An Exploration of Health, Human Capital and Growth

A thesis submitted for the Degree of Doctor of Philosophy

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DEDICATION PAGE

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General Declaration

I hereby declare that this thesis contains no material that has been accepted for the award of any other degree or diploma in any university or other institution. I further declare that to the best of my knowledge the thesis contains no material previously published or written by another person, except where due reference is made in the text of this thesis.

Contents

Abstract

This thesis investigates the importance of health as a primary policy target. In particular, it focuses on how health can improve investment and in turn economic growth; this probes the question of what determines improvements in health. The first major chapter (chapter 2) introduces information about the benefit of using life expectancy at age 10 rather than at birth for investment decisions. This emphasizes how health, at an age later than birth, is important for both educational and physical capital investment due to the elimination of infant and early childhood fatalities. The second major chapter (chapter 3) looks into the public and private financing of health expenditure. The chapter examines whether the financing of health care has an influence on health outcomes and offers some policy advice on the restructuring of healthcare systems. The third contribution (Chapter 4) questions the recent literature that claims empirical evidence shows health improves during recessions. This is a counter intuitive discovery, and a plausible explanation is offered here. It is important as it may highlight certain areas where government can target areas of health depending on the state of the economy. Thereby, helping to reduce the impact fluctuations in growth may have on health, and improving overall health.

1. Introduction

Growth theory has become a major part of economics and can explain a significant portion of the development of some countries and the poverty of others (Becker et al., 1990). Health plays a pivotal role in development and growth because of its important contribution to human capital and productivity. Improved health has previously shown to be highly correlated with a reduction in poverty and inequality (Ehrlich and Lui ,1991 and Mankiw, Romer and Weil, 1992). Health directly raises levels of human capital, which improves the productivity of the workforce and, in turn, the growth rate of the economy.

The importance of the relationship between health and economic growth has been increasingly recognized. It has only been over the past 20 years that the role health plays in the accumulation of human capital has been realized. Prior to this, human capital has mainly been linked to education alone. Preliminary work into the two-way interplay between health, human capital and economic growth includes Ehrlich and Lui (1991) and Mankiw, Romer and Weil (1992). These works emphasise the importance of considering health and nutrition to achieve a more complete analysis of human capital. Fogel (1994) and Barro (1996) pursued the relationship between economic growth and health and this has given rise to a substantial line of literature in the area. Health and its crucial role in the level of human capital and investment has become an important focus of this literature. This thesis concentrates on some of the components of this relationship as well as investigating some possible determinants of good health.

This introductory chapter will first provide a brief overview to recent growth theories and the role human capital plays in general. A discussion of the relationship between health and human capital follows. And finally, a synopsis of each of the three major chapters is given.

1.1. Three Major Chapters

This thesis consists of three major chapters. Each chapter considers a different aspect of public health. The first major chapter (chapter 2) explores the effect improvements in health can have on investment and therefore economic growth, whereas second and third major chapters explore some possible determinants of public health. Chapter three (second contribution) analyses the health outcomes from variations in the mix of public and private

health expenditures. Chapter four looks into the possibility that short run economic conditions can affect mortality rates. In the empirical analysis, health is an explanatory variable in the first chapter and the dependent variable in the second and third. The study adopts life expectancy and mortality rates as proxies for health.

The theoretical basis for chapter two is a simple growth model. The model is derived from Zhang and Zhang (2005) where agents choose schooling time and life cycle consumption. Both chapters three and four assume a basic health production approach where health at a particular point in time is the outcome of various inputs. Chapter three derives a general form of the model and uses private and public health care expenditures as the primary choice inputs into the production of good health. Chapter four simply assumes a health production function and focuses on unemployment and income as the main exogenous inputs into good health. Each chapter contributes an independent policy recommendation.

1.2. Chapter Two

Does Health Motivate Education and Saving?

The hypothesis of this chapter is, as the title suggests, that increased life expectancy encourages investment. Life expectancies have greatly increased since the mid 19th century and increased longevity is used to proxy the improvements in health that have occurred over this period. Both school enrolments in education and investment have also increased over this period. More specifically growth in tertiary enrolments (primary enrolments were already compulsory) and the investment ratio (the portion of GDP that is invested) has increased. This chapter will investigate whether there is significant correlation in the growth rates of these variables.

The theoretical section of this chapter is based on the Diamond overlapping-generations model and is a slightly adjusted version of Zhang and Zhang (2005). In this model time is discrete, and individuals are born in period t and live for three periods. Each agent maximizes the utility derived from the discounted value of their lifetime consumption, which is affected by both their level of education as well as their life expectancy. The optimal levels of consumption, savings and education are all found. Optimal savings and education are positively related to life expectancy which provides the theoretical groundwork for the empirical investigation.

Previous empirical studies have found a mixed and/or weak relationship between life expectancy at birth and human and physical capital investments (Shastry and Weil 2003; Lorentzen, McMillan, and Wacziarg 2005; Acemoglu and Johnson 2007). This chapter proposes that these results are sensitive to small adjustments in model specification or data because life expectancy at birth is being used as the dependent variable, where life expectancy at a later age would be more suitable. Life expectancy at age 10 has been collected across 21 OECD countries in order to revisit the questions pertaining to the impact health/ longevity has on investment decisions. Therefore, using a new, longer and broader data series this chapter repeats the investigations carried out by numerous authors, with the most similar being Arora (2001).

Strong positive relationships between longevity and both physical capital investment and tertiary enrolment rates have been found in this investigation so long as life expectancy at age 10 is used. Rising longevity increases incentives to invest in education because the returns to that investment increase with the longer the time horizon over which they can be earned. Furthermore, increased longevity encourages a higher savings rate because there is a higher need for greater future consumption to sustain a longer expected retirement. This chapter argues that causality should run from life expectancy to investment. Firstly, the longer a person expects to live, the more incentive they have to spend time getting a tertiary degree, and benefit from the increased wage income. This does not mean that better education doesn't also improve awareness of healthy lifestyle choices just that increased longevity comes first. Secondly, life expectancy should directly precede savings from expectations of a longer retirement.

The macroeconomic policy implications of this are very important for developing countries. Policy makers need to improve the health and in turn life expectancy of the population in order to encourage them to educate. Otherwise, pouring money into providing public schooling may not be successful, and children will either not be enrolled or not attend. Only once they think it is likely that they will live long enough to reap the benefit of this education, will they invest time in schooling. It is a similar story for investment. Only once the workforce foresee a longer life and foresees a need to fund a longer retirement, are they likely to save. Therefore, health's impact on life expectancy becomes a very important pathway via which economies can achieve human and physical capital accumulation, and is therefore fundamental for development.

1.3. Chapter Three

Public versus Private Health: Who provides better health outcomes?

Health care policies are at the forefront of most government policy. This study follows Pocas and Soukiazis (2010) and uses international data of 18 OECD countries from 1960 to 2008 to explore the link between health outcomes and health expenditure. The data is from the OECD and the countries are selected by the availability of health expenditure data for this entire period, although for some countries the data is a little shorter. Unlike Pocas and Soukiazis (2010), this chapter assumes public and private health expenditures are not perfect substitutes, and considers each separately. Life expectancy and infant mortality are used to proxy health status. The estimation also assumes income, education, and healthcare expenditures are endogenous and a unique set of instrumental variables are used to control for this problem.

The theoretical investigation is based on a basic health production model which includes the possibility that private and public health expenditures may affect health outcomes differently. In the model finitely lived agents maximize log utility subject to two budget constraints. There are two key constraints, an income constraint and a health production constraint. The outcome is optimal health that increases when either private or public health expenditures increase, however, not necessarily by the same magnitude.

Empirically the difference between this chapter and other recent literature is that it uses separate data for private and public health expenditures while at the same time using instrumental variables to deal with the inherent endogeneity. To the best of my knowledge, this has not been done before. The instruments that have been chosen are also unique to this chapter. The empirical evidence shows that public and private health expenditures are not perfect substitutes and should be considered separately when analysing policy options. The results do not give a compelling argument for policy makers to move away from the public provision of healthcare. The expenditure data does not provide conclusive evidence. Instead this chapter calls for more research into the provision of private healthcare, particularly into the qualitative aspects of healthcare systems before government moves into private provision of healthcare.

1.4. Chapter Four

Mortality and Unemployment: Recessions are Not Good for Health

Long term improvements in health and mortality are generally thought to be associated with upward trends in per capita income. Whether this relationship exists between short term income growth and health is not clear. More specifically, whether the relationship is actually positive or negative has become a contentious issue. This chapter further explores the short term interaction between fluctuations in unemployment rates and mortality rates over 138 years. This is a far longer time span than has been investigated before.

Short run fluctuations in growth are an important focus of economic policy for political and social reasons. Public health is important in short run policy for both social well being and short term labour productivity as well as long term economic growth. Empirically, the link between health and income has only been established in the long run. This triggered the works of Brenner (1973, 1979) to look at the short term cyclical effects of income and employment on health and found these effects to be positive as well. However, since then most research has opposed Brenner's findings [1]where all of these works argue the relationship is negative and claim that health decreases when income and employment rise in a cyclical upturn. Ruhm (2000) explains that this is because workers endure larger amounts of stress during economic expansion, and this causes them to develop unhealthy habits. The idea for this chapter came from a disbelief that stresses pertaining to extra work could outweigh the stress associated with unemployment. If this work-related stress did get too great you could simply chose not to work as much.

The data that is used in all of the above mentioned investigations are contemporaneous mortality rates, unemployment rates and income as well as other lifestyle variables such as tobacco consumption. The measures of income vary a little across papers, with the geofigureic regions and time periods usually different. The data in this chapter differs significantly from all of the previous work in this area. The chapter initially replicates the regressions using contemporaneous data. The main contribution of this chapter is to go on and construct cohort explanatory variables for unemployment rates, income and cigarette consumption. The proposal is that it is not contemporaneous unemployment rates and

[1] Eyer (1977), Gravelle, Hutchinson and Stern (1981); Stern (1983); Wagstaff (1985), Ruhm (2000) Granados (2005), Gerdtham and Ruhm (2006), Ruhm (2007), Economou et al. (2008), Miller et al. (2009), Gonazalea and Quast (2011), Douglas et al. (2008), and Stevens et al. (2012)

income that will affect current mortality rates. Except in exceptional circumstance, it would be the unemployment rates, income and lifestyle factors that a person has experienced over their entire working life that will affect their current mortality rate.

The purpose of this chapter has been to investigate the claim by Ruhm (2000), in which he finds that higher unemployment is associated with lower mortality. Ruhm's (2000) finding goes against medical research (Sandifer and Stein, 1985). There could be many reasons why losing one's job may affect mortality. Some of which may have positive effects on health such as increased leisure time and less work related stress, as suggested by Ruhm (2000), but these and other reasons may also have significant negative effects. The resulting fall in overall income due to job loss can reduce the ability to enjoy leisure activities and buy nutritional foods. Job displacement is very stressful and quite plausibly at least as stressful as work related stresses. Financial stress in particular has been shown to have detrimental health effects.

This chapter replicated Ruhm's results using contemporaneous data but found the results reversed when cohort data is used. That is, there is a significant and positive relationship between cohort unemployment rates and mortality rates, when cohort unemployment rates go up the mortality rate also increases. This indicates that government should try to identify the detrimental effects of economic down-turns and try to target policy at those likely to suffer from these down turns. This should improve the overall long run health in the economy as well as investments in human and physical capital and therefore economic growth.

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2. Does Health Motivate Education and Saving?

Abstract

Theory suggests that longer life expectancy encourages investment. Educational investment increases because of a longer time horizon; this increases the value of education as the returns pay out over entire working lives. Investment in physical capital is encouraged by increased savings to finance a longer period of retirement. To estimate the significance of this effect data from 19 OECD countries from 1870 to 2006 is examined. Over this period both life expectancy and investment have increased. This chapter assesses whether growth in life expectancy is associated with increased growth in tertiary enrolments and capital investments. It is found that life expectancy at age 10 is positively correlated with both.

2.1. Introduction

Over the past century life expectancy has increased dramatically in OECD countries. This improvement in life expectancy has wider benefits over and above that derived from greater consumption of good health and therefore greater social welfare. Increased life expectancy also encourages investment, both in education and physical capital. Any investment that is associated with a future income stream is more valuable the longer this stream of payments lasts. The implication of this is twofold: improvements in life expectancy should increase investments in human capital, since the returns to education may be higher. Increased life expectancy will also increase the length of retirement which means workers will want to save more, higher savings means more funds available for investment in physical capital. Theoretically, increased investment in both human and physical capital would stimulate growth. This chapter broadly examines whether an increased life expectancy encourages investment, and specifically, gross enrolment rates in education and investments in physical capital. If this is the case it would suggest (amongst other things) that improving a country's health would improve growth.

There have been many studies into the impact of health on labour efficiency and growth. Howitt (2005) identifies some of the possible channels such as improved cognitive abilities, learning capacity, productive efficiency, concentration, stamina, coping skills, creativity, new ideas etc. All of these improvements to health impact labour efficiency:

- Directly by improving the health of workers, and

- Indirectly by improving the learning ability of the next generation's labour force.

The focus in this paper is on whether increases in life expectancy can improve economic activity by increasing people's incentives to invest both in tertiary education for an extended working career and in their savings/ portfolio accounts for a longer future retirement. Most empirical studies use life expectancy at birth as an indicator and find mixed or weak relationships between life expectancy and human and physical capital investments (Shastry and Weil 2003; Lorentzen, McMillan, and Wacziarg 2005; Acemoglu and Johnson 2007). It is proposed here that this is because life expectancy at birth is not the most appropriate measure to use when examining schooling and savings decisions. This is because statistically, advances in life expectancy have, to a large degree, been driven by falling infant mortality rates. This is particularly true in the earlier years of the sample data (1870-1950), the entire period ends in 2006. Infant mortality does not affect the length of working life or retirement and therefore should have no impact on a rational investment decision.

Good health is determined by many factors that occur throughout life, beginning from before an infant is conceived right throughout life. These factors include hygiene, mother's health, nutrition, education, environmental factors, geofigureical factors, lifestyle habits, and social interactions. Good health has many benefits; it directly increases welfare itself since being healthy is far preferable to being unhealthy. It also generates many external benefits that feed back into the original determinants such as increased ability to think and learn, better social skills, sharper awareness (consciousness), higher productivity, better lifestyle choices and increased nutrition to name a few. The economic interest in health is primarily derived from it being a major component of human capital, the other component being education (either formal or informal).

The two main hypotheses being tested in this chapter are both related to how rising longevity affects investment incentives. The first hypothesis is that if people think they will live longer they will educate themselves at a tertiary level, due to their belief that it will increase their ability to earn income. It may enhance the incentive to increase salary potential by increasing educational attainment, as people will maximize the discounted value of lifetime income. With a longer working life, the income stream will be longer and therefore more valuable. The second hypothesis states that this more valuable retirement "nest egg" will promote the development of effective savings habits, incentives to increase skill levels in their jobs, which may facilitate the incentive to save during the working period to allow for a longer retirement, increasing both the incentive to attain a higher wage as well as the propensity to save during the working period. This will not only lead to a higher level of human capital in the economy but also an increase in the level of investment in the economy. Since neither of these reasons should be affected by infant mortality rates life expectancy at age 10 should be more closely related these investment decisions and is therefore used as an explanatory variable along with life expectancy at birth.

The results show strong positive relationships between longevity and both investment and enrolment rates. It is found that life expectancy at age 10 is significantly and positively related to gross enrolment rates. Life expectancy at birth is often negatively related to education and investment, and is not always empirically significant. Investment is also found to be positively related to life expectancy at age 10 with high degrees of significance and again, insignificance and often negative responsiveness to life expectancy at birth. The results are insensitive to violation of all reasonable assumptions regarding our estimation procedures. The data is drawn from 19 OECD countries from 1870-2006. Over this period primary education was largely compulsory in these countries and is therefore not included. The decision to increase enrolments should be reflected by changes in discretionary enrolments not compulsory enrolments. Hence, life expectancy at age 10 should predict enrolments in secondary and tertiary education, as well as savings.

The rest of the chapter is organized as follows: first a brief literature review. Second, the theoretical model is outlined; third the empirical methodology is explained followed by a discussion of the data. Fourth, the results are presented; the chapter is then concluded with a discussion of the results and policy recommendations.

2.2. Literature Review

Solow (1956) pointed out that the accumulation of physical capital alone cannot account for either rapid growth over time or the income disparities between countries. Since then growth theorists have been trying to extend the neoclassical growth model in order to explain other potential sources of growth. In particular, for the past two or more decades much effort has gone into developing endogenous (Ha and Howitt, 2007) and/or unified growth theories (Galor, 2005). Some of these models identify fertility or the demofigureic change as an underlying trigger for growth (Becker, Murphy *et al.* 1990; Galor and Weil 1996). Others explore the possibility that better institutions and more secure property rights lead to better investment and are therefore fundamental for economic development (Acemoglu, Johnson *et al.* 2001; Acemoglu and Johnson 2005). Trade is another channel via which education is identified as stimulating growth. This is because it allows technological spill overs and encourages imitation (Howitt and Mayer-Foulks 2002). Acemoglu *et al*. (2005) suggest that Atlantic trade encouraged urbanization which meant that cities in Western Europe grew significantly faster than Eastern Europe after 1500.

Friedman and Kuznets (1945) formulated a capital theory approach to explain how improved health increases incentives to invest in human capital which arise from increased returns to investment. The theory was developed by Becker (1962, 1964) and Mincer (1958, 1962) who viewed the life cycle of earnings by linking this to the time profile of investment in human capital. Fogel (1994) introduced the idea that improved nutrition caused improved physiological developments, life expectancy and therefore labour productivity and growth. Emphasizing that major issues of economic policy such as medical care, pensions and the challenges of globalization are all governed by long run processes. Howitt (2005) sets up a theoretical model that contains various different channels through which improvements to health can affect growth. These improvements include creativity and coping skills which affect the rate of innovation. A theme that is apparent throughout all of these theories is that human capital either through health, education or the transfer of knowledge via trade or work experience plays a central role in economic growth.

A main focus in this chapter is the idea that any investment that pays out a certain amount each year is more valuable if the stream of payouts lasts longer. An implication of this is that improvements in life expectancy should increase investment in human capital, which should

spur economic growth. A large literature has explored this idea theoretically (Ben-Porath 1967; Kalemli-Ozcan, Ryder and Weil 2000; Bils and Klenow 2000; Zhang 2005; Soares 2005: Howitt 2005; Murphy and Topel 2006). Most previous empirical research measures cross-country variation between life expectancy at birth and education or growth (Shastry and Weil 2003; Lorentzen, McMillan, and Wacziarg 2005; Zhang and Zhang 2005; Acemoglu and Johnson 2007). The empirical results from these studies are mixed. Shastry and Weil (2003) and Lorentzen, McMillan and Wacziarg (2005) find large positive effects on human capital and growth from the improvements in life expectancy. Acemoglu and Johnson (2007) find small effects. One problem with the approach in these papers is that a portion of the variation in life expectancy over time is driven by reductions in infant mortality. By using life expectancy at age 10 our empirical approach avoids this problem.

The other main focus of this chapter is that a longer life, particularly, longer periods of retirement must encourage higher savings. Either private or public savings would need to increase in order to fund the extra consumption required by the elderly. Sala-i-Martin (2005) identifies three effects that health has on physical capital. First is the increased incentive to save due to the expectation of living longer after retirement. Second, is complementarity across inputs. If human capital is complementary to physical capital then increasing human capital (by improved health) should increase the incentive to invest in physical capital. The final channel Sala-i-Martin (2005) drew attention to is what he called a "private poverty trap" where in very poor countries there can be limited access to private or public health. This can mean that families are forced to spend their life savings on curing one family member. This can force children out of school and reduce the ability of the family to earn income in the future. This chapter focuses on the first two points. Firstly, savings should increase to pay for a longer retirement and therefore fund more physical investment. Secondly, human and physical capital complement each other.

The theoretical models that inspired this investigation focus specifically on the impact life expectancy has on education, savings and therefore growth (Bils and Klenow 2000; Zhang 2005). Bils and Klenow (2000) examine an infinite horizon model which links schooling to growth via increased longevity. They derive the optimal level of education and find that there is a one to one relationship, between life expectancy and the optimal level of education. Zhang and Zhang (2005) construct a three period overlapping generations model that shows rising longevity reduces fertility, raises savings, schooling time and growth. Zhang and

Zhang (2005) also find some empirically significant evidence for this, but it is not quantitatively large. Arora (2001) does use life expectancy at different ages and finds that health related variables correlate positively with schooling. Arora (2001) also finds that schooling itself doesn't replicate the impact health has on growth. This should be the case since schooling is not the only source of human capital accumulation. On the job training and work experience is at least as important, if not more. As summarized by Howitt (2005) there are various channels, other than schooling, where health affects labour and therefore productivity. We used long run data as in Arora (2001) however; we have collected data on twice as many countries, and our life expectancy data is constructed differently. The theoretical model and empirical tests follow Zhang and Zhang (2005) but the model is modified and our data covers a far longer time horizon, again the life expectancy data differs.

The main contribution of this chapter is the empirical evidence supporting the proposition that life expectancy at age 10 should be better at explaining the impact increased longevity has on physical and human capital investments. The crucial reason for this is that life expectancy at age 10 eliminates the previously noted problem of infant/ child mortality and more accurately captures information that people would use in investment decisions.

2.3. Theoretical Model

To guide the empirical investigation the model of Zhang and Zhang (2005) is adapted to illustrate the effect of increased live expectancy on investment in both human and physical capital through the savings decisions of households. Agents are identical and live for three periods; childhood, where they work or go to school, middle age, where they work and save, and old age where they consume from their savings. The utility function is given by:

$$U_t = \varphi ln(1 - e_t) + lnc_{t+1} + \rho\delta lnc_{0,t+2}, \quad (1)$$

where e_t is the time spent at school, c_{t+1} is middle aged consumption and c_{0t+2} is old aged consumption. Therefore, φ is the preference for work over education, p is the probability of surviving to old age, and δ is the time preference discount factor (or the interest rate).

The constraints are that the time of a child, normalized to unity, is either spent in school, e_t, or at work, $(1-e_t)$, and that the child's educational attainment once they reach the workforce

depends on how much time was spent at school as well as the level of education of the parents.

$$h_{t+1} = Ae_t h_t, \tag{2}$$

here h_t is the human capital of the middle aged (parents) and h_{t+1} is the human capital of the child once formal education is completed i.e. the education level of the middle aged in the next period. The income constraints are straight forward, in middle age, consumption, c_{t+1}, must equal wage income, $w_{t+1}h_{t+1}$ less savings, s_{t+1}:

$$c_{t+1} = w_{t+1}h_{t+1} - s_{t+1}. \tag{3}$$

In old age consumption must equal savings, s_{t+1}, from the previous period plus the return on these savings, r_{t+1}, discounted by the probability that they will live to receive this income, p. Another simplifying assumption, as in (Zhang, 2005), is that there is an actuarially fair annuity market that transfers funds from those wishing to save to those wishing to invest without any leakage (zero transaction costs). In this model there are no bequest, old age survivors equally share the savings and interest of those who die prematurely; the rate of return to savings is therefore the interest rate over the probability of surviving to old age, r/p. Old age consumption simply equals the discounted return on middle aged savings:

$$c_{0,t+2} = {}^{r_{t+2}s_{t+1}}/_{p}. \tag{4}$$

Production is given by a Cobb-Douglas production function, where capital, K_t, and effective labour, $L_t h_t$, are used to produce a single final good, Y.

$$Y_t = AK_t^{\propto}(L_t h_t)^{1-\propto} \ , \tag{5}$$

where A is the productivity parameter, L_t, is the size of the labour force (or cohort t+1), and α represents the relative share that capital and labour contribute to production. Physical capital is assumed to depreciate fully in each period for simplicity. The economy is competitive; therefore, firms earn zero economic profits and pay factors their marginal products. The marginal products are the returns to factors of production, $w_t$being the real wage and $r_t$being the real interest rate. All other variables are as defined above.

$$w_t = (1-\propto)A\left({}^{K_t}/_{L_t h_t}\right)^{\propto} \text{ and } r_t = \propto A\left({}^{K_t}/_{L_t h_t}\right)^{\propto-1}.$$

Optimal middle age consumption is given by the equation:

$$c_{t+1} = \frac{1}{1+\delta p} w_{t+1} h_{t+1}. \qquad (6)$$

Let $\alpha_c = 1/(1+\delta p)$, now α_c represents the marginal propensity to consume from current income. Here the fraction of labour income spent on middle- age consumption decreases as survival to old age increases,

$$\frac{\partial c_{t+1}}{\partial p} = (-) w_{t+1} h_{t+1} \frac{\delta}{(\delta p+1)^2}, \qquad (6a)$$

This is one of the main hypotheses of this empirical investigation. Regardless of the magnitude of labour income the proportion of income consumed in middle age will decline as life expectancy increases (p is proxy). This proposition is basically explaining that regardless of how rich a person/ society is, the longer they expect to work and live after finishing work, the larger the proportion of their working income they will save. Or the literal interpretation of inequation 6(a) would be that middle (working) aged consumption reduces when life expectancy increases. That is the marginal propensity to consume falls, and the savings rate rises. The savings function which is simply labour income less middle age consumption is given by:

$$s_{t+1} = w_{t+1} h_{t+1} \left(\frac{\delta p}{\delta p+1}\right). \qquad (7)$$

The proportion of middle age income saved increases as the likelihood of surviving to old age increases,

$$\frac{\partial s_{t+1}}{\partial p} = w_{t+1} h_{t+1} \frac{\delta}{(\delta p+1)^2} > 0. \qquad (7a)$$

It also increases at a diminishing rate,

$$\frac{\partial^2 s_{t+1}}{\partial p^2} = (-2) w_{t+1} h_{t+1} \frac{1}{(\delta p+1)^3} < 0, \qquad (7b)$$

more is saved for old age consumption the higher the likelihood of survival, however, the closer that probability becomes to one, the smaller the increase in savings becomes. Clearly

there would be other factors that affect the propensity to consume and save such as tastes and demofigureics.

The solution for optimal schooling, e_t^* , depends on life expectancy, the preference for work over education, and the discount factor:

$$e_t^* = \frac{1+p\delta}{1+p\delta+\varphi}\,. \tag{8}$$

As the probability of survival increases so does the need for consumption. This raises the incentive to increase educational attainment in order to achieve higher future earnings.

$$\frac{\partial e_t^*}{\partial p} = \frac{\varphi\delta}{(1+p\delta+\varphi)^2} > 0. \tag{9}$$

Although it is not explicitly modelled here, it is proposed that the preference parameter regarding the trade-off between schooling and working, *φ*, which is the preference for work over schooling, would also depend on the discounted value of life time income. Bils and Klenow's (2000) model captures this aspect of the schooling decision where an increase in lifespan increases the optimal amount of education by providing a longer working period over which to reap the benefits of higher wage level yielded by a higher level of education:

$$\frac{\partial e_t^*}{\partial \varphi} = (-1)\frac{1+p\delta}{(1+p\delta+\varphi)^2} < 0. \tag{10}$$

There are two reasons for this negative relationship. Firstly, a longer life provides a longer working period over which to receive the higher wages yielded by more education. Secondly, life expectancy itself, *p*, directly results in a need for more consumption in old age or retirement and therefore an incentive to save more.

2.4. Methodology

The main aim of this exercise is to establish a relationship between life expectancy (health) and investment, and hence show that improved health that leads to longer life may lead to growth via increased incentives to invest. This relationship was established and found to be positive for investments in both human capital and physical capital, if life expectancy at age 10 was used to proxy health. This result can't be established for life expectancy at birth. To support this claim the investigation looks at both life expectancy at birth and life expectancy

at age 10 and tries to determine which is better at explaining the deviations in investment. The results support our proposition that it makes more sense for life expectancy at 10 to provide investment incentives. The following two equations are estimated:

$$\Delta GER_{it} = \beta_0 + \beta_1 \Delta lnLE_{it} + \beta_2 \Delta lnLE10_{it} + \beta_3 \Delta^2 lnY_{it} + \beta_4 \Delta^2 lnY_{it-1} + \beta_5 \Delta r_{it} + \varepsilon_{it} \quad (11)$$

$$\Delta I_{it} = \theta_0 + \theta_1 \Delta lnLE_{it} + \theta_2 \Delta lnLE10_{it} + \theta_3 \Delta^2 lnY_{it} + \theta_4 \Delta^2 lnY_{it-1} + \theta_5 \Delta r_{it} + \theta_5 \Delta q_{it} + \varepsilon_t \quad (12)$$

In this study, panel-data regressions test the impact of the independent variables over the dependent variables. The dependent variables quation 11 or growth in physical investment, in equation 12. This is either the change ΔGER_{it} and ΔI_{it} stand for the annual growth, and represent growth in tertiary enrolments in e in Gross Enrolment Rates (GER)[2] in tertiary educational enrolments or the growth rate in the ratio of total investment to GDP. The subscript $_i$ denotes the country and $_t$ the year. $\Delta lnLE_{it}$ and $\Delta lnLE10_{it}$ are life expectancy at birth and at age 10 respectively in country i at time t, extended life should be positively related to investment. $\Delta^2 lnY_{it}$ and $\Delta^2 lnY_{it-1}$ are the acceleration of per capita GDP and its 1st lag, these coefficients should also be positively related to investment. However this will depend on whether income growth precedes investment or the other way around. If income growth precedes growth in investment then $\Delta^2 lnY_t$ may be negative and its 1st lag positive. Δr_t is the change in the real interest rate which is the long term rate on government securities, this is expected to be negatively related to all types of investment. The annual difference in Tobin's q, Δq_t, is included as an explanatory variable when physical capital investment is the dependent variable. Tobin's q is the market value of a portfolio over the cost of replacing the portfolio's physical capital. This should be positively related to investment. If the market value is greater than its replacement costs then investment should increase.

Initially, annual differences were estimated and they form the basis of the analysis and discussion that follows. However, equations 11 and 12 were also estimated using 5 year differences to try to eliminate any noise. This also checks the robustness of the annual results. The results from the five year differenced estimations are reported. They are not nearly as significant and have not been discussed in much detail. They have been reported as

[2] The measure of education is the difference in gross enrolment rates from one year to the next. Gross enrolment rates (GER) are defined as the number of students enrolled at a particular level of education as a percentage of the total number of children in the official school aged population.

they do provide evidence supporting the sensitivity of the main results to annual fluctuations or "noise".

Good health itself is difficult to measure. Generally in macroeconomic studies, life expectancy or mortality rates are used as they are available for long time periods and they are relatively accurate. There are other measures that can give more precise information about health such as morbidity measures, however, overall morbidity is very difficult to measure and data are not readily available. In this chapter, the problem of selecting other measures of health has been largely avoided since we are focussing on life expectancy specifically (not health or the quality of life in the broader sense). Having said that, although health is not the focus of the empirical investigation, life expectancy is still considered a proxy for public health in general. In the absence of accidents, suicide, natural disaster, violence and war, better health will lead to longer life, so it is widely accepted that the two correspond.

There is a dummy variable that has been created for the major war periods, equals one if the country was affected by war in that year. This dummy was calculated individually for each country. If a country is at war life expectancy will fall for a relatively brief period causing a break in the data. This will bias the estimation if the dummy is not included. Year dummies were included to take account of serial correlation; however, these didn't affect the results. On top of this a dummy needed to be created for the extraordinary growth in tertiary education that is discussed below in the data section.

2.4.1. Potential Problems

The error term in equation 11 is likely to be capturing a variety of factors that would affect investment in physical and human capital; there must be some omitted variables. For example educational attainment may be affected by childhood health and nutrition as well as the quality of public schooling. The availability of public schooling should affect health, however, public primary schooling was already established and compulsory in the countries studied here, enrolment rates in primary schooling have therefore been deliberately excluded. Investment in physical capital is affected by government and/or central bank policy. Physical investment would also be influenced by the level of corruption and affected by cultural beliefs as well as the demographic structure of the economy. Fixed effects dummies automatically eliminate bias due to particular geographical or cultural differences between

the countries. However, these were excluded in the final estimations because they are not appropriate when growth rates are used and were not significant.

Time series OLS regressions were estimated in Stata, the standard errors are robust to heteroskedasticity and serial correlation. This estimator is consistent when the regressors are exogenous. The problem of endogeneity occurs when the explanatory factors that affect a particular outcome, depend themselves on that outcome, in other words the causality flows in both directions. For example, it is very likely that gross enrolments in tertiary education are positively affected by longevity. This is consistent with my main hypothesis. It is also highly likely that health variables themselves depend on tertiary enrolments. The more educated the adult population becomes; the more likely it is to develop advanced medical techniques and good hygiene practices, and therefore raise healthy children. Since enrolment rates depend on life expectancy at age 10, enrolment rates are not related to education in tertiary education that occurs in adulthood. Endogeneity (reverse causality) where better education leads to better health is not likely to be present here. This is because higher enrolment rates particularly at the tertiary level are only likely to affect life expectancy or health at some point in the future. That is, gross enrolment rates would need to be lagged for this endogenous argument to make sense.

Growth rates or log differences are used in these regressions so bias caused by serial correlation is not likely. When there is a certain amount of auto correlation or heteroskedasticity in the residuals, the OLS estimation on levels can be inefficient and the standard errors are biased. Growth rates have been used to eliminate some of the serial correlation, the growth rate data can be seen in Figure 3, which is relatively stationary. To further test the results GLS estimation has been used. It has been suggested this deals with serial correlation and heteroskedasticy better than standard OLS, and therefore should be more efficient and robust. The results from both OLS and GLS methods were similar, the coefficients only differ in magnitude not sign and they were all still statistically significant. The GLS results have not been reported. This reinforces that the inferences made from the OLS estimations are not misleading.

2.5. Data

The data used here is from 19 OECD countries: Canada, USA, Japan, Australia, New Zealand, Austria, Belgium, Denmark, France, Germany, Ireland, Italy, Netherlands, Norway,

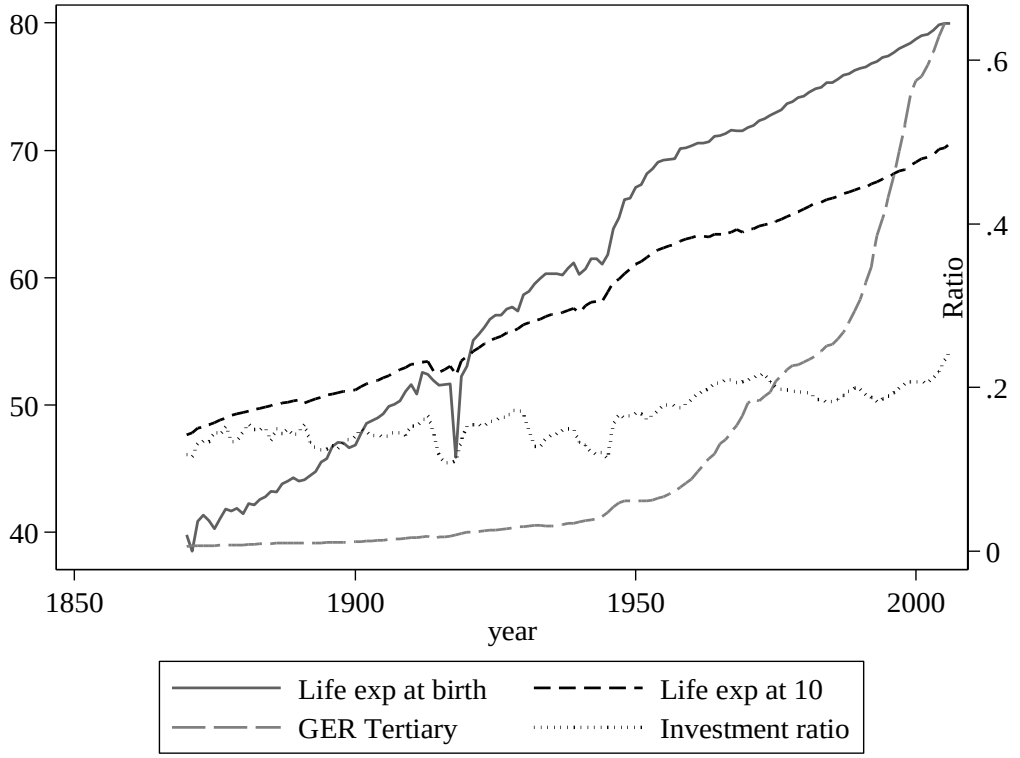
Portugal, Spain, Sweden, Switzerland and England. The sources of the data are detailed in appendix 1. Life expectancy is defined as the average remaining number of years a person of a particular age expects to live given the prevailing mortality rates. Life expectancy data are available for many countries calculated by their domestic statistical agencies. This data is available on the Human Life-Table Database at www.lifetable.de. Life expectancy data has been collected; for life expectancy at birth, aged 10 and age 65.

The measure of education is the difference in gross enrolment rates from one year to the next. Gross enrolment rates (GER) are defined as the number of students enrolled at a particular level of education as a percentage of the total number of children in the official school aged population. In countries where many children enter school late or repeat a grade the GER can exceed 100%. The data is separated into gross enrolments at the primary, secondary and tertiary. However, since particularly primary and to a certain extent secondary enrolments were largely compulsory in these countries over this period, GER in tertiary education is the most likely to reflect discretionary education choices. If there is no choice as to whether or not children are enrolled, enrolments are not reflecting investment decisions. Gross enrolments rather than years of schooling are used because it is the enrolment decision that reflects the desire to invest in education. Years of schooling attainment would reflect a decision that took place in the past.

The measure for investment is the difference in total investment as a proportion of GDP from one year to the next. The investment ratio is used because it eliminates the problem of different country size and allows cross country comparison. The interest rate used is the long term interest rate on government bonds. The difference in the interest rate is included as an explanatory variable in all regressions since theoretically there should be a significant and negative relationship between investment decisions and the interest rate. Finally, following the model of Bils and Klenow (2000) the acceleration of GDP (g_t) per hour worked from one year to the next is used as an explanatory variable. It is likely that GDP or at least its lags would have significant impact on investment and education.

The following figures the levels of life expectancy at birth and life expectancy at age 10 in years on the left hand axis and Gross Enrolment Rates (GER) in tertiary education and the Investment ratio on the right hand axis.

Figure 1: Life Expectancy in years, Gross Enrolment Rates, and Investment Ratio (1870-2006)



The investment ratio fluctuated around 8% from 1870 until after the first world war then it fluctuated around 12% until after the second world war. Investment then increased as a portion of GDP to around 18% and has recently increased to around 22%. Gross enrolment rates in tertiary education increase from 1870 to 1940's very slowly and are quite low, always below 5%. That means that total enrolment in tertiary education regardless of age is less than 5% of the tertiary aged population defined by the number of people within 5 years of leaving secondary school. After that enrolments increased at a faster rate from the 50's to the mid 80's and then increased very quickly in the early nineties, since then they have fluctuated between 57 and 60%. In OECD countries, enrolments in tertiary education rose an average of 40% in the first 6 years of the 1990s which has been largely driven by rising rates of youth participation. The main reasons cited for this increase are:

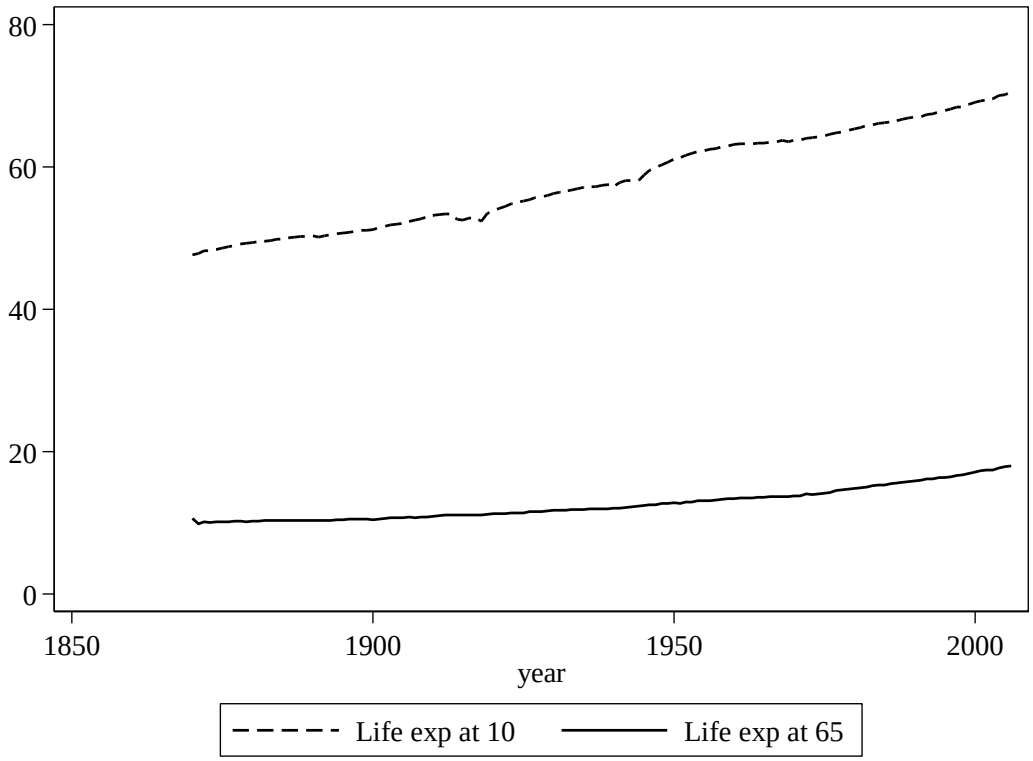
- Firstly, there has been an increase in secondary school completion.
- Secondly cultural preferences have changed as tertiary education becomes more desirable,

- Finally, there has been shift in the demand of employers towards employees with higher skills and knowledge (OECD, 1999).

Life expectancy at age 10 was around 48 at the begining of the period and actually above life expectancy at birth until 1920 relflecting the high magnitude of infant mortality. In other words, once a child reached age 10 it is much more probable they would outlive the average infant. Life expectancy at birth then fluctuated slightly above life expectancy at age 10 until the 1950's when it began to follow a very similar gradual upward trend that life expectancy at age 10 was following. The fact that life expectancy at birth was below life expectancy at age 10 at the begining of the period and above it by the end of the period shows the large reduction in infant and childhood mortality. The overall reduction in mortality is likely due to medical breakthroughs during the early part of the 20th century such as tuberculosis and polio vaccinations as well as the introduction of penicillin, improvements in public health and nutrition as well as other medical treatments (Cutler *et al*. 2006). All of these would have drastically improved the health of the young and vulnerable. The long-term reach of early health factors is considered one of the most important determinants of longevity (Barker, 1990). Of particular importance to the reduction in infant mortality is the improvement in the quality of the milk supply (Lee, 2007). Lee found that in the United States that cleaning up the milk market was the single most important contribution to the decline in the incidence of diarrhea in young children and over all infant mortality. He also reports that it was far more influential than increased family income, improved sanitation and better medical care.

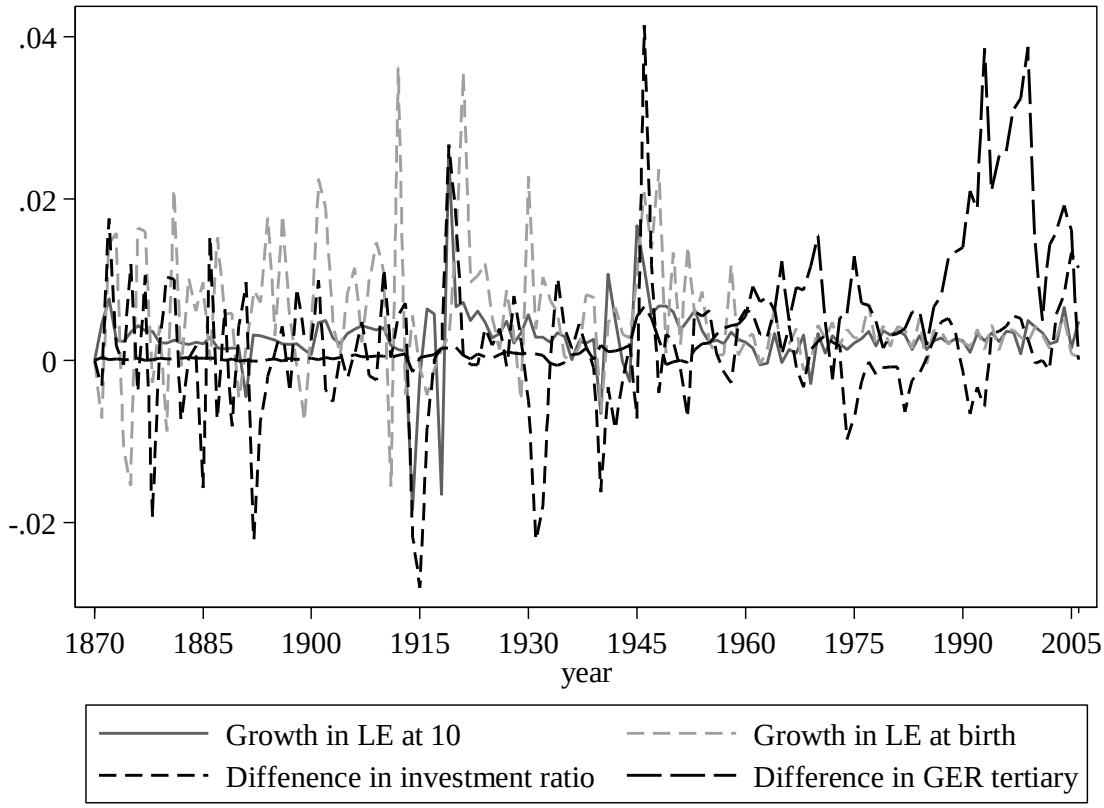
The problem with high infant mortality for this investigation is that it means the connection between a longer life and incentives to invest would be weaker. If there is a high likelihood of premature death, decisions about education would not be made until a child reached an age where life expectancy was more certain. Deaths that occur before schooling begins do not affect the expected time period over which the returns to education are earned. Investments in physical capital are also likely to be affected by life expectancy at a later date. Once the chance of infant and early childhood mortality have been avoided, life expectancy becomes highly correlated itself at different ages. The following shows the relationship between life expectancy at age 10 and life expectancy at 65. The correlation (covariance) coefficient is 0.93. Figure 2 shows the similar paths that life expectancy at age 10 and age 65 follow.

Figure 2: Life expectancy at aged 10 and 65 in years, (1870 - 2006)



All variables have increased over time and are therefore going to produce misleading estimates in regression analysis. For this reason all of the variables used in this analysis have been converted into growth rates (logged differences or just differences in the case of variables that are already expressed as ratios). As can be seen below in figure 3 the growth of each series is stationary over most of the period, and is therefore likely to produce less biased results. The extraordinary growth in GER tertiary 1990 and 1996 (OECD, 1999) is discussed in the next parafigure

Figure 3: Growth rates of GER, the investment ratio and life expectancy at aged 10



Life expectancy at birth is far more volatile than life expectancy at age 10 until the 1950's where it becomes more stable. The growth in life expectancy at birth ranges from a positive growth rate of around 8% (increased longevity) to a negative rate of over 5% (which occurred during the major war periods). There are other periods of rapidly declining longevity probably reflecting various epidemic bouts of disease or infection. These outbreaks reduced significantly with the medical breakthroughs that occurred during the 20th century (Cutler *et al*. 2006).

The growth in GER was positive, relatively constant, between 1 - 2%, until the 1920's. Then it fluctuated at a lower rate until after the Second World War. Since then the GER has generally grown at a positive rate with a very large growth spurt in the early 1990. This growth spurt has been corrected for in the regression analysis by creating a dummy variable that takes the value of 1 for each year from 1990 to 1996. This was the period that has been identified by OECD education policy analysis (1999), and previously mentioned as a time of

unusual growth in tertiary enrolments. On average, this growth rate was 40% across OECD countries over the first 6 years of the 1990's, and has declined since.

The growth in investment as a portion of GDP has usually fluctuated between positive and negative 2%. Larger growth rates occurred after each world war, which is expected due to the need for reconstruction and rebuilding of infrastructure. Life expectancy and investment are particularly affected by war and the dummy used was created for each individual country based on the length of time particular countries were affected by the war.

2.6. Empirical Results

2.6.1. Life Expectancy and Enrolment Rates

GERt is the gross enrolment rate in tertiary education, LE_t is life expectancy at birth, *LE10t*, Y_t is per capita GDP, $\Delta^2 lnY_t$ is the growth in per capita GDP growth or other words the acceleration or deceleration of income growth, g_t . g_{t-5} is a 5 year lag on Y_t. r_t is the real rate of interest. Time is denoted by the subscript *t* and ε_t represents the error term which collects omitted variables and noise, Δ represents change in the variable between two periods and *ln* for the natural logarithm. The first model (model 1) is an Ordinary Least Squares (OLS) regression that has robust standard errors. The second (model 2) is an OLS model that has robust standard errors and also a time dummy for the acceleration in tertiary enrolments in the 1990's. The third model (model 3) is a dynamic OLS with 1 lag and 1 lead. The fourth and the fifth model (models 4 and 5) are the same as models 1 and 2 except that the growth rates are in 5 year differences to eliminate any annual fluctuations or noise.

$$\Delta GER_t = \beta_0 + \beta_1 \Delta lnLE_t + \beta_2 \Delta lnLE10_t + \beta_3 \Delta^2 lnY_t + \beta_4 \Delta^2 lnY_{t-5} + \beta_5 \Delta r_t + \varepsilon_t$$

The results are reported in table 1 on the following page.

Table 1: Results of Life expectancy on Tertiary enrolments

GER	Model 1	Model 2	Model 3	Model 4	Model 5
Life exp. @ 0	-0.046***	-0.010***	-0.097***	-0.831**	-0.743**
	(-6.19)	(-2.54)	(-9.40)	(-1.95)	(-1.77)
Life exp. @ 10	0.013***	0.003***	0.033***	2.92***	2.855***
	(3.40)	(2.35)	(3.08)	(4.75)	(4.77)
Income	-0.001	-0.001	0.003*	0.084	0.088
	(-0.66)	(-1.15)	(1.43)	(0.54)	(0.56)
Income $_{t-5}$	0.002*	0.002**	0.004**		
	(1.52)	(2.15)	(1.67)		
Interest rate	-0.002***	-0.061	-0.228***	-0.005	-0.0004
	(-4.16)	(-1.19)	(-10.87)	(-1.19)	(-0.10)
R^2 (between)	0.07	0.087	0.024	0.099	0.095
observations	2470	2470	2470	494	494

Notes: Statistics are robust to heteroskedasticity and the numbers in parentheses are t statistics.

All estimated coefficients that are significant are of the sign economic theory would predict except for the growth in life expectancy at birth which is significantly negative. This indicates that something is relating prolonged life expectancy at birth to lower gross enrolment rates; this is contrary to widely accepted theory. The coefficients range from -0.046 to -0.831, the interpretation of this is that a 1 point increase in the growth of life expectancy at birth is expected to decrease the growth in enrolments in tertiary education by between -0.046 points and -0.831 points. Gross enrolments in tertiary education have fluctuated between 0.62 % and 65% over the period; In 2006 it is about 64.5% on average across these 19 countries. Recently, the annual models would predict a point increase in life expectancy at birth to decrease GER by between 1% and 9.7%. The magnitudes of the

coefficients on the 5 year differenced data are significantly larger than the annual data, and are more difficult to believe.

The growth in life expectancy at age 10, however, has positive and significant effects on gross enrolment rates. The coefficient on annual differences indicate that the models would expect a 1 point increase in the growth of life expectancy at aged 10 to increase GER by between 0.003 and 0.033 points. Currently the annual models predict a 1 point increase in life expectancy at age 10 to increase GER by between 1.3% and 3.3%. Again, the coefficients on the 5 year differenced data are unreasonably high. Ignoring the 5 year differenced data, this is one of the main results of this chapter; initially high and then declining rates of infant mortality have disrupted the correlation between longevity and tertiary enrolment decisions. Life expectancy at birth should not have the same effect on educational investment as life expectancy at age 10 does. It is not until a child has matured that decisions are made about their education.

The effect of the interest rate is negative as predicted by theory, since the price of investment should be inversely related to the investment rate. The change in the acceleration of the rate of income is not significant unless it is lagged. A 5 year lag was chosen since empirical evidence suggests the correlation between income and investment holds largely because high growth leads to high saving (investment), and not the other way around (Carroll, Overland and Weil, 2000). The fifth lag on the acceleration of per capita income is significant and positive. The 5 year differenced models are consistent with the annual models but only significant for the life expectancy variables, they are insignificant for income and interest rate variables. As previously mentioned 5 year differencing eliminates the noise but also reduces the significance of the results. The results have been recorded below.

2.6.2. Health on Investment

I_t/Y_t, is the investment ratio which is gross capital formation over GDP, LE_t is life expectancy at birth, $LE10_t$ is life expectancy at age 10, Y_t is the growth per capita GDP growth, or acceleration in income growth. Y_{t-5} is a 5 year lag on Y_t. r_t is the difference in the real rate of interest, and q_t is Tobin's q. Time is denoted by the subscript *t*, and ε_t represents the error term which collects omitted variables and noise, Δ represents change in the variable between two periods and *ln* for the natural logarithm. The first model is an Ordinary Least Squares (OLS) regression that has robust standard errors. The second is an iterated Generalized Least

Squares (GLS) autoregressive model that is lagged and is corrected for panel heteroskedasticity. The third model is a dynamic OLS with one lag and one lead. The fourth and fifth models are the same as models 1 and 2 except that the growth rates are in five year differences to eliminate any annual fluctuations or noise.

$$\Delta {}^{I_t}/_{Y_t} = \theta_0 + \theta_1 \Delta lnLE_t + \theta_2 \Delta lnLE10_t + \theta_3 \Delta lnY_t + \theta_4 \Delta lnY_{t-5} + \theta_5 \Delta r_t + \theta_5 \Delta q_t + \varepsilon_t$$

The results are reported on the following page in table 2.

Table 2: Investment responsiveness to life expectancies

Investment ratio	Model 1	Model 2	Model 3	Model 4	Model 5
Life exp. @ 0	0.005	-0.013	-0.007	1.187	0.287
	(0.08)	(-0.73)	(-0.50)	(0.77)	(0.16)
Life exp. @ 10	0.077***	0.083***	0.029***	0.785	0.856
	(3.71)	(4.32)	(3.75)	(0.61)	(0.59)
Income (g)	-0.008	-0.019***	-0.039***	-0.189	-0.282
	(-0.32)	(-5.31)	(12.36)	(-0.67)	(-0.82)
Income $(g)_{t-5}$	0.008	0.006**	0.018***		
	(1.20)	(1.67)	(5.98)		
Interest rate	-0.008	-0.025	-0.028	-0.003	-0.011
	(-0.16)	(-0.74)	(-1.05)	(-0.32)	(-1.26)
Tobins Q	0.005	0.006***	0.010***	0.048	0.139***
	(1.27)	(3.82)	(8.88)	(1.08)	(2.55)
R^2 (between)	0.01		0.098	0.25	0.172
observations	1861	1861	738	360	360

Notes: Statistics are robust to heteroskedasticity have fixed effects, and a time dummy. The numbers in parentheses are t statistics.

The coefficient of life expectancy at birth is not of a consistent sign across the models however it is not significant. Life expectancy at age 10 is highly significant and positive across the first three models; the models using five year differences are not significant. If the annual data is used, a one point increase in the growth rate of life expectancy at age 10 will increase the change in the investment ratio by between 0.029 points and 0.083 points. These coefficients are not very large. The investment (gross capital formation) has ranged between

6% and 45%. In 2006 it was 24.5% across these countries. The annual models predict that a one point increase in the growth of life expectancy at age 10 would increase the investment ratios by between 0.1% and 2%. However, as previously mentioned the results from the annual data are affected by short term economic fluctuations or "noise".

Income is either not significant or significant and negative; the lagged income variable is the opposite. The fifth lag on income is positive and significant. This supports Carroll, Overland and Weil, (2000), who have cited recent empirical evidence that high growth leads to high savings and not the other way around as standard growth models predict. The coefficients on the income variable are also consistent with the results from the regressions on tertiary enrolments. The interest rate is negative and significant across three models which is expected since decreases in the interest rate should encourage investment. These results are consistent with the results from GER. Tobin's q is positive and significant across three of the models and positive and insignificant on the other two. This means that when there is growth in the market value or price of an index in comparison to the cost of replacing it, investment increases. This result is also in line with theory.

Life expectancy at 65 was also used as a regressor; however, as its growth is so highly correlated with the growth in life expectancy at aged 10, the sign and significance of the coefficients were not changed. If both life expectancy at 10 and life expectancy at 65 were included at the same time usually only one would be significant. This is because they are so highly collinear; hence it did not make sense to report both results.

2.7. Discussion

Gross enrolment rates and the investment ratio have increased over time with life expectancy at all ages. The two grow contiguously with life expectancy at age 10 and the opposite direction with life expectancy at birth. The relationship between life expectancy and enrolment in education can be broken into two separate categories. Firstly, there is compulsory enrolment which by definition does not reflect individual decision making. Secondly, there is discretionary enrolment which would very likely be dependent on an individual's perception of the worth of education. Therefore, we find it is connected with individuals' investments and life expectancy. Enrolment in primary education was compulsory throughout most of the period in the 19 OECD countries examined. Gross enrolment in primary school is, therefore, unlikely to be a very informative indication of

changing investment decisions over this period. Secondary enrolment, although not entirely compulsory, has had a compulsory component for much of the period - that is a minimum age for leaving school. Tertiary enrolments have been and are entirely optional (fully discretionary). Hence, the impact life expectancy has on tertiary enrolments is most likely to provide information on investment choices and is the dependent variable that produces the results of this chapter. Life expectancy provides an incentive for prospective students to achieve a higher level of education. However, there are also forces arising from the fact that human and physical capital investments are complements. As economies develop they become more innovative both in production and financial sectors. As this happens there is a higher demand from firms for educated workers.

Investment/savings have increased as a percentage of income overtime. This is again likely to be driven by both increased demand by firms and increased savings from the private sector. As people get older the supply of savings increases, also as capital becomes more profitable and easier to acquire (financial innovations) investment increases. The focus here is the investment ratio increasing with longevity. This is because if people expect to live longer, they should save more of their income during their working life to finance longer retirement. The growth in the investment ratio is positively related to growth in life expectancy at age 10. This is consistent with the results from tertiary enrolments and seems to provide a robust argument for the relationship between longer life and increased investment in both human and physical capital. Even though the results from the physical capital model are not as significant as the tertiary enrolment model, they consistently support the premise that there is a relationship between both types of investment and life expectancy from an age (e.g. 10) where education and retirement seem inevitable.

2.8. Conclusion

Over the last century life expectancy and incomes have improved dramatically, however the relationship is inherently bi-causal. Healthier people are more likely to be more productive and better educated, just as people who have higher income are more likely to be well nourished and have access to better health care. There are a number of different channels through which health may affect growth. This chapter has investigated the effect of life expectancy on investment, focussing on the changes to tertiary gross enrolment rates and savings. The hypothesis tested is that rising longevity increases incentives to invest in

education because the returns to that investment increase with the longer the time horizon over which they can be earned. Furthermore, increased longevity encourages a higher savings rate because there is a higher need for greater future consumption to sustain a longer expected retirement. There is also complementarity between human and physical capital investments. Increases in education lead to better utilization of physical capital.

Previously investigations of this nature have used life expectancy at birth to relate life expectancy to education and growth; however, this measure suffers from bias due to changing levels of infant mortality rates. Deaths that occur before schooling do not affect the expected period over which returns to education are earned, nor do they affect the need to save for retirement. We proposed that life expectancy later in childhood or adolescence would be the most appropriate measure when investigating investment behaviours, particularly the schooling decision. Life expectancy at age 10 was chosen because primary schooling was compulsory throughout the period studied, life expectancy in early adolescence should be equally informative.

The results show there is a significant and positive relationship between life expectancy at age 10 and investment. If increased longevity does provide incentives to invest in both education and physical capital there are important macro policy implications for both developed and developing economies. Firstly, this chapter has argued that at least in the case of tertiary enrolments the causality should run from life expectancy to investment. Higher enrolment in tertiary education should improve the health of the generation following those who were better educated, since it is the education of the parents that affects the health of their children; therefore reverse causality does not make sense. Secondly, increases in life expectancy should increase savings and therefore investment due to the need to save for a longer retirement. However, we have also followed Carrol *et al.*(2000) and assumed that accelerated growth will precede increased capital investments. Both of these premises were confirmed by the empirical tests.

The policy implications from this investigation are very important for developing countries. To clarify the association between this study based on data from developed countries and the developing world, an assumption needs to be made. It was unavoidable here to relate tertiary enrolments to life expectancies because in developed countries tertiary enrolments are the only enrolment rates that have been totally discretionary. In developing countries, enrolments (or at least attendance) in all levels of education can be discretionary. To draw

policy recommendations for developing countries it is necessary to assume that the results here would hold for enrolments at the primary, secondary and tertiary levels as long as they were discretionary. If this is the case policy makers need to improve the health and therefore life expectancy of the population in order to encourage them to educate. Otherwise, pouring money into providing public schooling may be not be successful, children will either not be enrolled or not attend. Only once they think it is likely they will live long enough to reap the benefit of this education, will they invest time in schooling. This is not to say that education is not important, just that attendance and enrolments may not achieve satisfactory levels until malnutrition and serious disease such as HIV, malaria and dysentery are under control. For developed nations, because enrolments are already high the policy implications are beyond the realms of the empirical investigation here. Nonetheless since healthier children benefit more from education than less healthy children, this chapter contends for continuing and possibly increasing the public expenditure on campaigns and institutions that encourage healthier lifestyles.

2.9. Appendix 1: Data Sources

Education

Gross enrolment rates in tertiary education. The data is from Madsen (forthcoming)

Output:

Economy-wide real GDP. The data are from Madsen (2008)

Employment:

Total employment in persons. The data are from Madsen (2008)

Hours worked:

Annual hours worked on average per worker. The data are from Madsen (2010)

Investment:

Gross domestic capital formation. The data are from Madsen (2010)

Tobin's q

Is the market value of a group of companies divided by its book value. The data are from Madsen (2011)

Life Expectancy:

World Population, *An Analysis of Vital Data*, Nathan Keyfitz and Wilhelm Flieger, The University of Chicago Press Chicago and London

Life Expectancy at Age 10:

USA

1830-1860 Haines, M.R. and Avery, T.C.,1980, The American Life Table of 1830-1860: An evaluation, The Journal of Interdisciplinary History, Voll 11, No 1, pp 73-95 The MIT Press

1850 to 1900 from Historical Statistics of the United States, Millennial Edition, Volume 1 Population. The data was life expectancy at age 5. The difference between LE age 5 and LE age 10 in the UK was taken from World population (above) and the LE age 10 figure for USA is LE age 5 less the difference between LE 5 and LE 10 in the UK

1900 to 2000 Felicitie C. Bell and Michael L. Miller . Life Tables for the United States Social Security Area 1900-2100 Actuarial Study No. 116 http://www.lifetable.de/data/MPIDR/USA000019001999CY1.pdf

2000 Arias, E., United States Life Tables, 2000, National Vital Statistics Reports, Volume 51, Number 3, December 19, 2002, p. 1-39. www.lifetable.de

2001-2006 National Vital Statistics Reports http://www.cdc.gov

Canada

1831-1921 Statistics Canada, Demofigurey Division, Demofigureic Document No.3, Current Demofigureic Analysis, New Birth Cohort Life Tables for Canada and Quebec, 1801-1991 by Robert Bourbeau, John Légaré, Valérie Émond, September 1997, p.72 http://www.lifetable.de

1921-1996 Collection of Canadian Life Tables Recueil de tables de mortalité canadiennes 1801-1996 http://www.prdh.umontreal.ca/BDLC/data/pdfs/CAN.pdf

1996-2002 Statistics Canada, Life Tables, Canada, Provinces and Territories, , Complete life table, Canada, downloaded from http://www.statcan.ca/english

2002-2006 Extrapolated based on US data. US data was chosen due to geofigureical proximity.

Japan

1870-1894 Extrapolated based on Austrian data. This is because the life expectancy in Austria in 1895 was the most similar to the Japanese life expectancy in that year. 1895 was the first actual data recorded for Japan.

1895-2006Statistics and Information Department, Minister's Secretariat, Ministry of Health, Labour and welfare http://www.lifetable.de "Historical Statistics of Japan"

Australia

1860-1875 Australian Historical Statistics 1856-1875 , Victorian State Library, Melbourne

1891 - 1922 Statistical Register of South Australia 1963-1964, Part II - Demofigurey, Australian Life Tables http://www.lifetable.de

1923-1932 Interpolated

1932-1980 Census of the Commonwealth of Australia. Australian Life Tables. Commonwealth Bureau of Census and Statistics, Canberra. http://www.lifetable.de

1980- 2006 Australian Bureau of Statistics, Life tables of Australia http:www.abs.gov.au/AUSSTATS/abs
New Zealand
1870-1892. Extrapolated based on UK data
1876-1931. Statistics New Zealand, Tatauranga Aotearoa. Life expectancy by age, sex and birth cohorts: 1876-2011 http://www.stats.govt.nz
http://www.stats.govt.nz/browse_for_stats/health/life_expectancy/cohort-life-tables.aspx
1936-1970. New Zealand, Life Tables and Life Annuity Tables, Department of Statistics Publication, Wellington, N.Z.
1970 – 1996. New Zealand Life Table, Contribution from Dr. V. Kannisto's Life Table collection
1996-2006 Statistics New Zealand. New Zealand life table. website http://search.stats.govt.nz
Austria
1870-1933. The Societies of Europe. The European Population 1850-1945. Franz Rothenbacher. 2002. Palgrave MacMillan p. 104
1866 – 2009. Statistik Austria, Jährliche Sterbetafeln seit 1947, downloaded from http://www.statistik.at and available at http://statline.cbs.nl .
Belgium
1856-1880 – Collected estimates of early life expectancies and infant mortality rates from around the world, mostly pre-1900, Peter Lindert, gpih.ucdavis.edu
1885-1930. The Societies of Europe. The European Population 1850-1945. Franz Rothenbacher. 2002. Palgrave MacMillan p. 136
1930-1995 Annuaire Statistique de la Belgique, Institut National de Statistique, Bruxelles http://www.lifetable.de
1996-2005 Démofigureie mathématique, Tables de mortalité, Tables annuelles 2004, Direction Generale Statistique et Information Economique, Bruxelles, http://www.lifetable.de
2006 Service Public Federal, Economie, P.M.E., classes moyennes et energie, Statistique et Information économique, Tables de mortalité 1996-2007
http://statbel.fgov.be/fr/modules/publications/statistiques/population/table_de_mortalite.jsp
Denmark
1840-1943. The Societies of Europe. The European Population 1850-1945. Franz Rothenbacher. 2002. Palgrave MacMillan p. 188
1943-2006 Statistical Year Books and Life Tables of Denmark downloaded from http://www.lifetable.de
France
1806-1997. Vallin, J. and Mesle, F., (2001), Tables de mortalité françaises pour les XIXe et Xxe siècles et projections pour le XXIe siècle, Institut national d'etudes demofigureiques
1824-1935. The Societies of Europe. The European Population 1850-1945. Franz Rothenbacher. 2002. Palgrave MacMillan p. 254
1881-2000. http://www.lifetable.de
2000-2005 Insee, état civil Table de mortalité des années; Mortalité générale;
downloaded from http://www.ined.fr
2006 TABLEAU 68 - TABLE DE MORTALITÉ DES ANNÉES 2006 – 2008 données provisoires arrêtées à fin mars 2010 www.inee.fr downloaded from
http://www.ined.fr/fr/pop_chiffres/france/mortalite_causes_deces/table_mortalite/
telechargement_fichier_fr_sd2006_t68_fm.xls; (15.09.2008)
Germany
1871-1934. The Societies of Europe. The European Population 1850-1945. Franz Rothenbacher. 2002. Palgrave MacMillan p. 292

1870-1910. Bewegung der Bevölkerung im Jahre 1910, Statistik des Deutschen Reichs, Vol. 246, Berlin 1913, 16-17. Downloaded from http://www.lifetable.de
1986-1988 Periodensterbetafeln für Deutschland, Allgemeine und abgekürzte Sterbetafeln von 1871/1881 bis 2004/2006, Statistisches Bundesamt, Wiesbaden. Downloaded from: https://www-ec.destatis.de
1991-1999. Bevölkerung und Erwerbstätigkeit, Fachserie 1, Reihe 1, Gebiet und Bevölkerung Statistisches Bundesamt Wiesbaden, Metzler-Poeschel Verlag, Stuttgart. Downloaded from http://www.lifetable.de
2001-2003 Periodensterbetafeln für Deutschland, Allgemeine und abgekürzte Sterbetafeln von 1871/1881 bis 2001/2003, Statistisches Bundesamt, Wiesbaden 2004
2004-2006 Sterbetafel für Deutschland 2002/2004, Statistisches Bundesamt, Wiesbaden. Downloaded from http://www.lifetable.de

Ireland

1870-1900 Extrapolated backwards based on English data
1900-1942. The Societies of Europe. The European Population 1850-1945. Franz Rothenbacher. 2002. Palgrave MacMillan p. 418
1925-1937. Census of Population of Ireland, Vol.V, Part 1: Ed. Department of Industry and Commerce, Statistics Branch,Dublin. Downloaded from http://www.lifetable.de
1940-1942. Register of Population 1941: Ed. Department of Industry andCommerce, Statistics Branch, Dublin, 1944, 82-85. Downloaded from http://www.lifetable.de
1960-1992 Irish Statistical Bulletin, Central Statistics Office, Dublin. Downloaded from http://www.lifetable.de
1995-2002. Irish Life Table, Central Statistical Office, Dublin, 2001. Downloaded from http://www.lifetable.de
2006. Central Statistics Office, Irish Life Tables 2006 downloaded from http://www.cso.ie/px/pxeirestat/Dialog/Saveshow.asp

Italy

1870-1875. Extrapolated backwards based on French data
1876-1937. The Societies of Europe. The European Population 1850-1945. Franz Rothenbacher. 2002. Palgrave MacMillan p. 448
1930-1962. Italian Life Table, Contribution from Dr. V. Kannisto's Life Table collection. Downloaded from http://www.lifetable.de
1974-1991. Tivoli di mortalità della popolazione . Downloaded from http://demo.istat.it
1993-1994 Annuario Statistico Italiano 1996 - Statistical Yearbook of Italy 1996: Ed. National Statistical Institute, Rome, 73-74
1999-2005. Istat - Istituto Nazionale di Statistica. [Demo: demofigurey in figures]: Tavole di mortalità della popolazione italiana -Italia; downloaded from: http://demo.istat.it
2006-2007. Tavole di mortalità della popolazione italiana per provincia e regione di residenza – Anno 2007 – Italia, downloaded from http://demo.istat.it/unitav/download.html

Netherlands

1816-1940. The Societies of Europe. The European Population 1850-1945. Franz Rothenbacher. 2002. Palgrave MacMillan p. 510
1861-1980. Life Tables of Netherlands, downloaded from: http://statline.cbs.nl . and available at http://statline.cbs.nl
1979- 1999 Centraal Bureau voor de Statistiek. Downloaded from http://www.lifetable.de .
2000-2007 Overlevingstafels; geslacht en leeftijd, 2007. Downloaded from http://www.lifetable.de and available at http://statline.cbs.nl .

Norway

1821-1941. The Societies of Europe. The European Population 1850-1945. Franz Rothenbacher. 2002. Palgrave MacMillan p. 540

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1998-2000. Population Statistics, Statistisk Sentralbyrå, Oslo Downloaded from http://www.lifetable.de .
2001-2006. Life Tables 2001, Statistisk Sentralbyrå, Statistics Norway. Downloaded from http://www.lifetable.de .
2009 Statistisk Sentralbyrå, Statistics Norway, 2008: Population statistics. Table 5 Life 2009, downloaded from:http://www.ssb.no and available at http://www.lifetable.de
Portugal
1870-1920. Extrapolated from Spanish data.
1920 - Collected estimates of early life expectancies and infant mortality rates from around the world, mostly pre-1900, Peter Lindert, gpih.ucdavis.edu
1929-1942. The Societies of Europe. The European Population 1850-1945. Franz Rothenbacher. 2002. Palgrave MacMillan p. 600
1939-1942. Tábua de mortalidade da população portuguesa, 1939-1942, p.24-27. Downloaded from http://www.lifetable.de
1941-1975 Tábuas Abreviadas de Mortalidade, 1941-1975, Estudos 56, Portugal, 1980. Downloaded from http://www.lifetable.de
1979-1982. Tábuas Abreviadas de Mortalidade Distritos e Regiõs Autónomas 1979-1982, Caderno. N.° 7, Centro de Estudos Demográficos, Instituto Nacional de Estatística, Portugal, 1988, p. 76-77. Downloaded from http://www.lifetable.de
1990-2003 Tábuas de Mortalidade em Portugal, Tábua completa (or abreviada) de mortalidade. N.° 7, Centro de Estudos Demográficos, Instituto Nacional de Estatística, Portugal, 1988,
2005. Tábua Completa de Mortalidade para Portugal 2004-2006 http://www.ine.pt/
2006-2007 Complete Life Table for Portugal 2006 – 2008, Instituto Nacional de Estatística, Statistics Portugal, May 2009. Downloaded from: http://www.ine.pt
Spain
1870-1879. Extrapolated from English data
1880-1900 – Instituto Nacional de Estadistica. Statistical Yearbood Accessed from http://www.ine.es
1900-1940 The Societies of Europe. The European Population 1850-1945. Franz Rothenbacher. 2002. Palgrave MacMillan p.632
1900-1950 Presidencia del Gobierno, Instituto Nacional de Estadistica. Tablas de Mortalidad de la Población Española Años 1960- 1970, Madrid, 1977 Downloaded from http://www.lifetable.de
1961-2001 Llorens, A.B., (2007, "La mortalidand en la Espana del Siglo XX. Analisis demografico y territorial, Tesis Doctoral, Doctorado en Demografia, Departamento Geografia de la UAB
1975 – 1976. Ministerio de Economia y Comercio Instituto Nacional de Estadistica
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1985-1986. Instituto Nacional de Estadistica Tablas de Mortalidad de la Población Española Madrid, 1991 Downloaded from http://www.lifetable.de
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Sweden
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2001-2005Official Statistics of Sweden, Life tables for the period 2002-2006,
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1998-2003. Sterbetafel für die Schweiz 1998/2003, Bundesamt für Statistik, downloaded from http://www.bfs.admin.ch
2005-2008 Bundesamt für Statistik, Statistik Schweiz, personal communication.
http://www.bfs.admin.ch
United Kingdom
1841-1932. The Societies of Europe. The European Population 1850-1945. Franz Rothenbacher. 2002. Palgrave MacMillan p.744
1838-1972. English Life Table No. 14, Contribution from Dr. V. Kannisto's Life Table collection.
1980-2008. Interim Life Tables, produced by the Government Actuary's Department, England and Wales 1980-1982, downloaded from http://www.gad.gov.uk

2.10. Appendix 2: Mathematical Appendix

The objective is maximizing utility which depends on the leisure-labour trade off during childhood, consumption in middle age and consumption in old age which is discounted by the time preference parameter and the probability of survival to old age.

$$\mathrm{Max} U_t = \varphi \ln(1 - e_t) + \ln c_{t+1} + p\delta \ln c_{0,t+2}$$

s.t.

$$h_{t+1} = Ae_t h_t$$

$$c_{t+1} = w_{t+1} h_{t+1} - s_{t+1}$$

$$c_{0,t+2} = r_{t+2} s_{t+1}/\mathrm{p}$$

The Lagrangian is

$$\mathrm{Max} u(\mathrm{e}, \mathrm{c}, \mathrm{n}, \lambda) = \varphi \ln(1 - e_t) + \ln c_{t+1} + p\delta \ln c_{0,t+2} - \lambda_1 (h_{t+1} - Ae_t h_t) - \lambda_2 (c_{t+1} - w_{t+1} h_{t+1} + s_{t+1}) - \lambda_3 (c_{0,t+2} - r_{t+2} s_{t+1}/\mathrm{p}) \quad (1)$$

Partial derivatives equal to zero

$$\frac{\partial u}{\partial e_t} = -\frac{\varphi}{1 - e_t} + \lambda_1 A h_t = 0 \quad (2)$$

$$\frac{\partial u}{\partial c_{t+1}} = \frac{1}{c_{t+1}} - \lambda_2 = 0 \quad (3)$$

$$\frac{\partial u}{\partial c_{t+2}} = \frac{p\delta}{c_{t+2}} - \lambda_3 = 0 \quad (4) \text{ and } (5)$$

$$\frac{\partial u}{\partial h_t} = \lambda_1 A e_t = 0 \quad (6)$$

$$\frac{\partial u}{\partial h_{t+1}} = \lambda_1 w_{t+1} - \lambda_2 = 0 \quad (7)$$

$$\frac{\partial u}{\partial s_{t+1}} = -\lambda_2 + \frac{\lambda_3 r_{t+2}}{p} = 0 \quad (8)$$

$$\frac{\partial u}{\partial \lambda_2} = c_{t+1} - w_{t+1} h_{t+1} + s_{t+1} = 0$$

$$\frac{\partial u}{\partial \lambda_3} = c_{t+2} - \frac{r_{t+2} s_{t+1}}{p} = 0 \quad (9)$$

$$\frac{\partial u}{\partial \lambda_1} = h_{t+1} - Ae_t h_t = 0 \quad (10)$$

From (4)

$$\lambda_3 = \frac{p\delta}{c_{t+2}} \tag{11}$$

From (9)

$$c_{t+2} = \frac{r_{t+2} s_{t+1}}{p} \tag{12}$$

From (8) and (11)

$$\lambda_2 = \frac{\lambda_3 . r_{t+2}}{p}$$

$$\lambda_2 = \frac{p\delta}{c_{t+2}} \frac{. r_{t+2}}{p} \tag{13}$$

$$\lambda_2 = \frac{\delta . r_{t+2}}{c_{t+2}}$$

Therefore,

$$\lambda_3 = \frac{\delta p}{s_{t+1}}$$

From (3)

$$\frac{1}{\lambda_2} = c_{t+1}$$

and from (8)

$$s_{t+1} = w_{t+1} h_{t+1} - c_{t+1}$$

$$s_{t+1} = w_{t+1} h_{t+1} - \frac{1}{\lambda_1}$$

$$s_{t+1} = w_{t+1} h_{t+1} - \frac{s_{t+1}}{\delta p}$$

$$s_{t+1} + \frac{s_{t+1}}{\delta p} = w_{t+1} h_{t+1}$$

$$s_{t+1}\left(\frac{1+\delta p}{\delta p}\right) = w_{t+1} h_{t+1}$$

$$s_{t+1} = \frac{\delta p}{1+\delta p} w_{t+1} h_{t+1} \qquad (14)$$

Let $\alpha_s = \frac{\delta p}{1+\delta p}$ which is the marginal propensity to save, because consumption in middle age is equal to income less savings

$$c_{t+1} = w_{t+1} h_{t+1} - s_{t+1}$$

From (14)

$$c_{t+1} = w_{t+1} h_{t+1} - \frac{\delta p}{1+\delta p} w_{t+1} h_{t+1}$$

$$c_{t+1} = w_{t+1} h_{t+1}\left(1 - \frac{\delta p}{1+\delta p}\right)$$

$$c_{t+1} = \left(\frac{1}{1+\delta p}\right) w_{t+1} h_{t+1}$$

$$c_{t+1} = \frac{1}{1+\delta p} w_{t+1} h_{t+1} \qquad (15)$$

$\alpha_c = \frac{1}{1+\delta p}$ which is the marginal propensity to consume from current income

To derive the optimal level of education, from (2)

$$-\varphi = \lambda_1 A h_t (e_t - 1)$$

$$e_t = \frac{-\varphi}{\lambda_1 A h_t} + 1$$

Substituting the formula for human capital accumulation

$$h_{t+1} = Ae_t h_t$$

$$e_t = 1 - \frac{Ae_t \varphi}{\lambda_2 A h_{t+1}}$$

From (7)

$$e_t = 1 - \frac{e_t \varphi}{\lambda_1 w_{t=1} h_{t+1}}$$

Substituting (3) in for lambda

$$e_t = 1 - \frac{e_t c_{t+1} \varphi}{w_{t=1} h_{t+1}}$$

$$e_t + \frac{e_t c_{t+1} \varphi}{w_{t=1} h_{t+1}} = 1$$

$$\left(\frac{w_{t=1} h_{t+1} + c_{t+1} \varphi}{w_{t=1} h_{t+1}} \right) e_t = 1$$

$$e_t = \frac{w_{t=1} h_{t+1}}{w_{t=1} h_{t+1} + c_{t+1} \varphi}$$

$$e_t = \frac{w_{t+1} h_{t+1}}{w_{t+1} h_{t+1} - \frac{1}{1+p\delta} w_{t+1} h_{t+1} \varphi}$$

$$e_t = \frac{1}{1 - \frac{\varphi}{1+p\delta}}$$

$$e_t = \frac{1}{\frac{1+p\delta-\varphi}{1+p\delta}}$$

$$e_t = \frac{1+p\delta}{1+p\delta-\varphi} \qquad (16)$$

And then using quotient rule

$$\frac{\partial e_t}{\partial p} = \frac{\delta(1-\varphi+p\delta)-\delta(1+p\delta)}{(1-\varphi+p\delta)^2}$$

$$\frac{\partial e_t}{\partial p} = \frac{\delta(1-\varphi+p\delta 1-1-p\delta)}{(1-\varphi+p\delta)^2} = \frac{-\varphi\delta}{(1-\varphi+p\delta)^2} < 0 \qquad (17)$$

The real wage and interest rate are:

$$w_t = (1-\propto)A\left({K_t}/{L_t h_t}\right)^{\propto} \quad \text{and } r_t = \propto A\left({K_t}/{L_t h_t}\right)^{\propto-1}$$

2.11. Appendix 3: Country codes (fcode) for Stata

1. Canada
2. USA
3. Japan
4. Australia
5. New Zealand
6. Austria
7. Belgium
8. Denmark
9. France
10. Germany
11. Ireland
12. Italy
13. Netherlands
14. Norway
15. Portugal
16. Spain
17. Sweden
18. Switzerland
19. UK

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3. Public versus Private Health: who provides better health outcomes?

Abstract

With health care policy at the forefront of public concern, governments worldwide want to address the future and sustainability of their current health care systems. This study uses international data of 18 countries from 1960 to 2008 to explore the link between health outcomes and health expenditure. Public and private health expenditures are not assumed to be perfect substitutes, and are therefore considered separately. Life expectancy and infant mortality are used to proxy health status. The estimation assumes income, education, and health care expenditures are endogenous and a unique set of instrumental variables are used to control for this problem. The evidence shows that public and private health expenditures are not perfect substitutes and should be considered separately when analysing policy options. The results do not find a compelling argument for policy makers to move away from the public provision of healthcare.

3.1. Introduction

This chapter investigates data from 18 OECD countries from 1960 to 2008. The purpose is to establish empirically if historical data suggests that public and private health expenditures are imperfect substitutes; that is increasing the expenditure in one sector may have greater effects on health outcomes than increasing expenditures in the other. The importance of this point is twofold. Firstly, from a theoretical perspective health expenditure is generally treated as a whole, that is, it is assumed that health outcomes depend on health expenditures regardless of where the funding comes from. This implies that they are perfect substitutes and means that model outcomes may not be as accurate as they would be if health expenditures were treated separately based on the funding source. Secondly, from a practical perspective if one source of funding has previously been more effective than the other, having a larger impact on social health, then governments need to consider this when reforming healthcare systems. There is a large body of literature on healthcare and healthcare reform however there is very little

empirical work that separates health effects of public and private health expenditures. This chapter proposes to do this as well as addressing the problem of endogeneity in the model, which has not been done before.

Healthcare reform is a key challenge for the majority of countries. Most developed countries are grappling with the problems of ageing populations, increased demand for healthcare, rising medical inflation and competition for tax revenues. Every developed country faces similar cost pressures. In the late 2000s these pressures have increased with global economic conditions, this has accelerated the need for reform as governments try to reduce their exposure to healthcare costs. One way of achieving this is to shift costs from the state to employers and individuals.

Historically, there have been two main models for the funding and delivery of healthcare: the Bismark model, and the Beveridge model. Since their introduction most developed countries have implemented a mixture of the two. The Bismark model (often referred to as Social Health Insurance or SHI) is based on the German social protection system introduced by Otto von Bismarck in the 1880's. In this model, health coverage is provided by a social insurance fund that is funded either privately, publicly or a mixture of both. The Beveridge model originated in the UK in the 1940's; it is based on healthcare funded purely out of general taxation, where government manages both the funding and delivery of health services through a national health system

Generally, health systems consist of a mix of public and private sector health services and a range of funding and regulatory mechanisms. Service providers include: private medical practitioners, other health professionals, public and private hospitals, clinics, and other government as well as non-government agencies. Funding is provided by federal governments, state or local governments, health insurers, individuals and a range of other sources (e.g. not for profit). The analysis of expenditure data may be inconsistent because much of the information regarding health outcomes is not represented by expenditures alone such as the structure and incentive schemes of the healthcare system. There is a lot of variety in the mix of public and private expenditures across countries as well as in the mode of delivery. While the differences in financing undoubtedly affect behaviour and outcomes, the differences in delivery, or in other words, the design of the healthcare system are potentially of equal or more significance to health outcomes than funding. Varying the structure and

delivery of healthcare alters the incentives given to both the providers of healthcare and the receivers of the care (patients) in as many ways as there are different systems.

Countries such as Australia, Austria, Belgium, France, Germany, Netherlands, Switzerland and the United States have predominately SHI (Bismark) based healthcare systems. The systems are funded through various mixes of public and private contributions to the health fund. Denmark, Finland, Ireland, Norway, Spain, Sweden, Canada and the United Kingdom have predominately "Beveridge" style tax based national health provision (Saltman and Dubois, 2004). In Australia, there has been an oscillation between public and private health systems that is unique among OECD countries. These ups and downs of healthcare funding derive from the prevailing political party competition.

The US is experiencing somewhat of a crisis in its healthcare system paying roughly twice as much per capita for healthcare than Canada, France and the United Kingdom yet experiencing lower life expectancy and higher infant mortalities. The US has by far the lowest public contribution to healthcare of all the countries mentioned above. Is this one reason for their poorer health? This paper will try to shed some light on this question by focussing on the source of funding and its health outcomes amongst the following 18 countries: Canada, USA, Japan, Australia, New Zealand, Austria, Belgium, Denmark, Finlnd, France, Germany, Greece, Ireland, Italy Netherlands, Norway, Portugal, Spain, Sweden, Switzerland, and the United Kingdom.

Increasing expenditure on healthcare should improve health outcomes regardless of how it is financed. It is the magnitude of this impact and the effect of any efficiency losses that should differ depending on the financing. Public provision of a good or service requires taxation and then subsidised supply. This interferes with the market pricing mechanism, alters incentives and usually the quantity and quality demanded. This implies that public financing of a good is not necessarily a perfect substitute to private financing of that same good. The public provision of healthcare is no different. Hence, altering the concentration of public health provision would affect prices and therefore consumption and savings decisions, which may impact health outcomes, the broader economy, and economic growth.

Public provision of healthcare promotes social equality because it is available to every citizen. But it also has the disadvantage of tax distortion in discouraging work and other distortions associated with bureaucracies and public supply. This includes inflating demand

due to subsidised (cheaper/free) prices on many services, such as people attending medical clinics for un-required treatments simply because they have the time and it is not costly.

Along with the advantages of equity there are also important areas of healthcare that are public goods by nature because of the social benefits of supply being far greater than private benefits (i.e. positive externalities). These include research, the reduction of infectious diseases through increased vaccinations, clean water, hygienic waste disposal, and encouraging healthy lifestyles. Examples of encouraging healthy lifestyles include promotion of exercise, and healthy eating and the discouragement of damaging activities such as smoking, and excessive consumption of alcohol or fatty foods.

Private healthcare is not available to everyone as some cannot afford it, however, it may be less of a social burden because using it may avoid the inefficiencies of public supply. Having said that, private providers and healthcare professionals create a different type of inefficiency; they attempt to inflate healthcare prices and over supply care, where on the other hand, governments have incentives to seek ways to moderate health inflation and reduce hospital stays.

Previously, much of the academic literature (empirical and theoretical) has assumed that public and private healthcare providers are the same, and have therefore used aggregate health expenses in their studies. Now with ageing populations and associated fiscal pressures, governments are pushing towards private provision of healthcare even though, from a purely social welfare perspective, it may be better to continue with public provision. It is important to question this assumption and therefore try to establish whether or not one form of healthcare provision is more effective.

After a brief literature review and theoretical overview, the empirical investigation will try to answer these questions. This will include an explanation of the empirical methods used as well as the technical problems encountered with the data and specifications. Most importantly the chapter will use an Instrumental Variable (IV) approach to alleviating the endogeneity issues that are inherent in the health literature as this has not been done before in this context, nor with such sound instruments. The chapter will conclude with a discussion of the results and suggestion of any policy implications that may be drawn from this study.

3.2. Literature Review

Auster et al. (1969), in their pioneering study on health production functions, investigated the effects of healthcare on mortality inspiring further research into the determinants of life expectancy and mortality rates. This line of literature focuses on macroeconomic data and estimates the overall effect of healthcare on the health status of the population. Examples are: Cochrane et al. (1978), Leu (1986), Wolfe and Gabay (1987), Hitiris and Posnet (1992), Babazono and Hillman (1994), Grubaugh and Rexford (1994), Elola et al. (1995), Barlow and Vissandjee (1999), Grossman (2000), Cremieux et al. (1999), Lichtenberg (2000), Robalino et al. (2001), Berger and Messer (2002), Asiskovitch (2010), Halicioglu (2010), and Pocas and Soukiazis (2010). None of these studies treat healthcare expenditures separately they all assume public and private healthcare are perfect substitutes.

In empirical research, health outputs are either measured by mortality rates as in Babazono and Hillman (1994) and Berger and Besser (2002) or by life expectancy. Life expectancy is either measured at birth (which captures infant mortality) as in Lichten (2000) or at various ages as in Miller and Frech (2002). All of these studies consider a number of determinants of a nation's health status such as national income, health expenditure, and some include others such as medical innovation (Lichtenberg, 2000), food expenditure and pharmaceutical expenditure (Shaw *et al*. 2005**)**. They also include life style measures such as cigarette smoking and alcohol consumption (Cochrane *et al*., 1978) as well as education (Cremieux et al. 1999), fat consumption (Berger and Messer, (2002), and obesity (Miller and Frech, 2002). Environmental determinants are also considered such as, urbanization (Leu, 1986) and pollution (Or, 2000)**.**

With the exception of Leu (1986) and Asiskovitch (2010) none of these empirical studies examine the health outcomes from private health expenditures separate to those from public expenditures. This assumes that life expectancy depends on gross health expenditure and implies that the effect on life expectancy from an increase in public health expenditure would be identical to that from an increase in private health expenditure. In other words they assume the two are perfect substitutes. There is no logical justification for this assumption; there are many differences. Public funding is the incentive mechanisms surrounding the two modes of funding. Leu (1986) examines the effect on post-neonatal mortality in 19 OECD countries and differentiates between public and private expenditure on healthcare. Leu (1986) finds that health expenditure does not have a significant impact on health outcomes when income is

controlled for. The study also suggests that third party (private) financing may have an adverse influence on post-neonatal mortality rates.

Asiskovitch (2010) investigates the impact of healthcare systems and their mix of public-private funding. This study finds that a public mode of funding has a greater impact on health outcomes than private and also that health expenditure benefits the old more than the young.

3.2.1. Endogeneity

The econometric methodologies and procedures between these studies vary from simple descriptive statistics to more complex panel data econometrics. However, very few studies of the determinants of health status consider the issue of endogeneity and health expenditures together. There is consensus in the health literature on the existence of a positive correlation between health, education and income. However, the direction of causality is not clear and there may also be other factors that influence all three simultaneously. The existence of this problem means the estimated results may be biased and inconsistent.

Halicioglu (2010) is concerned with endogeneity when determining the factors affecting life expectancy in Turkey. The empirical estimation is conducted on the basis of the Autoregressive Distributed Lag (ARDL) approach to cointegration. The finding is that nutrition and food availability along with health expenditures are the main factors improving longevity, however, there is no differentiation between public and private health expenditure. When dealing with endogeneity, it is necessary to use a more specified approach than the more general ARDL. The instrumental variable method is the only way to deal properly with endogeneity.

Pocas and Soukiazis (2010) analyse determinants of life expectancy using data from 17 OECD countries. The aim is to estimate a health production function, to analyse the impact of education, income, medical care, and cigarette and alcohol consumption on health improvements. They also address the endogeneity by using instruments. The paper adopts a two-step GMM (fixed effects) estimation method using instruments to deal with the endogeneity of education and income. The instruments were chosen based on a significance of 5% of the correlations between the endogenous variables and other variables that can serve as instruments. The appropriate instruments they identified were total expenditure on health,

number of medical graduates per practicing physicians and the number of patents per million. The problem is that these instruments are likely to be highly endogenous. The instruments are validated using the Sargan-Hansen test to rule out over identification, and revealed no correlation with the error term. Inferences are also made about error autocorrelation and homoskedasticity using the Wooldridge test. The study uses total healthcare expenditure, as most other studies do, and therefore assumes the impact on longevity of a one dollar increase in private health expenditure is equal to the impact of a one dollar increase in public health expenditure. The outcome of this investigation is that income, education and health resources (consultations per capita, but not expenditure) all positively affect life expectancy.

So far there exists no previous study that addresses the endogeneity issues whilst analysing the debate on public versus private financing (provision) of healthcare. Therefore, the aim is to investigate the differential impact of public and private spending (using different life expectancy data) and adopting the more rigorous methods of Pocas and Soukiazis (2010). Hence, providing a more reliable answer to the question of whether public spending has a more beneficial impact on health outcomes than private provision, or not. From a social welfare perspective, public health expenditure should be more effective since it is available to the whole population while private is only available to those who can afford it. Also, private healthcare is often cosmetic or elective types of expenditures which would not improve life expectancy, whereas public expenditures are limited to necessary treatments.

3.3. Theoretical Outline

This investigation is guided by a basic health production model adapted to include the possibility that private and public health expenditures will have different impacts on health outcomes. Agents are identical and time is discrete. The individual derives utility from the consumption of good health, h_t, and the consumption of all other goods, c_t. The stock of good health is inherited initially and then depreciates over time but may be augmented by acts of health investments. Health investments include healthcare expenditure and all other health promoting activities such as good food, exercise, clean air and water etc. The model has log utility that is a function of the consumption of good health, h_t, and the consumption, c_t, of all other goods.

$U(c_t, h_t) = \ln(c_t) + \propto \ln(h_t)$,

(1)

With $\propto \in (0,1)$.

The instantaneous utility function is continuous, twice differentiable, strictly increasing (i.e. $U'(c_t, h_t) > 0$) and strictly concave (i.e. $U''(c_t, h_t) > 0$) and satisfies the Inada conditions $\lim_{c\to 0} U'(h_t) = +\infty$; $\lim_{h\to+\infty} U'(h_t) = 0$ *and* $\lim_{c\to 0} U'(c_t) = +\infty$; $\lim_{c\to+\infty} U'(c_t) = 0$.

Utility is maximized subject to income and health production constraints. The income constraint on the consumption of all other goods is:

$$c_t = P_{vt}.M_{vt} + I(1-\tau) \ , \qquad (2)$$

where P_{vt} is the price of private medical services, M_{vt}, is the quantity of private medical services consumed, and *I*, constitutes the remainder of income. Part of this income is actually saved and part is taken in tax, represented by the tax rate, τ. All tax is spent on public medical expenditures. That is total national public medical consumption, M_{bt}, is given by the equation $M_{bt} = I.\tau$. Therefore,

$$c_t = P_{vt}.M_{vt} + I - M_{bt}. \qquad (3)$$

The second constraint is on the production of health which follows a Cobb Douglas production function in its general form; hence, it can exhibit increasing, decreasing or constant returns to scale. Health is produced with the financial resources devoted to medical expenditures according to:

$h_t = A.{M_{bt}}^{\beta_1}.{M_{vt}}^{\beta_2}$,

where *A*, β_1 *and* $\beta_2 > 0$. (4)

Here, A, is an exogenous constant representing the technology in the production of health which must be positive. β_1 and β_2 are parameters allowing the impact of private expenditures on health to differ to those of public expenditures. The production function is continuously differentiable, strictly increasing in both arguments (M_{bt} and M_{vt}) and strictly concave. Furthermore the function satisfies the Inada conditions.

Substituting the constraints into the utility function gives:

$$U(c_t, h_t) = \ln(P_{vt}.M_{vt} + I - M_{bt}) + \propto ln\,(\mathrm{A}.{M_{bt}}^{\beta_1}.{M_{vt}}^{\beta_2}\ ). \qquad (5)$$

Maximization of this problem requires that

$\frac{\partial U}{\partial M_v} = 0 \; and \; \frac{\partial U}{\partial M_b} = 0.$

For an optimal result this implies that

$F_1(M_{vt}, M_{bt}) = 0,$

and

$F_2(M_{vt}, M_{bt}) = 0.$

The solution will give the optimal levels of public and private medical expenditures as functions of the parameters and prices. That is the optimal level of health spending will depend on the returns to each type of expenditure (the β's) and the price of private health.

$M_{vt}^* = F_{vt}(\beta_1, \beta_2, p_{vt}),$ (6)

and

$M_{bt}^* = F_{bt}(\beta_1, \beta_2, p_{vt}).$ (7)

Therefore the optimal level of health will be given by:

$h_t{}^* = \mathrm{A}.\, M_{bt}{}^{*\beta_1}.\, M_{vt}{}^{*\beta_2}$. (8)

The derivatives with respect to medical expenditures are:

$\partial h_t{}^* \Big/ \partial M_{vt}^* = \mathrm{A}.\, M_{bt}{}^{*\beta_1}.\, \beta_2.\, M_{vt}{}^{*\beta_2 - 1},$ (9)

and

$\partial h_t{}^* \Big/ \partial M_{bt}{}^* = \mathrm{A}.\, \beta_1.\, M_{bt}{}^{*\beta_1 - 1}.\, M_{vt}{}^{*\beta_2}.$ (10)

Given the restrictions on A, β_1 and β_2 below equation (4) the solutions to both (9) and (10) must be positive. That is:

$\frac{\partial h_t{}^*}{\partial M_{vt}^*} > 0 \; and \; \frac{\partial h_t{}^*}{\partial M_{bt}^*} > 0$. (11)

This makes sense. Any additional medical care must have some positive impact on health. However, due to the fact the tax rate, τ, cannot exceed one, this model assumes either decreasing or constant returns to scale. The only alternative is that additional medical services will have a negative impact on health which is ridiculous. It would be very difficult to explain any increase in real medical expenditures reducing health. The inequalities in (11) give the theoretical premise that underlies this empirical investigation.

The logarithmic transformation of the health production function is

$$\log h_t^* = \mathrm{A} + \beta 1 \log M_{bt}^* + \beta 2 log M_{vt}^* \qquad (12)$$

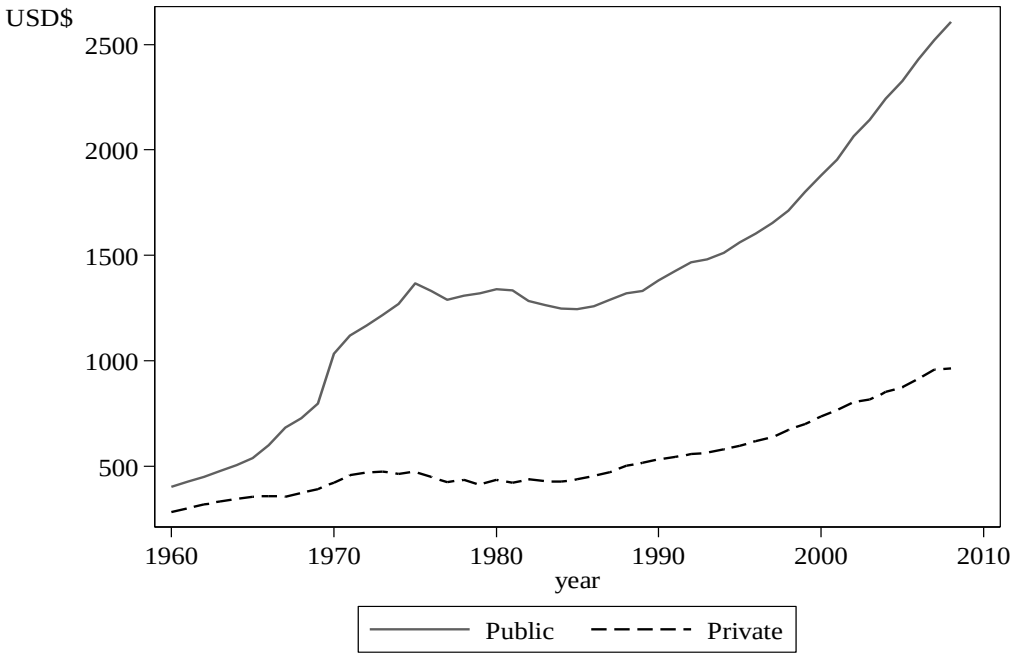
Equation (12) is the basis of the empirical estimation that follows.

3.4. Overview of Data

In this study health status is measured by life expectancy at different ages which are constructed from mortality rates. Ideally morbidity would be a better measure since it measures the incidence of ill health. However, morbidity is far more difficult to measure than mortality. This is because morbidity can recur whereas mortality cannot. The duration of morbidity can vary from seconds to years where mortality is instantaneous and again, can only occur once. Because of this, measures of morbidity such as the Disability-Adjusted Life Year (DALY) have only been developed recently and data is only available as far back as the 1990's, where life expectancy data goes back centuries.

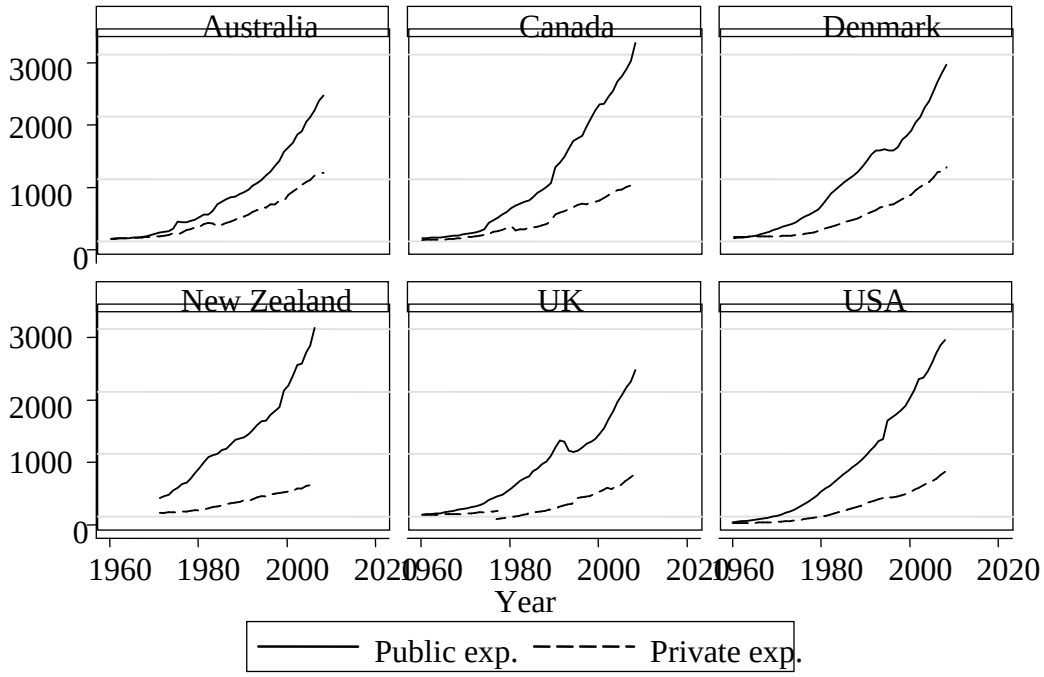
The average life expectancy at birth across these countries has risen from 66.75 years to 76.27 years, which is an average growth of 14% (See figure 1 Appendix). This growth was relatively consistent across countries. Over the same period, total healthcare expenditure per capita rose from $70 to $3800 ($US PPP). In most countries there was substantial growth in both public and private financing; in most countries expenditure on public health has exceeded private expenditures and has grown at a faster rate see figure 1 below.

Figure 1: Unweighted Average across all Countries of Real Per Capita Health Expenditures, 2005 USD PPP



There is, however, a lot of diversity in the public/ private mix of healthcare funding across these countries. The healthcare spending by funding source of a few key countries are illustrated below for comparison and discussion.

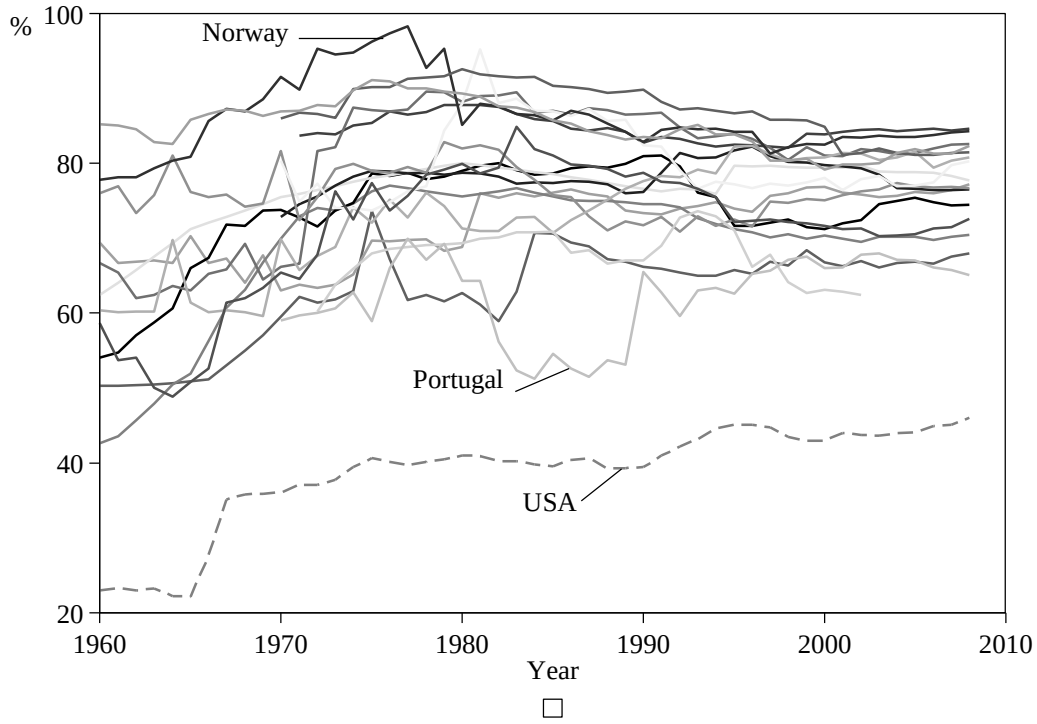
Figure 2: Per capita real health spending in current US$ by funding source for six countries.



In all countries other than the USA the growth in public spending outstripped that of private health expenditure. The USA is also the only country where private health expenditure exceeds public expenditure (see figure 4). Interestingly, the total per capita spending on health in the US is higher than average and they also have higher public spending than some countries, but they still have a lower life expectancy and higher infant mortality when compared to the rest of the group. This should be further investigated; particularly now when populations across OECD countries are aging and current governments, afraid of the rise in demand for healthcare and associated larger budget deficits, are pushing for greater private provision.

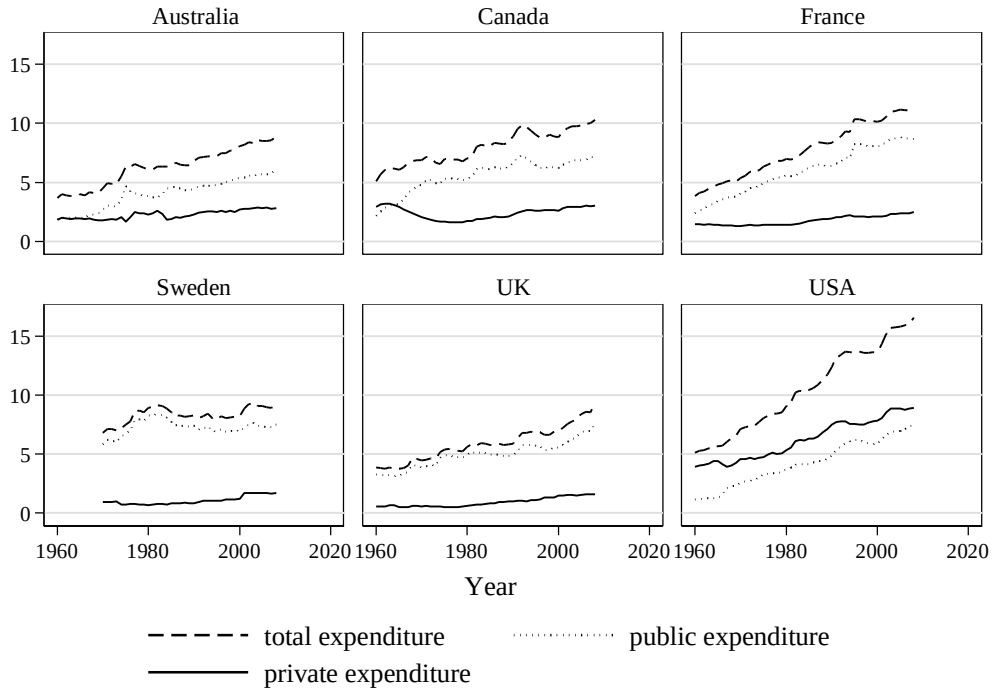
Figure 3 shows public health expenditures as a percentage of total health expenditure over the period. This highlights both the diversity across countries in the proportion of total health that is funded by the government as well as volatility of this ratio within countries across time. The range is quite large the minimum being 20% public provision in the USA during the early 1960's to a maximum of well over 90% in Norway during the 1970's and early 80's.

Figure 3: Public Contribution to Heath Expenditure (% of total expenditure on health)



There is more volatility in the public health ratio within countries in the early half of the sample. After the 1980 the variation in the ratio smooths out, excepting Portugal. This indicates that the policy variations within countries have stabilised. It is also apparent that the gap in the ratio between countries is larger in the earlier periods. The difference between countries seems to have converged toward a public contribution to healthcare of between 60 to just over 80% across these countries, excepting of course the USA. The public contribution to total healthcare spending has remained the lowest in the US throughout the entire period and is currently at around 40% of total spending. The Scandinavian countries tend to be at the higher end of the scale for example Norway, currently just over 80%. The following is a figure of the data aggregated and expressed as a percentage of GDP. All of the health expenditure variables used in the panel analysis later in the paper are expressed as percentages of GDP.

Figure 4: Health expenditures percentage of GDP (Current US$ PPP)



In most countries total health expenditure is around 10% of GDP and private health expenditure is well below 5%. Excepting the US of course where total health spending is just over 15% of GDP with private contributions far exceeding public.

3.5. Basic Specification and Explanation of Data

The output of medical services is improved health. Most empirical studies that use health as the dependent variable adopt a production function approach (Auster et al, 1969, Grossman, 1990, Nixon and Ulmann, 2006). That is good health, H_t, is produced by a production function which is composed of various inputs.

$H_t = h_t(m_t, z_t, e_t)$.

This function has as its inputs into the production of health, m_t, medical expenses, z_t, other inputs that produce good health such as income and therefore nutrition, hygiene, time and effort put into good health as well as things that depreciate good health such as stress, cigarette and alcohol consumption. e_t, is education which contributes to the efficiency of producing good health. Previously empirical work in this field focuses on total medical

inputs rather than distinguishing between public healthcare and privately financed healthcare. The following logarithmic transformation of the health production function separates medical expenditures according to their source of funding as well as specifying the other inputs included in this investigation:

$$lnLE = \beta_0 + \beta_1 \ln(y_t) + \beta_2 \ln(e_t) + \beta_3 \ln(pub\, m_t) + \beta_4 ln(priv\, m_t) + \beta_5 ln(c_t) + \beta_6 ln(a_t) + \varepsilon_t,$$

where $\varepsilon_{ij} = \alpha_i + \mu_{ij}$ where α_i denotes the country-specific effects or measurement errors and μ_{ij} refers to the idiosyncratic error term. c_t is cigarette consumption per capita, a_t is the per capita consumption of alcohol, and y_t is per capita income. The following is an explanation of the variables and why they have been chosen. The exact sources and description of the data is in the data appendix.

3.5.1. Health Status

The dependent variable, life expectancy (LE), proxies health status. Life expectancy is defined as the probable number of years a person of a specific age would be expected to continue living. The cohorts considered here are life expectancy at birth, at age 10 (which eliminates infant mortality) and at 65 which is an indication of the expected length of retirement. As previously mentioned, morbidity would actually be a better measure of health as it captures the incidence of illness and not just the instance of death. However, morbidity is difficult to measure and the data is not available.

Another good indication of total population health is infant mortality, so this will also be investigated as a possible dependent variable. This measure of health is of particular interest because it is more likely to be derived from current health expenditures than life expectancy. The reason for this is that good maternal health, access to prenatal and postnatal health facilities and maternal education all contribute to lower rates of infant mortality and are all determined largely by current health expenditures. Life expectancy is dependent on these expenditures further back in time. Infant mortality is measured by the Infant Mortality Rate (IMR), this is the number of deaths per year of children below the age of 12 months per 1000 live births.

The determinants of health status or life expectancy considered here are:

3.5.2. Medical Care (m)

To measure medical care, the dollar amount of real healthcare expenditures per capita is used (refer figures 1 and 2). This should indicate the quantity of care received per person per year. Total expenditures can be split into expenditure by government (public expenditure) and all other expenditure on health (here referred to as private expenditure). Since 1960 where available the OECD has collected private expenditure and public expenditure data separately for many countries which is the data used here. In the regression analysis, for reasons discussed later the expenditure variables are divided by GDP. Therefore, nominal values of both the expenditure and GDP data are used to ensure consistency this is because there can be no discrepancy due use of different deflators.

3.5.3. Socioeconomic Status

Income and Education are included to represent socioeconomic status. Income is real per capita GDP for each period. The relationship between income and life expectancy is expected to be positive. Higher levels of income can finance improvements in health that come with higher living standards. Better sanitation and nutrition are often cited examples as well as exercise, access to clean water, the absence of infectious disease, and foetal and maternal health. However, it is possible that beyond some threshold level of affluence increasing income may no longer buy increased life expectancy, and may in fact lead to increased stress and other unhealthy behaviours that could adversely affect the health status of the population (Fuchs, 1994).

Education is measured as the average years of education of the working age population. Populations with a higher level of education are expected to have higher life expectancy. Grossman (1972) theorized that higher levels of education increase the efficiency with which individuals are able to produce health by increasing health knowledge and improving the ability to process health information. This explanation implies a direct effect of education that is independent of income and unobservable characteristics that may be correlated with both health and years of schooling (Thorton, 2002).

3.5.4. Lifestyle factors

The impact on life expectancy of other lifestyle variables across countries is captured by cigarette and alcohol consumption. The negative relationship between cigarette consumption and health is well established. While excessive alcohol consumption is known to lead to cirrhosis of the liver and other alcohol related diseases which negatively affect life expectancy, moderate consumption may have beneficial health effects or be correlated with other beneficial behaviours such as relaxation. Thus, the net effect of alcohol consumption in the population is uncertain.

Country fixed-effects were introduced to take into account cross-country heterogeneity not reflected in the other explanatory variables. This is to account for the fact that countries differ in the characteristics that determine the life expectancy of their population, for example the institutional features of their healthcare system, culture, crime rates, pollution or climate.

3.6. Problems with the Specifications and Data

3.6.1. Correlation

There is a number of specification and data related problems. First, the health production function has determinants that are highly correlated such as healthcare spending per capita and per capita income. For this reason all expenditure variables are expressed as a percentage of GDP. The magnitude of these ratios is therefore is also not influenced by country size.

3.6.2. Endogeneity

Secondly, there are endogeneity issues. Health spending, income and education levels would also be affected by life expectancy, that is, there is reverse causality. Healthier people live longer and the longer people expect to live the more incentive they have to invest in education and physical capital hence improving education levels and per capita capital stocks and therefore per capita income. The endogeneity problem is addressed by instrumenting these variables. The instruments suggested for income (GDP per capita) were foreign direct investments (FDI over GDP) and trade openness (total imports and exports over GDP). For 620 observations the coefficients for FDI and GDP were over 0.15 which is statistically significant at one per cent for a sample this size. The instrument selected for education was the patent ratio which is the number of patents per 100,000 persons aged between 15 and 64.

The coefficient is 0.19 for 823 observations which is highly significant (level 0.0005). Instrumenting the health expenditure data was a little more difficult.

While it is should be the case that extra spending on health would lead to better health, better health means people live longer and since older people demand more medical care, better health should also increase the demand for healthcare expenditure. Hence the need to find instruments for the health expenditure data. The instruments tested for public health spending are government expenditure excluding health over GDP and the proportion of public spending as a percentage of total health spending. Public health expenditure needs to be instrumented since demand for public health expenditures is likely to increase when life expectancy increases. It is proposed here that demand for government expenditure over GDP would be reflective of the size of the government within the economy and not the demand for public expenditures on health. Therefore; it is unlikely to be correlated with the error term.

Public spending excluding expenditure on health over GDP is an excellent instrument since it is highly correlated with government spending on health. The correlation coefficients on this instrument are higher between the logged transformations of these ratios, which is the variable used in this chapter. The coefficient is 0.11 for 661 observations which is significant at the 5% level.

Government spending excluding health is also exogenous. It is difficult to imagine that government spending excluding health suffers the same endogeneity problem that public health spending does. Life expectancy should not affect total government expenditures since they should be derived from the size of the labour force, income and the tax rate. Total government spending has to be related to the amount of revenue they can generate either in the current period or in the future. This instrument is unique to this paper in that it has not been used in this context before.

Private health expenditures were instrumented by the credit to GDP ratio which represents the financial resources provided to the private sector which is not likely to be driven by changes in life expectancy and it is therefore uncorrelated with the error term and hence, exogenous. The financial resources provided to the private sector are likely to be closely related to the private provision of healthcare. This instrument is again unique to this paper and an excellent choice. The credit ratio is highly correlated with private health expenditures and the coefficients are highly significant (well within the 0.0005 level).

All instruments were assessed for over or under identification with the Hansen J statistic which is recorded with the results.

3.6.3. Heteroskedasticity and Serial Correlation

Another problem is that the residuals estimated by Ordinary Least Squares are both heteroskedastic and serially correlated. This does not affect the consistency of the IV estimates but the standard IV estimates of the standard errors are affected by heteroskedasticity and are inconsistent which prevents valid inference. The usual diagnostic tests for endogeneity and over identification of the instruments will also be invalid if heteroskedasticity is present. Robust standard errors are obtained to take care of error heteroskedasticity. To further increase the efficiency a two-step GMM estimation of the model is carried out in Stata using the ivreg2 command as in Pocas and Soukiazis (2010) and guided by Baum and Schaffer (2002). This corrects for the serial correlation by using lagged dependent variables on the left hand side of the estimation. However, once this is done the results are far less significant since the lagged dependent variable explains so much of the variation in the dependent its conclusion detracts from the explanatory value of income, education, healthcare expenditures, and cigarette and alcohol consumption. Its inclusion also changes the sign on many of the variables including income which doesn't make any intuitive sense. Income should not be negatively related to life expectancy, income represents too many positive lifestyle and environmental factors in OECD countries.

6.4 Lag Length

Selecting lag lengths for the explanatory variables is difficult. It is not clear which lags would be appropriate. Of particular concern is lagging the expenditure variables (which are the main topic of the paper). Unfortunately unless the expenditure data is lagged by 70 years or more, to capture foetal and maternal health, there does not seem to be any reasonable justification for selecting shorter lag lengths. This is not possible as data is only available back to 1960 or 1970 for most OECD countries. Any other lag selection would be arbitrary and therefore should not be included, although different lag lengths were investigated with mixed results.

3.7. Method and Results

The reported estimations have life expectancy at birth as the dependent variable; estimations using life expectancy at 10 and at 65 are reported in the appendix. The following results from table 1 are discussed below.

Table 1

LE	Model 1	Model 2	Model 3	Model 4	Model 5
LE (t-1)	N/A	N/A	N/A	1.146***	0.965***
				(7.43)	(12.78)
GDP	0.002*	-0.014 (I)	0.079 ** (I)	-0.033 * (I)	0.020 (I)
	(1.36)	(-0.67)	(2.14)	(-1.26)	(0.91)
Education	0.01***	0.24*** (I)	0.071** (I)	0.181 * (I)	-0.023 (I)
	(2.75)	(2.59)	(1.82)	(1.52)	(-0.63)
Public health exp.	0.069***	0.023***	0.070*** (I)	0.025 *	-0.02**(I)
	(3.62)	(2.32)	(4.13)	(1.58)	(-1.77)
Private health exp	-0.087 ***	-0.007***	0.008* (I)	-0.002 *	0.015**(I)
	(-3.11)	(-4.26)	(1.28)	(-1.31)	(2.23)
Alcohol consumption	-0.0002	-0.022***	-0.008**	-0.009	0.007**
	(-0.28)	(-3.355)	(-1.72)	(-1.21)	(-1.92)
Cigarette consumption	-0.003***	-0.032 ***	-0.005	-0.0003	0.0003
	(-3.01)	(-4.26)	(-0.63)	(-0.06)	(0.07)
No. of Obs.	581	474	502	458	474
No. of countries	17	16	16	16	16
R squared	0.99	0.88	0.887	0.96	0.94

F value	45041	481.18	562.75	337.57	234.66
Hansen J	N/A	2.276	0.957	0.2	0.598

Notes: Statistics are robust to heteroskedasticity, the numbers in parentheses are t statistics and (I) denotes the instrumented variables.

3.7.1. Model 1

$lnLE = \beta_0 + \beta_1 \ln(y_t) + \beta_2 \ln(e_t) + \beta_3 \ln(pub\, m_t) + \beta_4 ln(priv\, m_t) + \beta_5 ln(c_t) + \beta_6 ln(a_t) + \varepsilon_t.$

This is a simple time series least squares (OLS) model with fixed effects and robust standard errors. The hypothesis being tested is that public and private health expenditure are not perfect substitutes; if this hypothesis were true, the coefficients on the variables for private and health expenditure would not be equal. This is true for all models tested. It follows, these results lead to the conclusion that the impact of public expenditures on life expectancy at birth is significantly different to that of private expenditure. Public expenditure has a positive effect on life expectancy while the coefficient on private expenditure is negative. A negative coefficient on health expenditure is crazy and must have something to do with model misspecification. However it does suggest that heal Unemployment that expenditures are imperfect substitutes and they should be treated as such in theoretical and empirical studies.

The results from the OLS model (model 1) are more significant for life expectancy at birth than they are for life expectancy at aged 10 and 65. The results for life expectancy at age 10 and age 65 are shown in tables 2 and 3 in appendix 3 (A3). Consistent across all three life expectancy variables is that GDP per capita is significantly and positively related to life expectancy but the coefficient is very small.

- A 1% change in per capita GDP is expected to increase life expectancy at birth by 0.2 or less than 1 day this is not a very big impact on life expectancy. The health expenditure and education variables are significant for life expectancy at birth but not the other models.
- A 1% increase in the average years of schooling of the working age population is predicted to increase life expectancy by 1% or by 3.65 days.

Alcohol and cigarette consumption are negative across all three dependent variables but vary in significance. Interpretation of the expenditure coefficients for model 1 on life expectancy at birth is as follows:

- A 1% change in per capita public health expenditure will result in a 7 % increase in life expectancy in other words an increase of 25.5 days.
- On the other hand, a 1% increase in private health expenditure will reduce life expectancy by 8.7% which is equivalent to taking 31.8 days off life expectancy at birth! This result is a bit ridiculous.

  Perhaps this result is due to the fact that a large proportion of private health expenditures occur later in life when the patients are more likely to die.
- Both of these coefficients are highly significant, however, since the first model has not dealt with any endogenous variables, it is not appropriate to take these interpretations too seriously or to make any further causal inferences.

3.7.2. Model 2

As previously mentioned, model 1 suffers from endogeneity problems that need to be adjusted. Following Haclicioglu (2010), the next step was to instrument income and education which are widely accepted in the health literature as endogenous. Income and education should affect health; however, since life expectancy also affects investment and savings decisions it also increases the incentives to acquire human and physical capital which in turn affects income and education levels. The second model uses a two stage least squares approach again with fixed effects and robust standard errors. The instruments selected are foreign direct investment over GDP and the patent ratio to instrument for income and education respectively. The first stage OLS estimate for income is predicted as by equation ():

$$ln\hat{y}_{t-1} = \beta_0 + \beta_1 \ln(y_{t-2}) + \beta_2 \ln(FDI_{t-2}) + \beta_3 ln(alc) + \beta_4 ln(cigg) + \varepsilon_t.$$

Giving the following results:

Dependent variable ($ln\hat{y}_{t-1}$)	Coefficients

$\ln(y_{t-2})$	0.961***
	(294.28)
ln (FDI_{t-2})	0.001***
	(2.50)
$ln(alc)$	0.019*
	(1.34)
$\beta_3 ln(cigg)$	-0.013***
	(-3.03)
F value	43,483.92

Notes: Statistics are robust to heteroskedasticity, the numbers in parentheses are t statistics

The F statistic is positive and high (above 10) and the coefficient β_2 is positive and significant. These results are sufficient to justify the use of the foreign direct investment to GDP ratio to be used to instrument GDP per capita.

The equation for OLS estimate for education using the patent ratio as an instrument is:

$$ln\hat{E}_{t-1} = \beta_0 + \beta_1 \ln(E_{t-2}) + \beta_2 \ln(Pat_{t-2}) + \beta_3 ln(alc) + \beta_4 ln(cigg) + \varepsilon_t.$$

And the results:

Dependent variable ($ln\hat{e}_{t-1}$)	Coefficients
$\ln(e_{t-2})$	0.983***
	(135.48)
ln (Pat_{t-2})	-0.00001**
	(-1.79)
$ln(alc)$	0.004

	(0.93)
$ln(cigg)$	-0.003
	(-0.80)
F value	24,953.85

Notes: Statistics are robust to heteroskedasticity, the numbers in parentheses are t statistics

The F statistic is again high and the coefficient for the patent ratio is significant, however, it is negative. This is a little puzzling since one would expect the number of patent applications per capita within a country to be positively related to the level of education. Further to this, the correlation coefficient between average years of schooling and the patent ratio is positive. The second stage of the OLS estimation method is the health production function run again using the estimated values for income and education.

$lnLE = \beta_0 + \beta_1 \ln(\hat{y}_t) + \beta_2 \ln(\hat{e}_t) + \beta_3 \ln(pub\, m_t) + \beta_4 ln(priv\, m_t) + \beta_5 ln(c_t) + \beta_6 ln(a_t) + \varepsilon_t.$

The results from the two stage least square regression, model 2, shown in table one indicate that once instrumented, the estimated variable for income is not significant at all for life expectancy at birth. The instrumented education variable is positive and significant.

- A 1% change in the average years of education increases life expectancy at birth (instrumented by the patent ratio) by 24% which is equivalent to 87.6 days.
- The coefficient for public health expenditure is positive and highly significant; a 1% increase in public health expenditure increases life expectancy at birth by 2.3% approximately 8 ½ days.
- Unfortunately the coefficient for private health is again negative and significant, which is very difficult to logically explain. However, this counter intuitive result is slightly offset by the coefficient being very small. A 1% increase in private health expenditure would suggest a 0.7% decrease life expectancy at birth, equivalent to a reduction by 2 ½ days. Again this may be because private health expenditure is more highly correlated with morbidity amongst people who are more likely to pass away, such as the elderly.

- The coefficients for cigarette and alcohol consumption are both negative and highly significant. Increasing cigarette consumption by 1% decreases life expectancy by about 12 days and increasing alcohol consumption reduces it by 8 days.

Excepting the change in the significance of the alcohol coefficient which changes from none in model 1 to highly significant in model 2 these two specifications are fairly consistent. Income is significant at the 10% level in model 1 and not significant in model 2 when it is instrumented, however, income is not expected to greatly affect life expectancy across the developed nations investigated here. Perhaps increases in income affect private health expenditure more than increases in public expenditure. That is as people become wealthier they choose to take private health.

3.7.3. Model 3

Health expenditure is also likely to be endogenous and it is therefore necessary to instrument health expenses since demand for healthcare increases as health declines and hence also as life expectancy increases (since older people require more care). The instruments for healthcare expenditures were constructed as follows:

$$ln\widehat{PubM}_{t-1} = \beta_0 + \beta_1 \ln(PubM_{t-2}) + \beta_2 \ln(Gov\ exp_{t-2}) + \beta_3 ln(alc) + \beta_4 ln(cig) + \varepsilon_t.$$

The results of this first stage regression are:

Dependent variable ($ln\widehat{PubM}_{t-1}$)	Coefficients
$\ln(PubM_{t-2})$	0.885***
	(42.04)
$\ln(Gov\ exp_{t-2})$	-0.004***
	(-2.69)
$ln(alc)$	0.031*
	(1.50)
$ln(cigg)$	-0.035***

	(-4.64)
F value	1720.55

Notes: Statistics are robust to heteroskedasticity, the numbers in parentheses are t statistics

The F statistic for this regression is high and the coefficient on government expenditure to GDP is significant however it is negative, suggesting that as the portion of public health expenditure to GDP rises as the government expenditure to GDP ratio falls, even though the correlation coefficient is significant and positive. Also cigarette consumption is negatively related with public medical expenditures while alcohol consumption is positive. This means that an increase in cigarette consumption decreases public expenditures and increases in alcohol consumption increases it. This probably doesn't have much meaningful interpretation since both cigarette and alcohol consumption in the past would be affecting public expenditures not current consumption habits.

And then the construction of the private health instrument is as follows:

$$ln\widehat{PrivM}_{t-1} = \beta_0 + \beta_1 \ln(PrivM_{t-2}) + \beta_2 \ln(Cred_{t-2}) + \beta_3 ln(alc) + \beta_4 ln(cig) + \varepsilon_t$$

The results:

Dependent variable ($ln\widehat{PrivM}_{t-1}$)	Coefficients
$\ln(PrivM_{t-2})$	0.957***
	(55.81)
$\ln(Cred\ exp_{t-2})$	0.037***
	(2.53)
$ln(alc)$	0.085***
	(2.92)
$ln(cigg)$	-0.055*
	(-1.54)

F value

Notes: Statistics are robust to heteroskedasticity, the numbers in parentheses are t statistics

The F statistic is high and the coefficient on the credit ratio is positive and significant.

The fully instrumented specification is:

$$lnLE = \beta_0 + \beta_1 \ln(\hat{y}_t) + \beta_2 \ln(\hat{e}_t) + \beta_3 \ln(pub\,\hat{m}_t) + \beta_4 ln(priv\,\hat{m}_t) + \beta_5 ln(c_t) + \beta_6 ln(a_t) + \varepsilon_t$$

The hat (^) above the variable indicates it is an estimated or instrumented variable. All of the coefficients in the 3rd model are significant with the exception of that on cigarette consumption.

- The model has estimated that the elasticity of life expectancy to income is 0.079 in other words a one per cent increase in per capita income will increase life expectancy 7.9% or by 28 days. This seems very high.
- A one per cent increase in the average years of education improves life expectancy by 7.1% or approximately 26 days.
- Improving per capita public spending on health increases life expectancy by 7% or 25.5 days,
- Per capita spending on private health improves life expectancy by 0.8% equivalent to 2.92 days.
- Increasing alcohol consumption per capita by 1% reduces life expectancy by 0.8% or 2.92 days.

This specification (model 3) is probably the best model in terms of explaining the theory. Income is more significant and the coefficient on private health expenditure is positive. Private health expenditures should have a positive impact on health. That this coefficient was negative in the previous two models was a major concern. The change in sign of the instrumented variable for private health could be explained by a negative relationship that runs from life expectancy to private health expenditures. This doesn't mean it is a full explanation, just that there is correlation. Other than on cigarette consumption all coefficients are significant, the model is robust and has exogenous explanatory variables. However, this model has not dealt fully with serial correlation.

3.7.4. Model 4

The 4th model run using a two-step GMM estimation with the ivreg2 command in Stata. It is the same as Model 2 in that income and education are instrumented and year fixed effects are corrected for. The main difference is that in the second stage of the regression the dependent variable is lagged and again year fixed effects are used. This model is robust to heteroskedasticity and serial correlation. The lagged dependent variable is highly significant as expected considering the likely impact the past value of a variable has on its current value. The instrumented income coefficient is negative which is counter intuitive.

- A 1% increase in income coincides with a 12 day reduction in life expectancy. This must largely be due to the interaction between income and education. Since education and income are highly correlated one will negate the affect of the other in regression analysis..
- The education variable is positive and significant. Increasing education by 1% correlates to a 66 day increase in life expectancy.
- Neither alcohol consumption nor cigarette consumption are significant.
- The model predicts the elasticity of life expectancy to a change in the public health ratio is 0.025 that is a one unit increase in the public health ratio will increase life expectancy by 9 days.
- The coefficient was negative on private health and although very small -0.002 it was statistically significant. However, this does not represent much economic significance, a one unit increase in the private health ratio decreases life expectancy less than a day. Again since a negative coefficient doesn't make sense in this case the logical conclusion is that there is interaction between the private health variable and other explanatory variables that is driving counterintuitive results.

3.7.5. Model 5

This is model 3 using the same procedure as in model 4.

$$ln\widehat{PubM}_{t-1} = \beta_0 + \beta_1 \ln(\widehat{PubM}_{t-2}) + \beta_2 \ln(\widehat{Gov\ exp}_{t-2}) + \beta_3 ln(alc) + \beta_4 ln(cig) + \varepsilon_t.$$

That is, all 4 endogenous regressors are instrumented and it is robust for heteroskedasticity and serial correlation. This is achieved using the 2 step GMM estimator lagging the

dependent variable, robust standard errors and using time fixed effects. The lagged dependent variable is again large and highly significant.

- Neither the income or education variables are significant.
- This time the coefficient on the private health is positive and relatively large, 0.015, and the coefficient on public health was negative, -0.02. A one per cent increase private health over GDP leads to 5.5 extra days of life expectancy while the same increase in the public health ratio reduces life expectancy by 7.3 days.
- Alcohol consumption was again negative and significant and cigarette consumption was not significant.

The fifth model is not as significant as the third, partly due to the lagged dependent variable and the coefficient on public health expenditure is negative which doesn't make sense. The negative coefficient on public health expenditure may be due to the fact that life expectancy was increasing much slower than health expenditure data over the period and that there has been huge increases lately with little change to life expenditure.

3.7.6. Infant Mortality

There is an issue with health expenditures affecting life expenditures far into the future. For this reason infant mortality rates are also investigated. This is because infant mortality rates should reflect health expenditures now rather than of the importance of foetal and maternal health far back in time which is a major determinant of life expectancy.

Table 2

Infant Mortality (IMR)	Model 1	Model 2	Model 3	Model 4	Model 5
IMR (t-1)	N/A	N/A	N/A	0.675***	0.687***
				(7.5)	(11.65)
GDP	-0.427***	-0.149	-0.545	-0.18	-0.323
	(-4.92)	(-0.83)	(-1.05)	(0.907)	(-1.04)
Education	-0.944***	-0.179***	-0.107***	-0.928*	-0.007
	(-2.51)	(-3.34)	(-2.48)	(-1.50)	(0.02)
Public health exp.	-0.037	-0.945***	-0.767***	-0.102	0.283*
	(-0.39)	(-3.53)	(-2.90)	(-1.02)	(1.37)
Private health exp	0.047	-0.129**	-0.262***	0.017	-0.192*
	(0.83)	(-1.71)	(-3.20)	(1.06)	(-1.47)
Alcohol consumption	0.246**	-0.31*	1.09	0.158***	0.179**
	(1.86)	(-1.29)	(1.03)	(2.98)	(1.94)
Cigarette consumption	0.072	0.591***	-0.952	-0.007	-0.004
	(1.14)	(5.33)	(-0.83)	(-0.29)	(-0.06)
No. of Obs.	586	556	494	474	458
No. of countries	17	17	17	16	16
R squared	0.93	0.80	0.844	0.95	0.95
F value	226.41	31.58	64.85	2295.65	391.21
Hansen J				1.253	0.262

Notes: Statistics are robust to heteroskedasticity, the numbers parentheses are t statistics and (I) denotes instrumented variables.

Infant Mortality Rates (IMR) measure the number of deaths of children aged less than 12 month per 1,000 live births. When this is used as the dependent variable the signs on the coefficients for health expenditures are negative and consistent. These coefficients should be negative since increasing health expenditures should reduce infant mortality. These models provide some evidence that both public and private health expenditures negatively affect infant mortality rates and also that the impact of public health is statistically more significant than private health. However, there is obviously going to be some serial correlation and this is evident when the significance falls when the GMM method is used. This suggests that to some extent the results in models 1,2 and 3are being driven by time trends.

3.8. Discussion

The initial aim of this study is to determine whether private or public healthcare provision produce the same health outcomes. They do not. One important result beyond the scope of this thesis, or the available data, is that further research is required to determine which type of healthcare provision is better. The reason for this is that with ageing populations and associated rising demands for healthcare, we need to determine the best policy for governments. The choice governments are facing is whether to push towards private provision of healthcare or not. The reason governments are moving in this direction is to alleviate fiscal budget pressures and the associated increase in taxation on a diminishing workforce. The other less practical but equally important result is that researchers should be considering public and private health expenditures separately not in total.

Figureically, the data shows public and private health expenditures have both risen substantially over time. There has also been quite a lot of variation in the mix of the two both within and between countries. Life expectancy on the other hand has been gradually increasing within all countries without much volatility, nor much variation between countries. A notable point arising from data is that the USA (even though its total health expenditure is higher than all of the other countries) its life expectancy is lower than most and infant mortality higher. This is interesting because it has the lowest percentage of public contribution to total health expenditure of all the countries. And further, private health expenditure has always exceeded public expenditure compared to all of the other countries

where this is the opposite. This fact suggests that for some reason privately financed healthcare may not be performing as well as publicly financed care. Investigating the reasons for this would involve qualitative data and is beyond the scope of this study.

A problem with discussing the above regression results is that they are very sensitive to slight changes in model specification. A glance at the regression results in appendix 4 shows that the results on life expectancy at age 10 are only really significant using model 3. At age 65 all of the models show some significance but not one of the coefficients is of consistent sign across all models. The 4^{th} and 5^{th} models that correct for both endogeneity and serial correlation are not at all significant for either of these dependent variables. The lag of variables with serial correlation can explain this lack of significance due to inertia in all these measures.

Another reason for the sensitivity of the coefficients on health expenditures to life expectancy across these models is that much of the health input data that is vital to current life expectancy is not captured by the health expenditure data here. The health expenditure data would need to be lagged 70 to 80 years which would mean back to the 1880s to capture infant health. Foetal and maternal health is well established as being vital to adult health and life expectancy (Barker, 1990). Human foetuses and infants may face limited food supplies which they will adapt to but in doing so they permanently change their physiology and metabolism. These changes can be linked to the likelihood of suffering a number of diseases later in life that cause death including coronary heart disease, stroke diabetes and hypertension. Barker (1990) observed that areas in Britain which now have relatively high rates of death from cardiovascular disease previously had high infant mortality. Noting that poor nutrition in early life forces the human body to adapt to survive, allowing the baby to continue to grow but doing so at the price of a shortened life.

A more explicit explanation of this problem in terms of the how life expectancy rates are constructed follows: Life expectancy is defined as the remaining lifetime (in years) for a person who survives to the beginning of the indicated age interval (e.g. life expectancy at age 10 is how many years a person that has survived to age 10 is expected to live). These figures are calculated by dividing the total number of person-years lived from that age by the number of persons alive at that age. The person-years lived at the beginning of the age intervals are calculated from age specific mortality rates. These rates come from the probability of dying, which is derived from: the number of persons alive in a specific cohort, the number of

persons alive in a specific cohort of 100,000 and the number of persons dying. Most of the people that have been born since 1960 (when this study began) would still be alive today. Since foetal and early childhood (infant) health is vitally important to life expectancy, the impact of health expenditure since 1960 would not yet be properly captured by the life tables. This is because they are based on current mortality rates which would largely have been established in the pre-1960 era when the current old age cohort were very young, hence these expenditures are not actuarially included in the life expectancy data.

In relation to the information given by the regression analysis the variables of major concern here, health expenditures, were significant across all specifications reported in the main body of the paper. That is when life expectancy at birth was the dependent variable. The coefficients were consistently positive across the first 4 models for public health expenditure and highly significant excepting model 4 where the significance dropped to within the 10% level. However, the coefficient was negative in the 5^{th} model. The drop in significance would suggest that the t-stats were partially inflated in the first three models due to some serial correlation. For private health expenditure, the coefficients were consistently significant across all 5 models but positive only in models 3 and 5 where it was instrumented. This supports the fact that private healthcare does not provide the same health outcomes as public healthcare does, but it does not tell us with very much certainty which one provides better outcomes. Beyond the scope of this study and data, it is also likely that this has a lot to do with the structure of each of the systems and healthcare delivery not just their financing. In this case health outcomes have to do with health inputs that have not been measured here. An investigation of the structure of healthcare systems and delivery would require more micro/case level data. This is not the intention of this macro based study.

A main reason behind the different outcomes generated from private and public health expenditures must be the different incentives they provide. These incentives will be affected by the delivery model as well as the type of financing. The incentives concerning the supply side of health provision are probably efficient in the delivery of public health due to the lack of an incentive to over provide services since they are not paid more. However, public delivery is probably inefficient in the funding of healthcare due to the dead weight losses involved with taxation and the efficiency losses associated with bureaucracy. On the other hand, private supply is likely to be more efficient in collecting the funding but less efficient in the supply due to the incentives to keep patients longer and inflate prices. From the

demand side public provision is likely to be less efficient due to under-pricing of certain services but more efficient in the provision of equally distributed health and other public good aspects of the provision of healthcare. There are also many other incentive mechanisms that both public and private can bring into healthcare systems such as charging higher premiums for people with unhealthy habits.

3.9. Conclusion

This chapter sought to answer two questions: firstly, whether private and public health expenditures are perfect substitutes, and secondly, what conclusions can be drawn as to which one provides better health outcomes.

In relation to the first question there is no evidence found to suggest that private expenditure can be treated as a perfect substitute for public expenditure. On the contrary, public health has often positive and significant relationships with life expectancy where private health has far weaker correlations, often insignificant and of seemingly fortuitous sign. Additionally, the public health proportion of total health spending is positively related to life expectancy. This suggests that the higher the public provision of healthcare is relative to private provision the longer people live.

To address the second question, there are serious problems arising from this empirical investigation. These problems prevent a compelling argument for public health expenditures being more beneficial than private expenditures. There are many aspects of the health system not reflected in this expenditure data such as: the quality of the service, and the maternal and foetal health of the population the life expectancy data is based upon. Nonetheless a number of important results can still be drawn. Firstly, it is likely that without far more extensive qualitative data analysis it is not going to be possible to make a conclusive policy argument based on expenditure data alone.

Secondly, because only 50 to 60 years of health expenditure data is available across most countries it is not possible to investigate expenditure far enough back in time to capture the expenditure on foetal and maternal health of the old age people today. In addition, life expectancy data is constructed from mortality rates at a point in time; there is persuasive evidence indicating mortality today would be significantly impacted by the foetal health of the people dying today who have an average life expectancy of over 80; based on this data

and evidence, health expenditure data needs to be investigated back in time for more than 80 years.

From a policy perspective this study can only caution governments against moving away from public provision of health if they hope to achieve the same or better health outcomes. The results here suggest private healthcare systems are not superior. However, the results are not strong enough to provide a conclusive policy argument either way. Extensive country specific qualitative investigations need to be carried out before any such recommendation for healthcare reform policy.

3.10. Appendix 1: Data Sources

Life expectancy

Natural log of life expectancy at birth, age 10 and age 65 available at www.lifetable.de

Tobacco

Log of Tobacco consumption (grams per capita 15+) data extracted on 05 Apr 2012 05:25 UTC (GMT) from OECD.Stat

Alcohol

Log of Alcohol consumption (Litres per capita (15+)) data extracted on 05 Apr 2012 05:25 UTC (GMT) from OECD.Stat

Health Expenditures

Health expenditure private and public /capita, US$ purchasing power parity data extracted on 09 Mar 2012 01:51 UTC (GMT) from OECD.Stat

Education

Education - average years of schooling of work force aged 25-64. The data are from Madsen, (forthcoming)

Patents

Patents total number of Patents sourced from WIPO statistics database, December 2011. Patent ratio is the number of patents per million inhabitants of working age. Counts are based on the patent filing date.

Population

Population is inhabitants aged 15 - 64 data extracted on 23 May 2012 01:16 UTC (GMT) from OECD.Stat

International trade

The measure of International trade is total trade (imports plus exports) to GDP ratios (current prices, US$) data extracted on 23 May 2012 01:43 UTC (GMT) from OECD.Stat

Foreign Direct Investment

FDI is Foreign Direct Investments (Net Inflows) over GDP in current US prices collected from Source: WDI world bank website. data.worldbank.org

Credit

Credit to GDP ratio credit data from Madsen and Ang (revise and resubmit)

Capital

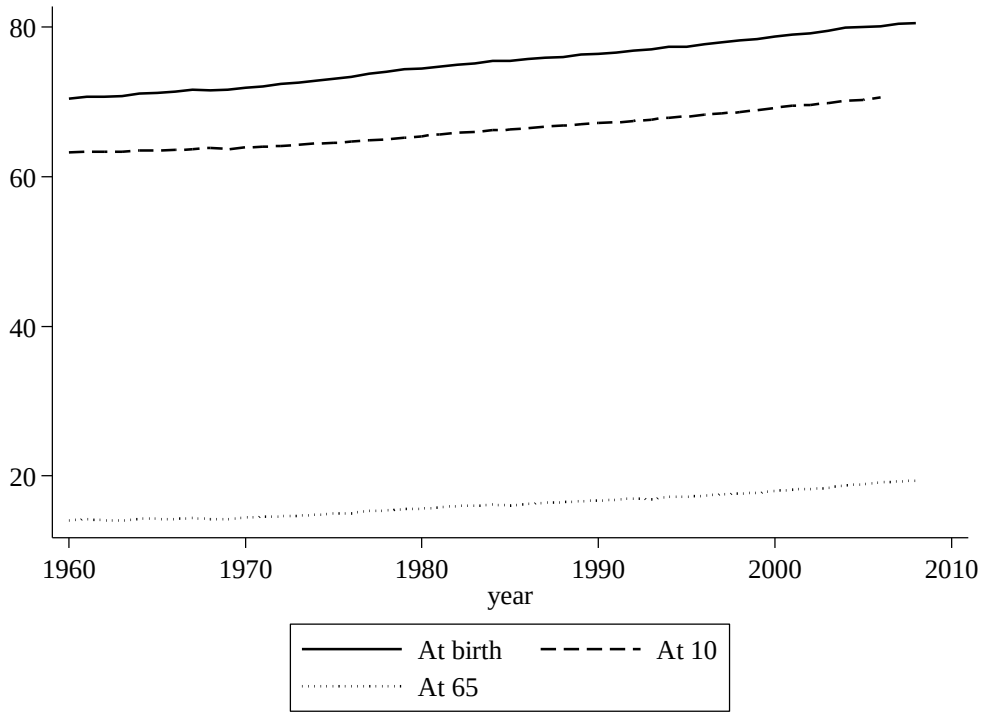
Gross National Capital formation is in % of GDP or in current US $ Source: WDI World Bank website. data.worldbank.org

Health

The public health ratio is public health expenditure (in millions of domestic currency) over total government expenditure excluding health expenditure (in millions of domestic currency) data extracted on 23 May 2012 07:09 UTC (GMT) from OECD.Stat

3.11. Appendix 2: Life expectancy data figures

Figure 4: Average life expectancy across all 18 countries



3.12. Appendix 3: Alternate Estimations

Table 3: Life expectancy at 10

Life Expectancy at age 10	Model 1 OLS	Model 2 IV 1	Model 3 IV 2
GDP	0.030***	0.120***	-0.020
	(3.03)	(2.93)	(-1.19)
Education	0.026	-0.022	0.216***
	(0.67)	(-0.34)	(03.25)
Public health exp.	-0.005	0.005*	0.120***
	(-0.50)	(1.35)	(3.22)
Private health exp.	-0.003	0.002	0.056**
	(-0.85)	(0.77)	(2.32)
Alcohol consumption	-0010*	-0.017***	-0.017**
	(-1.33)	(-3.91)	(-1.89)
Cigarette consumption	-0.034***	-0.032 ***	0.032*
	(-4.24)	(-7.72)	(1.58)
No. of Observations	569	458	458
No. of countries	18	16	16
R squared	0.49	0.88	0.52
F value	59.08	403.32	147.57
Hansen J	N/A	5.26	0.946

Table 4: Life expectancy at 65

Life Expectancy at age 65	OLS	IV 1	IV 2
GDP	0.117***	0.170	0.014
	(4.25)	(0.99)	(0.17)
Education	-0.106	0.191	-0.194**
	(-1.14)	(1.03)	(-1.56)
Public health exp.	-0.031	0.034**	0.125**
	(-0.95)	(1.87)	(2.04)
Private health exp.	-0.024*	-0.018***	0.011
	(-1.51)	(-2.30)	(0.71)
Alcohol consumption	-0048	-0.050***	0.010
	(-1.05)	(-2.97)	(0.55)
Cigarette consumption	-0.088***	-0.074 ***	-0.027
	(-3.54)	(-7.27)	(-0.96)
No. of Observations	514	407	432
No. of countries	16	15	15
R squared	0.34	0.876	0.82
F value	34.97	365.92	264.29
Hansen J	N/A	7.755	0.837

Notes: Statistics are robust to heteroskedasticity, the numbers in parentheses are t statistics and (I) denotes the instrumented variables.

Table 5: Total health expenditures

	Life at birth	Life at 10	Life at 65
GDP	0.020	0.099**	0.85***
	(0.40)	(2.05)	(2.85)
Education	0.118**	0.011	-0.54**
	(1.78)	(0.18)	(-1.70)
Total health	0.128***	0.072**	-0.210
	(4.23)	(2.44)	(-0.89)
Alcohol consumption	-0.012**	-0.005**	-0.031
	(-1.86)	(-2.54)	(-1.2)
Cigarette consumption	0.009	-0.013	-0.148**
	(0.64)	(-0.89)	(-1.69)
No. of Observations	458	458	407
No. of countries	16	16	15
R squared	0.81	0.84	0.52
F value	411.23	384.88	96.88
Hansen J	1.305	12.5	6.451

Notes: Statistics are robust to heteroskedasticity, the numbers in parentheses are t statistics and (I) denotes the instrumented variables.

3.13. Appendix 4: Variable list

'h' is the consumption of good health

'z' is the consumption of all other goods

'U' denotes a utility function

'α' is a parameter on the consumption of good health

'$M_{v'}$ is private medical services or expenditure

'$P_{v'}$ is the price of private medical services

'I' is the remainder of income not spent on private health expenditure. Part of this is saved and the other is taken in tax to be spent on public health expenditure.

'τ' is the tax rate

'$M_{b'}$ public medical expenses given by $M_b = I.\tau$.

'A' is an exogenous constant representing technology

β_1 and β_2 are parameters allowing the impact of private and public expenditures on healthcare to differ.

superscript * denotes the optimal level of a variable

'H_t' is good health

'$m_{t'}$ is medical expenses in aggregate

'z_t' is all other inputs that help to produce good health

'e_t' is education

'y_t' is per capita output

'a_t' is per capita consumption of alcohol

'c_t' is per capita consumption of cigarettes

$pub\ m_t$ is per capita public health expenditure

$priv\ m_t$ is per capita private health expenditure

3.14. Appendix 5: Country identification codes (fcode for Stata)

1. Australia
2. Austria
3. Canada
4. Denmark
5. Finland
6. France
7. Germany
8. Iceland
9. Ireland
10. Japan
11. Netherlands
12. New Zealand
13. Norway
14. Portugal
15. Spain
16. Sweden
17. UK
18. USA

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4. Mortality and Unemployment: Recessions are Not Good for Health

Abstract

In Western countries mortality dropped throughout 19th and 20th Centuries. Even so, overlooking the long-term falling trend, it has also oscillated over time. It has been supposed that these short term oscillations are related to economic conditions. Furthermore, Ruhm (2000) and others have postulated that economic expansions are associated with increased mortality. To ascertain if these short-term oscillations are related to fluctuations in the economy, mortality rates for specific age groups were regressed on economic indicators (unemployment rates and income), across 21 OECD countries over 138 years. Statistically significant results show that mortality rates are pro-cyclical if current economic indicators are used, and counter cyclical if cohort indicators are used. The conclusion is the probability that a 65 year old person has of dying, decreases the better the economic conditions during their working life. This implies that the material and health effects of economic upturns and downturns develop over entire working careers not over short term periods.

4.1. Introduction

Long term improvements in health and mortality are generally thought to be associated with upward trends in per capita income. Whether this relationship exists between short term income growth and health is not clear. More specifically whether the relationship is actually positive or negative has become a contentious issue. This chapter further explores the short term interaction between fluctuations in unemployment rates and mortality rates over 138 years.

Short term employment fluctuations are a major focus of economic policies as is public health. However, most policy makers tend to ignore any interaction the two effects may have on each other. Public health is a very important component of human capital which is vital for income growth and should be an integral part of policy. On the other hand, increased income should afford better health. Statistically, the link between health and employment

(income) has only been found in the long run. This justifies the short term focus of this empirical study.

The seminal works of Brenner (1973, 1979) found powerful positive effects of unemployment rates on mortality rates. That is increases in unemployment, increase the mortality rate. Eyer (1977); Gravelle, Hutchinson and Stern (1981); Stern (1983); and Wagstaff (1985) all found a negative relationship. They found increased unemployment rates are associated with lower mortality rates, and claim there are serious flaws in Brenner's analysis.

Over the past 40 years most researchers have failed to replicate Brenner's positive results. Ruhm (2000) fails to find a positive relationship between unemployment and mortality rates, and believes it does not exist in the short term. Ruhm (2000) also provides a number of arguments supporting his belief that recessions are actually good for health. Empirically, there is more evidence that current unemployment rates are negatively related to current mortality rates. This indicates less people die in recessions, hence, Ruhm's conclusion that recessions are good for health. Ruhm's paper argues increased stress and unhealthy habits developed due to increased work lead to higher death rates during economic expansion. This suggestion of a negative relationship between unemployment rates and mortality has triggered others to search for reasons that could support less employment making people healthier. Not the other way around as is commonly thought.

Ruhm (2000) renewed the mortality/ unemployment debate, further arguing temporary growth could cause mortality rates to increase. He identifies four possible mechanisms through which fatalities could vary pro-cyclically: First, there is the opportunity cost of time. Leisure time declines as economic activity increases this makes it more costly to undertake exercise and other health promoting activities. Second, health is an input into production. The physical exertion of employment may have negative effects on health if job hours are extended. Also pollution increases when production increases. Third, there may be external sources of death. Work-related accidents are likely to be more common during temporary expansion as are other accidents such as motor vehicle fatalities. Finally, he notes that migration flows will increase. Localized fluctuations in population in response to local economic conditions have the potential to increase death rates because of increased crowding, importation of disease or negative health issues if the migrants are unfamiliar with traffic

conditions or medical infrastructure. Ruhm concludes that recessions are good for health emphasizing this does not call for contractionary policies.

This chapter will investigate Ruhm's arguments relating to the negative stress related health effects of economic expansion. The proposal is that the only reason the spurious positive relationship occurs between recessions and mortality rates in Ruhm and other recent papers is because they run regressions on contemporaneous data. It is difficult to explain rationally how current unemployment rates could affect current mortality rates. Life expectancy in OECD countries is approximately 80 years of age. This means a large majority of the people dying have been retired for 15 years, and would therefore be unlikely to suffer from fluctuations in employment (since they are not working anyway). The use of current unemployment rates is therefore not an appropriate choice of data to investigate the relationship.

Since most deaths that contribute to the mortality rate data are from those retired for 15 years or more, the health of those dying today shouldn't be affected by detrimental effects of current economic expansion. More explicitly, the current unemployment rate is giving deceptive results. Further to this, it is not reasonable to expect the likelihood of death now to be affected by stress induced unhealthy lifestyle habits developed in only one period. It must be the unhealthy effects of behaviours developed in the past. If someone gets stressed and starts smoking today, they will not die from smoking related illness today, tomorrow or for some time into the future. There are two feasible reasons that the current death rates of those currently working could be affected by current economic activity. Firstly, suicide rates go up when people lose their jobs. Secondly, as economic activity increases and employment rises there will be more cars on the road, leading to more motor accidents and on the job accidents. The first reason supports a negative relationship and the second a positive one.

In order to test the effects of economic activity on particular lifestyle variables, such as increased smoking and less leisure, the method that is appropriate involves calculating the average incidence of unemployment over the person's entire working life. This is because the impact of any unhealthy habits developed during a person's working life will affect mortality rates in the future. Further to this, it will be the total amount of unemployment or over employment (that is, duration and levels of stress) a person has experienced over his/ her entire career that ultimately affects their health as represented by mortality rates.

If the current health effects of current economic activities are sought, the measure of health needs to reflect something that is immediately affected by economic activity, such as actual stress levels, happiness or incidence of depression. No such measure is available over long periods of time. This chapter will reproduce Ruhm's (2000) results using contemporaneous regressions on a panel data set that covers 21 countries over a far longer time span (1870-2008), and show that Ruhm's results do hold. It will then go on to run the same regressions using a cohort unemployment rate and show that Ruhm's results are reversed.

To rectify the problem of using contemporaneous unemployment rate, it is necessary to construct an unemployment rate for a cohort of a particular age. The mortality rate of 65 year olds is the mortality rate chosen here. Therefore, the cohort unemployment rate is a weighted average of the unemployment experienced for each year of this sage groups 49 years of working life. Cohort cigarette consumption and per capita income growth is also used. These variables far more accurately estimate the impact unemployment and negative lifestyle effects have on mortality rates. When these constructed values of unemployment and lifestyle effects are used in the regressions, Ruhm's results are significantly and consistently reversed. The cohort investigation in this chapter considers the effects of income, unemployment and smoking. There is not enough data to construct cohort alcohol consumption. This study, unlike Ruhm, also explores cyclical income, not just per capita income.

The chapter begins with a brief review of the literature, a look at the data, and a discussion of the data construction. Following this is an explanation of the econometric methods and issues including different specifications. The results aim to clarify the above mentioned hypothesis that short term fluctuations in unemployment do not affect current mortality rates. This result has previously been found due to an inappropriate use of contemporaneous data in recent literature.

4.2. Literature Review

Brenner (1973) reopened the discussion of the detrimental health effects of recession, this issue had attracted quite a bit of attention post the Great Depression earlier in the 20th century but little since. The hypothesis is that economic instability, recession followed by rapid growth, is potentially harmful to individual health. Brenner (1979) emphasizes the impact of recessions on low skilled workers, workers in industries for nonessential services,

and those whose skills become redundant, is particularly harsh. The reason for this is, a lack of income security makes social and family structures breakdown, and can lead to bad habits. These health effects of recession (unemployment) can mean that recessions are more costly than is measured by income loss alone.

Brenner (1979) uses data from studies from the U.S. as evidence and backs this up with data from England and Wales from (1936-76). Brenner and Money's (1983) empirical study uses similar data as Brenner (1936-76) of mortality rates and business cycles (income and unemployment rates), and they still find an inverse relationship. They also explore measures of rapid economic growth to see if it is harmful to specific minorities: firstly, the deviation of actual from trend income and secondly the annual change in income. Brenner and Money's (1983) paper spends quite a lot time backing up Brenner's earlier papers which had received quite a lot of criticism (Eyer, 1977, Gravelle, Hutchinson and Stern, 1981, Stern, 1983).

Eyer (1977) was one of the first to find results contrary to Brenner's view and argued the general death rate rises during business booms and falls during depression. Using macro data they show that mortality rates are procyclical. His reasoning for this is the role of social stress. Overwork and fragmentation of communities through migration are two important sources of stress which rise with an economic boom. Eyer believes this to be demonstrably related to the causes of death which show the variation of the death rate that coincides with business cycles.

The critique of Gravelle et al (1981) is based on the specification of the model, particularly omitted variables (discussed next parafigure) and inappropriate measures of growth. The use of real per capita personal disposable income rather than real per capita GDP suggests that other uses of national output (pre tax income) are not expected to reduce mortality. As Gravelle et al (1981) point out; the use of personal income instead of GDP implies that increased public health expenditures (a reduction in personal income) increases mortality. On the other hand, decreased expenditure on public housing reduces mortality. Gravelle et al (1981) also discusses an issue with the measures of rapid growth, they are differenced in levels and are not proportionate to high and low income groups. The reliability of the data is also questioned. Gravelle et al (1981) claim that the time series are geofigureically inconsistent, some of the variables are for Wales and England and others for the U.K. The mortality data includes civilian mortality due to the operations of war which have nothing to do with business cycles.

Gravelle et al (1981) conclude that the period over which his model examines is vital to the robustness of his results. There is nothing in Brenner's (1973) model to suggest the period over which he has tested his hypothesis is crucial. In order to provide some evidence of this suggestion they compare Brenner's results with others and find dramatic differences using different time periods. They also tested Brenner's results using the Chow test and found considerable structural instability - the argument is he has not allowed for the fact that his time series has a significant break in 1951.

Further criticism of Brenner includes his selection of independent variables. In particular, the authors have argued that the exclusion of variables such as education, (McAvinchey, 1983), and cigarette and alcohol consumption, (McAvinchey, 1984, and Sogard, 1983) lead to omitted variable bias. Leaving out these variables means you could have either positive or negative effects on health, which may lead to the model incorrectly over or underestimating the effect of one of the other explanatory factors. Sogard (1983) also argues that Brenner's model suffers due to its exclusion of environmental factors that may affect health. Stern (1981) voiced concerns over the strength of the causal relationships underlying Brenner's hypothesis. Brenner's procedure for selecting lag structures has been criticized for being arbitrary (Stern, 1981). Wagstaff (1985) replicates Brenner's regressions emphasizing the problems of the pattern of the lag coefficients and structural stability. Wagstaff (1985) concludes that time series analysis may produce convincing evidence at the macro level in the future, but believes Brenner has not yet done this. Since these discussions between Brenner and his critics in the 1980's, interest subsided from the issue of health and business cycles for roughly a decade.

Stern (1983), while criticizing Brenner does believe unemployment is a likely determinant of ill-health; the problem is it is very difficult to test. There are multiple socio-economic determinants of mortality rates and their relationship to both poor health and unemployment. The data rejects the view that unemployment has no effect on health but Stern did not find reliable evidence in favour of the view that unemployment is major determinant of mortality. In particular, he states that Brenner's claim that unemployment is a major factor in the annual changes in mortality rates is rejected by the data if proper statistical methods are applies.

Ruhm's (2000) empirical investigation uses data from 1972-1991. He uses Census data from the US bureau of Statistics which is based on America, across 50 states. The econometric method uses simple ordinary least squares models (OLS) with fixed effects to control for

unobserved, time-invariant characteristics. The estimations are mainly of current mortality associated with current economic conditions but do include four-year lags on unemployment rates and personal incomes to provide information on dynamics. Ruhm also includes other health inputs such as height adjusted body weight, and the consumption of alcohol and tobacco. He believes the analysis provides strong evidence that health improves when the economy temporarily declines. Specifically, a one percentage point rise in the state unemployment rate, relative to its historical average, is associated with a 0.5 to 0.6 percent decrease in total mortality, alternatively a reduction of approximately 11,000 fatalities per year. Unemployment rates in states across the USA are negatively related to total mortality rates; alternatively, increases in unemployment reduce mortality, this finding is the opposite of Brenner (1973, 1979, 1983).

Ruhm's (2000) results and arguments have proven to be popular throughout the early 2000. A number of authors have been inspired to investigate the seeming paradox and report similar positive health affects arising from recession. Examples of these are: Granados (2005), Gerdtham and Ruhm (2006), Ruhm (2007), Economou et al. (2008), Miller et al. (2009), Gonazalea and Quast (2011), Douglas et al. (2008), and Stevens et al. (2012). On top of mostly reaffirming Ruhm's results that there are positive, or at least non negative health effects from recession, these authors look to offer explanations for these results that differ somewhat from Ruhm's.

Granados (2005) noted that mortality has dropped throughout the 20th century, but that over and above the long-term falling trend the death rate has oscillated over time. He finds that statistically significant results show the decline of total mortality and mortality for different groups, ages and causes accelerated during recessions and was reduced or even reversed during periods of economic expansion—with the exception of suicides which increase during recessions. Granados suggested this deceleration or even reversal of the secular decline in mortality during economic expansion include both maternal and psychosocial effects. He noted that the expansion traffic and industrial activity would directly raise injury-related mortality, that decreased immunity levels (owing to rising stress and reduction of sleep time, social interaction and social support), and increased consumption of tobacco, alcohol and saturated fats.

Gerdtham and Ruhm (2006) use data from 23 OECD countries from 1960-1997. The specification is slightly different from Ruhm (2000) as they use a weighted least squares

estimation (weighting the observation by the square root of the national population. They also allow for first order autocorrelation with country specific AR(1) coefficients. Otherwise, the model is basically the same as Ruhm (2000). The main result is the same, total mortality and deaths from several common causes increase when labour markets strengthen. He emphasizes that increased mortality associated with transitory strengthening of the labour market does not necessarily imply negative effects of permanent economic growth. Temporary increases in output usually involve more intensive use of labour and health inputs with existing technologies, whereas permanent growth results from increased technologies or expansions in capital stock which push out the production possibility frontier and thus attenuate (reduce) the costs to health. Gerdtham and Ruhm (2006) emphasize that evidence of health deterioration during economic expansion is not an argument for inducing recessions. Rather that there should be intervention designed to ameliorate the health risks of expansions and further that it should be microeconomic in nature and vary with the specific problem addressed.

Ruhm (2007) again uses similar methods to his 2000 and 2006 papers, this time on US data but the main focus is mortality from coronary heart disease (CHD) not total mortality. The results are that deaths from heart disease rise when the economy strengthens because of increases in CHD. Most of the fluctuations in death involve senior citizens, and thus the important pathways extend beyond workers. Examples include air pollution and traffic congestion and also a reduction in medical attention. He also notes that fatalities from CHD respond faster to changes in macroeconomic conditions than most other disease-related causes of death but also return more rapidly to near baseline levels. Again, he mentions that these results do not justify contractionary policy and that there is a need for intervention to alleviate the negative health effects of short term economic expansion.

More recently studies are questioning Ruhm's results and reasoning. Economou et al. (2008) use data from 13 European countries over a 20 year time period (1977-1996) and find no strong relationship between unemployment rates and mortality rates. They find in contrast the evidence from Europe supports the opposite view that economic downturns are harmful to health. However, when they closely replicate Ruhm's study, although usually insignificant, they do find a negative relationship. In particular, when they include in the regressions additional demofigureic, health prevention and lifestyle factors the relationship is negative. This indicates that short term economic fluctuations may affect health through their effect on

lifestyle and other factors. The suggestion is unemployment rate acts as a surrogate of the effect unemployment rates have on demofigureic, health prevention and lifestyle factors.

Gonzalez and Quast (2011) aim to find whether the pro-cyclical nature of mortality and business cycles found in developed countries is present in Mexico. The main results are firstly, total mortality rates increase (decrease) during economic expansions (contractions) during this time period. Secondly, a larger effect of business cycles is found on the mortality rates of those aged 20-49 than on older cohorts. Thirdly, increases in per capita income are associated with lower mortality rates for cancer and higher mortality rates for suicide. They provide some insight into these results for Mexico. Firstly, changes in per capita income produce social changes such as automobile accidents, homicides and suicides. Secondly, public health spending and the number of doctors do not have a statistically significant effect on the mortality rate in Mexico.

A number of papers have given support to Ruhm's empirical results but question his explanation for these results such as Douglas et al. (2008), and Stevens et al. (2012). Douglas shows that most of the additional deaths arising from reductions in the unemployment rate are concentrated among the non-working age population, those under 18 and over 65. Thus, mechanisms involving direct effects of individual work hours or behaviour on one's own health are unlikely to be driving these results. This supports the hypothesis that the mechanisms connecting current economic activity and current mortality do not involve an individual's own current employment status or hours of work.

Stevens et al. (2012) focus on mortality figures in the USA and where and when the extra deaths are occurring. A key initial finding is that most of the additional deaths that are occurring during economic slowdowns are elderly people and especially elderly women. They find that approximately ¾ of the additional deaths are elderly (over 65). Elderly women accounted for 55 percent of the additional deaths. Elderly women typically outlive their husbands and are more likely to reside in nursing homes or other care facilities at the end of their lives. Since these people are usually retired, factors related to current working conditions are unlikely to explain rising mortality. Noting that the elderly are likely to use more medical services as they get older Stevens et al. (2012) examine employment in the health care system. Health care facilities find it more difficult to hire staff when the economy is strong. Low-paid low skill health care workers find better jobs elsewhere. Labour

shortages also occur in hospitals during strong job markets. Stevens et al. (2012) offer interesting counter explanations to the more popular stress and social arguments of Ruhm.

It appears, even though some writers have pointed out that additional current deaths are not occurring predominately amongst the working population, no one has noted that the use of contemporaneous variables that relate to business cycles is unlikely to be appropriate. A more realistic investigation of Ruhm's (2000) would be to follow Sullivan and von Wachter (2007, 2009). This research links an individual's job loss (displacements) to that individuals own probability of dying.

Sullivan and von Wachter (2007, 2009) offer the following theory and have backed it with empirical results. Health stock, H_{it}, evolves according to

$$H_{it} = \alpha y_{it} + (1 - \delta) H_{it-1}$$

Where y_{it} is income, α_{it} is the fraction of income invested in health stock and δ is the constant rate at which health stock depreciates. This implies that the stock of health is a function of current and previous income.

$$H_{it} = \sum_{s=0}^{t-1} \alpha y_{it} (1 - \delta)^s + (1 - \delta)^t H_{i0}$$

This suggests that mortality depends on an individual's employment over their entire working life. The equation above provides a justification for constructing a variable that represents an individual's entire income (present and past), in this case the unemployment rate experienced over a cohorts entire working life. This is the basis for the empirical investigation that follows.

Sullivan and von Wachter (2007) find that job displacements increase the probability of dying. Current death rates are dependent on many factors, most of them historical and many reaching far back into the past. For example, current mortality, and in particular deaths from coronary heart disease, have been found to be largely determined by foetal and maternal health (Barker, 1990). Current business cycles that affect stress and lifestyle cannot affect current deaths; it must be business cycles that caused these detrimental health habits over the working period of those dying. That is deaths of the elderly (65+) will be affected by the stress and bad habits that they picked up during their working career. The only sensible uses

for contemporaneous death rates on current business cycles are those that have immediate impacts such as suicide, homicide or road accidents or workplace accidents. Other sensible explanations must be current in nature also, such as Stevens et al. (2012) where the effect of a strong labour market does affect current aged care facilities and therefore the elderly death rate

4.3. Theory

The basic theoretical approach adopted here is similar to that of chapter two. Where health is the dependent variable most empirical studies will adopt a production function approach. Good health, H_t, is the outcome of various inputs at time *t*. In this chapter the main input is the state of the macro economy measured by the unemployment rate and per capita income. The following is the assumed production of health:

$H_t = h_t(u_t, y_t, c_t, z_t)$.

Where, H_t, good health is the mortality rate at time *t*, u_t, represents the cohort unemployment rate at the same time, y_t, is per capita income at *t*, c_t, tobacco consumption and, z_t, captures other variables that produce good health at that time such as foetal and early childhood health, reduced alcohol consumption, education, nutrition and other environmental factors.

Assuming some sort of Cobb Douglas function, the basic logarithmic transformation of this health production function is:

$$\ln \quad H_{jt} = \alpha_t + \beta_1 lnu_{jt} + \beta_2 lny_{jt} + \beta_3 lnc_{jt} + \beta_4 lnz_{jt} + S_j + \varepsilon_{jt} \qquad (4)$$

Where *j* and *t* are subscripts to index the country and the year, *H* is the natural log of the mortality rate, u and y proxies economic conditions and are the unemployment rate and per capita GDP respectively, c is cigarette consumption, *z* is a vector of supplementary regressors and, ε ,measures the error. The fixed-effect of *S* controls for time-invariant country characteristics, and α represents a year specific-intercept. Due to the collinearity between the unemployment rate and per capita income the above regression was also estimated without income:

$$\ln \quad H_{jt} = \alpha_t + \theta_1 lnu_{jt} + \theta_2 lnc_{jt} + \theta_3 lnz_{jt} + S_j + \varepsilon_{jt} \qquad (5)$$

All of the regressions in this chapter have been estimated using the variables in levels, log levels and growth rates. The results were not sensitive to different measures therefore levels have been used for consistency and comparison with the literature. The literature discussed above uses log level data.

4.4. Data

This chapter offers two data contributions to the literature. The first is the collection of a far longer time series data set for the consumption of tobacco than has previously been used. This covers 21 countries from 1870 to 2008 and provides a large cross section. Secondly, the calculation and use of the average unemployment a person has experienced over their working life (cohort unemployment,e_{jt}^{c}) is introduced, rather than just using the annual unemployment rate as a regressor. Cohort aggregate per capita income, y_{jt}^{c} , and tobacco consumption, c_{jt}^{c} , has also been calculated.

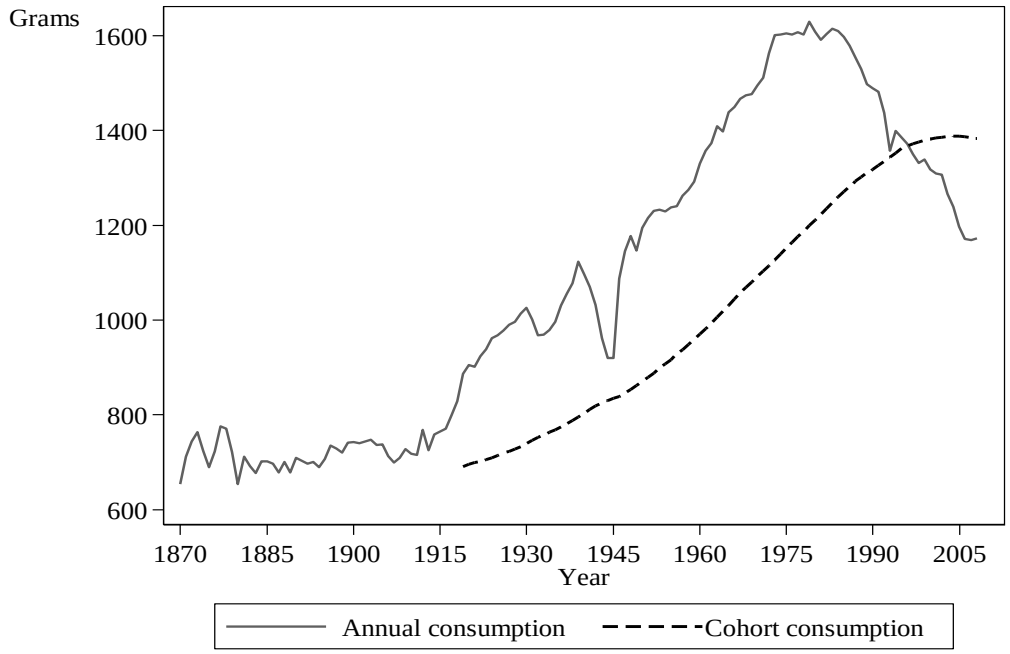
4.4.1. Tobacco

Tobacco consumption data has been collected since 1870 or even earlier for some countries. The data sources for each country are explained in the data appendix and have been measured in two ways;

1. The current tobacco consumption for the population aged 15+

2. Cohort tobacco consumption. This is the average consumption for a person over the period that they were of working age (15-65).

Figure 1: Annual Tobacco consumption per capita (15+) and average lifetime consumption at 65



Tobacco consumption increased from 653 grams per person per year in 1870 to 1629 grams per year in 1979 it has since declined to 1172 grams in 2008. Tobacco consumption increased sharply after both World War I and World War II. The post-World War II increase occurred at a time where there was high income growth and a reduction in trade restrictions, particularly amongst the OECD countries. It then declined after 1975 probably due to increased awareness of the detrimental health effects of smoking.

4.4.2. Unemployment

The unemployment rate has been calculated in a way that represents the amount of unemployment a person would have experienced over his/her entire working life. This is done to capture the overall affect unemployment would have on human health. The data that is appropriate here is the unemployment rate of a particular cohort averaged over their entire working life (15 - 65). A number of things need to be considered. Firstly, younger people tend to be laid off work before older people in economic downturns. There is therefore a higher incidence of unemployment amongst the young relative to the middle aged. Secondly, between countries there are different proportions of the workforce in different age groups (i.e. some countries will have a younger workforce and others older). To accurately calculate the

average unemployment rate for a person of a particular age the following data was collected: the annual unemployment rate, U_{jt} , the average incidence of unemployment amongst different age cohorts, I_i, and the percent of the workforce that is in that age group, e_{it}.

In the following; subscript *i* denotes the different age groups within the workforce, which are; the young 15-24, middle aged 25-54, and old 55-64. And subscript *t* denotes the year. The superscript *c* represents cohort. The calculation was carried out for each country to achieve the cohort tobacco consumption for the entire panel. The percentage of the workforce aged 15-24, 25-54, and 55-64 (e_{it}) is from the OECD website.

The incidence of unemployment (I_i) is the annual, harmonized unemployment rate from the OECD labour force statistics. It shows the average unemployment from 1995 to 2010 for persons in age groups 15-24, 25-54 and 55-64 for each year. This has been used as it was the only available data. The average over this period (1995-2010) was used to calculate the incidence of unemployment for each age group when calculating cohort unemployment. The estimation of alpha is the incidence of unemployment for a particular cohort divided by the average total unemployment rate for the same period (1995-2010).

$$\propto_{ji} = \frac{I_{ji,t=av(1995-2010)}}{U_{jt=av(1995-2010)}} \quad (1)$$

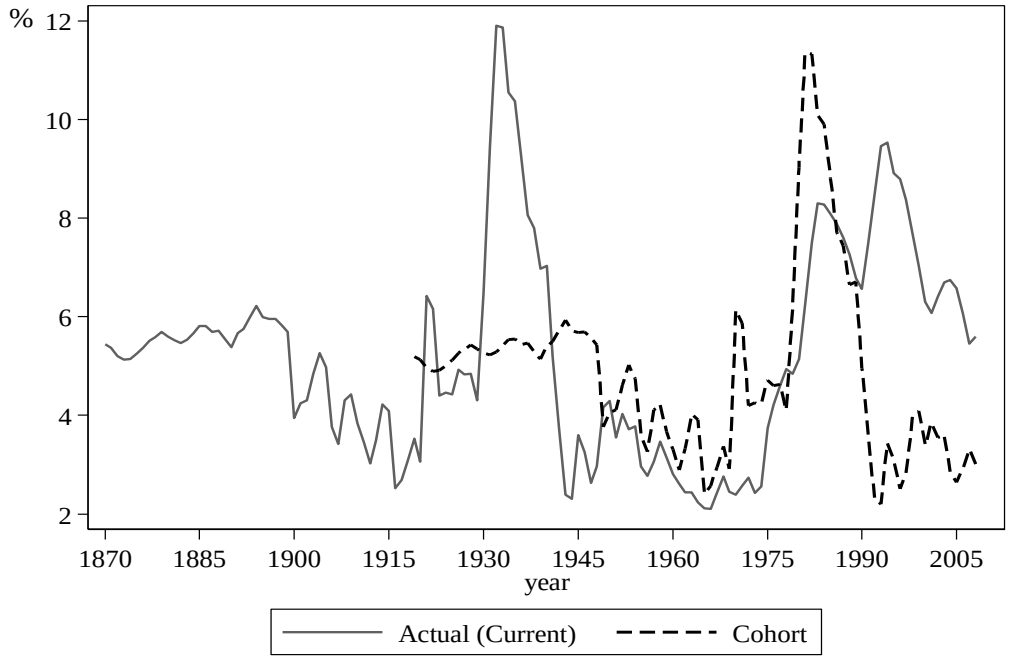
The calculation for the estimated unemployment for each country and each cohort, (U_{it}^{c}), is equal to the percentage of the workforce that is in that age group (e_{it}) multiplied by the ratio of the incidence of unemployment over average unemployment for that cohort (α_i) multiplied by the actual unemployment rate for each year (U_{jt}) in each country.

$$U_{jit}^{c} = e_{jit}. \propto_{ji}. U_{jt} \quad (2)$$

The next step is to estimate the average unemployment rate for a person of a particular age cohort, for their entire working period, U_{jt}^{c}. For example a person aged 65, from country *j*, at time *t*, would be calculated over the past 49 years. Since the data starts in 1870 the first estimated cohort unemployment rate for those aged 65 is for the year 1919. The calculation for this is simply the sum of the average estimates for each year

$$U_{jt}^{c} = av.\ U_{j,t-49,\ldots,40}^{c} + av.\ U_{j,t-39,\ldots,11}^{c} + \ U_{j,t-10,\ldots,1}^{c} \quad (3)$$

Figure 2: Actual and cohort unemployment rates



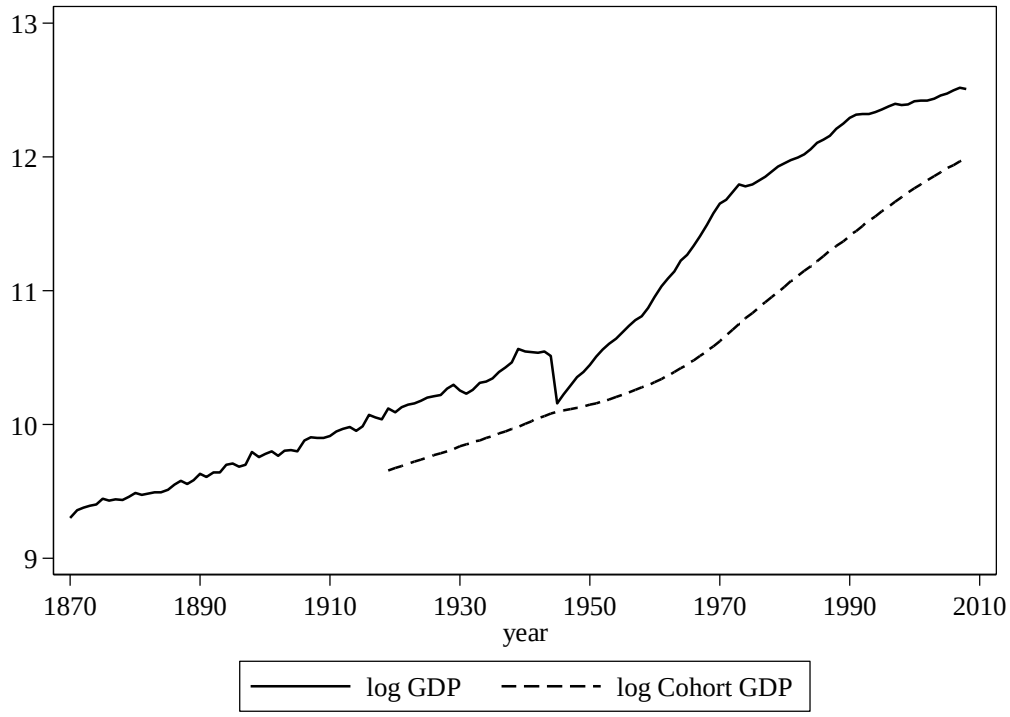
The cohort unemployment rate (dashed line) does not follow the same path over time as the current unemployment rate; current rates can be high when the cohort rates are very low, they have often moved in opposite direction. This shows there is plenty of scope for differences in the regression results depending on whether current or cohort unemployment rates are used.

4.4.3. Output

Output has been measured in three ways

1. Current real GDP per person for each year.
2. The cohort per capita output for a 65 year old each year for the period over which they were aged 15-65, or cohort per capita GDP. Because the data series starts in 1870 the cohort (average working life) series must start 49 years later in 1919.

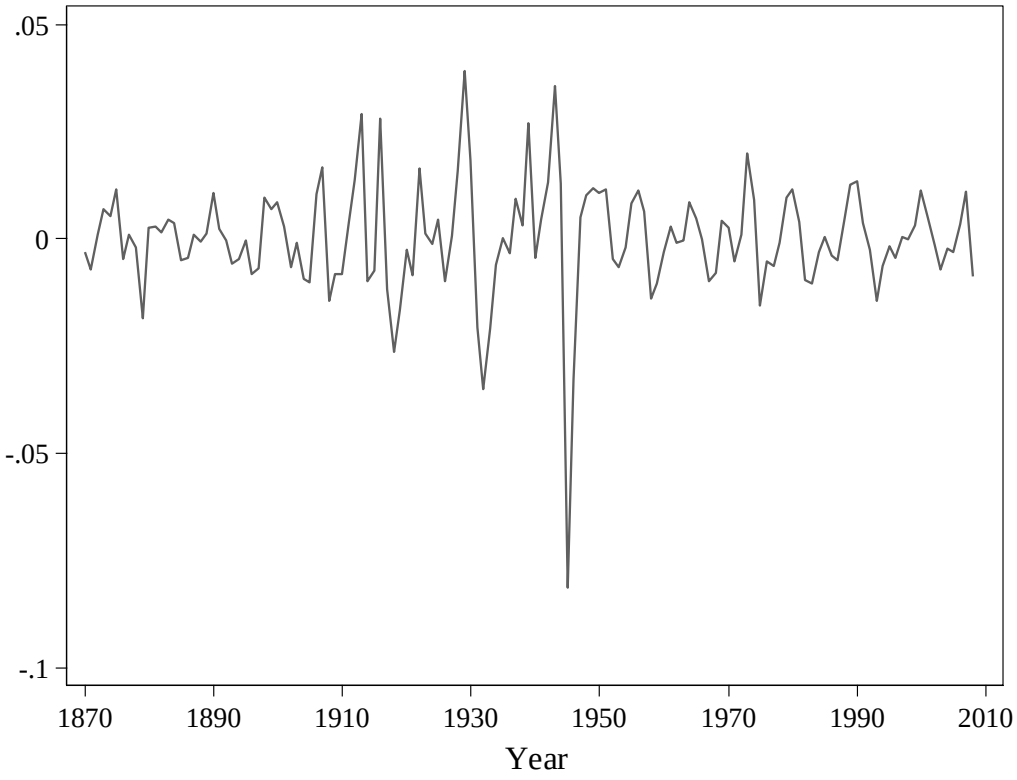
Figure 3: Log of Real GDP per capita and for the 65 year old cohort (Int. GK$ Maddison)



The average annual per capita income across countries in 1870 was just over 2,000 dollars and has risen to nearly 25,000 dollars. Figure 3 shows that cohort per capita GDP, that is the average income a 65 year old earned over their 49 year working career, is slightly smoother than annual per capita GDP. However they both follow a relatively consistent upward trend.

3. Cyclical income. This is cohort per capita GDP with the trend taken out using the Hodrick-Prescott filter.

Figure 4: Natural log of cyclical per capita GDP (average over all countries)



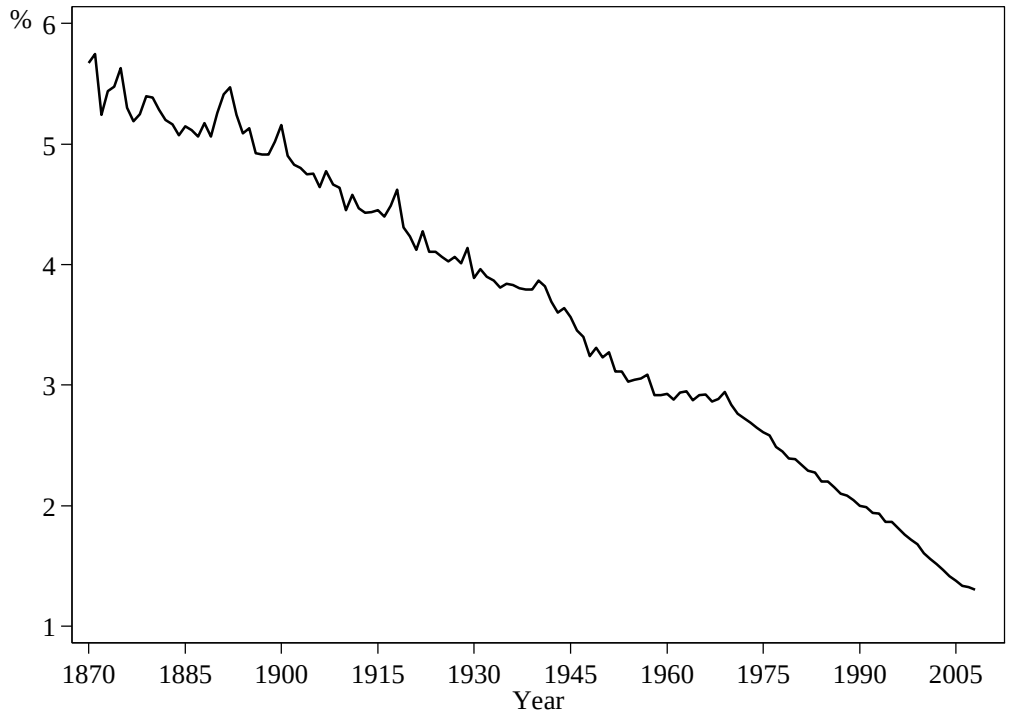
The cyclical component of the natural log of per capita GDP ranges from on average across all countries from 0.03 to - 0.08 and appears to ocillate around zero in a similar pattern across countries. The overall average for the entire period is -0.000025, which is very close to zero. This is expected, cyclical income should reflect the short term ups and downs in per capita income and not have any long term bias. There is a major drop in average cyclical income in 1945.

4.4.4. Mortality rates

Death rates for those aged 65 to 69 are used for this analysis and figureed below. Other age groups were also investigated with little difference to the results. Mortality rates are the best available proxies for health available over this time period. Unfortunately mortality which is the term used for the number of people who have passed away does not give direct information about the state of health of the living population per se. We need to assume the two are closely related. Morbidity, which refers to the state of being diseased or unhealthy within a population, is really what we would like to capture. The reason this is not used is that morbidity is much more difficult to measure and the data is not available. Morbidity has

only been measured since the 1990's and not for many countries. Mortality rates are available for far longer time horizons.

Figure 4: Average Mortality Rate across 21 countries



The average mortality rate across these countries has fallen from 5.7% amongst the 65 to 69 age group in 1870 to 1.3% in 2008. The downward trend also becomes very smooth after 1970.

4.5. Results

4.5.1. Ruhm's results

The first step was to investigate whether the negative relation Ruhm found between mortality and unemployment rates using contemporaneous data could be replicated using a longer data set. The panel used here covers the years 1870- 2008 across 21 OECD countries which is quite a different sample to Ruhm's 1972-1991 across the 50 states of America. Following Ruhm, equations (4) and (5) from page 11 were estimated. All of the models are Ordinary Least Squares (OLS) with country fixed effects. Ruhm (2000) also estimated the model without fixed effects; the results were very similar so they have not been reported below.

Table 1: Regression Results from Contemporaneous Data

Mortality rate	(A)	(B)	(C)	(D)
Unemployment rate	-0.009	-0.009	-0.053**	-0.48**
	(-0.67)	(-0.73)	(-2.03)	(-1.78)
Income		-0.14		-0.022
		(-0.75)		(-0.79)
R^2	0.038	0.25	0.039	0.025
Observations	2945	2903	2945	2903
Year effects	Yes	Yes	No	No

Notes: Statistics are robust to heteroskedasticity; the numbers in parentheses are t statistics.

The results for the unemployment rate are similar to Ruhm's, so long as year effects are excluded (models C and D). In model D, the coefficient on the unemployment rate is -0.053 and significant at the 5% level. The coefficient on per capita income is negative which is not what Ruhm found. It is also not consistent with a negative coefficient on the unemployment rate; income and unemployment should be of opposite sign, increased income should coincide with decreased unemployment. The regressions were consistent for different aged mortality rates and lagged unemployment rates. Ruhm used absolute per capita income not cyclical income; other authors have done the same. Cyclical income is arguably just as, if not more informative when investigating business cycles and the impact they have on health. If cyclical income is used the coefficient is still negative and therefore adds nothing to this replication.

To briefly investigate two of the contemporaneous mechanisms that Ruhm suggested could lead to the paradoxical result he found, that is bad lifestyle habits, the unemployment rate is regressed on both cigarette and alcohol consumption. The following OLS regressions were estimated:

$$c_{jt} = \alpha_t + \beta_1 u_{jt} + S_j + \varepsilon_{jt} \text{ and,} \quad (6)$$

$$alc_{jt} = \alpha_t + \theta_1 u_{jt} + S_j + \varepsilon_{jt}. \quad (7)$$

Where, *alc*, is alcohol consumption and all other variables are as defined above.

The results are in table 2 below.

Table 2: The Effect of Unemployment on Lifestyle

	Cigarette consumption	Alcohol consumption
Unemployment rate	-0.0438*	-0.017***
	(-1.42)	(-4.78)
R^2	0.006	0.08
Observations	2938	1027
Country fixed effects	Yes	Yes

Notes: Statistics are robust to heteroskedasticity; the numbers in parentheses are t statistics.

The unemployment rate was negatively related to both cigarette and alcohol consumption. Growth in the unemployment rate reduces the growth rate of cigarette consumption by 0.044 at the 10% significance, and reduces alcohol consumption by 0.017 at the 1 % significance level. These results are in line with Ruhm's argument that increased employment causes more stress and therefore more unhealthy habits such as smoking and drinking.

The proposal in this chapter is that it is not current business cycles (or unemployment) that effect current mortality. Rather, it is factors such as unemployment, foetal and infant health, as well as lifestyle habits that have occurred or been developed far into the past will affect current mortality. Therefore, the unemployment rate (and income levels) over the entire working life of a particular cohort (in this case 65-69 year olds) would be determining current mortality rates.

4.5.2. Cohort effects on Mortality with Income

Equation 8 is analogous to equation 4 except it is based on cohort data hence the superscript, *c*, on the appropriate variables and is including infant mortality, z_{jt}. The following was estimated over the period from 1870 to 2008 across 21 OECD countries:

$$lnH_{jt} = \alpha_t + \beta_1 \ln u_{jt}^c + \beta_2 \ln y_{jt}^c + \beta_3 lnc_{jt}^c + \beta_4 lnz_{jt-64} + S_j + \varepsilon_{jt} \quad (8)$$

Here, H_{jt} , represents the current mortality rate of country *j* at time *t*, as it did previously. The unemployment rate variable has changed, u_{jt} is now the cohort unemployment rate. Defined previously as the average unemployment rate in country *j*, experienced at time *t*, by those aged 65, over the previous 49 years. y_{jt} is cohort income or the average income experienced in country *j* that has been experienced at time *t*, by those aged 65, over the previous 49 years. The cigarette consumption of the aged 65 cohort, c_{jt} , is the average tobacco consumption experienced in country *j*, by those aged 65, over the previous 49 years. The only other explanatory variable is, z_{jt-64}, which is infant mortality lagged 64 years to capture early childhood health of the 65 year old cohort; data for foetal health has not been obtained.

Table 3: Regressions of unemployment and income on Mortality

Mortality Rate	(A)	(B)	(C)	(D)	(E)	(F)
Mortality					0.926***	
(1^{st} lag)					(36.36)	
Unemployment	0.043***	0.028***	0.043***	0.025**	0.003*	0.06***
	(4.44)	(2.35)	(4.34)	(2.19)	(1.55)	(9.23)
Income	-0.58***	-0.171	-0.29***	-0.068***	-0.003**	0.015
	(-6.34)	(-1.08)	(-5.25)	(-2.47)	(-1.75)	(0.30)
Cigarette	0.044	-0.080	-0.237*	-0.106	0.003	-0.061
	(0.52)	(-0.82)	(-1.63)	(-1.10)	(0.42)	(-0.46)
Infant Mort.	0.043	0.126*	0.227***	-0.462*	-0.005*	0.29***
	(0.47)	(-1.29)	(4.70)	(-1.39)	(-1.43)	(8.54)
No. of Obs.	1601	1601	1601	1601	1580	1241
R^2 within	0.89	0.929	0.872	0.928		0.56
Country Fixed effects	Yes	Yes	No	No	No	No
Year effects	No	Yes	No	Yes	1 lag, 1 lead	No
AR(1)					-3.58	
($p \leq 0.1$)					(0.000)	
AR(2)					2.22	
($p \geq 0.1$)					(0.027)	
Hansen J test					0.00	
(Chi2 $\geq$0.1)					(1.00)	

Notes: Statistics are robust to heteroskedasticity; the numbers in parentheses are t statistics and there is a year dummy for war periods for each country.

Unemployment

The coefficients on the cohort unemployment rate are consistently positive and significant across all specifications of the model. The OLS model without an adjustment for year fixed effects anticipates a one percent increase in the unemployment rate would bring about a 4.3 percent increase in the mortality rate; these coefficients are highly significant at the 1% level. This is the opposite of Ruhm's results. The size of the coefficient generated by the OLS estimator drops by 0.02 when year fixed effects are included by. However, the model still predicts an increase in mortality of over 2.5% if the unemployment rate increases by 1%, and the significance is still high, although slightly higher when country fixed affects are included than when they are not.

To check the sensitivity of the model to the estimation method chosen a Dynamic OLS (DOLS) regression and Arellano-Bond (A-B) estimations have also been considered and reported. The significance drops to 10% on the coefficient for unemployment in the Arellano-Bond and its size falls to 0.003, this is to be expected since the effect of the lagged dependent on the mortality rate is huge and highly significant - mortality rates have steadily declined over the period as seen in figure 3. The inclusion of a highly correlated regressor will always subtract the significance of the other regressors. Further to this, lagged dependent variables would usually be expected to explain a large proportion of the dependent. The DOLS estimator had the largest coefficient on the unemployment rate predicting a 1% increase in the cohort unemployment rate to increase the current mortality rate at age 65 by 5.6%. This result is the most significant, and also suggests the opposite conclusion to that found by Ruhm; recessions are not good for health.

Income

The coefficients on the per capita income are negative across 5 of the models but only significant across 4. If per capita income increases models 1, 3, 4 and 5 predict a decrease in the mortality rate which is consistent with a positive relationship with the rate of unemployment. Increased real cohort income should indicate higher production and therefore more demand for labour and less cohort unemployment. These results are consistent and are suggesting that the short term fluctuations in cohort output and cohort unemployment have a procyclical effect on health. That is, the lower the income a particular cohort has experience over their working life, the higher the cohort mortality rate. Specifically, economic upturns

are associated with lower mortality rates and downturns with higher mortality rates. Since income and unemployment are so highly correlated, income is not included in the regression results in table 4. This is because there is a possibility income and unemployment are essentially capturing the same effect.

Cigarette Consumption

Cohort cigarette consumption doesn't appear to explain very much of the variation in mortality rates. This is probably due to other variables that are related to both, for example discretionary income, health education and healthcare improvements. When it is slightly significant it is negative which means increased smoking decreases mortality rates. It is unlikely that smoking reduces mortality; it is more likely that better healthcare practices and improved nutrition have vastly increased public health in general over the past 100 years. And these improvements occurred at the same time as discretionary income and smoking increased. In reality, the increases in tobacco consumption have probably somewhat negated the positive effects of increased healthcare. Increases and decreases in cohort cigarette consumption have developed alongside improvements in medical care, income, education as well a far greater awareness of the importance of maternal and childhood health, nutrition and exercise.

Infant mortality

The expectation is that increased foetal and early childhood health increases life expectancy and therefore the mortality rate at any given age. In particular, it reduces the likelihood of coronary heart disease (Baker, 1990, Gonzalez and Quast, 2011). The infant mortality rate that is used is lagged by the age of the particular cohort, in this case 64 years. It is hoped this will capture the health of the cohort when they were in early childhood. This infant mortality coefficient was a little sensitive to model specification. While normally significant the effect was positive in three of the models and negative in two. This does not provide a conclusive result.

Alcohol

Alcohol consumption has not been included in the above regressions as its inclusion severely reduces the number of observations. There is also not enough data to obtain cohort

consumption figure as has been done with the unemployment, income and tobacco consumption data and is therefore not equally comparable.

Table 4: Regressions on Mortality rates without income

lnH_t	OLS fe r	OLS r	OLS yr* r	OLS yr* fe r	DOLS	Arellano-Bond (1 2)
ln H_{t-1}						0.93***
						(44.84)
ln u_{t65}	0.046***	0.046***	0.025**	0.024**	0.060***	0.003**
	(4.20)	(4.46)	(2.09)	(2.01)	(7.61)	(1.74)
ln c_{t65}	-0.522***	-0.48***	-0.134*	-141*	-0.49	0.007*
	(-3.12)	(-3.10)	(-1.38)	(-1.35)	(-0.57)	(1.39)
ln z_{t-64}	0.436***	0.438***	-0.147*	-0.159*	0.394***	-0.003
	(5.18)	(5.68)	(-1.35)	(-1.36)	(10.02)	(-0.69)
No. of Obs.	1601	1601	1601	1580	1241	1580
R^2 overall	0.424	0.429	0.824	0.819	0.413	
AR(1)						-3.57
($p \leq 0.1$)						(0.00)
AR(2)						2.20
($p \geq 0.1$)						(0.028)
Hansen J test						0.00
						(1.00)

(Chi2 ≥0.1)

Notes: Statistics are robust to heteroskedasticity; the numbers in parentheses are t statistics

If GDP per capita is dropped from the regression, the coefficient on the unemployment rate remains consistently positive across each model specification. This provides robust evidence that the unemployment rate a 65 year old has experienced over their working life is positively related to the mortality rate. This result is opposite to that of Ruhm (2000) and others of late (Miller et al. 2009; Granados, 2005). The cigarette consumption is negative and more significant than in table 3, again this result is justified by the probable coincidence of increased tobacco consumption with increases in discretionary income and health care in general. Infant mortality is positive and highly significant across three models (A, B and E), this should be the case since mortality rates tend to move together regardless of the age cohort. However, it is negative and slightly significant at the 10% level in two of the models so again the lagged effect infant mortality has on the mortality rate of 65 year olds is sensitive to model specification.

4.5.3. Cyclical Income

The analysis was additionally conducted replacing the natural logarithm of per capita income with that of cyclical income. Cyclical income should, if Ruhm (2000) is correct, be positively related to mortality rates. That is increases in cyclical income, like decreases in unemployment, will reduce health and therefore increase mortality rates. On the other hand, if the hypothesis of this chapter is correct and cohort data is used the relationship will be negative. Increases in cyclical income over a person's working life will improve health and therefore reduce mortality.

Table 5: Contemporaneous Data Regressions with Cyclical Income

Mortality rate	(A)	(B)	(C)
Unemployment	-0.183***		-0.179***
	(-5.00)		(-4.93)
Cyclical Income	-0.0546	0.239*	
	(-1.21)	(1.44)	
Cigarette	-0.613***	0.446***	0.387***
	(2.42)	(2.49)	(2.89)
Alcohol	0.095	-0.28	0.085
	(0.55)	(-0.13)	(0.50)
Observations	984	984	984
R^2	0.148	0.148	0.44

Notes: Statistics are robust to heteroskedasticity; the numbers in parentheses are t statistics

The contemporaneous results support Ruhm (2000). The coefficient on the annual unemployment rate is negatively related to the mortality rate and highly significant. The models above would predict a 1% increase in the unemployment rate to coincide with a 0.18% increase in the mortality rate. The coefficient on cyclical income is negative in model A which is contradictory to the negative coefficient on the unemployment rate, however, it is not significant. In model B the coefficient on cyclical income is positive and significant which is consistent with mortality rates rising when income increases, that is mortality rates fall (health increases) when the economy is in recession. The cyclical component of income is likely to be more highly correlated the unemployment rate than income in general; it therefore doesn't make much sense to include both the unemployment rate and cyclical income at the same time. Cohort regressions of cyclical income were likewise investigated with the unemployment rate.

Table 6: Cohort Regressions with Cyclical Income

lnH_t	OLS	OLS	OLS	OLS	Arellano-Bond	DOLS
	Fe r	Fe, r, y*	r	R, y*	1 lag, 1 lead	
ln hpy_{t65}	1.63	0.813	1.51		-1.10	-2.13
	(1.26)	(0.79)	(1.16)		(-0.99)	(-0.49)
ln c_{t65}	-0.625***	-0.143	-0.556***		-0.010	-0.307***
	(-2.80)	(1.20)	(2.56)		(-0.07)	(-3.13)
ln z_{t-64}	0.345***	-0.233*	0.351***		-0.379	0.421
	(3.13)	(-1.42)	(3.34)		(-0.92)	(6.85)
No. of Obs.	1155	1155	1155		1155	71
R^2 within	0.718	0.87	0.717			0.985
AR(1) ($p \leq 0.1$)					(0.007)	
AR(2) ($p \geq 0.1$)					0.757	
Hansen J test (Chi2 ≥0.1)					0.00	

Notes: Statistics are robust to heteroskedasticity; the numbers in parentheses are t statistics

The coefficient on cyclical income is not significant across any of the estimated models. These results do not seem to be useful due to the lack of significance of the cyclical income coefficient. However, this fact itself may imply that the significance of the income coefficient in table 3 may be partly due to the trend component in the GDP series. The insignificance of the cyclical income coefficient may also bring into question the possibility

of omitted variables driving the correlation between the unemployment rate and mortality rates. Cyclical income should in theory move in a similar way to the unemployment rate, although it is also likely unemployment would lag cyclical income since it should take time for employers to realise a fall in revenue and make layoffs. These lags have not been investigated. This section remains since it reinforces the results obtained using contemporaneous data, when unemployment rates decrease, mortality rates increase. For the cohort regression the implication is that it is not cyclical income that is generating a pro-cyclical relationship between the unemployment rate and mortality rates. There is a relationship between unemployment and health that is somewhat independent of fluctuations in income.

4.5.4. Endogeneity

There are of course other causes of changing mortality rates that are not included in the model. Education, technology and advancements in medical treatments are some of the more important. Other variables that may affect mortality are geofigurey, climate, genetics, population density, and pollution to name a few. Firstly, it is pointed out here that it is not entirely necessary to include education in the regression analysis since it moves with unemployment. When unemployment increases amongst the young they stay at school longer or decide to spend their otherwise free time obtaining tertiary education. Secondly, technology and advancement in medical treatment are variables have improved vastly over the past 140 years. Advancements in medical treatment also explain a large portion of reduced mortality, but like education is also likely to move with income and therefore employment. The presence of omitted variables means one should be careful not to draw causal arguments from these results.

The point is not that omitted variables are not important there are always going to be excluded variables that have some explanatory power. However, in this case the inclusion of the above mentioned variables would not contribute to the analysis. This is because the conclusion is not that higher unemployment *causes* higher mortality; rather that the cohort unemployment rate is highly and positively correlated with current mortality rates. Further to this, there is no evidence the relationship is negative unless contemporaneous unemployment rates are used.

There should be no reverse causality between the dependent variable and the main explanatory variable, the cohort unemployment rate, since the cohort unemployment rate is lagged average over 49 years. It is not possible for the current mortality rate or health to affect previous unemployment rates.

4.6. Discussion

Long-run evidence shows that rising living standards decrease average mortality all over the world. However, evidence over the short to middle term fails to show the same relationship. The purpose of this paper has been to investigate the claim by Ruhm (2000, 2003, and 2007), where he asserts that higher unemployment is associated with lower mortality. This goes against medical research (Sandifer and Stein, 1985). There could be many reasons why losing one's job may affect mortality. Some of which may have positive effects on health such as increased leisure time and less work related stress, as suggested by Ruhm (2000), but these and other reasons may also have significant negative effects. Losing ones' job would reduce either short or long-term income, and could even lead to early retirement, the resulting fall in overall income can reduce the ability to enjoy leisure activities (because they may be expensive) and buy nutritional foods for the same reason. Job displacement is often very stressful, and stress is generally thought to be detrimental to health. Financial stress in particular has been shown to have detrimental health effects Job loss may also lead to the loss or reduction of health insurance, which can reduce the quantity of medical care a person is able to receive.

A precise response to Ruhm (2000) and the four main mechanisms via which he believes recessions may have positive effects on health (negative effects on mortality) follows. Ruhm's (2000) arguments outlined on page 2 are briefly restated for clarity:

First, there is the opportunity cost of time. Leisure time declines as economic activity increases this makes it more costly to undertake exercise and other health promoting activities.

Second, health is an input into production. The physical exertion of employment may have negative effects on health if job hours are extended. Also pollution increases when production increases.

Third, there may be external sources of death. Work-related accidents are likely to be more common during temporary expansion as are other accidents such as motor vehicle fatalities.

Fourth, he notes that migration flows will increase, increasing death rates because of increased crowding, importation of disease or negative health issues if the migrants are unfamiliar with traffic conditions or medical infrastructure.

The following comments on each of these four mechanisms:

Firstly, the opportunity cost of time (the real wage) does go up when the economy temporarily improves. However, unless workers fear they will lose their jobs if they do not work more hours, they are choosing to work more. If the decision to work more is a personal choice (preference), workers will be just as likely to benefit from more healthy activities, due to a rise in income, as they are to suffer detrimental effects. After all, we assume the economy adjusts to fluctuations in the business cycle, by re-optimizing labour/ leisure choices and hence consumption choices. An increase in economic activity is an increase in production and consumption possibilities. As consumption possibilities increase and wages increase, the price of leisure (as Ruhm points out) does become relatively expensive; however, in response to this, individuals adjust their allocations between labour and leisure to maximize welfare. If we make all the usual assumptions on rational behaviours, it is not theoretically possible for the economy to end up at a lower level of well being. Welfare must increase if consumption possibilities expand otherwise people are behaving irrationally.

Secondly, the argument about health as an input into production does have some merit. 'On the job' accidents may increase if more work is taking place with the same technology. Pollution may also increase. It is doubtful that work related stress could outweigh the stress of not being employed and as the previous parafigure pointed out, if this stress did get that great you could simply chose not to work as much.

The third argument is to do with external sources. One of the reasons is that there are more car accidents due to increased traffic and increased drink driving. Increased cars on the road could increase the likelihood of collision however it is unlikely that extra work leads to increased drink driving. It is just as easy to imagine people who don't have to work the next day, would be more likely to go out drinking and driving. On the other hand, it is also possible that increased traffic due to more economic activity, would increase the incidence of motor accidents. His acknowledgement that suicides increase as the economy deteriorates

due to stress is in direct conflict with his second argument above that stress decreases during recession.

Finally, Ruhm points out that increased migration flows or urbanization, in response to economic fluctuations, are detrimental to health because of increased crowds. This does not seem a likely situation in developed countries. However, importation of disease and inadequate urban infrastructure, could increase mortality or at least decrease health in certain countries. Ruhm's very last point to do with migration is the reasonable considering the use of contemporaneous data. The movers (migrants), who are usually young and therefore relatively healthy, would induce a spurious negative correlation between economic conditions and mortality in the migrant's country of origin.

There needs to be a clear distinction between the effect of over employment, under employment and unemployment. Employee's work/leisure/ consumption preferences are very important here. Both over and under employment levels are likely to be less desirable and have larger detrimental effects on health than full employment. However, so long as there is freedom of choice, if there was a persistent occurrence of over employment (such that it reduced welfare), we would witness businesses having trouble finding staff and/ or wages increasing rapidly during good times to attract otherwise reluctant staff. We do not see this. Further to this, over the business cycle, we assume, during times of increased economic activity, that the real wage actually falls due to rising prices. Workers will take time to negotiate increases in the nominal wage in response to the price rise. And once they do the real wage increases, ultimately they will shift the economy back to its long run level of employment or full employment. This process is always expected to happen during increased economic activity and the opposite for a contraction unless the disturbance is permanent.

4.7. Conclusion

It has been found here that there is a significant positive empirical relationship between the cohort unemployment rate and the mortality rate. This makes sense for the following reasons:

- People who are unemployed or under employed are just as likely to be stressed and develop unhealthy habits as those who are over employed.

- If people accept more work during higher growth periods they have chosen to do so in response to the changing economic environment. And if they are rational they will have made the choice that best suits their welfare.
- It does not make sense to regress current unemployment on current mortality rates. This is because the impact on health will be lagged over the entire working life of those suffering or benefiting from the state of the economy.

The negative relationship between contemporaneous unemployment rate and mortality rates that Ruhm and others have found is both negative and significant some of the time. However, the results from various studies appear to be sensitive to the estimation method used, the time period over which the investigations were conducted, as well as the geographical location. The models can also produce a positive significant relationship with contemporaneous data. The data set used in this chapter offers some evidence for current recessions leading to lower mortality rates under certain conditions, i.e. where annual data is used. This, however, is likely to be connected to the current economic conditions affecting the elderly, not the current work force. It does not seem reasonable to suggest as Ruhm (2003) and others have, that *"Good times make you sick"*. In particular, it is difficult to believe lifestyle reasoning provided by Ruhm (2000) could lead to negative habits that develop from higher employment out-weigh the positive effects of having work. It is far more plausible that factors that are related to current unemployment and the mortality rate of the elderly are driving this result. For example poorer aged care during economic expansions, due to a lack of staff.

Finally, the main findings are two-fold. Firstly, contemporaneous unemployment, income and lifestyle variables are not appropriate statistics to use to question how unemployment or over employment affects health (and therefore mortality rates). Cohort statistics for unemployment, income and cigarette consumption have demonstrated opposite the results achieved by contemporaneous data. It is therefore concluded in answer the question of whether recessions are good or bad for health via stress and lifestyle mechanisms, cohort data must be used, not contemporaneous data. Secondly, if contemporaneous data is being used the mechanisms must be related to the deaths that are actually occurring currently. That is the deaths of the elderly, not causes of stress or death amongst the current work force.

4.8. Appendix 1: Data Sources

Cigarette consumption

The cigarette consumption data has been used in two ways

1. The current cigarette consumption for the population aged 15+

2. The cigarette consumption for the working life of a particular cohort. This is just the average for a person for the period that they were of working age (15-65)

General notes on the construction of the cigarette data:

- Cigarette consumption is the consumption of all tobacco products in grams per person (15+) per year.
- Most of the data was collected in aggregate tobacco consumption in tonne per nation per year. This was then converted to tonne per person by dividing by the population multiplied by the proportion population aged 15 and above. This was then multiplied by 1 million to get grams per adult aged 15 and above.
- If the data was recorded in pounds (often in the earlier years) this was converted to tonne based on a metric tonne equalling 2204.62262 pounds. Unless otherwise specified. Eg. USA 1 Ton = 2000 pounds.
- If tobacco consumption was recorded in older measures these are specified and converted to tonne also, i.e. France: milliers dequitaux = 100 pounds
  Switzerland: q = 100 pounds
  The hundredweight or centum weight (cwt) = 100 pounds
- If it was measured in number of cigarettes, it was converted to tonne based on the average cigarette containing 0.76 grams of tobacco
  The calculation is number of cigarettes multiplied by the grams per cigarette divided by 1 million.
- Where the data was recorded in monetary units the prices of tobacco per pound for that year were also collected and this was then used to convert the data into tonne. Where the prices of tobacco for each period were not available the CPI was used to convert tobacco into real prices and then into tonne.

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Unemployment

The aggregate unemployment rate is in percent is from: Madsen (2003)

The data that is appropriate here is the unemployment rate of a particular cohort averaged over their entire working life (15 - 65). A number of things need to be considered here. Firstly, younger people tend to be laid off work before older people in economic downturn. There is therefore a higher incidence of unemployment amongst the young relative to the middle ages. Secondly, between countries there are different proportions of the workforce in different age groups (i.e. some countries will have a younger workforce than others). To accurately calculate the average unemployment rate for a person of a particular age the following data was collected:

In the following subscript, *i*, denotes the different age groups which are the young 15-24, middle aged 25-54, and old 55-64. And subscript, *t*, denotes the year. The subscript, *c*, represents cohort.

The percentage of the workforce aged 15-24, 25-54, and 55-64 (e_{it}) extracted from the OECD website.

The incidence of unemployment (I_i) is the annual, harmonized unemployment rate from the labour force statistics at OECD.stat. It shows the average unemployment from 1995 to -2010

for persons in age groups 15-24, 25-54 and 55-64 for each year. Unfortunately the incidence of unemployment amongst different age groups has only been recorded since 1995. This is why the average over this period was used in the following first calculation.

The estimation of alpha is the incidence of unemployment for a particular cohort divided by the average total unemployment rate for that period.

$$\propto_i = \frac{I_{i,t=av(1995-2010)}}{U_{t=av(1995-2010)}} \quad (1)$$

The calculation for the estimated unemployment for each cohort, (U_{it}^e), each year is equal to the percentage of the workforce that is in that age group (e_{it}) multiplied by the ratio of the incidence of unemployment over average unemployment for that cohort (α_i) multiplied by the actual unemployment rate for each year (U_t).

$$U_{it}^e = e_{it}. \propto_i . U_t \quad (2)$$

The next step is to estimate the average unemployment rate for a person of a particular age cohort (65), for their entire working period, U_{ct}^e, for example a person aged 65 at time, *t*, would be calculated over the past 49 years. Since the data starts in 1870 the first estimated unemployment rate is for those aged 65 in the year 1919. The calculation for this is simply the sum of the average estimates for each year

$$U_{ct}^e = av.\ U_{y,t-49,40}^e + av.\ U_{y,t-39,11}^e + av.\ U_{y,t-10,1}^e \quad (3)$$

This then adjusted by multiplying with the ratio of the estimated cohort unemployment to the actual unemployment rate.

$$adjusted\ U_{ct}^e = U_{ct}^e * \beta\ where\ \beta = \frac{U}{U_{ct}^e} \quad (4)$$

Output

Is calculated as the average growth rate in output over the working life of a person aged 65 in attempt to capture the exposure to economic growth a person aged 65 has experienced.
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5. Conclusion

This thesis was inspired by an interest in the relationship between health and economic growth, and explores the mechanisms via which health can impact economic growth. The search to understand the processes via which health affects economic growth is complex. Like all macroeconomic fields of study there are multiple factors constantly interacting with each other which makes it difficult to estimate causal effects. Theoretically, the relationship is more straightforward, with the interaction between technological progress and human capital occupying a prominent place in recent growth literature. The part health has to play in human capital is becoming increasingly recognized.

Different theories of economic growth consider technological change, human capital accumulation and the savings rate to be either exogenous or endogenous depending on the model. This thesis assumes an endogenous accumulation of human capital that derives from better health as well as education. In chapter one, the outcome variable is investment and the main input variable is life expectancy as a proxy for health. In chapters two and three, the outcome variable is health so only the inputs pertaining to the production of good health are considered. Further to this, due the many factors contributing to good health, only particular inputs into health production are considered at any one time.

The empirical approach in chapter two estimated the effects of health on investment using Cobb Douglas aggregate production and considers good health and education as the main components of human capital accumulation. Consumption and savings are both dependent on health, education, the real wage and the interest rate (discount factor). Unlike traditional neo-classical growth models the inclusion of the function explaining the evolution of human capital allows the model to be partially endogenous. Theoretically this is not an uncommon assumption. The data underpinning the analysis in chapter one is unique; the data set extends over a far longer time period than most studies, and looks at the relationship between investment and life expectancy at age 10 (rather than at birth as in most studies). The analysis in chapter three is unique due to the introduction of previously unused instruments for public and private health expenditures. The empirical method is also new in that private and public health expenditures have been considered separately as in Leu (1986) and Asiskovitch (2010). Additionally, endogeneity issues have been addressed as in Pocas and Soukiazis (2010). The contribution from the data in chapter four is two-fold. Firstly, instead

of using contemporaneous data, this chapter presents the construction and use of cohort variables. These cohort variables make more sense (than contemporaneous data) when analysing the impact unemployment has had on mortality rates. This is because they take into account the impact overall unemployment has on life expectancy and health, rather than just the impact contemporaneous unemployment has had. As far as I know from this literature, this is unique. Secondly, a long panel of tobacco consumption data across 21 countries has been collected.

Chapter two focuses on how health may contribute to growth through investment. This chapter reports the significant and positive relationships found between life expectancy at age 10 and investments, both in tertiary enrolments and physical capital. The results show strong negative relationships between investment and life expectancy at birth, this negative relationship stemming from the deferment of investment decisions until children have survived infancy. As soon as the child reaches an age where survival to old age is likely, there is a greater incentive to educate them. Due to the strong correlation between life expectancy at age 10 and life expectancy at older ages, this also indicates that once people expect to live longer they will save more. And that investing in education will only be worthwhile if life expectancies are great enough to realize the payoffs to education, and therefore warrant participation. This implies health should be a fundamental component in any policy aimed at improving educational and investment levels in developing countries.

Chapter three reports inconclusive evidence as to whether public health expenditures or private health expenditures produce better health outcomes. Life expectancy at birth is found to be positively related to public health expenditures and both positively and negatively related to private expenditures, depending on the model specification. Infant mortality rates have been found to be negatively and positively related to public health expenditures (depending on model specification) whereas private health expenditures are always negative. This is to be expected due to the complexity of healthcare systems and the need for qualitative information as well as a longer time series of financial data. Longer data time series are necessary to account for the foetal and maternal health of those dying today, which is the basis of life expectancy calculations. The qualitative data are needed as it is the delivery as well as the funding that produces good health. There is evidence, based on the empirical results, that private health care systems are not superior for health outcomes.

However, the results are not strong enough to provide any conclusive recommendation for healthcare reform policy.

Chapter four opposes recent findings in the main literature that unemployment is good for health and finds that recessions are not good for health[3]. This chapter contends that short run down turns in economic activity (e.g. recessions) are associated with increased mortality rates. What has been established is that there is a relationship with cohort unemployment rates and current mortality rates (as a proxy for health). Cohort unemployment rates are significantly and positively related to current mortality rates while cohort income is negatively and significantly related. These results are consistent across different model specifications and indicate that current mortality is positively related to cohort unemployment rates. In other words, recessions are not good for health as Ruhm (2000) claims. Governments recognise the importance of short term employment fluctuations and the importance of public health, but often fail to link the two in policy discussions. Moving forward, policy makers need to recognize that recessions are bad for health. If health is related to business cycles it is important for government to focus on how economic fluctuations are affecting people's health and try to alleviate the sufferance through, for example, countercyclical fiscal policies.

[3] Ruhm (2000), Granados (2005), Gerdtham and Ruhm (2006), Ruhm (2007), Economou et al. (2008), Miller et al. (2009), Gonazalea and Quast (2011), Douglas et al. (2008), and Stevens et al. (2012).

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