.01	0.10	0.08	0.01
		(3.13)			(2.13)	(0.40)	(2.23)	(2.09)	(0.11)	(0.62)	(2.17)	(0.21)
ln(PTRATIO)			-0.09		-0.19		-0.16	-0.10			-0.08	
			(-1.71)		(-1.81)		(-2.09)	(-1.91)			(-2.07)	
ln(PE)				0.05		0.05			0.02	0.02		0.01
				(0.85)		(0.89)			(0.31)	(0.66)		(0.17)
Dln(HEXP)							-0.05		-0.02		-0.05	-0.02
							(-2.07)		(-0.71)		(-1.92)	(-0.72)
Dln(PTRATIO)								0.06			0.06	
								(1.41)			(1.53)	
Dln(PE)										-0.03		-0.01
										(-0.94)		(-0.35)
CONSTANT	0.26	0.27	0.81	0.40	1.37	-0.89	1.01	0.84	-0.85	-0.94	0.58	-0.87
	(2.27)	(0.95)	(2.72)	(2.52)	(1.93)	(-2.76)	(2.02)	(1.90)	(-2.69)	(-2.82)	(1.73)	(-2.74)
NOB	943	480	687	604	323	286	312	320	285	285	309	285
Adjust R^2	0.89	0.88	0.91	0.88	0.87	0.46	0.9	0.86	0.54	0.41	0.89	0.52
F-value	59	269	584	592	296	575	489	318	598	547	496	590

Table 4.7 Two-stage fixed effect panel data estimation for the world for the augmented Mankiw, Romer and Weil (1992) model

Independent Var.	1	2	3	4	5	6
ln(RGDP1)	0.91	0.817	0.88	0.90	0.77	0.89
	(67.33)	(20.38)	(43.74)	(43.56)	(10.04)	(20.92)
ln(INV)	0.17	0.134	0.16	0.17	0.16	0.24
	(11.22)	(3.66)	(10.12)	(7.49)	(4.25)	(4.25)
COM	-0.06	0.023	-0.05	-0.04	-0.12	-0.12
	(-2.32)	(0.25)	(-1.05)	(-0.71)	(-1.16)	(-0.72)
ln(HExp)		0.491			0.38	0.21
		(3.32)			(2.22)	(1.19)
ln(PTRATIO)			-0.10		-0.18	
			(-1.79)		(-1.75)	
ln(PE)				0.02		0.18
				(0.36)		(2.83)
CONSTANT	0.36	0.241	0.90	0.39	1.56	-0.17
	(2.86)	(0.75)	(3.12)	(2.02)	(2.06)	(-0.35)
NOB	943	480	686	604	323	287
Adjust R^2	0.85	0.65	0.88	0.85	0.80	0.77
F-value	1549	172	385	382	177	198

Table 4.8 GMM estimation for the world for the augmented Bassanini and Scarpetta (2001) model

Independent Var.	1	2	3	4	5	6	7	8
ln(RGDP1)	-0.03	-0.06	-0.04	-0.06	-0.10	-0.13	-0.07	-0.10
	(-0.67)	(-1.01)	(-0.91)	(-1.37)	(-1.72)	(-2.03)	(-1.31)	(-1.24)
ln(INV)	0.18	0.17	0.24	0.24	0.17	0.17	0.22	0.20
	(4.97)	(4.69)	(3.84)	(3.29)	(4.36)	(3.81)	(3.69)	(2.82)
POP	-0.02	-0.01	-0.02	-0.01	-0.01	-0.01	-0.01	-0.01
	(-1.49)	(-0.88)	(-0.59)	(-0.78)	(-0.28)	(-0.41)	(-0.32)	(-0.24)
ln(HExp)	0.11	0.10	0.17	0.17	0.16	0.16	0.22	0.27
	(1.91)	(1.55)	(2.16)	(2.71)	(2.67)	(2.23)	(2.68)	(2.13)
ln(PTRATIO)	-0.03	-0.05			-0.10	-0.14		
	(-0.45)	(-0.53)			(-0.95)	(-1.11)		
ln(PE)			0.13	0.15			0.03	0.08
			(1.84)	(1.92)			(0.42)	(0.55)
Dln(Hexp)					-0.09	-0.09	-0.13	-0.12
					(-2.61)	(-2.68)	(-2.49)	(-1.43)
Dln(PTRATIO)					0.13	0.14		
					(1.22)	(1.53)		
Dln(PE)							0.13	0.09
							(1.97)	(0.88)
NOB	230	230	194	194	230	230	194	194
F-value	6.32	5.23	7.64	19.27	5.63	4.97	5.67	5.64

Table 4.9 GMM and LSDVC estimation for the world for the augmented Mankiw, Romer and Weil (1992) model

Independent Var.	1	2	3	4	5	6
ln(RGDP1)	0.85	0.87	0.91	0.94	0.93	1.08
	(10.19)	(12.37)	(25.63)	(15.27)	(15.19)	(30.83)
ln(INV)	0.22	0.20	0.22	0.28	0.27	0.36
	(5.34)	(4.03)	(6.01)	(4.27)	(3.78)	(4.16)
COM	-0.19	-0.18	-0.08	-0.09	-0.07	0.03
	(-1.81)	(-1.93)	(-0.89)	(-0.53)	(-0.31)	(0.19)
ln(HExp)	0.15	0.11	0.03	0.19	0.20	0.02
	(2.17)	(1.75)	(0.59)	(2.24)	(2.22)	(0.58)
ln(PTRATIO)	-0.15	-0.12	-0.07			
	(-1.08)	(-0.79)	(-0.91)			
ln(PE)				0.15	0.21	0.06
				(2.04)	(2.37)	(1.65)
NOB	230	230	230	194	194	192
F-value	90.95	129.55	12.94	118.51	122.23	9.12

Figure 4.1 Microeconomic links among health

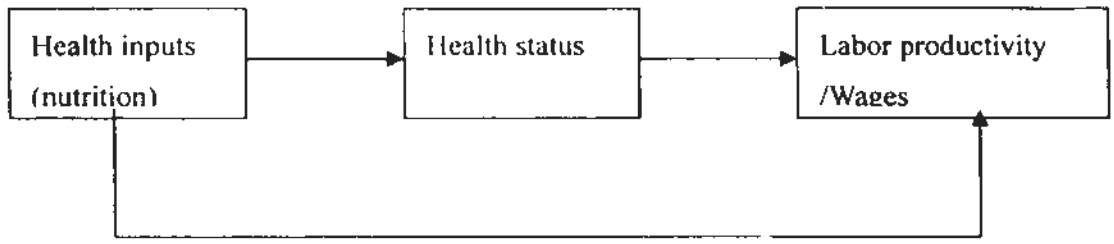
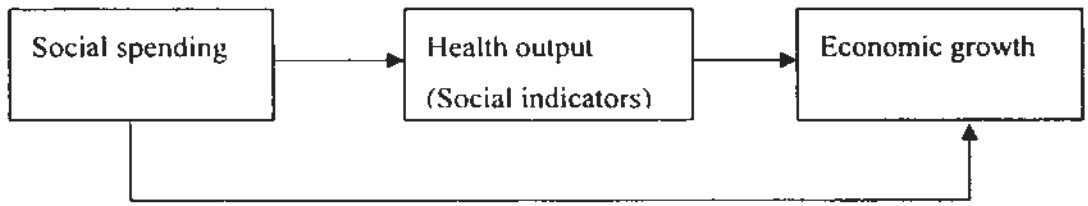


Figure 4.2 Macroeconomic links among health



Appendix 4.4 Countries included in the world dataset

Austria	Cote d'Ivoire	Italy	Romania
Bahamas, The	Cyprus	Jamaica	Rwanda
Bahrain	Czech Republic	Japan	Saudi Arabia
Bangladesh	Denmark	Jordan	Senegal
Barbados	Dominica	Kenya	Seychelles
Belgium	Fiji	Korea, Rep.	Sierra Leone
Belize	Finland	Kuwait	Singapore
Benin	France	Latvia	South Africa
Bhutan	Gabon	Lesotho	Spain
Bolivia	Gambia, The	Libya	Sri Lanka
Botswana	Germany	Mauritius	Sudan
Brazil	Ghana	Mexico	Suriname
Brunei Darussalam	Greece	Morocco	Swaziland
Bulgaria	Grenada	Mozambique	Sweden
Burkina Faso	Guatemala	Nepal	Switzerland
Burundi	Guinea	Netherlands	Uganda
Cameroon	Guinea-Bissau	New Zealand	United Arab Emirates
Canada	Guyana	Nicaragua	United Kingdom
Cape Verde	Haiti	Niger	United States
Central African Republic	Honduras	Norway	Uruguay
Chad	Hong Kong, China	Oman	Vanuatu
Chile	Hungary	Pakistan	Venezuela, RB
China	Iceland	Panama	Vietnam
Colombia	India	Paraguay	Yemen, Rep.
Comoros	Indonesia	Peru	Zambia
Congo, Dem. Rep.	Iran, Islamic Rep.	Philippines	Zimbabwe
Congo, Rep.	Ireland	Poland	
Costa Rica	Israel	Portugal	

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