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PhD Monograph

A Dual-Phase Health Capital Model and Its Application to Health Co-benefit Modelling of Decarbonisation

Yifeng (Philip) Chen

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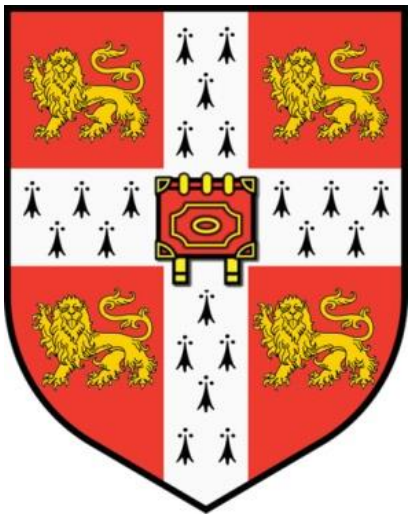

Cambridge Centre for Climate Change Mitigation Research (4CMR),

Department of Land Economy, University of Cambridge

Email: yc318@cam.ac.uk

This dissertation is submitted for the degree of Doctor of Philosophy

For Mo Jia

Declaration of integrity

This dissertation is the result of my own work and includes nothing which is the outcome of work done in collaboration except as declared in the Preface and specified in the text. It is not substantially the same as any that I have submitted, or, is being concurrently submitted for a degree or diploma or other qualification at the University of Cambridge or any other University or similar institution except as declared in the Preface and specified in the text. I further state that no substantial part of my dissertation has already been submitted, or, is being concurrently submitted for any such degree, diploma or other qualification at the University of Cambridge or any other University of similar institution except as declared in the Preface and specified in the text. It does not exceed the prescribed word limit for the relevant Degree Committee. In total, the thesis is approximately 62,000 words, and therefore does not exceed the regulation length, including footnotes, references and appendices.

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Table of Contents

List of Tables

List of Figures

Nomanclature

British Household Panel Survey (BHPS)

Carbon Dioxide (CO_2)

Consumer Price Index (CPI)

Disability Adjusted Life Years (DALY)

Greenhouse gases (GHG)

General Certificate of Secondary Education (GCSE)

Global Burden of Disease (GBD)

Gross Domestic Product (GDP)

Gross National Income (GNI)

Gross Value Added (GVA)

Intergovernmental Panel for Climate Change (IPCC)

Parts per million (ppm)

Particulate Matter (PM)

Particulate Matter less than 2.5 micrometer in diameter ($PM_{2.5}$)

Particulate Matter less than 10 micrometer in diameter (PM_{10})

Quality Adjusted Life Years (QALY)

Relative Risk (RR)

Sulphur dioxide (SO_2)

Two-staged-least-squares (2SLS)

United States Dollar (USD)

Years Lived with Disability (YLD)

Years of Life Loss (YLL)

Summary

This thesis is developed in the context of investigating the health co-benefit of decarbonisation. Health co-benefit refers to the collateral benefit which arises from decarbonisation policies external to the main intended benefit of climate change mitigation via the reduction of Greenhouse Gases (GHG). Health co-benefit of this kind often arises via the corresponding reduction in air pollutants when GHG is reduced. This is because GHG and air pollutants such as particulate matter are often derived from the same source – the combustion of fossil fuels which drive economic activities. Existing literature in the health co-benefit of decarbonisation fail to give consider the effect of socio-economic variables such as income and education on the expected health co-benefits, and this is where the thesis begins.

The backdrop of health co-benefit modelling and the need to incorporate socioeconomic considerations provide the impetus to develop a health economics model. However, in many ways this health economic model deviates from the health co-benefit studies methodologically and instead follows the tradition of the Health Capital Model developed by Grossman (1972). This is due to the micro-economic nature of this health economic model which employs standard economic theory and technique of optimisation, which differs from the fundamentally empirically driven approach of health co-benefit studies. The health economic model developed here is an opportunity to address some of the short-comings of the Health Capital Model. The health co-benefit background however provides some concrete context and inspiration for the application of the theoretical insights which can be drawn from this model.

The main contribution of the model develop in this thesis from the theoretical point of view lies in the division of the lifecycle analysis of health into two distinct but related phases of childhood and adulthood. The two phases are specified with different assumptions reflecting the differing characteristics of childhood and adulthood. The most important distinction between the two phases is the manner in which investment in health capital (using time and goods resources) enters the modelling framework. In the childhood phase, health investment augments or increases the existing stock of health capital, while during the adulthood phase health investment prevents the decline of health but does not increase its stock. I believe this better reflects the biological behaviour of health over one's life than the HCM which implicitly assumes that new stock of health and existing stock are perfectly substitutable. In my model,

this substitutability is possible only during the childhood corresponding with the body and mental development. On the other hand, during adulthood when them body no longer grows, health investment may only preserve health.

After developing the model, I went about to test it empirically. I used the Understanding Society youth questionnaire to test the child model and the British Household Panel Survey (BHPS) to test the adulthood model. Due to the way that optimisation problem was specified, the terminal end time conditional in the optimal control model became another endogenous variable. This variable is treated empirically as the life expectancy at the national level. I find that in general the empirical data strongly supports the theoretical propositions of my model. It should be noted here that since the main contribution of this thesis is in theoretical development, the empirical efforts were designed primarily with the intention of validating the propositions of the model, and not really for direct policy application. This is also reinforced by the use of ordered logit models where the coefficients of the independent variables on the dependent variable generally have no meaning, where we only concentrate on the signs of the relationship.

Having successfully developed the model, it is applied in two policy settings. Firstly, through reformulation of the model gives the inclusion of socio-economic variables in the measure of Relative Risk (RR) a theoretical grounding. We utilised the Global Burden of Disease (GBD) data to compute RR across 180 countries in the world and regressed with World Bank data on ambient particulate matter pollution as well as GDP per capita. The former variable represents the exogenous rate of depreciation while the latter socio-economic variables, particularly income. I find that the RR is negatively associated with the GDP per capita at the national level. Using the estimated coefficients with the help of Professor Crawford-Brown we attempted to forecast how GDP per capita will interact with the health co-benefits of decarbonisation under a range of future scenarios.

The second application of the model is in its use to predict the inequality implications of decarbonisation policy. This is performed by taking the second order partial derivative of an endogenous variable such as health, as will be described in detail later. This approach is sufficiently flexible to accommodate the prediction of inequality over range of policies and variables. The inequality implications and predictions according to this model are not tested empirically here. However, they are perhaps the most fruitful area for future research.

CHAPTER 1 – Introduction

The Intergovernmental Panel for Climate Change (IPCC) predicted that if global emissions of Greenhouse gases (GHG) continue to grow at the current rate, the earth will experience considerable global warming by 2100, perhaps up to 5°C.[1] This represents approximately 50% increase compared to the pre-industrial Carbon Dioxide (CO_2) atmospheric concentrations (Tans and Keeling, 2014) which poses significant risk to the environment, society and development. With the poorest nations likely to bear the majority of climate change associated dangers due to being located in already warmer climates and having less financial resources to adapt, the situation could be detrimental to global equality, reversing any development progress made to-date and put future goals in jeopardy. In light of this, it has been suggested that global emissions should be restricted so that the probability of exceeding 2°C may be capped below 50%. For this to be achievable, it is necessary to stabilize the atmospheric concentration of CO_2 at 450-550 parts per million (ppm). This is an ambitious decarbonisation strategy and it appears that it is unlikely to be achievable. Nonetheless it would be prudent to reduce the growth of GHG emissions wherever possible within the priorities of satisfying social and economic development goals. An effective argument to propel the reduction in GHG emission consistent with social and development is the generation of health co-benefits[2] due to decarbonisation. The most prevalent anthropogenic source of GHG emission is the combustion of fossil fuels and this chemical process also releases various kinds of air pollutants detriment to human health. It should be noted that whilst GHG contributes to climate change, they are generally not considered harmful gases to health. By contrast most air pollutants most harmful to human health are often neutral in their effect on the climate or at least are not major causes of climate change.

Although this proposition of improving public health or reducing health hazards via decarbonisation is founded on a legitimate and plausible rationale, there is a crucial limitation which is inadequately addressed in current studies of health co-benefits from decarbonisation. Existing studies on this subject have not accounted for the interaction of socio-economic variables with either the environment or decarbonisation policies. For a given unit of ambient

[1] The IPCC Fifth Assessment Report that under the worst case scenario of RCP8.5, global mean surface temperature by the end of the 21st century (2081-2100) is expected to be 4.8°C higher relative to 1986-2005. https://www.ipcc.ch/pdf/assessment-report/ar5/syr/AR5_SYR_FINAL_SPM.pdf

[2] Co-benefits refer to the collateral benefit of a policy external to the main intended benefit.

air pollution exposure, richer individuals are likely to experience lesser impact than poorer individuals due to a number of reasons. First, richer individuals can afford better nutritional intake and/or possess the means to engage in healthier lifestyle resulting in stronger resilience to the harmful effects of air pollution. Secondly, richer individuals can purchase more protective measures against air pollution, for example the installation of air filtering systems at homes and travel to work in private transport instead of walking or cycling, leaving them less exposed to the direct effects of air pollution. Thirdly, richer individuals are generally involved in occupations which are relatively sheltered from air pollution. These differences caused by individual variations are often reflected at the national level between countries of different economic development. More developed countries have higher calorie intake per capita (FAO, 2014),[3] invest more in health infrastructure (WHO, 2010), and their economic systems are less reliant on environmentally polluting industries such as heavy manufacturing and instead more on 'clean' activities such as services. Given it is expected that most if not all countries, especially developing countries, to grow substantially in economic terms during the modelling period from 2010 to 2050, a period in which not only national incomes but other socio-economic variables such as education are likely to improve, it can be expected that the health co-benefit resulting from decarbonisation policies will be reduced generally speaking.

The thesis therefore seeks to incorporate the role of socio-economic variables into the modelling of health co-benefit from decarbonisation. In this process, I take the opportunity to construct a health economics model through the modification of Grossman (1972)'s 'health capital model'. Grossman (1972) and subsequent authors who built upon his work utilised the economic methodology of dynamic optimisation, treating health as a form of capital, which in economic terms possesses the following three characteristics:

1. Durable stock generating a flow of services or benefits per unit of time.

2. Possesses a long or even infinite lifespan (at least theoretically).

3. Subject to depreciation but can be increased through investment.

The above features of capital are typically reflected in physical capital such as plants or machinery, where such a framework was first applied by economists. Later however, such a

[3] Although for developed countries, the relevant concerns are now with the 'quality' of calorie intake, or balanced nutrition, rather than the actual physical energy intake.

conceptual framework became increasingly applied to more non-tangible assets such as financial capital. Becker (1962) employed this concept to develop a model for 'human capital', which reflected individual lifetime decisions in choosing the stock of education or knowledge capital to acquire. According to Becker (1962), although investment in human capital or education require substantial time and monetary resources, it would increase the individual's employment prospects and income later in life, which can be regarded as a flow of services or benefits derived from a unit (increase) in human capital. Upon such a model, Grossman (1972) argued that health should be a component of human capital along with education and thereby developed the first health capital model. Just like education, the investment in health would require time and money, but better health reflected by a higher stock of health capital would reduce sick days per unit of time, thus acting as the flow of benefit or services. The reduction in sick days may be employed to work, earning higher monetary returns or utilised as leisure time, increasing the individual's utility.

Viewing health as a form of capital certainly possesses many advantages, among which include the convenience of economic modelling. Nonetheless since this concept was borrowed from the analysis of physical capital which are lifeless objects, the analogy drawn to human health 'capital' which is a form of biological organism no doubt possesses certain limitations. It is the view of the author that some analogy when applied to health have been taken to extremes by the current literature in health capital models of which Grossman (1972) is the pioneer. For example, the third feature of capital as mentioned above when formulated into economic models almost always imply that a given unit of capital depreciation can in all instances be recovered fully by sufficient investment. Whilst this may be the case for many physical capital or financial capital where the substitutability of newly added stock with existing stock is high, the same cannot be said for health capital. Certain damage to health such as serious injuries leading to disability for example, can never be restored fully.

The thesis takes this opportunity to develop a health capital model following the tradition of Grossman (1972), addressing some of the inadequacies of applying capital theory to the modelling of health which are prevalent in the existing literature. This health capital model is then used as the theoretical basis in an epidemiological setting to include socio-economic variables in the computation of Relative Risk (RR) of air pollution. The RR is a common metric

in epidemiology employed to denote the degree of public health hazard from a given source.[4] The coefficient of socio-economic variables on the RR can be estimated empirically to determine quantitative relationships which can then be used to augment estimates of health co-benefit from decarbonisation, accounting for the effect of likely economic development during the modelling period. In order to conduct such an empirical exercise, I use compute RR values using Global Burden of Disease (GBD, 2010, 2013) data. Data for the socio-economic variables come from the World Bank database (World Bank, 2010).

The health capital model developed in this thesis also possesses the feature of predicting theoretically how the health co-benefit of decarbonisation policies would vary along socio-economic lines and thus whether such policies are likely to be equitable or otherwise. In fact, the model is sufficiently flexible to predict theoretically how any policy associated with the exogenous variables specified in the model would a) impact on health and b) how such impact would vary along the lines of other exogenous variables many of which can be considered socio-economic variables and thus derive (in)equality implications. The (in)equality implications however will not be tested empirically since they are not the core focus of the thesis. They are however areas fruitful for future research. Since health is not the only endogenous variable in the health capital model developed, the same procedure for predicting effects of a policy, and how that policy will vary along socio-economic lines can be applied to other endogenous variables, such as education. The predictions generated would also be suitable for future empirical research.

The model developed as well as its application to the modelling of health co-benefit from decarbonisation raises some interesting policy implications. If economic development of countries ceteris paribus reduces the expected health co-benefit, then it raises the question of how much and whether there is any net health improvement at all. If a government pursues decarbonisation at the expense of forgoing economic development and/or reducing investment in healthcare for example, even though there may be direct observable health improvement, it will be reduced by forgoing the opportunity to improve health using economic means. In extreme cases the net health co-benefit from decarbonisation can even be negative. This finding would also suggest that if economic development remains unchanged, poorer countries should have greater incentives to invest in decarbonisation as well as other policies which reduce air

[4] I compare the incidents of air pollution related mortality with the background incidents.

pollution. This would be exactly what is required in order to meet the IPCC's decarbonisation targets since non-Annex I, which are developing countries will be the source of the bulk of emission increase. However, low income countries would seek to improve health not via decarbonisation particularly if such measures are costly and/or if they lack the necessary technological capabilities, but instead improve health via economic growth. The strategy of 'pollute first clean up later' whereby countries focus on economic development first resulting in pollution, before later engaging in environmental amelioration after sufficient level of economic development is reached, would seem very attractive and may be difficult to avoid for most developing countries. Furthermore, the 'clean up later' part will perhaps be delayed for a long time or never occur. This is because as countries develop economically moving from low to high income, the incentives to decarbonise or reduce pollution actually decrease progressively from the perspective of the expected health co-benefits generated. Instead, countries may easily become locked into a pattern of striving for perpetual growth to improve health and to further increase living standards, neglecting environmental amelioration. The policy section of this thesis in Chapter 6 discusses further some of the dilemma faced by countries seeking on the one hand to reduce air pollution and to develop economically on the other hand.

The remaining sections of this thesis are as follows. In Chapter 2 the relevant literature regarding the modelling of health co-benefits and health capital models are presented. The underlying theoretical framework of this thesis, which is the health capital model developed is laid out in Chapter 3 with an Appendix showing in detail the procedures of the model derivation. Chapter 3 also formally lists a number of testable hypotheses which are the product of the mathematical model solutions derived under the first order optimality conditions. In Chapter 4 the empirical tests of the main hypotheses presented in Chapter 3 which are relevant (not all the hypotheses are tested empirically) are shown, and how well the results fit with the hypotheses are discussed. In Chapter 5 a methodology is outlined for applying the health capital model developed in Chapter 3 to the context of health co-benefit modelling. As mentioned earlier, Chapter 6 discusses the policy implications of the thesis' findings. Chapter 7, the final chapter concludes this thesis, summarising all the analyses presented, discusses the limitations of this piece of work and how future research may build upon them.

CHAPTER 2 – Literature Review

The thesis attempts to develop a health economics model for the purpose of incorporating socio-economic variables into the health co-benefit modelling framework. As such it is necessary to be aware of two strands of literature, one regarding the modelling of health co-benefit via decarbonisation strategies, and the other in relation to health economics models. The health co-benefit literature is described in section *2.1*. The literature regarding health economic models can be divided into those which analyse health at the microeconomic (individual) level, and those at the macroeconomic (aggregate) level. Microeconomic health models are described in section *2.2* while macroeconomic health models are shown in section *2.3*. Section *2.4* discusses the knowledge gap in the literature and how this study can contribute to the existing field of knowledge.

2.1. Studies on Health Co-benefit of Decarbonisation Policies

Policies which reduce greenhouse gases (GHG) have the potential to simultaneously offset the emission of various air pollutants including particulate matter (PM), sulphur dioxide (SO_2) and ozone. The combustion of fossil fuel is a major source of both GHG and air pollutants of anthropogenic origin. It has therefore been argued that decarbonisation strategies have the potential to generate significant health improvements, often known as health co-benefits, via the co-reduction in these air pollutants (Smith and Haigler, 2008; Younger et al., 2008; Bollen et al., 2009; Anenberg et al., 2012). PM has been identified by medical evidence as perhaps the most hazardous form of air pollutant to public health, contributing substantially to cardiovascular and respiratory diseases (Pope et al., 2002; Pope et al., 2004; Riediker et al., 2004). PM refers to any small solid or liquid particles suspended in the atmosphere as aerosols (USEPA, 2004). PM_{10} refers to PM of 10 micrometer in diameter or less. As this size is the critical level at which penetration into the respiratory system becomes likely, PM_{10} is responsible for the majority of PM related health effects. A sub-category of PM_{10}, $PM_{2.5}$ refers to fine particles of 2.5 micrometer in diameter or less. $PM_{2.5}$ is believed to be twice as hazardous as PM_{10} per unit of exposure (WHO, 2006). Coarse particles[5] originate naturally yet finer

[5] PM with diameter exceeding 10 micrometer.

particles[6] are primarily the result of combustion processes in urban environments, which are also the main sources of anthropogenic GHG. Existing studies of health co-benefits do not give sufficient attention to the potential influence of socio-economic variables. In particular, the impact on health co-benefit estimates and their distributional implications due to socio-economic variations remain unexplored. This thesis provides a framework for incorporating socio-economic factors into health co-benefit modelling.

The impact pathway analysis provided a logical framework to model health co-benefits from reduced air pollution (Curtiss and Rabl, 1996). The procedure included the following four steps:

1. Characterisation of the relevant technologies and the environmental burdens they impose;
2. Calculation of increased pollutant concentration in all affected regions;
3. Estimation of physical impacts using an exposure-response function;
4. The economic valuation of these impacts (an optional step).

The following studies utilised the above framework, converting cases of mortality and morbidity into monetary values. Burtraw et al. (2003) demonstrated that by applying a tax rate of $25 per metric ton of carbon to the US power sector, the resulting decrease in pollutant emissions leads to health co-benefits of $12-14 per metric ton of carbon mitigated. The marginal benefits appeared to be constant and independent of the tax rate. Although the marginal cost of the carbon tax outweighed the benefits, there may be net benefits if reduction in climate change risks are also factored in. Moreover, revenue from the carbon tax can be re-distributed for welfare enhancing purposes hence should not be regarded solely as a societal cost.

Aunan et al., (2004) investigated six carbon abatement options in Shanxi province, China, concluding that sizable co-benefits prevail, with the elderly being the main beneficiary age group.[7] The abatement strategy options included ‘co-generation’, ‘modified boiler design’, ‘boiler replacement’, ‘improved boiler management’, ‘coal washing’ and ‘briquetting’. With the exception of ‘improved boiler management’ in the scenario of using low economic valuation of health, there existed substantial net benefits. Even without considering the health

[6] PM_{10} and $PM_{2.5}$

[7] The elderly as a group are more vulnerable to ambient pollution.

co-benefits, some measures, such as co-generation, would produce net economic benefit solely due to their improvement in energy efficiency (London et al., 1998; Aarhus et al., 1999).

Nemet et al. (2010) surveyed previous health co-benefit studies and found that the marginal benefits ranged between $2 and $196 per ton of CO_2 mitigated, with a mean of $49. The values include both mortality and morbidity reductions and are similar to marginal abatement costs. However, Nemet al., (2010) argued that these estimates were rarely incorporated into integrated assessments of climate policy when considering their costs and benefits. Li and Crawford-Brown (2011) estimated that a PM related vehicle inspection and maintenance programme in Thailand, which reduced CO_2 emissions by 0.4 tons per person per year, could generate substantial net health and economic benefits. The net annual cost of the programme was estimated at 147 million USD yet the health co-benefits in terms of the avoided mortality and morbidity may have reached over ten times this amount by 2015. The mortality's economic benefit is measured by the Value of Statistical Life (VSL) while the morbidity by the Cost of Illness (COI) method. The COI includes the treatment cost and economic loss due to recuperation time and is an underestimate since the disutility of illness is not included in the study. Crawford-Brown et al., (2012)'s results showed that a policy, which reduced GHG emission in Mexico by 77% relative to the baseline level, would decrease annual mortality and morbidity by 3,000 and 417,000 cases, respectively. The economic equivalent of avoided annual morbidity would be $0.6 billion per year, measured by the cost of illness method.

Crawford-Brown et al. (2013) modelled the health co-benefit of a 100% global reduction in GHG emission, uniformly implemented across all countries. Various scenarios as well as differential decarbonisation targets for Annex I and II countries[8] could also be modelled easily under their framework. Decarbonisation would be achieved via pure reduction in usage (demand management) and not from technical substitution, as was the main approach in other studies.[9] Analysis of health co-benefit from demand management compared to technical substitution possesses an important advantage of being applicable across a larger geographical

[8] Annex I and Annex II countries are designated by the United Nations where the former group of countries take on specific decarbonisation targets. The latter group is exempt from rigid targets as they tend to be developing countries which lack the resources and technology.

[9] The other studies considered how technical substitution, i.e. replacing current GHG emitting technology with more efficient technologies with less GHG emissions, would result in health improvement. Crawford-Brown et al., (2013) considered how reduction in demand or energy usage, with current technology can result in health improvement.

area. This is because the potential for a policy measure involving technical substitution are highly specific to certain geographical regions, such as a city, and to certain industries and sectors, such as the power or transport sector, requiring substantial amount of considerations for the unique local situation or characteristics, and as such would not be suitable in general for application across a nation, given the large non-uniformities which usually exist. Demand management policy recommendations and scenarios on the other hand, are generally applicable across all sectors, households, firms, and across larger geographical areas, such as at the national and international level. Since decarbonisation policies, targets and agreements are frequently set at the national and international levels, it is vital that the modelling results, policy recommendations and scenarios can be used at this level.

Health co-benefits of decarbonisation can arise through channels other than reduction in ambient air pollution. The articles from the series on climate change and health published on the Lancet examined the potential for decarbonisation strategies to improve health in four areas: household energy, urban land transport, low carbon electricity generation and agriculture (Markandya et al., 2009; Friel et al., 2009; Woodcock et al., 2009; Wilkinson et al., 2009; Haines et al., 2010). Greater use of public transport for example may reduce risk of accidents, while active transport would foster more physical activity beneficial for bodily circulation, hence reducing rates of obesity, high blood pressure and heart attacks. The results of the Lancet series supported the general finding that health improvement potentials would be larger in developing compared to developed countries. In many developing countries, indoor rather than outdoor air pollution is the main health threat, which arises from biomass cooking fuel. Certain decarbonisation policies may therefore result in negative health co-benefit if they encourage greater use of biomass in substitution for electrical appliances, even though the former emits lower levels of GHG. This may be the case especially where energy efficiency results in reduced air exchange rates in homes.

2.2. Health Economic Models at the Microeconomic Leve

Microeconomic models of health analyse how individuals make decisions concerning their own health and the exogenous factors which influence their decisions. Grossman (1972) pioneered the use of formal economic models which explained socio-economic determinants of health, known as the 'demand' for health. Lifecycle analysis of health capital was formulated as a dynamic optimisation problem, in which individuals selected the optimal quantity of health investment to undertake at any point in time or age t, using medical care and time as inputs, in order to maximize an inter-temporal utility function, over a time horizon $[0, T]$, where T

denotes the length of life, or the time from birth to death.[10] Health investment increased the stock of health capital, which in turn increased the flow of healthy time, directly raising utility.[11] In addition, consumption opportunities would be expanded since the extra healthy time can be employed in either the market sector to earn income or the non-market sector to further increase health investment and/or consumption. The inputs required for health investment, however, can alternatively be used for consumption; hence health investment would directly compete with consumption for scarce resources.

The solution to the above optimal control problem involved the identification of the optimal time path of health investment, through which the equilibrium time profile of health capital can be inferred. The comparative static analysis modelled the effect of variations in income, education and age on the equilibrium stock of health capital to derive theoretical and empirical predictions of the determinants of health.[12] The results predict that the optimal stock of health capital is positively associated with income and education, but negatively linked to age. Aside from these three variables, Grossman (1972) also analysed the effect of a change in the rate of health depreciation. An increase in the rate of health depreciation lowers the optimal stock of health capital. Health depreciation also played an important function in that it was assumed to be a positive function of age beyond some stage in lifecycle hence increasing age would eventually result in an optimal stock of health which is below the stock necessary to sustain life, leading to death or termination of life.

Grossman (1972) presented two variants of his model with restrictive assumptions. The first was the 'pure investment' model in which the marginal utility of healthy time was assumed to be zero, or that the stock of health capital and healthy time did not enter into the utility function at all. This implied that the benefits of good health would not be enjoyed in any way by the individual and that health capital like other forms of capital should be viewed entirely as a

[10] The intertemporal utility function was of the form $\int_0^T U\big(C(t), h(H(t))\big)dt$, where $C(t)$ and $h(H(t))$ were consumption and the flow of healthy time at the instant $t \in [0, T]$, respectively, with the later being a positively monotonic function of the stock of health capital at t, $H(t)$. $U(C(t), h(H(t))$ was assumed to be continuously differentiable and concave in its arguments. An additional assumption of diminishing returns to health capital was also imposed such that $h''(H) < 0 \; \forall \; H$. Grossman's original model was in discrete time, though he presented a continuous time variant in the appendix. Subsequent models mostly adopted the continuous time analysis.

[11] The flow of healthy time was an argument in the inter-temporal utility function.

[12] i.e. how the equilibrium stock of health capital any point in time or age t is altered by changes in the three exogenous variables – income, education and age.

commodity to enhance monetary return or its equivalent. The monetary returns would be generated by a reduction in sickness time or a correspondingly equivalent increase in healthy time as a result of investing in health capital, which could be devoted to work thus increasing incomes.[13] The second variant was the opposite extreme known as a 'pure consumption' model in which health capital yielded only utility.[14] The pure investment model provided clear and empirically testable predictions and therefore had been the focus of empirical studies (Wagstaff, 1986, 1993; Nocera and Zweifel, 1998). The mix model whereby health capital would be demanded for both investment and consumption reasons was avoided by Grossman (1972) in order to avoid model complications, even though this would have clearly been more realistic.

The work of Grossman (1972) was considered a major breakthrough in the field of health economics. Rather than studying the market for healthcare and health insurance, the focus of health economics shifted towards the study of factors which caused people to 'demand' more or less health, i.e. engage in health promoting or damaging behaviour. Healthcare and health insurance were relegated to the realm of derived demand, seen as a means to the end of good health and driven fundamentally by an individual's desire to achieve that end. This was considered as an improvement in health analysis since the standalone analysis for healthcare and health insurance were not very meaningful as these goods and services do not contribute to consumer utility directly.[15]

Becker (1962) and Ben-Porath (1967), examined the lifecycle variation in human capital, which consisted only of educational or knowledge capital. Grossman (1972) employed this framework and methodology to assess lifecycle variations in health capital, which he also argued should constitute an important component in human capital along with educational or knowledge capital. The original Grossman (1972) model suffered from several limitations, which subsequent models attempted to address. Firstly, Grossman (1972)'s adoption of

[13] It was assumed that productivity and wages were independent of the stock of health and the flow of healthy time, but more healthy time would become available for working as a result of a higher stock of health capital. This was differentiated from the human capital model of Becker (1962) and Ben-Porath (1967) which assumed that human capital, mostly educational and knowledge capital, would increase the wage rate but not the time available for work.

[14] The 'pure consumption' model was not published in Grossman's 1972 seminal paper but rather in his monograph book titled 'The Demand for Health: A Theoretical and Empirical Investigation'.

[15] In fact most likely they cause disutility hence without the motivation of improving health, they would not be demanded at all.

household production theory to the non-market sector lacked sufficient theoretical and empirical justification. It was reasoned that since more educated individuals were generally more efficient converters of factor inputs, most notably of time, into monetary returns,[16] the same relationship would prevail in the non-market sector in like manner. Specifically, Grossman (1972) assumed that more educated individuals produced greater units of health investment and consumption, for any given level of input.[17] Whilst the positive relationship between health and education has been widely observed (Winkleby et al., 1992; Cutler and Muney, 2006; Conti, et al., 2010), there was little evidence that education operated as an efficiency parameter affecting general production relationships for health investment. The assumption that education affected the efficiency of consumption inputs required even greater justification both theoretically and empirically.[18] Muurinen (1982) modified Grossman's (1972) model by letting education affect the rate of health capital depreciation rather than health investment. Muurinen (1982) argued that the rate of health depreciation should be a function of a vector of general environmental variables[19] of which the level of education was an element,[20] since it would likely affect life decisions and behaviour such as alcohol and cigarette consumption, which in turn would affect the rate of health depreciation. This formulation can be employed to include environmental variables such as exposure to ambient pollution in the analysis to health, which can be included in the vector of general environmental variables. Muurinen (1982) also included financial capital or wealth as an additional state variable in the optimization problem, thus more accurately reflecting the (opportunity) cost of health investment in terms of foregoing financial investment. This approach was adopted by

[16] The more educated have higher wages.

[17] The inputs were time and goods. The consumption and health investment functions were given by $C(X, \tau_C; E)$ and $I(M, \tau_I; E)$, respectively, in general form, where X, M, τ_C, τ_I and E were the quantities of consumer goods, medical goods or services, time devoted to consumption (may be interpreted as leisure time), time devoted to producing health, and education, respectively. Since $\frac{\partial C}{\partial E}$ and $\frac{\partial I}{\partial E} > 0$, more educated individuals were more efficient converters of time and goods into units of consumption and health.

[18] This assumption essentially implied that for a given unit of time or goods, a more educated individual generates higher level of 'consumption', or happiness. Not only was this implication difficult to justify, sometimes anecdotal evidence would suggest otherwise, with those uneducated knowing how to spend their time and whatever they have to achieve better satisfaction and happiness in life.

[19] By 'Environment' Muurinen (1982) was not explicitly referring to the atmospheric or natural environment, but the set of environmental conditions which encompass a particular person, such as his or her social, economic, familial environment and other surrounding elements.

[20] Though not the only element.

subsequent studies (Ehrlich and Chuma, 1990; Johansson and Löfgren, 1995; Galama and Van Kippersluis, 2010; Galama and Kapteyn, 2011).

Secondly, health capital was assumed to always reside upon its optimal time path. Any discrepancy between the initial and optimal stock can be eliminated instantaneously, due to the assumptions of constant returns to scale in the health investment function and zero adjustment costs. This formulation of the dynamic optimization problem according to Ehrlich and Chuma (1990) would result in a 'bang-bang' solution with infinite health investment in the initial period following the discrepancy between actual and optimal stock, followed by zero net investments in subsequent periods, once equilibrium is reached. Furthermore, there may not even exist an equilibrium quantity of health investment since both the marginal benefit and cost were independent of the quantity of health investment. Ehrlich and Chuma (1990) reformulated the Grossman (1972) model using optimal control theory in continuous time. Health investment was assumed to exhibit diminishing marginal productivity denoted by rising marginal costs for generating an additional unit of health investment, thus guaranteeing the existence of an optimal quantity of health investment. Grossman (2000) responded to Ehrlich and Chuma (1990), claiming that under the assumptions of his original 1972 model, the marginal benefit of health investment declined rather than remained constant, as was alleged by Ehrlich and Chuma (1990). This was because although I_t, the health investment at any instant did not affect H_t, the stock of health capital at that instant,[21] all future health stocks would be increased as a result of that investment decision ($\frac{\partial H_s}{\partial I_t} > 0, \forall s > t$). Since health capital was assumed to possess diminishing marginal product,[22] the marginal benefit of health investment should decline.

Given that health capital cannot be sold on an open market, health investment must be nonnegative. The original Grossman (1972) model was therefore not able to process corner solutions, thereby restricting health investment to be strictly positive. [23] This effectively excluded individuals who possessed health stocks above the optimal level, for which the

[21] In Grossman's original model, the equation of motion for health capital was given in discrete time by $\Delta H_t = H_{t+1} - H_t = I_t + \delta_t H_t$. Therefore, a one period lag is introduced between health investment and the resulting change in health capital.

[22] Further increase in the stock of health capital would generate progressively less flow of healthy time per unit of time.

[23] Although health investment was required to be nonnegative rather than strictly positive, a value of zero (the corner solution) would lead to indeterminacy of the stock of health capital.

optimal health investment should be zero for at least some stages of life.[24] The nonnegative constraint placed upon health investment also implied that optimal health could not be reached immediately. Muurinen (1982) analysed and contrasted the situation between $I_t > 0$ and $I_t = 0$, using the 'complementary slackness' condition. In the former case, the optimality condition held just as in Grossman's (1972) original model. In the latter, the marginal benefit of health investment was strictly below the marginal cost, resulting in zero health investment. Galama and Kapteyn (2011) expanded upon this feature, arguing for a 'missing threshold', which would separate persons with $I_t > 0$ and $I_t = 0$ into two groups. They argued that Grossman's (1972) model explained only persons who consume medical care ($I_t > 0$). Since most samples employed in empirical studies consist mainly of healthy individuals characterized by $I_t = 0$, they fall out of Grossman (1972)'s explanation, thus leading to results which contradict Grossman (1972)'s predictions.

Thirdly, the terminal time T in the original model was criticised for not being truly endogenous (Ehrlich and Chuma, 1990; Ried, 1996, 1998). If T the length of life was not an endogenous process but exogenously given, it would be independent of the stock of health capital and health investment for all t. Health capital therefore affected only the quality of life and not the length of life,[25] since Grossman (1972) did not develop the necessary 'transversality condition'[26] to determine the optimal length of life. Ried (1996, 1998) employed complex discrete control theory to develop this transversality condition. On the other hand, Ehrlich and Chuma (1990) through structuring the optimization problem in continuous time, utilised a relatively simple transversality condition – by setting the value of the Hamiltonian function to zero at time T, the optimal length of life T^* may be solved.[27] By applying the comparative dynamic analysis method developed by Oniki (1973), parametric changes, which alter entire life cycle paths can be traced. The demand for health was revealed to be more sensitive to initial wealth or stock

[24] Given that negative health investment or 'selling' access health investment/stock on the open market were ruled out.

[25] Capturing only the 'morbidity' and not the 'mortality' aspect of investment in health.

[26] The specification of end time conditions. Death occurs when $H(T) \leq H_{min}$ or $h(H_{min}) = 0$. Individuals would choose a value of T at which these conditions became satisfied. The variable T became an endogenous choice variable to be optimised since the increase of T would entail costs – time and money must be devoted to health investment throughout life, and a rational individual must select a T whereby marginal increase in it would no longer be desirable, i.e. less than the marginal benefit of living longer.

[27] Though T^* may not always be solved as an explicit function of the exogenous variables, it can be solved implicitly.

of financial capital[28] than income. In defense, Grossman (2000) explained that in his original model individuals could engage in an iterative process of health adjustment, thus endogenising the terminal time T, making it a choice variable.

Fourthly, Grossman's (1972) original model did not factor in the uncertain nature of health. Individuals were assumed to possess perfect knowledge regarding the health investment and depreciation functions, as well as the death stock H_{min} and by implication the time of death.[29] Cropper (1977) presented two models which incorporated elements of uncertainty. In the first model, the probability of illness in the subsequent instant followed a normal distribution.[30] An increase in the stock of health capital can shift the entire distribution of sickness in subsequent instant to the left but cannot guarantee a reduction in sick time, thus introducing uncertainty regarding the payback of health capital.[31] In the second model, individuals allocated time between hazardous and safe occupations. Hazardous occupations commanded higher wages but exposed the individual to greater ambient pollution, which increased the probability of death. Individuals possessed no knowledge regarding the lethal dose of pollution but if they remained alive at any instant, would conclude that the exposure level they experienced was below the critical dose. The amount of hours devoted to hazardous occupations was modelled as a control variable to be optimized, given the uncertainty surrounding the lethal dose, which was subjected to progressive revelation.

Dardanoni and Wagstaff (1987) constructed a two-period version of the Grossman (1972) model. Grossman (1972)'s original model showed that health investment in any instant was independent of initial conditions, yet through the assumption of absolute risk aversion in the utility function, Dardanoni and Wagstaff (1987) demonstrated that health investment would be a function of initial wealth. Therefore, inheritance and endowed wealth can greatly affect health inequality. According to Dardanoni and Wagstaff (1986), the introduction of uncertainty with

[28] Which can be interpreted as inherited/endowed wealth.

[29] Death would be reached when the stock of health capital declined to H_{min}.

[30] In a model without uncertainty, a given stock of health capital would always generate a given flow of healthy time per unit of time, via which the flow of unhealthy or sick time may be inferred. In Cropper (1977)'s model, a given stock of health capital generated on average, or an expected level of flow of healthy time, but was subject to a random standard variation component which could increase or decrease the flow of healthy time for a given stock of health capital. The distribution was assumed to be symmetrical or normal around the mean or expected flow of healthy time.

[31] It would possible for those who invest heavily in their health to nonetheless be sick often, while for those who invest little in their health to be rarely sick. However, this would not be the general rule but only the exception.

regards to the return of health investment also reduced its attractiveness for the poor. However, Selden (1993) showed that the results of introducing this form of uncertainty were sensitive to "subtle distinctions concerning the way uncertainty enters the model" (p.110) hence such conclusions must be treated with caution. Assuming an additive rather than multiplicative utility function, Selden (1993) reached the opposite conclusion to Dardanoni and Wagstaff (1987) – in light of uncertainty regarding the return to health investment, the poor would have an incentive to invest more in health rather than less, since although health investment may carry the risk of the benefit of less sick time failing to materialize, the risk of not investing may be even more detrimental to the poor, since frequent sickness would result in heavy loss of income and thus be even more damaging to the poor than to the rich. Chang (1996) specified the returns to health investment as a stochastic process while the return to financial investment as certain, dictated by a constant exogenous rate of interest alone. Chang (1996)'s optimization problem thus became a variant of Arrow's (1965) portfolio choice model in which there was a risky (health investment) and a safe asset (financial investment) in a portfolio, and the individual must decide the optimal allocation of investment between to the two assets, subject to a budget constraint.

Zweifel et al. (2009) were completely dissatisfied with Grossman's (1972) implication that individuals can possess full control over the status of their health, and so they abandoned the use of optimal control theory altogether. Instead, they reformulated the problem as one in which the health status might take only one of two states – 'healthy' and 'sick'. Health states as a function of time followed a stochastic process. If an individual was healthy in any time period, a higher stock of health capital reduced the probability of transition into sickness in the subsequent period. On the other hand, if the individual was sick, it was assumed that only usage of medical care would be effective in increasing the probability of transition back to good health during the subsequent period. Zweifel et al. (2009) concluded that the values individuals placed on health and medical care would be dependent on the initial state of health – when individuals were healthy, they attach a low value to health but would devote all resources to cure should they enter a state of sickness, since the marginal utility of consumption was assumed to be zero in the event of illness.[32]

[32] Irrespective of how rich, a sick individual would derive absolutely no utility from his or her financial assets and time. Thus he or she would have an incentive to devote a large amount of resources to improve health if sick and be free of illness as soon as possible, subject to the uncertainty of whether he or she can move back to the state of health in the subsequent period.

Despite the modifications made by studies mentioned above, the major propositions of the original Grossman (1972) model remained largely unaltered. Health was still predicted to vary positively with income, education and the use of medical care and negatively with age. The prediction of positive relationship between health and medical care had been consistently rejected by empirical findings (Cochrane et al., 1978; Wagstaff, 1986; Leu and Gerfin 1992; Zweifel and Breyer, 1997; Nocera and Zweifel, 1998),[33] which revealed very significant negative relationships. Grossman (2000) defended his model by suggesting that endogeneity may be the cause for the observed negative relationship.

Several studies from the University of Lund presented interesting variations to the Grossman (1972) model. Bolin et al. (2001, 2002a, 2002b) modelled families rather than individuals as producers of health in a situation where spouses were Nash bargainers. Family structure and the possibility of divorce affected the distribution of health capital within family members. A later paper modelled situations where spouses behaved strategically in the production of their own health and the health of other family members, while another paper investigated situations where employers have an incentive to increase the health capital of employees. Bolin et al. (2003) included the interaction of health capital with social capital and found positive correlation between health and social capital. Health and social capital acted as both substitutes and complements to each other.

Dardanoni (1986) presented a simplified Grossman (1972) model in order to increase its accessibility. Most of the analytical conclusions could still be obtained. Forster (1989) mentioned that Dardanoi (1986)'s simple approach could only be used to analyse the steady state condition but provided no information regarding the optimal level of health investment to undertake when the individual's health deviated from the steady state stock of health. Forster

[33] The negative relationship between health and medical spending was reported in micro studies using individual data. Even from anecdotal evidence it was apparent that frequent users of medical care and health services tend to be those who have health problems and thus the relationship would negative. However, macro studies using aggregate or national data generally report positive and not negative relationships. Countries and regions which invest more in medical care also have better health metrics in terms of life expectancy and infant mortality. This dilemma between micro and macro data in terms of the relationship between health and medical usage/spending will be discussed more thoroughly later. For now it suffices to say that measures of health at the micro level tend to focus on morbidity while at the macro level, indicators of health relate more to mortality measures. Thus it suggests that medical care is effective at preventing deaths, injuries and other crisis which would cause serious health damage, but may not be effective at fundamentally eliminating many diseases which contribute to poor health or morbidity. Many degenerative and non-communicable diseases for example, have no proven effective treatments.

(1989) expanded Dardanoni (1986)'s model to investigate the dynamics and the 'turnpike' property of health capital.[34]

Models which followed the inspiration of Grossman (1972) by viewing health as a durable stock are often termed the 'health capital model'.[35] Daalgard and Strulik (2012, 2014) attempted to develop an alternative class of model, which they named the 'health deficit model'. They modelled the process of ageing as the accumulation of health deficit, which would result in death when a certain threshold was reached. The rate of deficit accumulation could be reduced by medical care, which exhibited diminishing product. Unlike the human capital model, medical care could not increase the stock of health, but merely reduced its rate of decline.[36] It was argued that the equation of motion for health in health capital models implied that health deterioration would be greater during early years when health stocks were large,[37] which Dalgaard and Strulik (2014) deemed contrary to medical science. By contrast in the health deficit model, once a process of deficit accumulation began, it had a tendency to perpetuate at an accelerating rate, creating health destabilization.[38] Dalgaard and Strulik (2015) also criticized health capital for being an elusive concept, which would be difficult to measure quantitatively with appropriate metrics, even with the use of latent variables. On the other hand, they proposed a 'frailty index' for measuring the stock of health deficit, rooted in medicine and

[34] 'Turnpike' refers to the behaviour whereby the optimal path remains within a neighbourhood of the steady state for the majority of time before digressing to satisfy the transversality condition. See Cass (1966) for more detail.

[35] The term 'health capital model' encompasses all the models mentioned so far in this section, with the exception of Zweifel et al. (2009).

[36] The stock of health may be inferred indirectly from the degree of deficit accumulation

[37] In absolute terms.

[38] The equation of motion for health deficit accumulation was given by $\dot{D} = \mu(f(D) - g(M))$, where $\dot{D}$, D, M and μ were the time derivative of deficit, stock of deficit, the quantity of medical care employed and the biological rate of ageing, respectively, with μ, $f'(D)$ and $g'(M) > 0$. Since $\frac{\partial \dot{D}}{\partial D} = \mu f'(D) > 0$; the rate of deficit accumulation would increase further with a higher stock of deficit, resulting in an unstable system. On the other hand, the equation of motion for health capital is in general given by the form $\dot{H} = f(I) - \delta H$ where $\dot{H}$, H and δ are the time derivative of health capital, the stock of health capital and the rate of depreciation, respectively, with δ and $f'(I) > 0$. Therefore $\frac{\partial \dot{H}}{\partial H} = -\delta < 0$, implying that a larger stock of health would increase the loss of capital hence health capital would converge to a steady state stock and the system would be stable.

gerontology (Mitnitski et al., 2002), which would "leave no degrees of freedom for the researcher" (p.1).[39]

Dalgaard and Strulik (2015) compared and contrasted the differences between the health capital and health deficit models. They casted doubt over the transversality condition of health capital models once more, questioning the approach of Ehrlich and Chuma (1990). Dalgaard and Strulik (2015) argued that there was no inherent reason for T to be finite, thus replacing Ehrlich and Chuma (1990)'s transversality condition with that for the infinite time horizon.[40] Under general circumstances, the optimal T would be infinite, if I assume that H_{min} the stock at which life can no longer be supported was less than the steady state stock of health capital. Therefore, according to Dalgaard and Strulik (2015), the health capital model would predict that individuals choose to live forever for typical parameter values, which is clearly unrealistic. The only viable solution was to make the rate of health capital depreciation δ a positive function of time, such that $\dot{\delta} > 0, \forall\, t > s$, where s is some stage in the life cycle and $s \in (0, T)$.[41] Although this was actually proposed originally by Grossman (1972), Dalgaard and Strulike (2015) argued that it would drastically complicate the model of Ehrlich and Chuma (1990) rendering it difficult to obtain analytical solutions for the optimal time path for health capital. This was because the comparative dynamic method of Oniki (1973) cannot easily be applied to such a problem.[42] It was therefore alleged that Ehrlich and Chuma (1990) made an implicit assumption that δ, the rate of health depreciation was constant, which according to Dalgaard and Strulik (2015) would result in the prediction of eternal life.[43] Other writers who employed Oniki (1973)'s method also made this simplifying assumption (Eisenring, 1999; Meier, 2000; Forster, 2001). Furthermore, Dalgaard and Strulik (2014, 2015) argued that the rate of biological ageing has no relationship with the chronological age as evidenced in gerontology

[39] The text statement means arbitrary judgement on the part of the economist designed to let the theory fit the data.

[40] Ehrlich and Chuma (1990)'s transversality condition for finite T was given by $J(T) = 0$, where J was the Hamiltonian function. The transversality condition for infinite time horizon of Daalgard and Strulik (2015) would be modified to $\lim_{T \to \infty} J(T) = 0$.

[41] $\dot{\delta}$ possesses no sign restriction for $t \in [0, s]$.

[42] Known as the problem of variable or non-constant coefficient. It is also difficult to apply Oniki's (1973) method to problems of multiple state variables, since the dimensional restriction limits the use of phase diagrams.

[43] Under typical parameter values, individuals are able to invest in health to the extent of offsetting the constant rate of health depreciation δ, so that the steady state of health capital exceeds H_{min}, the minimum stock of health required to sustain life.

(Mitnitski et al., 2002a, 2002b, 2002c) and deemed the absence of a specific functional form for $\delta(t)$ as unsatisfactory.

2.3. Health Economic Models at the Macroeconomic Level

The models in the previous section analyzed health from the microeconomic perspective. Macroeconomic studies of health were designed to answer different sets of questions to microeconomic studies. Instead of examining variables which cause variations in health, macroeconomic studies tend to be concerned with the effect of societal investment in health and health infrastructure, often from a planner or the government's perspective, and thus frequently focused on the relationship between health and economic growth. Whilst empirical findings strongly suggested a positive relationship between health and economic growth (Knowles and Owen, 1995; Bloom, Canning and Sevilla, 2001, 2004; Weil, 2005),[44] the channels of causation were complex and required more rigorous theoretical support. It was not even apparent which direction of causation was dominant – does better health result in faster economic growth or faster economic growth result in better health?

The main advantage of macroeconomic models of health compared to microeconomic models was the ability to model interactions effects with other economic sectors and mechanisms not accounted for by microeconomic models.[45] Two interactions with health at the macroeconomic level were of particular conceptual significance to this study. First, investment in health capital may crowd out the investment in physical and human capital[46] by directly competing for scarce resources, thus retarding economic growth (Barro, 1996; Van Zon and Muysken, 2001, 2005; Gong and Wang, 2012). Government investment in infrastructure or public capital would also

[44] The indicator for health (capital) at the macroeconomic level for the purpose of cross country comparison was often taken to be the life expectancy. This was because life expectancy as a metric has the advantage of capturing both the mortality and morbidity dimensions of health. It captures entire episodes of morbidity throughout life since frequent onslaught of illness and injuries often reduce lifespan, while average life expectancy would also be very sensitive to premature mortality metrics such as infant mortality. Weil (2005) discussed some indicators at the microeconomic level including adult height, adult survival rate and age of menarche, which can be applied to cross country comparison of health. Most empirical studies at the microeconomic or individual level utilise self-reported health status or medical conditions to measure health status (or the stock of health capital).

[45] Microeconomic models are often considered 'partial equilibrium' models in that only the equilibrium conditions of one particular sector or market, in this case the health sector/market, are considered. The potential interactions of the sector/market of interest with other sectors/markets are ignored. Macroeconomic models instead are interpreted as 'general equilibrium' models where a number of sectors/markets interact and jointly determine the equilibrium conditions in all sectors/markets.

[46] Throughout this text, unless otherwise stated 'human capital' will be used to specifically denote educational, knowledge or capital embodied in skills, even though health capital is arguably also a form of human capital.

be diverted, reducing growth (Agenor, 2008). However, these drags may be offset by an increase in labour productivity (Muysken et al., 2003). Hosoya (2012) developed an endogenous growth model whereby consumption and the level of public health infrastructure comprised a non-separable utility function. In addition to affecting the utility at the macroeconomic level,[47] public health infrastructure enhanced labour productivity growth. It was revealed that multiple equilibria of consumption, health infrastructure and physical capital would exist under such conditions, and the equilibria were all dynamically stable.[48] If health policies are inadequate, a country's economy would converge towards a sub-optimal equilibrium of low economic growth, which would be difficult to escape, thus falling into a growth trap. Howitt (2005) identified six channels of economic growth which can be brought about by the improvement of public health: increase in labour productive efficiency; rise in life expectancy; enhanced learning capacity; creativity; coping skills and reduction in inequality. For certain countries which lag far behind the technological frontier, an increase in health may be of sufficient magnitude to escape the growth trap, initiating a catch-up.

The life expectancy channel has been extensively studied (De la Croix and Licandro, 1999; Aisa and Pueyo, 2004; Chakraborty, 2004; Cervellati and Sunde, 2005; Leung and Wang, 2010; Agenor, 2014). A longer life expectancy would increase the payback period of human capital investment, thus raising the net present value of such investment. A longer life expectancy would also be associated with a lower mortality rate, which would reduce the risk of human capital investment being completely destroyed, hence the expected net present value would also be increased. If health increased life expectancy, then human capital accumulation would be encouraged. Human capital in turn would raise the economy's productivity, innovation and technological advancement, which would all enhance economic growth. However, Zon and Muysken (2001, 2005) showed that mortality reduction will raise the dependency ratio,[49] which

[47] Macroeconomic models like microeconomic models also employ utility functions. However, such utility functions represent not the welfare of an individual but that of a society, or country. It is assumed that the preferences or utility functions of individuals can be aggregated in some way (usually via summation) to form the utility function at the macroeconomic level. The macroeconomic utility function is often known as the social welfare function.

[48] The various factors and endogenous variables have a tendency to converge to a dynamically stable equilibrium.

[49] The dependency ratio refers to the economically inactive population by age as a percentage of the economically active population by age. The economically inactive population is generally considered to be those younger than 16 years of age and those older than 65 years of age, while the economically active population are those between 16 and 65. As people live longer with a rising life expectancy, those over 65 years of age are likely to increase, thus raising the dependency ratio.

would divert resources away from capital accumulation and growth. This phenomenon would be more pronounced in countries with low marginal productivity in health investment and high or rapidly rising demand for health.

The empirical relationship between life expectancy and economic growth has been clearly documented, and illustrated via the Preston Curve (Preston, 1975). From the cross-sectional dimension, high income countries in general have longer life expectancy, though the increase in life expectancy as a result of higher GDP per capita gradually diminishes for very high income countries where life expectancy approaches the biological upper limit. The Preston Curve has been shown to shift upwards over time for almost all countries so that life expectancy became higher for all countries at whatever level of economic development. The theoretical explanations of the Preston Curve relationship however generally suggested the opposite direction of causation to studies which investigated how life expectancy enhanced economic growth (Bloom and Canning, 2007) – i.e. that life expectancy growth was the product of economic development, rather than economic growth being attributable to life expectancy increase. Fogel (2004) argued that higher income facilitated better nutritional intakes which contributed to the observed rising life expectancies. Deaton (2006) on the other hand emphasized the role of clean water, sanitation (Cutler and Miller, 2005) and better medical care (Cutler and McClellan, 2001) being provided as a result of rising national incomes.

A second interaction not accounted for by microeconomic models was environmental pollution. Economic activities consumed energy resources, which generated ambient pollution, detriment to health, as was described by the co-benefit literature. The environment and growth relationship has often been hypothesised by the 'Environmental Kuznets Curve', where pollution and growth exhibit an inverted U shape (Grossman and Krueger, 1993).[50] The message to policy makers was simple – 'pollute first and clean up later'. The priorities of low income countries were employment, industrialisation and economic growth, which must not be hindered by environmental policies. Only when the country has reached middle to high income status, should it pursue stringent environmental regulations. The validity of this hypothesised

[50] For undeveloped economies, the relationship between economic growth and environmental damage is positive – economic growth results in further pollution. However, when a certain level of economic development is reached, the relationship between economic growth and environmental becomes negative – economic growth at this point results in less pollution.

relationship was not universally accepted and widely contested both by those who refuted such relationships and those who disagreed regarding the policy implications (Dasgupta et al., 2002).

Shafik (1994) argued that there were three types of pollutants which differed in terms of their relationship with economic growth. The first type of pollutant has a negative relationship with economic growth at all levels of income. Economic growth once in motion would immediately begin to lower the emissions of this type of pollutant. Examples include contaminated water and pollution which causes sanitation issues. The second type of pollutant displays the typical environmental Kuznets curve relationship where economic development would initially cause an increase in the level of pollution before declining. Examples include ambient particulate matter and other forms of air pollution. The third type of pollutant has a positive relationship with economic growth at all levels of income. Economic growth at all levels continue to increase the pollutant which shows no sign of declining. Examples of this type of pollutant including CO_2 and other GHG.

There were however relatively few studies which simultaneously modelled the interaction of the environment, health and economic growth, since most concentrate on either the relationship between the environment and economic growth, or health and economic growth, but not the effect of environmental damage on economic growth via the negative impact on health. Such studies tended to assume that environmental damage reduced the quantity and/or quality of labour supply, since pollution would cause higher mortality and morbidity, and may negatively impact on labour productivity in the workplace. Environmental regulations therefore would promote economic growth by remedying such market distortions, where pollution often operates as a negative externality from the economic activities of private unregulated firms. Pautrel (2007) showed that the greater the health effect of pollution and the more health contributes to productivity, the more likely environmental policy would be positive for growth. Schwartz and Repetto (2000) found that the optimal level of environmental tax increased when health interactions were considered, due to a reduction in negative tax distortions. However, they only modelled health implicitly in the utility function. Nonetheless Mohajan (2011) argued that the optimal environmental tax should be less than the marginal damage of environmental pollution, given the presence of pre-existing taxes which distort the free market competitive equilibrium. Williams (2003) by explicitly modelling health effects reached a contrary conclusion to Schwartz and Repetto (2000). Williams (2003) argued that environmental improvements induced via tax would reduce the cost of medical care, resulting in an income or wealth effect leading consumers to substitute labour for leisure, thereby exasperating the

market distortion. However, by incorporating into the model the subsidization of medical care and a social security system, Caffet (2005) reverted Williams (2003) conclusion in support of Schwartz and Repetto (2000).

Aloi and Tournemaine (2011) suggested that tighter environmental policy will induce greater spending in R&D to facilitate long-run growth. In a more recent paper, Aloi and Tournemaine (2015), predicted a win-win situation whereby tighter environmental policy would lead simultaneously to better environment, health, economic growth and lower inequality, in a model where pollution exposure and vulnerability were unequally spread. Likewise, Chen et al., (2008) also predicted that more ambitious environment regulation can result in higher environmental quality, growth and employment.

Gupta and Barman (2010) extended upon the model of Agenor (2008), specifying the general equilibrium relationship between health, environment and infrastructure investment in an endogenous growth model. The optimal fiscal policy with regards to taxation and government spending (in public infrastructure and health) were proposed. It was found that in the long term, the fiscal policy to maximise economic growth and social welfare were the same. There may however be conflict in the short term, as the transitional dynamics converging to the steady state may not satisfy saddle-point stability.[51] The market economy growth rate[52] was not necessarily lower than the socially efficient growth rate, and the difference between the two was governed by the emission-economic output ratio.

2.4. Knowledge Gap and Contribution

This thesis seeks to provide a framework for incorporating socio-economic factors into the modelling of health co-benefit which arises from climate change mitigation and air quality policies.[53] Currently this is a deficiency in the health co-benefit literature. The methodology developed here is applicable to both health co-benefit policies involving technical substitution as well as those concerned with demand management. This is because I simply analyse how a change in the emission of air pollution interact with socio-economic variables resulting in a certain health impact, and are not concerned at all in the mechanisms bringing about such a change in air pollution. Aside from augmenting the socio-economic dimension, the

[51] i.e. the equilibrium is dynamically unstable.

[52] The economic growth rate in the absence of any fiscal policy or distortions.

[53] Environmental policies which aim to reduce air pollution.

methodology I develop also provides a toolkit to predict whether the health co-benefit arising from decarbonisation strategies vary along socio-economic lines and thus whether it has implications for (in)equality. In fact, the toolkit can be used to predict the (in)equality implications of any public policy on a number of endogenous variables, provided I know how a given policy in question is likely to affect those endogenous variables. The toolkit therefore has policy applications beyond the assessment of climate change mitigation and air quality policies.

The development of the methodology involves the construction and utilization of a health capital model based on Grossman (1972). This model is used to predict how variations in the socio-economic variables income, education and age, affect health. Existing health capital models would not be very suitable for this purpose since they have not adequately modelled the role of health capital depreciation, which is an important part of both the theory and empirics in the model developed. Therefore, through the endeavour of developing this health capital model, I provide an improvement to existing health capital models which follow the tradition of Grossman (1972).

A crucial innovative aspect of my model compared to existing models, is the separation of the life cycle analysis into two connected stages – childhood and adulthood. Existing models treat the life cycle process as a single optimization problem from either birth to death as the initial and terminal time, respectively, or from adulthood to death.[54] The model developed here analyses the life cycle by specifying two dynamic optimization problems, one for childhood and the other for adulthood. The two phases are distinct in that the decision making process during childhood and adulthood are assumed to be independent from each other. The two phases however are connected in that the terminal conditions in the childhood model act as the initial conditions in the adulthood model. For example, if a child's health reached $\bar{H}$ at the end of his/her childhood, then for the adulthood the initial health condition would be $\bar{H}$. The childhood and adulthood optimisation problems are also specified differently, with the most important difference being the way the health production function operates. During the

[54] Wolfe (1985) considered the case of early retirement where pre and post retirement could be considered two different phases in life. Nonetheless Wolfe (1985)'s model remained one single optimisation problem whereby the age of retirement was an endogenous variable to be optimised. In other words, individuals were assumed to possess a choice regarding when to enter the next phase in life i.e. from working to retirement. My model on the other hand assumes that the time from childhood to adulthood is a fixed or exogenous process where the individual has no choice. This is the main conceptual distinction between my model and Wolfe (1985).

childhood phase, health investment increases the stock of health capital but for the adulthood phase health investment instead reduces the rate of health depreciation but can no longer increase the stock of health capital. Furthermore, during the childhood phase, decisions must be made by the child regarding optimal investment in jointly determining the stock of health and education, while for adults only health is relevant and education becomes exogenous.[55] Furthermore, during the childhood stage consumption and investment in medical care[56] are assumed to be governed by parents and guardians hence treated as exogenous, while for the adulthood phase these become endogenous variables subject to a budget constraint.

The approach of my model possesses several advantages over existing health capital models. Firstly, the division of life cycle into two distinct phases allows the most important features of each phase to be identified, which is realistic since situations and priorities often change in different seasons of life. The existing health capital models assumed that individuals have extremely long planning horizons which span their entire life. This is not realistic even from casual observation and would especially be the case for children, for some teenagers and young adults whose ability to make informed decisions for themselves have not yet formed. Frequently such individuals plan relatively short term, having only in mind the childhood world as they know it. The inability of individuals to plan for the very long term, the cultural importance of certain life milestones such as age 18 and going to university/college, as well as unknown and even fear regarding life beyond childhood as an adult may reinforce a myopic view in such a way that young individuals plan only for the childhood phase. Even though decisions taken at this stage will affect the life after, it may be difficult for young individuals to fully grasp the consequences of their actions or inactions until they become much older. It is possible and in fact frequent for adults to regret what they did or did not do as a child, suggesting that the life planning as a child had not been longer term covering the adulthood life. In my model, the health and educational capital accumulated up to the end of the childhood phase become the initial stocks for the adulthood phase. Upon becoming an adult, he or she may find that the stocks are lower than what he/she would have liked. The person may have regretted that as a child he/she did not take good care of their health, engaged in health

[55] For the child there are two state variables – health and education, yet for the adult there is only one state variable – health.

[56] As will be argued shortly and later in the text, the variable in the model should not be seen as 'medical care' but instead as goods and services which promote good health.

damaging behavior such as cigarette consumption, and did not study enough so that the educational capital could be higher.

Secondly, the distinction of phases permits the effect of health investment and health behavior to be treated differently for childhood and adulthood. This will more realistically reflect the different biological characteristics of humans between childhood and adulthood, and how individuals respond accordingly. I assume that health investment can increase the stock of health capital for those in the childhood stage since the biological process of bodily and mental development is undoubtedly a mechanism which augments the stock of health capital. During the adulthood phase however, since the bodily and mental development ceases, it is no longer possible to increase the stock of health capital. The process of aging begins and the individual's investment in health may only reduce the decline in health capital by reducing the rate of health depreciation, which is always positive. In other words, only the bodily and mental development process and any health investment taken during that stage to enhance or shepherd that process are considered health augmenting/increasing health investment in my model. Health investment taken after the development process are instead considered only as health maintenance. My model is therefore more realistic compared to existing health capital models which regard health investment at all stages of life to be investments which add to the stock of health capital.

In addition, existing health capital models implicitly assume that health damaging or negligent behaviors can be substituted inter-temporally. For example, an individual can work very hard, working long hours frequently, and/or expose themselves to hazardous occupations as in the case of Cropper (1977) in order to earn higher income. These activities all raise the rate of health depreciation so that health declines faster. However with higher income, the individual can devote more time and monetary resources to health investment at later stage in life to compensate for the rapid decline in health earlier in life.[57] The opposite scenario would also be possible though perhaps less common, whereby individuals invest heavily in health early in life and deplete them later in life for financial gain.

Grossman (1972) argued that since there does not exist a tradable market for health capital whereby those who wish to convert health into wealth and vice versa can freely exchange, only

[57] The individual will also have more time since there is less need to spend time working after accumulating sufficient wealth.

this type of inter-temporal substitution would be possible to accommodate for different inter-temporal preferences between health and wealth between individuals. Whilst this type of behavior is clearly existent in the real world, there are limits to which this is possible or feasible. Evidence suggests that health investment early on in life, particularly at childhood, have greater potency compared to later in life, and strongly determine the health and socio-economic status of adulthood life (Case et al., 2005; Shonkoff et al., 2009). The model I develop explicitly highlights the limit to such substitution. It implies that this type of substitution is only fully possible during the childhood phase since like existing health capital models, health investment increases the stock of health capital during this phase. For example a child may consume high levels of cholesterol causing a high rate of health depreciation, but 'make up' for it later through aerobic exercises. On the other hand, the child may accumulate health capital through exercise and utilize it by reducing health investment as major examinations approach when they have little time to exercise or even eat proper meals. During the adulthood phase the adult is no longer able to increase his/her stock of health capital. Therefore, at any point in time it is necessary to 'keep in check' the rate of health depreciation. The adult for example can no longer allow the intake of high levels of cholesterol like when he/she was a child since the high rate of health depreciation would substantially reduce the stock of health and irrespective of how much health investment is placed afterwards, the damage would have taken its toll. The substitution of health capital between the two phases is also limited. Only excess rather than deficient health capital can be transferred from the childhood to the adulthood phase. The child can invest heavily in his/her health capital as a child, and utilize it during adulthood. However, if health investment is neglected during childhood, there is no way to recover the deficit as an adult. Given this, it is possible that in light of uncertainty and risk aversion the child may hold on to extra health capital and over-invest in it prior to entering adulthood, although I do not explicitly model such behaviour.

Thirdly, the model I develop makes an attempt at addressing the joint determination of health and education which is absent in existing models. Grossman (2000) had lamented that his plea for the development of such a model went unanswered and to this day it remains the case. Although my model probably is not what Grossman (2000) had envisaged given its relatively little focus on education, it is a step in the right direction. In some ways my model is superior to what was envisaged since in all likelihood the conceptual understanding would have been the joint determination of the stock of health and educational capital spanning the entire life cycle. In my model however, the joint determination of health and educational capital takes

place only during the childhood stage and at the adulthood stage the stock of educational capital becomes fixed and is an exogenous variable. Educational capital should not be regarded as a variable throughout the entire life but should instead be restricted to the childhood phase, at least if by education I refer to formal education and schooling. To be fair and precise, the type of education or 'human capital' envisaged, following the human capital models developed prior to Grossman (1972) were broader concepts than formal education and schooling, encompassing continuous knowledge accumulation, on the job training and work experience. This type of education or human capital should probably be modeled as a continuous development in life rather than assumed to cease at the end of the childhood phase. Nonetheless the concept of human capital from Becker (1962) clearly encompassed formal education and schooling, which form an important if not the central part of human capital at large. Therefore, beginning to formally treat education and schooling[58] in an economic model, to distinguish it from other forms of knowledge capital and experience, is potentially useful leading us in the future to develop a more comprehensive model encompassing different forms of human capital and health capital.

The model I develop is tested empirically for validation. The childhood and adulthood phases produce two different sets of model solutions and thus are treated as two separate models even though conceptually they are linked. The two phases are also separately tested empirically. Only the reduced forms rather than the structural form equations are tested.[59] This is because in order to account for the endogeneity in structural form equations, it is necessary to apply econometric techniques such as Two-Staged-Least Squares (2SLS) regression, and General Methods of Moments (GMM). This is difficult if not impossible to perform when the dependent variable is non-continuous categorical, and the dataset is panel data.[60] Appropriate unbiased and consistent estimators have not yet been developed, tested with confidence and widely

[58] Which may thus be viewed as a subset of human capital exclusive of health capital.

[59] Reduced form equations are equations where the final dependent variable is expressed as a function of exogenous variables. Structural form equations on the other hand involve a set of connected equations. The final dependent variable is expressed as a function of exogenous variables and at least one endogenous variable, which in turn is a function of exogenous variables.

[60] More will be described about the data in Chapter 4. Most individual data on health rely on self-reported health, where the responses are often assigned categories such as 'good', 'fair' and 'poor'. These variables are categories and clearly non-continuous. Usually, only medically developed metrics of health such Body Mass Index (BMI), blood sugar levels etc are continuous. Self-reported health has the strong advantage of being more encompassing, capturing a fuller dimension of general health level than any medically produced health indicators and I therefore select them for my empirical analysis.

applied in such a setting. This means that I am unable to test the most controversial proposition of health capital models involving the relationship between health and medical care usage, since medical care usage is an endogenous variable which explain health status in my adulthood model. Nonetheless for the childhood model, as I have assumed that medical care usage is dictated by parents, guardians and perhaps even the government via mandatory health programs such as vaccination, it can be included into the regression analysis as an exogenous variable.

It should be pointed out that I do not use medical care usage in the data, for it is one of the aims of this thesis to propose an alternative view to the commonly held perception that medical care improves health.[61] I question Grossman (1972) and other health capital models' mechanical assumption that medical care usage contributes to health expressed as an increase in the stock of health capital. Instead I postulate that the medical care usage modelled in the health capital models, including my own should be interpreted strictly as goods and services which are conducive to good health, such as vegetable, fruit, gym membership, exercise equipment, holiday travels etc. Clearly most if not all of these goods and services require the input of time in order to generate health investment.

The definition of medical care usage which is improves health may be stretched to alternative medicine practices such as Chinese medicine and acupuncture, which focus on enhancing the immune system, and certain types of preventive medicine, including vaccination. This is because Western or mainstream medicine in general as it has developed today, contrary to other types of medicines are based on treating a particular disease rather than to improve health generally. Heavy users of Western medicine are often those with severe underlying health issues which may be long-term. They are certainly in most cases not individuals who seek to become more 'healthy' in the broadest sense. Therefore from a conceptual perspective, medical care usage should signal poor health, high rate of health depreciation, rather than being regarded as a mechanism which augments the stock of health capital. As an example, Western medicine has undoubtedly saved many lives which would have been taken away by heart attacks, yet it can do little if any to fundamentally solve the health issue which caused the symptom in the first place, such as obesity, being a lifestyle issue. The traditional Grossman framework would regard medical care usage here as health investment, while I propose that

[61] The author strongly believes this to be the case if 'health' is defined to mean general health or healthiness, which is what is being tested empirically. The author does not deny the tremendous progress made in medical science which have treated many diseases and saved lives. Nonetheless it is the view of the author that such use of medical care should not be regarded as an improvement of general health.

the usage of medical care for this purpose should signal a high rate of health depreciation, while reserving health investment to activities which reduce obesity. Whilst not a central goal of this thesis, it is hoped that in the process of developing the health capital model and validating it empirically, a challenge can be raised regarding the interpretation of the component which is commonly modelled as medical care usage, as well as the data suitable for their empirical test.

The health capital model I developed is then applied to the modelling of health co-benefit arising from decarbonisation and air quality policies, incorporating socio-economic variables. This is performed by setting the optimal rate of health depreciation as the dependent variable[62], which will be functions of the socio-economic variables, most importantly income, education and age. Aside from the socio-economic variables, the endogenous rate of health depreciation is also assumed to possess an exogenous rate of health depreciation. This exogenous component may be interpreted as environmental pollution exposure experienced by the individual, such as ambient PM exposure. Therefore, arranged in this way my model is able to predict how individual health risks (to pollution) vary, given the level of pollution exposure and variations in socio-economic factors. Existing empirical studies in epidemiology have begun to explore how socio-economic status affect health risk due to air pollution (Gouveia and Fletcher, 2000; Evans and Kantrowitz, 2002; O'Neil et al., 2003; Jerret et al., 2004). However, there are no formal economic models which attempt to explain differential health risks across socio-economic groups via an economic or utility maximization perspective. The observed empirical relationship between higher health risk and lower socio-economic status requires further theoretical explanation regarding the mechanisms which are not yet fully understood. This is especially important given that the data often cannot separate higher health risk for a given unit of exposure among lower socio-economic groups or that such groups face higher health risk due to higher exposure via some form of self-selection process (Forastiere, 2007).[63] An advantage of using an economic model to explain this observed empirical relationship is that a substantial amount of other explanations from a wide range of disciplines, such as biological, nutritional and sociological can be subsumed under the modelling

[62] The health depreciation rate is an endogenous variable only in the adulthood phase of the model (although it contains an element of exogenous health depreciation), hence only this portion of the model can be used for health co-benefit modelling.

[63] It is often not possible or very expensive to obtain accurate data on actual level of exposure. Instead, geographically identified air pollution information often at relatively low spatial resolution are used to proxy for ambient air pollution exposure in any given region. It is assumed that all individuals in a given geographical unit experience the same level of ambient pollution, which may not be the case.

mechanisms and the umbrella criterion of utility maximization. For example, biologically some people are naturally more susceptible to air pollution, possibly as a result of old age, others are nutritionally deficient and therefore have less immunity, whilst still others are wealthier and can afford better protective measures. The biological explanation can be explained by my model's dynamic properties in that those who are older, being nearer to death (their 'optimal' length of life) have more incentive to let health decline. The nutritional explanation could be explained by those spending more in goods and services which promote good health, while the sociological explanation involves high income, highly educated individuals having more resources to purchase such goods and services. Whilst the economic utility maximization explanation does not offer direct 'scientific' explanation for the observed empirical relationship, it is consistent with much of the explanations which may be put forward by any discipline, or at least not mutually exclusive. As a result, the economic approach has the advantage of being broader in its interpretation, application and more encompassing than any highly specific mechanism of explanation.

The next chapter sets out this thesis' theoretical framework by explaining the construction of a health capital model following the traditions of Grossman (1972). The details of the derivation however are put in the Appendix for conciseness.

CHAPTER 3 – Theoretical Framework

As the basis of the theory, I construct an economic model which can be used to analyse the effect of socio-economic variations on health. The strength of economic models lies in their underlying principle of utility maximisation, which as mentioned acts as a flexible framework incorporating the explanations of health and socio-economic variables from various theories and disciplines. The model developed here follows the basic structure of the Grossman model (Grossman, 1972) and this type of model is often termed the health capital model. Continuous time optimal control theory is used to derive a dynamic model, which characterises the behaviour of a representative agent operating under certainty and perfect information, seeking to maximise his or her lifetime utility.[64] The model also assumes additively separable utility functions similar to Dardanoni (1986) and Selden (1993).

A major advance of my approach compared to existing health capital models which followed Grossman (1972) lies in the division of the life cycle analysis into two interconnected phases of childhood and adulthood. The division of the model into two phases has the advantage of being able to treat childhood and adulthood separately, yet still connected, emphasizing the most important features in each stage, and specifying different sets of conditions which govern the two phases separately. It yields two empirical model propositions which can be used to test the health behavior of children or young individuals, and another for testing adults or the general population at large. This distinction also allows the incorporating of both health capital models and the health deficit models proposed by Dalgaard and Strulik (2014), both of which have their distinct advantages. In short, I believe that the childhood phase is better represented by a health capital model while the adulthood phase more closely resembles the health deficit model.

The advantages for a dual-phase model become apparent when I examine the specific differences in model mechanisms between the two phases. Many differences between the two phases are best resolved by separating the models, since key aspects which influence the decision process operate differently during the childhood and adulthood phases. Firstly, in addition to maximizing the inter-temporal utility function, a child also seeks to maximize the accumulated educational capital and thus allocate resources between the two aims accordingly.

[64] I do not incorporate uncertainty into the model as it is not a central focus of the thesis though arguably it is a substantial limitation to my model. The division into two stages suggest that planning is relatively short-term compared to other models and hence the agents may not factor in uncertainty, at least to the extent when compared to other health capital models.

This is more realistic in that education is often an important aspect of a child's life, determining future socio-economic prospects as an adult. Even if the child does not fully understand the consequences of investing or not investing effort into education he or she should certainly grasp some aspects of future utility or opportunities related to a good education. Secondly, certain decisions such as the use of medical care and consumption for the child are treated exogenously since such decisions are often made by parents and guardians on behalf of the child. It is also possible that the government via certain mandatory health programs force children to consume a given quantity of medical care.[65] Thirdly, I assume that whilst investment in health capital increases the stock of health capital for the child, such investment does not increase the stock for the adult but merely reduces the decline of health capital. This assumption reflects the different biological features at the two different phases. During childhood as the body is still developing, it is possible for more stock of health capital to be augmented to the existing stock. On the other hand, during the adulthood phase the process of ageing leads to progressive irreversible health decline which should be interpreted as a decline in health capital and any health investment during this phase of life should be regarded as health preservation rather than health enhancement. Furthermore, utility maximization across the two phases are assumed to be independent processes meaning that a child seeks to maximize his or her childhood utility without regard for the utility in the adulthood stages. This is to reflect the inability of children or adult to plan for the very long term. However, the educational capital[66] accumulated during the childhood phase influences adulthood utility via the effect on income or wages and it is assumed that the stock of educational capital is fixed at the adulthood phase or at least treated as exogenous.

I first construct a general function model to lay out the foundation of my model and assumptions. General functions specify the relationship between the dependent and independent variables and the direction of influence but not the exact functional forms. The general function model serves to illustrate the key concepts and is widely applicable. However, specific relationships cannot be identified and its applicability in many cases is limited without further assumptions. Once the general function model is specified I develop a model with

[65] As already pointed to early, 'Medical care' is the interpretation of the mechanism which enhances health in the health capital models, a point which this thesis questions. Instead that mechanism should be interpreted as health promoting goods and services such as fruit and vegetables etc.

[66] Educational capital in this context refers solely to formal education or schooling, which become fixed predominantly when the child leaves school or college.

specific functions, assuming functional forms to the general functions. This allows me to generate a solution for the model by finding the optimal control and equilibrium relationships which form the basis of my testable hypotheses. It should be noted that a general function model can be interpreted using a number of specific function models and I have simply chosen one I feel most suitable which yields analytically solvable solutions. The rationale behind the specific functions which constitute the model is explained as they are introduced. Section *3.1* describes the general function model while section *3.2* describes the specific function model. For details regarding how the model solutions are obtained, please refer to the Appendix. Once the specific functional forms are chosen, I proceed to derive the model solutions which are optimality or equilibrium conditions, and these form the basis of my testable hypotheses. This is described in section *3.3*.

3.1. General Function Model

At both stages, whether during childhood or adulthood, I assume that a person seeks to maximize an inter-temporal utility function over some planning horizon. Even though utility maximisation for adults has been more or less accepted as a fundamental axiom in economic modelling, the same cannot be said for the child because much of the decisions of the child's life particularly very early in life are taken by parents or guardians. Therefore, in the model developed here the child is able to control only certain aspects, and the variables frequently treated as endogenous in other models, such as the amount of consumption, is treated here as exogenous variables. The utility function at any instant may be represented by:

$$U(t) = U\big(X(t), \tau(t)\big) \qquad \frac{\partial U}{\partial X} > 0, \frac{\partial U}{\partial \tau} > 0 \tag{3.1}$$

Where $X(t)$ is the quantity of (composite) goods consumed and $\tau(t)$ the amount of free time, which may be interpreted as leisure time.

This interpretation of the inter-temporal utility function is already at variance with most health capital models which followed Grossman (1972). In most other models, the utility function possessed two arguments – the flow of healthy time and a composite consumption known as 'commodity'. This composite consumption or commodity in turn was produced by input of time and the quantity of (composite) goods consumed, governed by an efficiency parameter which the original Grossman model stated as the stock of education.[67] In fact, this 'commodity'

[67] Though many subsequent models did not continue to use education as an efficiency parameter in this way.

is essentially equivalent to the entire utility function as shown by equation (3.1). It is my opinion that modelling the production of a composite 'commodity' which then becomes an argument in the utility function is rather onerous and can cause conceptual confusion or misunderstanding. There is no apparent rationale to create this 'commodity' which enters the utility function as an argument, and in the process cause the entire utility function to become a composite function. This is unnecessary given that the 'commodity' was but a flow variable in Grossman (1972) and other health capital models, which unlike health capital, which functioned as a stock variable. For this reason, I simplify the utility function by making it essentially equivalent to the composite consumption of commodity. Only two factors drive one's utility – the amount of material goods consumed and free (healthy) time. This is easy to understand and makes intuitive sense. The health investment function,[68] describes how a unit of health capital is produced given factor inputs. The two factor inputs are $M(t)$ and $\tau_H(t)$, which represent the amount of medical goods/services employed and time devoted to health improvement, respectively.[69] The latter includes but is not limited to time spent in hospital check-ups, exercise and vacation.[70] A general form for the health investment function is given by:

$$I(t) = I(M(t), \tau_H(t); A) \tag{3.2}$$

A is an efficiency parameter governing the input-output relationship and is similar to the role played by education in Grossman (1972). The stock of health capital increases the flow of healthy time in any instant, which can be devoted to work, health improvement or free time/leisure:

$$h\big(H(t)\big) = \tau_W(t) + \tau_H(t) + \tau(t) \qquad h' > 0 \tag{3.3}$$

[68] Sometimes known as the health production function.

[69] $M(t)$ was interpreted primarily as medical care in Grossman (1972), though as is argued here in this thesis, should instead be interpreted as health promoting goods and services. Medical care in general, other than preventative medicine, vaccination and regular health inspections performed independently of health conditions, signal poor health or the existence of some underlying health issues.

[70] I ignore possible overlap between certain aspects of $\tau_H(t)$ and $\tau(t)$ for the sake of simplicity. It is not clear for example whether vacations which improve health constitute time inputs in the creation of health capital or directly to increase the utility.

Here h is a function which maps the stock of health $H(t)$ to the flow of healthy time, while $\tau_W(t)$ is time devoted to work. By rearranging (3.3) making $\tau(t)$ the subject and substitute the result into (3.1) I obtain the following equation:

$$U(t) = U\left(X(t), h\big(H(t)\big) - \tau_H(t) - \tau_W(t)\right) \qquad (3.1')$$

Equations (3.1) – (3.3) and (3.1') should be the same for both the childhood and adulthood phases.

In many health capital models diminishing returns to factors or inputs were often assumed for the above equations or their equivalents. I make no such assumption since it is not clear whether it reflects reality in my case. For example, in the case of equation (3.1) and (3.1'), it is not obvious that $\tau(t)$, the amount of free time actually leads to diminishing marginal utility due to the fact that free time in this day and age is often a superior commodity in short supply and may be employed in countless ways flexibly to prevent diminishing marginal utility from setting in. For equation (3.3) it is also not obvious that the marginal product of a stock of health capital in terms of its generation of the flow of healthy time, is subject to diminishing returns and so I avoid stating the second order derivative. Another potential problem with specifying diminishing returns is that it restricts the type of specific functional forms which corresponds to the general functions, which may in turn prevent model solutions to be obtained analytically.

3.1.1. The child's problem

A representative child who is rational seeks to maximise his or her intertemporal utility function as expressed in equation (3.1) aggregated over the childhood years, which I define as from $t = 0$ to $t = q$, where q is the time when childhood ends. In addition to maximising the utility function, I assume that the child seeks to maximise the stock of education obtained at the end of his or her childhood years, at q. This is because the higher the stock of education at that point in time, the better is his or her employment prospects and income as an adult. The child realises this to an extent and knows that even though education does not yield any childhood utility, adulthood utility will be strongly influenced by it and so have an incentive to invest in education. This is consistent with the casual observation that educational attainment

is a major goal of most children.[71] I specify the equations of motion for the accumulation of health and educational capital by equations (3.4) and (3.5) below, respectively.

$$\dot{H} = I(t) - \delta H(t) \tag{3.4}$$

$$\dot{E} = f(\tau_W(t), H(t); A) \tag{3.5}$$

Where δ is a constant rate of health depreciation, f is the production function for educational capital, dependent on the flow of time input and the stock of health, and subjected to the same efficiency parameter A as the health production function.[72] $\tau_W(t)$ is time devoted to work, but in the context of the child, it is time devoted to increase the stock of educational capital or in other words study time.

If I assume that the dual criteria of maximising childhood utility and final stock of education can be combined via an additive weighted function, where θ is the relative weight attached to the final stock of education, then the child's optimisation problem can be expressed as the maximisation of the following objective function (3.6), subject to (3.4) and (3.5), with the initial conditions for health and educational capital given, and their terminal states free to vary.

$$\int_0^q U(t)e^{-\beta t}\,dt + \theta E(q)e^{-\beta q} \tag{3.6}$$

β is the subjective rate of discount. The subjective rate of discount is conventionally applied in dynamic optimization models, based on the assumption that utility in the future are discounted at an exponential rate.

An optimal control problem where the objective function takes the above form is often known as the problem of Bolza (Bolza, 1913). Nonetheless this type of problem can be easily converted into a standard form problem where the objective function to be maximized is incorporated into a single definite integral, which is shown by (3.6') below.

$$\int_0^q [U(t) + \theta(\dot{E} - \beta E(t))]e^{-\beta t}dt \tag{3.6'}$$

Substituting (3.5) into (3.6') I obtain the following objective function:

[71] The extent to which this is true may be influenced by the child's family upbringing, culture or even genetics. These influences will be described later.

[72] The parameter A includes many factors in the 'health education gradient' (Grossman, 2006; Conti et al., 2010), which influences both health and education.

$$\int_0^q [U(t) + \theta(f(\tau_W(t), H(t); A) - \beta E(t))]e^{-\beta t} dt \qquad (3.6'')$$

The child's optimal control problem is then to maximise equation (3.6'') subject to the dynamic constraints imposed by equations (3.4) and (3.5) as well as the initial and terminal conditions for health capital and educational capital, which are known as the state variables in optimal control theory. The initial condition for health is given (H_0), and the initial stock of educational capital is assumed to be zero. The control variables in this problem are $\tau_H(t)$ and $\tau_W(t)$. Unlike most other health capital models which treated $X(t)$ and $M(t)$, consumption and the use of medical services respectively, as choice variables, I treat them here as exogenous since in most cases they are determined by parents or guardians rather than by the child. The child must choose the optimal time paths of $\tau_H(t)$ and $\tau_W(t)$, which will yield a corresponding time path for the stock of health and education, maximising equation (3.6'') in the process.

3.1.2. The adult's problem

When adulthood commences, the person becomes responsible for his or her own life. The person must decide how much goods to consume and the medical goods and services to employ, subject to some financial constraint. $X(t)$ and $M(t)$ therefore become endogenous choice variables during the adulthood stage, like in other health capital models. However, unlike in other models which treated $\tau_W(t)$ as a choice variable and one of the main rationales for investing in health capital is so that there is more time available for work which in turn increases income, I treat $\tau_W(t)$ as an exogenous variable for the adult stage. This is because working hours in the modern world are frequently determined or at least heavily influenced by the employer and/working environment and culture. In practice, the adult may have little or no control over working hours hence I treat it as exogenous during the adult phase.

An important difference between the child and adult, a feature which I have specifically built into my model as an improvement compared to other health capital models, is the different manner in which health investment affects health accumulation. Unlike children, an adult's bodily development process had ceased. It is therefore not possible for the adult conceptually to increase health and his or health is subjected to progressive decline until death, when the health capital falls to or below H_{min}, the minimum stock of health capital necessary to sustain life. In the modelling context I specify this feature by making it impossible for the individual during the adulthood stage to augment his or health capital beyond the childhood stage. Instead, health investment is treated as a means to decrease the rate of health depreciation and prolong

the time it takes for the stock of health to fall to or below H_{min}. The adult's equation of motion for health capital 'accumulation' can be shown by equation (3.7) below:

$$\dot{H} = -\delta\big(I(t)\big)H(t) \quad \delta' < 0, \delta > 0 \; \forall \; I(t) \tag{3.7}$$

In order to simplify the analysis, if I assume that all income is consumed in each instant and that there are no savings. The adult's budget constraint can be shown by equation (3.8) below.

$$P_X(t)X(t) + P_M(t)M(t) = y\big(H(t), E(q)\big)\tau_W(t) \tag{3.8}$$

where $P_X(t)$ and $P_M(t)$ are prices of consumption goods and medical goods/services, respectively.[73] y is the wage rate, which is a function of the stock of health at time t and educational capital accumulated during childhood. Unless otherwise stated, I refer to the wage rate as y and omit expressing it as a function with its arguments.

I assume that the adult's stock of education is fixed and in most cases is simply the education he or she has accumulated as a child up to the end of the childhood at $t = q$. This is a realistic assumption so long as $E(q)$ is interpreted solely as the educational capital acquired from formal schooling and not experiences acquired during work, which also strongly influences the wage rate. Furthermore, I exclude the possibility of retraining, though this aspect may be interpreted as an exogenous increase in $E(q)$ and hence if so wished can be incorporated into this model in this manner. Since the adult no longer possesses the option of adding to the stock of education, his/her aim is simply to maximise remaining lifetime utility, denoted by the equation (3.9) below:

$$\int_q^T U(t)\, e^{-\beta t} dt \tag{3.9}$$

where T is the time or age of death.

The adult seeks to maximise equation (3.9) subject to the constraints imposed by equations (3.7) and (3.8). The former is a dynamic while the latter is a static constraint. The initial condition of the adult's problem is determined exogenously by the terminal states of the child's problem at $t = q$.

[73] P_M shold be interpreted as the price of a unit of goods and services which promote good health.

As for the transversality (terminal condition) which refers to any condition which must be satisfied at the end of the time path when $t = T$, there are two possible specifications. For the first possibility T is treated as an exogenous variable and the terminal state of health capital, $H(T)$ is free to take on any value subject to no constraint, save perhaps that it must be non-negative or at least not fall below H_{min}.[74] This is the transversality condition adopted for the childhood phase where q, the time when childhood ends (and by implication the beginning of adulthood) is exogenously given. A potential problem with stating this transversality condition is the implication that the adult's decision to invest in health cannot increase the expected length of life by postponing death and the only benefit is the generation of more healthy time or reducing sick time during this fixed lifespan. With regards to the second possibility, the transversality condition can be stipulated as $H(T) = H_{min}$ or $H(T) = 0$. This makes T an endogenous variable to be optimised since given the exogenous variables, I am in search of a T or T^* which satisfies the transversality condition that the final stock of health capital is equal to the minimum stock required to sustain life or zero. Given the assumption that $\delta > 0 \; \forall \; I(t)$ as seen in equation (3.7), it implies that the optimal T^* is a finite number and there is no possibility that T^* however large can be infinite. In other words 'eternal life' is impossible, which is obviously true in this lifetime at least.

3.2. Specific Function Model

Having established the general function relationships, I attach specific functional forms to the general functions in order to analyse the problem and obtain model solutions which form the basis of the testable hypotheses. It should be emphasised once more that multiple functional forms can be given to any general function. The criteria for the selection of functional forms are a) the assumptions on the forms are realistic and b) ultimately I seek a set of specific functions which would allow us to derive an optimal/equilibrium solution, acting as the foundation of the testable hypotheses. Unless otherwise stated, I assume all exogenous variables to be positive.

For the intertemporal utility function of (3.1) I make three simplifying assumptions which help us to specify the function form:

1. The arguments in the utility function are additively separable.

[74] It may be appropriate to apply a state space constraint whereby $H(t) > H_{min}$ or $H(t) > 0 \; \forall \; t \in [q, T)$ i.e. the stock of health capital must not at any point in time fall below 0 or H_{min}, save at the terminal time where $t = T$.

2. Consumption of goods is subject to diminishing marginal utility, expressed by a logarithmic function. A logarithmic function also implies that goods consumption below a minimum threshold (e.g. the poverty line) *reduces* utility. As $X(t) \to 0$, $U(t) \to -\infty$. This rules out corner solutions and implies that a minimum amount of goods consumption is fundamental to all individuals.
3. Free time yields constant (unity) rather than diminishing marginal utility.

By applying the above assumptions, (3.1) can be written as the equation below:

$$U(t) = (\log\big(X(t)\big) + h\big(H(t)\big) - \tau_W(t) - \tau_H(t))e^{-\beta t} \qquad (3.1')$$

I further assume that the stock of health capital linearly increases the flow of healthy time and that $h\big(H(t)\big) = 0 \leftrightarrow H(t) = 0$. Equation (3.1') can be altered as follows.

$$U(t) = (\log\big(X(t)\big) + GH(t) - \tau_W(t) - \tau_H(t)))e^{-\beta t} \qquad (3.1'')$$

Where G is the constant marginal product of healthy time per unit of health capital.

For the child's equation of motion for health capital expressed in (3.4) I also adopt three assumptions:

1. Time and medical goods/services (input of health promoting goods and services) as inputs to health production are additively separable.
2. Time input into health production is subject to diminishing product but always remains positive, which can be expressed by a logarithmic function of the form $\ln(x + 1)$, where $x \geq 0$.
3. The exogenous medical care (input of health promoting goods and services) increases health accumulation at a constant rate (unity), though its influence declines exponentially as t increases. This assumption is adopted into the model to reflect recent medical evidence which strongly supports the importance of early childhood and even neonatal care on the health and development of children. Medical care (input of health promoting goods and services) usage is constant throughout the child's life, unless subjected to an exogenous shift.

By applying the above three asumptions, the equation of motion for the child's health capital accumulation as written in (3.4) takes on the following specific form.

$$\dot{H} = A(\ln(\tau_H(t) + 1) + Me^{-t}) - \delta H(t) \qquad (3.4')$$

For the child's equation of motion for the accumulation of educational capital, shown by equation (3.5), the following three assumptions are made:

1. Time input into education (learning) and the effect of health capital on the accumulation of educational capital are additively separable.
2. Time input into the accumulation of education capital is subject to diminishing product but always remains positive, which can be expressed by a logarithmic function of the form $\ln(x+1)$, where $x \geq 0$.
3. A unit of health capital increases the rate of educational capital accumulation at a constant rate.

The application of the above three assumptions lets us write the equation of motion for educational capital accumulation as follows:

$$\dot{E} = A\ln(\tau_W(t)+1)) + \omega H(t) \qquad (3.5')$$

Where ω is the constant marginal product of health capital in the production of educational capital.

The adult's health capital accumulation equation of motion is fundamentally different from that of a child's in that health investment no longer increases the stock but instead reduces the rate of decline, as can be contrasted between equations (3.4) and (3.7). In order to attach a specific functional form to equation (3.7) I make the following four assumptions:

1. There is an exogenous rate of health capital depreciation which is independent of both time input and the use of medical care (health promoting goods and services).[75]
2. Time input and the use of medical care are subject to diminishing return.
3. The marginal return of time and medical good is dependent on each other.
4. The endogenous rate of health depreciation is positive for all t.

One possible functional form for the depreciation function consistent with the above assumptions incorporated into the equation of motion for health capital accumulation is shown below:

[75] For the purpose of theoretical modelling, I treat medical care and health promoting goods and services as synonymous. However, as argued previously medical care should not be considered as health investment, and this would be reflected in my empirical model.

$$\dot{H} = -\delta\big(I(t)\big)H(t) = -\frac{\delta_0 e^{A\tau_H(t)}}{AM(t)+1}H(t) \qquad (3.7')$$

Where δ_0 is an exogenous rate of health depreciation. It is necessary for $\tau_H(t)$, $M(t) \geq 0 \; \forall \; t \in [q, T]$ and $\delta_0 > 0$ in order for the third assumption to be satisfied.

The above functional form as shown in equation (3.7') satisfies all the four assumptions. It has the advantage of allowing analytical solutions to be obtained relatively easily. Another possible form is the Cobb-Douglas function. However, this would not be as straightforward since the factors in the equation contribute to reduction rather than an increase, as would be the case in a typical production function.

Equation (3.7') however poses problems for the optimal control problem in that solutions for the optimal time path of the control and state variables ($\tau_H^*(t)$, $M^*(t)$ and $H^*(t)$) rarely exist, due to the need for simultaneous solving of a non-linear system of higher order differential equations. For simplicity I assume that health depreciation is stock independent thus modifying equation (3.7') to (3.7'').

$$\dot{H} = -\delta\big(I(t)\big) = -\frac{\delta_0 e^{A\tau_H(t)}}{AM(t)+1} \qquad (3.7'')$$

Not only does the problem become considerably easier to solve if (3.7'') replaces (3.7') as the equation of motion, there is also an important justification for this simplification based on Dalgaard and Strulik's (2012, 2014, 2015) health deficit model. It was reasoned that stock dependent health depreciation implies that healthy individuals (those with a high stock of health capital) lose greater health capital per unit of time due to depreciation compared to unhealthy individuals (those who a low stock of health capital), an implication which Dalgaard and Strulik (2012, 2014, 2015) deemed contrary to the evidence in medical science and argued that the reverse is true – those who are unhealthy face more severe deterioration of health per unit of time. Dalgaard and Strulik's (2012, 2014, 2015) argument however was somewhat redundant when applied to most health capital models which assumed the diminishing product of health capital as a feature. In these health capital models, despite individuals with higher stocks of health losing greater quantities of health through depreciation, the values of such losses were diminishing. In contrast to other health capital models, I do not adopt diminishing but instead assume constant product to health capital. Therefore, the assumption of stock independence in health depreciation is both necessary and complementary to the assumption of constant product

to health capital. Most importantly such an assumption would permit a unique solution of optimal time path to be obtained.

By analogous reasoning, the equation of motion for children as shown by equation (3.4') should also be altered to (3.4'') below.

$$\dot{H} = A(\ln(\tau_H(t) + 1) + Me^{-t}) - \delta \qquad (3.4'')$$

I prefer (3.4'') over (3.4') for the reason that the assumption of constant marginal product of health capital should go hand in hand with the assumption that the depreciation of health capital is independent of the stock, even though adopting (3.4') would not significantly complicate the solution process for the childhood model.

I make two simplifying assumptions regarding the budget constraint of (3.8):

1. The wage rate y is independent of the stock of health at any instant.
2. The exogenous variables $P_X(t)$, $P_M(t)$ and $\tau_W(t)$ are constant.

Equation (3.8) can be re-written as (3.8') below[76]:

$$P_X X(t) - P_M M(t) = y\tau_W(t) \qquad (3.8')$$

Via the introduction of the first assumption mentioned above, the adult model can be considered a form of 'pure consumption' model corresponding to Grossman (1972), since it is implied that health does not increase income but only utility via the increase in healthy time. However, an 'investment' element is still retained since y is a function of the stock of educational capital, and its accumulation had been part of the decision process when the person was a child. During childhood, part of the reason for investing in health capital would be to enhance the learning process since I assume that the accumulation of educational capital is dependent on health as seen in equations (3.5) and (3.5'). It is not obvious at that stage what such investments are for, yet when adulthood comes it becomes clear that such investments increase the wage rate y. Therefore, my model on the surface appears to be a pure consumption model by Grossman (1972)'s standard but in reality is a 'mixed' model in which both the

[76] Although the wage rate is no longer a function of health, it is still a function of the stock of education accumulated during childhood hence it is more accurately written as $y\big(E(q)\big)$. However, for simplification, I omit $E(q)$ from now on unless otherwise stated.

consumption and investment aspects of health investment are retained, and expressed when the two phases of adult and childhood are examined together.

3.3. Model Solutions

I employ optimal control theory in order to derive the model solutions. There are in general three branches of mathematical methods available to solve dynamic problems such as those I state in section *3.1* and *3.2* – calculus of variations, optimal control theory and non-linear (dynamic) programming. The first two methods are very similar, with the latter being the extension of the former. In comparison to the calculus of variations, optimal control theory produces more succinct solutions and generates results on the optimal control variables which may be of interest to us. The final method of non-linear programming is a more generalised method compared to optimal control and therefore has a wider range of application. However, it is more computationally demanding and would only be necessary if the problem is stated in discrete time, yet I state my problems in continuous time.

I apply the Pontrayagin's Maximum Principle, which is by far the most commonly used method in optimal control theory, yielding solutions to the state, co-state and control variables as functions of time and the exogenous variables. Movements or variations in exogenous variables are regarded as parametric changes which alter not only the state, co-state and control variables at any particular time t but generally the entire time path is altered. Comparing the time path which is subjected to a change in exogenous variable(s) is known as comparative dynamics,[77] which is widely employed in my study to guide empirical efforts.

Even though the childhood and adulthood phases are connected and should be taken as a whole, for the purposes of deriving equilibrium/optimal solutions, the two phases are treated as separate optimal control problems. The terminal conditions for the child when $t = q$ are taken as the initial conditions for the adulthood control problem. Section *3.3.1* details the solutions to the model for the childhood phase while section *3.3.2* details the solutions to the model for the adulthood phase. For details regarding the derivation procedures for the solutions, please refer to the Appendix.

[77] The dynamic equivalent to 'comparative statics', which analyses how equilibrium conditions are changed in static optimisation problems.

3.3.1. Model Solutions to the Childhood Phase

The solution for the control variables are shown below, assuming non-negative values:

$$\tau_H^*(t) = \max\left\{A\left((\omega\theta(q-t)-G)e^{-\beta(q-t)} + G\beta^{-1}\right) - 1{,}0\right\} \tag{3.10}$$

$$\tau_W^*(t) = \max\left\{A\theta e^{-\beta(q-t)} - 1{,}0\right\} \tag{3.11}$$

I assume non-negativity in the time inputs since conceptually it does not make sense that time devoted to improving health and to work (study) can be negative. If this constraint is not imposed then there can be multiple solutions to the co-state and state variables, which may be problematic. The alternative to imposing this constraint is to assume that the parameters are of a combination such that the control variables are positive for all t, which is not unreasonable.

The solutions to the co-state variables are:

$$\lambda_H^*(t) = (\omega\theta(q-t)-G)e^{-\beta(q-t)} + G\beta^{-1} \tag{3.12}$$

$$\lambda_E^*(t) = -\theta\left(1 - e^{-\beta(q-t)}\right) \tag{3.13}$$

Assuming interior and excluding corner solutions,[78] the solutions for the state variables are:

$$H^*(t) = A\left(M(1-e^{-t}) + \int_0^t \ln\left(A\left((\omega\theta(q-u)-G)e^{-\beta(q-u)} + G\beta^{-1}\right)\right)du\right) - \delta t + H_0 \tag{3.14}$$

$$E^*(t) = \frac{1}{2}At\left(\ln\left((\omega\theta(q-t)-G)e^{-\beta(q-t)} + G\beta^{-1}\right)\omega t + 1\right) + \omega\left(AM\left(t-(1-e^{-t})\right) - \frac{1}{2}t(\delta t - H_0)\right) \tag{3.15}$$

3.3.2. Adult Model Solutions

In order to simplify the analysis, I rescale the adult's planning horizon from $[q, T]$ to $[0, V]$, where $V = T - q$. The initial state of health in the adult model H_0 is equivalent to the terminal state of health attained in the child model $H(q)$. Furthermore, I assume that $\beta = 0$, i.e. there is no discount in the case of adult. This assumption significantly reduces the complexity of the solutions at little or no cost to their analytical power. The omission of β simplifies greatly the functional forms of the model solutions but does not alter the direction of partial derivatives

[78] i.e. $\tau_H^*(t), \tau_E^*(t) > 0 \; \forall \; t \in [0, q]$. I may make the assumption that the exogenous variables take on values such that the control variables are always positive.

for any exogenous variables. Therefore, the discount rate is omitted since there are no suitable data for adults hence its effects are not testable empirically, without which there would be no point in including in the theoretical model given that it would not alter any theoretical predictions. Assuming non-negative values, the solutions for the control variables are:

$$\tau_H^*(t) = \max\left\{\ln\left(\frac{A\delta_0 P_M(c_1 - Gt)}{y}\right)^{\frac{1}{A}}, 0\right\} \tag{3.16}$$

$$M^*(t) = \max\left\{\frac{y - P_M}{AP_M}, 0\right\} \tag{3.17}$$

$$X^*(t) = \frac{y}{P_X} \tag{3.18}$$

The solution to the co-state variable is:

$$\lambda_H^*(t) = c_1 - Gt \tag{3.19}$$

c_1 is an arbitrary constant to be definitized.[79] The exact definitization of c_1 depends on the transversality condition. If I specify the optimization problem as a fixed terminal time problem where T is fixed and exogenously determined but $H(V)$ free to vary (subject to non-negativity), then c_1 can be solved rather quickly. Under such a condition because there is no constraint imposed upon the state variable, the co-state variable (sometimes known as the 'shadow price') have no value during the terminal period such that $\lambda_H^*(V) = 0$. Using this condition c_1 is immediately solved from (3.19):

$$c_1 = GV \tag{3.20}$$

Substituting equation (3.20) into the optimal time path for state variable yields the following equation:

$$H^*(t) = (GA)^{-1}\ln\left(\frac{V - t}{V}\right) + H_0 \tag{3.21}$$

As can be seen from equation (3.21), the optimal stock of health should decline steadily but as $t \to V$, $\frac{dH^*(t)}{dt} \to -\infty$, since the log function approaches $ln(0)$. This is expected since the sole

[79] The constants are endogenous variables. By 'definitisation' I refer to the process of expressing the constants as a function of the model's other exogenous variables. This is achieved by substituting known values into the model solutions and solving for the constants. For example the initial and terminal values for the solutions, when $t = 0$ and $t = V$, are known, can be used.

purpose of health capital is for the generation of the flow of healthy time $\tau(t)$ to increase utility. When life is nearly overly, the individual has no incentive to retain any health capital and any excess health capital above H_{min} represents a waste of resources i.e. resources which could have been consumed when the individual was still alive.

However, if I specify the endogenous terminal time problem where T^* or $V^* = T^* - q$ are endogenous variables to be optimized, then c_1 takes on a different value. For the optimality condition to be satisfied in the terminal period, the Maximum Principle states that it is necessary for the discounted Hamiltonian function (see Appendix) to be zero, implying all benefits or utility have been exhausted to the extent that the marginal cost of extending the terminal time is greater than the marginal benefit. The condition is stated mathematically as $Z^*(V^*)e^{-\beta V^*} = 0$, where $Z^*(t)$ is the Hamiltonian function at time t evaluated at the optimum.

The solution for the state variable in the case where T and V are endogenous is shown in equation (3.21') below:

$$H^*(t) = (GA)^{-1}\ln(Gt - c_1) + c_2 \tag{3.21'}$$

c_2 is yet another arbitrary constant to be definitized.

c_1, c_2 and V^* need to be jointly solved by three simultaneous equations. The three equations are the initial condition for the state variable, the terminal condition for the state variable, and the transversality condition, written by $H^*(0) = H_0$,[80] $H^*(V) = H_{min}$ and $Z^*(V) = 0$, respectively. The solutions are shown below in equations (3.20'), (3.22) and (3.23).

$$c_1 = \frac{y}{A\delta_0 P_M}\left(\frac{y}{P_X}\right)^A e^{A(GH_0-1)-2+\frac{P_X}{y}} \tag{3.20'}$$

$$c_2 = (GA)^{-1}\left(A + 2 - \frac{P_M}{y} - \ln\left(\frac{A\delta_0 P_M}{y}\left(\frac{P_M}{y}\right)^A\right)\right) \tag{3.22}$$

$$V^* = (GA\delta_0 P_M)^{-1}\left(1 - e^{-GA(H_0-H_{min})}\right)\left(\frac{y}{P_X}\right)^A e^{A(GH_0-1)-2+\frac{P_M}{y}}y \tag{3.23}$$

[80] The 'initial condition' is equivalent to the terminal condition for the childhood phase where $t = q$. For simplicity I write here $t = 0$. It should be reminded that H_0 in the adulthood model is equivalent to $H^*(q)$, the stock of health capital which is accumulated during childhood.

Substituting equations (3.20') and (3.22) into (3.21'), I have a solution for the state variable in the case of variable terminal time:

$$H^*(t) = (GA)^{-1}\left(\ln\left(\frac{A\delta_0 P_M}{y}\left(\frac{P_X}{y}\right)^A\left(e^{A(GH_0-1)-2+\frac{P_M}{y}} - Gt\right)\right) + A + 2 - \frac{P_M}{y}\right) \qquad (3.21'')$$

Unlike equation (3.21'), equation (3.21'') no longer contains the variable V^*, the terminal time is no longer consider an exogenously given variable but instead will change given changes in other exogenous variables.

3.3.3. Testable Hypotheses and Econometric Models

The optimal solutions shown in sections *3.3.1* and *3.3.2* form the basis of my testable hypotheses. The testable hypotheses are therefore equations (3.10) – (3.15) for the childhood phase and equations (3.16) – (3.19), (3.20'), (3.22), (3.23) and (3.21'') for the adulthood phase.[81] This thesis due to the scope and focus is interested primarily in the stock of health capital, how it changes throughout one's life, as well as the effect of other socio-economic variables on health. The testable hypotheses therefore are a subset of the above equation – (3.14) for the case of the childhood phase and (3.21'') for the adulthood phase. In addition, equation (3.23) regarding the optimal length of life or life expectancy, which is a by-product of the adulthood phase of the model, is also tested empirically.

All the model solutions which function as the testable hypotheses can potentially be tested empirically. However most of these equations have extremely complicated functional forms and may be difficult to test with data in their original form. It is pragmatic therefore to alter the testable hypotheses into simplified linear econometric models for the purpose of empirical testing. I assume that the testable hypotheses are adequately represented by the linearly transformed econometric models. For example, equation (3.10) may be converted to equation (3.10') below.

$$\tau_H^*(t) = \alpha_0 + \phi_1 A + \phi_2 \theta + \phi_3 G + \phi_4 \beta + \phi_5 q + \phi_6 t + u \qquad (3.10')$$

Where α_0 is an intercept, ϕ_{1-6} are coefficients to be estimated using empirical data and u is the error term.

[81] The thesis explores only the case of variable terminal time.

Equations such as (3.10') may be estimated using cross sectional, time series or panel data, depending on appropriateness and data availability. Panel data is likely to be the ideal given that it captures both the cross-sectional variations which arise between individuals as well as the time series dimension reflecting the changes over time or age for the same individual. It is not immediately obvious from equation (3.10) whether changes in any of the exogenous variables increases or decreases the dependent variable $\tau_H^*(t)$ and by implication it is not obvious what signs I should expect for ϕ_{1-6}. I can apply comparative dynamic analysis by differentiating the optimal solution partially with respect to a given exogenous variable in order to predict the signs of the ϕ coefficients. For example, if I partially differentiate $\tau_H^*(t)$ as shown in eequation (3.10) with respect to the exogenous variable A,[82] I obtain the following results shown in equation (3.24):

$$\frac{\partial \tau_H^*(t)}{\partial A} = \omega\theta(q-t)e^{-\beta(q-t)} + \frac{G}{\beta}\left(1 - e^{-\beta(q-t)}\right) \tag{3.24}$$

As can be seen from equation (3.24), the partial derivative of the child's optimal time devoted to health improvement with respect to A, the efficiency parameter is positive in most cases, $\frac{\partial \tau_H^*(t)}{\partial A} > 0 \; \forall \; t \in [0, q)$, $\frac{\partial \tau_H^*(t)}{\partial A} = 0$ if $t = q$. I can therefore conclude that ϕ_1 the coefficient showing the relationship between $\tau_H^*(t)$ and A should be positive. This entire process can be repeated for all the models and variables I wish to test.

Starting with the control variables, their solutions are shown by equations (3.10) and (3.11) for the childhood phase and equations (3.16), (3.17) and (3.18) for the adulthood phase. Using equation (3.10) one can test the relationship between the time a child spends improving health, such as exercise, with the exogenous variables listed on the right hand side. For equation (3.11), one can examine the relationship between the time children devote to studying with the same set of variables. Data on these variables at an individual level are relatively uncommon and normally available in surveys only.

For equations (3.16), (3.17) and (3.18) the dependent variables are adult's time devoted to improving health, consumption of medical goods and services, and consumption of normal goods and services, respectively. The list of exogenous variables is affected by the selection of the transversality condition. I propose to select the transversality condition of variable terminal

[82] Assuming interior solution or that $\tau_H^*(t) > 0$.

time where length of life is endogenously determined. This is because such an assumption is clearly more realistic. If I assume that individual actions or investments in health have no bearing on how long they live, then health capital is used only to reduce sickness for however long the arbitrarily defined timespan is.[83]

Equations (3.12) and (3.13) show the optimal time paths for the co-state variables during the childhood phase. For the adult phase it is shown by (3.19) and the exact value c_1 takes on is subject to the transversality condition specified. As stated I prefer the assumption that the terminal time is endogenous, hence c_1 should take on the value shown in equation (3.20'). The co-state variables in optimal control theory as well as other dynamic optimization methods are merely means to an end. They are used to derive solutions to the state variables and in and of themselves have little or no value. However, just like the role the Legrange multiplier plays in static optimisation, the co-state variable reflects the dynamic constraint of the state variable and is often termed the 'shadow price' of the state variable (Heckman, 1974). In other words it measures the value of a unit of the state variable at any point in time. Empirically the co-state variables can be interpreted as the willingness to pay for the state variable in question. For example $\lambda_H^*(t)$ can be interpreted as the willingness to pay for a unit of health capital or its associated benefit, the flow of health time generated by a unit of health capital $GH(t)$.

The state variables are usually of primary interest in most models. In my case, the childhood phase possesses two state variables – the stock of health and educational capital. During the adulthood phase only health capital remains a state variable, with the stock of educational capital set to be exogenous, taking on whatever value at $t = q$. Given the scope of this thesis, I am interested only in the relationship between the stock of health capital and other exogenous variables. Therefore for the childhood phase, the equation I wish to empirically verify is (3.14) and for the adulthood phase given my assumption of variable terminal time I wish to test for equation (3.21''). Under the assumption of variable time, I produce an alternative equation shown by (3.23). The 'optimal length of life' may be interpreted as life expectancy, given the exogenous variables.

The data for such measurements are usually reliant on survey questions. However, it can be conceptually difficult to frame the concept of health capital and usually a survey question asks

[83] For the childhood model it is permissible to define a set time q when childhood ends since it is often associated with natural events in life such as being fully grown, completing full time education, turning 18 or 21, becoming legally an adult and no longer receiving parental support.

the respondent to rate their own level of health status. It is assumed here that respondents' understanding of their own health status reflects the theoretical or conceptual notion of health capital, or at least is strongly correlated with health capital. Other measures of health include Quality Adjusted Life Years (QALY) and Disability Adjusted Life Years (DALY), which are frequently used in epidemiological studies and facilitates comparison as well as capturing both the morbidity and mortality aspects of health. Again, it is necessary to assume that such measures of health reflect health capital. This is plausible if the stock of health capital always resides on its equilibrium path, which the following section argues should be the case by definition.

3.4. Equilibrium Paths

The equilibrium condition for health in my model is not dynamically stable unlike many health capital models. For the childhood phase there is only one instant in which $\dot{H} = 0$ and it does not persist. For the adult model health continuously declines for all times/ages.

It is assumed that the actual stock of health capital held by all individuals always reside on the optimal path as was also implied by Grossman (1972) in his model. This is for two reasons a) a direct shock to health not only alters the actual stock of health but also the underlying equilibrium time profile of health and b) the equilibrium of health for at all times/ages is a function of initial health, H_0 and $\overline{H}$, hence it is not possible for a discrepancy to exist between initial and desired/equilibrium health.

All direct shocks[84] to the equilibrium health can be conceptually framed into three categories:

1. Shock to initial stock of health.
2. Shock to the stock of health at $t \in (0, T^*)$.
3. Shock from an increase in the exogenous rate of health depreciation,

For succinctness I analyse the shocks to the childhood and adulthood phases, with the full knowledge that the two phases are connected via the terminal condition in the childhood model. I analyse only negative exogenous shocks which are more relevant.

[84] By direct shock I refer to an exogenous shock in which the stock of health is directly affected, in contrast to indirect shocks which are alterations to other exogenous variables which cause the target/equilibrium stock of health to change.

3.4.1. Negative Shocks in the Childhood Case

The effect from the first type of health shock can be obtained by partially differentiating equation (3.14) with respect to H_0, i.e. $-\frac{\partial H^*(t)}{\partial H_0}$. If the negative health shock occurs at some point after $t = 0$, say at $t = s$, where $s < q$, then I may substitute s into equation (3.14) and call the resulting stock of health H_s. H_s is then treated as the initial health for the remaining period of optimisation. This can be represented by shifting the time path forward by s in equation (3.14) produces the following equation:

$$H^*(t-s) = A\left(M\left(1-e^{-(t-s)}\right) + \int_s^t \ln\left(A\left((\omega\theta(q-u)-G)e^{-\beta(q-u)} + G\beta^{-1}\right)\right)du\right) - \delta(t-s) + H_s \qquad (3.14')$$

The negative health shock can be obtained by $-\frac{\partial H^*(t-s)}{\partial H_s}$.

The effect from the third type of shock can be obtained by partially differentiating equation (3.14) with respect to δ or $\frac{\partial H^*(t)}{\partial \delta}$. If the shock occurs at $t = s$, then differentiate (3.14') instead of (3.14), or $\frac{\partial H^*(t-s)}{\partial \delta_0}$. The different types of shocks which can occur in the childhood phase are illustrated by *Figure 3.1*.

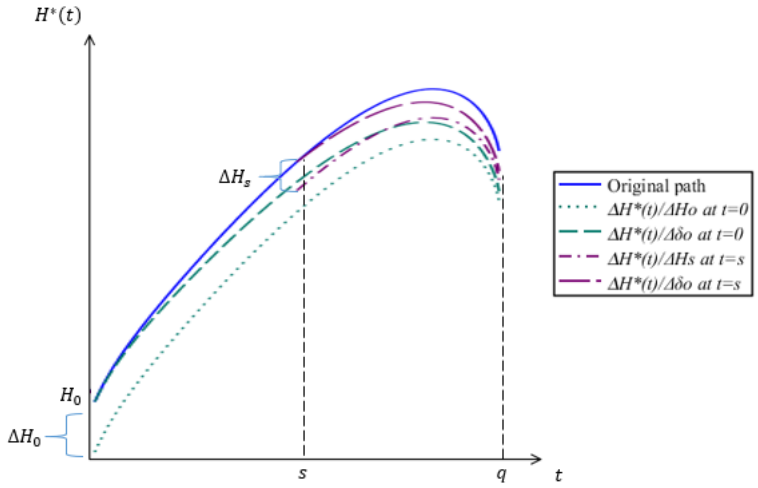


Figure 3.1. Direct shocks to health during childhood

Figure 3.1 shows the expected time path of health capital for a typical child under typical parameter values, obtained using calibration. This is shown by the 'Original path' or solid blue line. The various types of health shocks which alter this time path are shown by the other lines. Health shocks which occur at $t = 0$ are shown by the dark green lines while those which occur later in life say at $t = s$ are shown by purple. The general pattern is for the child to accumulate health capital throughout the majority of childhood years, before depleting it shortly before q,

the end of the childhood phase. This reason for this general relationship is due to the specification of my model where health capital yields utility. However, the value of health capital diminishes at the end of planning period, hence it is allowed to decline.

3.4.2. Negative Shocks in the Adulthood Case

A health shock during the adulthood phase may occur from either a direct loss in the stock of health capital at any age, or through an exogenous increase in the rate of depreciation, δ_0. If a negative shock to health occurs at the beginning of the adulthood phase then it may be expressed by partially differentiating equation (3.21'') with respect to $\bar{H}$, or δ_0, if the shock is of the nature where the rate of depreciation increases. A shock which occurs at any point beyond the initial age in adulthood but before the time of death, say at $t = z$, where $z \in (q, T^*)$, can be represented algebraically by setting $t = z$ in equation (3.21''). The remaining time path is then shown by equation (3.21'''):

$$H^*(t-z) = (GA)^{-1}\left(\ln\left(\frac{A\delta_0 P_M}{y(\bar{E})}\left(\frac{P_X}{y(\bar{E})}\right)^A\left(e^{A(G\bar{H}_z-1)-2+\frac{P_M}{y(\bar{E})}} - G(t-z)\right)\right) + A + 2 - \frac{P_M}{y(\bar{E})}\right) \quad (3.21''')$$

Where $\bar{H}_z$ is the level of health reached at $t = z$ in the absence of any shock.

A direct health shock and a shock via higher depreciation are obtained by $-\frac{\partial H^*(t-z)}{\partial \bar{H}_z}$ and $\frac{\partial H^*(t-z)}{\partial \delta_0}$, respectively. The different types of health shocks possible are illustrated graphically in *Figure 3.2* below. Note that in general the optimal length of life T^* denoted by when health falls below H_{min} will also decrease following a health shock.

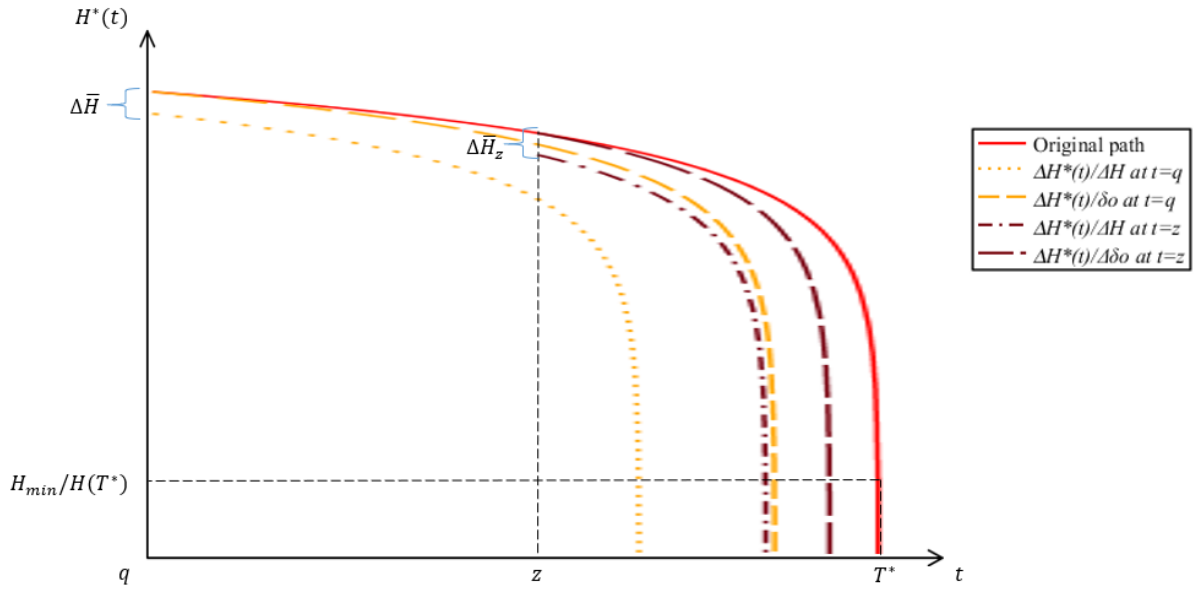


Figure 3.2. Direct shocks to health during adulthood

The typical adult health time path is given by the 'Original path' or red line in *Figure 3.2*. The various shocks to the adult's health are shown by the other lines. Shocks which occur immediately during adulthood are shown by the light orange lines while those which occur later say at $t = z$ by the brown lines.

3.4.3. Interpretation of the Different Forms of Shocks

A significant fall in initial childhood health at $t = 0$ may represent poor neo-natal care, inherited diseases or other events which adversely affect the person early on in life so that potential development becomes limited. A higher rate of depreciation may characterize exposure to health hazardous environments. In extreme cases the higher rate of depreciation may mean that the optimal time path of health falls below H_{min} prior to $t = q$ and the person never survives to adulthood. Children born in countries with high infant mortality rates may experience exceptionally high δ. On the other hand, the shock may happen at $t = s$. A direct health shock may represent a serious accident or injury to which full recovery is not possible or that the growth and development phase is impaired so that health at $t = q$ is reduced. A higher δ at $t = s$ may be brought about by health abusive behavior associated with adolescence, such as beginning to drink, smoke or use drugs. Similar reasoning applies to health shocks during the adulthood stage.

The above scenarios cover all the manifestations of a negative shock one is likely to encounter in real life. Combined with the assumptions which suggest that the 'equilibrium' stock of health is never dynamically stable, and will shift accordingly to exogenous shocks, this means that by conceptual *definition* all observed health resides on the optimal health path. Health variations between individuals or between time for the same individuals are caused entirely by different optimal/target health conditions influenced by the exogenous variables.

CHAPTER 4 – Empirical Investigation

The theoretical model in *CHAPTER 3* – Theoretical Framework generates a set of solutions which form the basis of my testable hypotheses. These solutions are shown by equations (3.10) – (3.15) in the case of a child, and by equations (3.16) – (3.23) in the case of an adult. Whilst all of these equations can be investigated empirically, I am primarily interested in exploring the effect of exogenous variables on the optimal stock of health. The exogenous variables as described later may be divided into environmental, such as exposure to ambient air pollution, and socio-economic such as income and education. Therefore equations (3.14) and (3.21'') are of primary interest to this thesis.[85] The former equation describes the optimal time path of health capital for a representative child, while the latter for the case of an adult under the assumption that the length of life is endogenously determined by the adult's choices. The hypotheses to be tested empirically are rooted in equations (3.14) and (3.21''). The relationships between health and the various exogenous variables can be predicted by applying comparative dynamic analysis, which investigates how shifts in exogenous variables alters the entire life cycle paths of health capital. In addition, equation (3.23) which shows the optimal length of life or life expectancy as a function of the exogenous variables, is also tested in this chapter.

It should be noted here that the empirical testing in this chapter is mostly for the purposes of validating the theoretical models developed in the previous chapter. This is achieved by checking whether the associations of the variables and health capital are in the expected direction. The coefficients or magnitude of the relationships are not the focus, nor am I using them to make quantitative estimates for the purpose of policy.

Error! Reference source not found. below illustrates the lifecycle path of health for a representative individual, constructed via calibration with typical parameter values. It is the graphical representation of equations (3.14) and (3.21'') assuming that childhood is subsequently followed by adulthood. The blue and red portions represent childhood and adulthood phases, respectively. The stock of health increases steadily from H_0 but declines at some point prior to $t = q$, when childhood ends. During adulthood, the stock of health decreases at a progressively faster rate until $t = T^*$, when the stock of health reaches H_{min}, the

[85] I choose to investigate the case of variable terminal time for the adult model.

minimum level necessary to sustain life. Parametric changes alter the lifecycle path as well as the length of the time path.

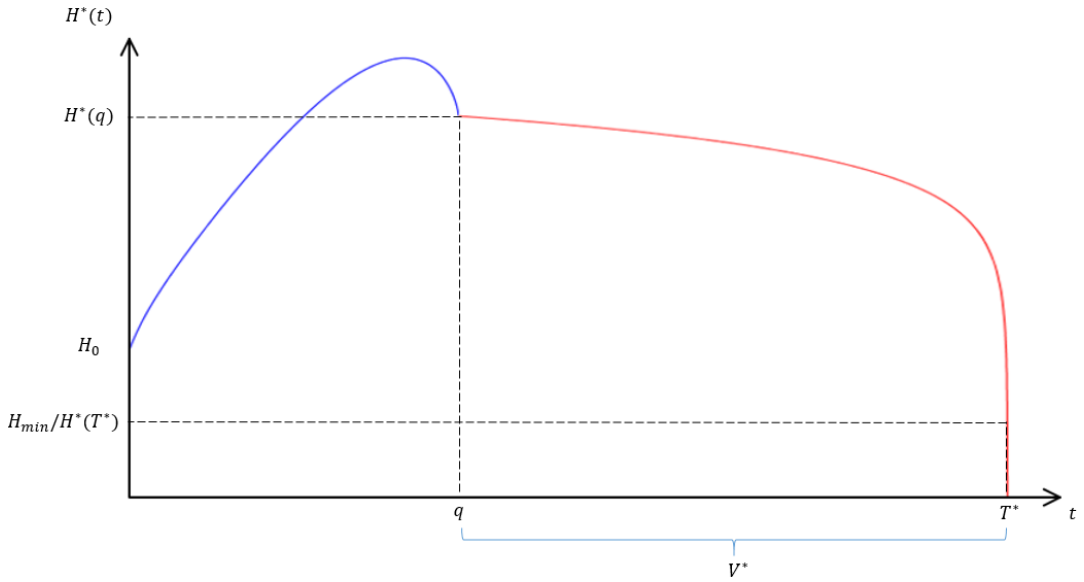


Figure 4.1. Lifecycle path of health capital

For the purpose of the empirical investigation, the childhood and the adulthood phases are treated as two separate models and tested separately in turn. Section *4.1.* describes the childhood model rooted in equation (3.14), its conversion to a linearlised econometric model, the expected signs of the parameters in the econometric model derived using comparative dynamic analysis, the data used as well as the results of the empirical estimation. The same procedures are repeated for the adult model rooted in equation (3.21''), shown in section *4.2.* Section *4.3* empirically tests for equation (3.23), a specific part of the adult model namely the relationship between the optimal length of life, which I interpret as the life expectancy of a representative individual, and various exogenous variables. Section *4.4* briefly summarises the empirical findings and discusses how well they match the theoretical framework and hypotheses described in *Chapter 3.*

4.1. Child Model Empirical Investigation

The child model's theoretical predictions are presented in section *3.3.1.* I am primarily interested in the prediction of child health shown by equation (3.14) – the relationship between various exogenous variables and the health of a child. I use data from the Understanding Society Survey. The details of the survey can be accessed online.[86] The data documentation

[86] https://www.understandingsociety.ac.uk/documentation/mainstage/dataset-documentation

includes a 'Youth' datafile, which contains variables derived from self-completed questionnaires of youths (those under the age of 18). The Understanding Society data may be obtained from the UK Data Service.[87]

Questionnaire variables are selected to represent the theoretical variables in equation (3.14). The dependent variable, the (optimal) stock of health capital $H^*(t)$, is represented by the questionnaire variable 'gets head-aches, stomach-aches or sickness'. The survey responses fall into the following categories: 'missing', 'not true', 'somewhat true' and 'certainly true', which are coded by the numbers -9, 1, 2 and 3, respectively. I remove the missing values before beginning data analysis. It can be inferred that the higher the numerical value representing the response, the truer the statement that the interviewed youth 'gets head-aches, stomach-aches or sickness', denoting a lower stock of health capital. Strictly speaking the response to this question reveals the flow of health time per unit of time $h(t)$ or $GH(t)$ rather than the stock of health capital. Nonetheless, the flow of healthy time is no doubt highly correlated with the stock of health capital and for my purpose suffices for its representation. I switch the numerical coding for 'not true' and 'certainly true' so that a value of 1 represents 'certainly true' while 3 represents 'not true'. The number 2 remains as before representing 'somewhat true'. In this way, a higher value denotes better health or a higher stock of health capital, which is more convenient for interpretation.

It is not possible to find questionnaire variables which would suitably represent all the exogenous variables in equation (3.14). There is therefore no way to test whether these variables significantly affect the stock of health capital. It is not possible to account for G (the marginal product of health capital in generating the flow of health time) and ω (the marginal product of health capital in the accumulation of educational capital). These variables are highly conceptual and difficult to capture empirically. They are therefore omitted in the empirical estimation and attention is devoted to the other exogenous variables shown in equation (3.14). The Understanding Society survey generates a panel data set, spanning from the year 2009 to 2014. It is a continuation of the British Household Panel Survey (BHPS), which is used to empirically test the adult model in section *4.2*. Even though the BHPS contains far larger datasets over longer time periods (18 years) compared to the newer Understanding Society

[87] University of Essex. Institute for Social and Economic Research. (2017). *Understanding Society: Innovation Panel, Waves 1-9, 2008-2016*. [data collection]. *8th Edition.* UK Data Service. SN: 6849, http://doi.org/10.5255/UKDA-SN-6849-9

survey, the latter is more up-to-date and contains some innovative experimental variables which are particularly suited to my task. For example, the response to the question 'restless and cannot stay still for long' can be used to represent the child's subjective rate of discount, β. The Understanding Society therefore allows us to test more variables in equation (3.14) than would be possible using the BHPS.

Error! Reference source not found. below lists the variables in the Understanding Society questionnaire dataset which are chosen as the empirical counterpart to the variables in equation (3.14). The letter 'w' in front of the questionnaire variable names as shown in column one denotes temporal 'wave', which runs from 1-5 in the survey and are denoted by letters a-e. Each wave spans two years, with wave 1 spanning from 2009-2010 and wave 5 from 2013-2014. With one exception, all the variables are categorical and non-continuous, separated into dummy categories and coded. If a variable is categorical, the various categories and their numerical representations are contained in {} brackets in column two. The missing category is dropped, and the remaining categories and numbers are used to generate dummy variables. The dummy variables generated from each categorical questionnaire variable and the numbers each dummy variable represents are shown in column 5, contained in {} brackets. A reference dummy category is stated, which does not enter any regression equation but acts as the category to which all other categories are compared. The term w_yplvhm is a continuous variable and so does not generate any dummy variables. The final column in *Table 4.1* shows the equivalent theoretical variable which the questionnaire variable is chosen to represent.

Table 4.1. Questionnaire variables selected from Understanding Society survey for empirical testing of the child model in equation (3.14)

Questionnaire variable	Variable description	Possible values	Data summary	Dummy variables	Associating theoretical model variable
w_ypsdqc	gets head-aches, stomach-aches or sickness	-9=missing,1=not true,2=somewhat true,3=certainly true	N: 10597; Groups: 7651; Average group size: 1.39 Category distribution: -9: 173 (1.63%), 1: 6401 (60.40%), 2: 3164 (29.86%), 3: 859 (8.11%)	-	$H(t)$ or $GH(t)$ the stock of health capital or flow of healthy time
w_ypsdqb	restless and cannot stay still for long	-9=missing,1=not true,2=somewhat true,3=certainly true	N: 10597; Groups: 7651; Average group size: 1.39	{beta1=1, beta2=2, beta3=3};	β the subjective rate of discount (reflecting people's tendency to

			Category distribution: -9: 169 (1.59%), 1: 2902 (27.39%), 2: 4662 (43.99%), 3: 2864 (27.03%)	reference category: beta1	discount future utility and costs)
w_ypsdqo	is easily distracted. is difficult to concentrate	-9=missing,1=not true,2=somewhat true,3=certainly true	N: 10597; Groups: 7651; Average group size: 1.39 Category distribution: -9: 174 (1.64%), 1: 3617 (34.13%), 2: 4591 (43.32%), 3: 2215 (20.90%)	{A1=1, A2=2, A3=3}; reference category: A3	A an efficiency parameter which denotes how the input of time and other resources translate into improvement in health (and education)
w_ypacvwell	importance of doing well in gcse's or standard grades	-9=missing 1=very important,2=important;3=not very important;4=not at all important	N: 10597; Groups: 7651; Average group size: 1.39 Category distribution: -9: 173 (1.63%), 1: 6401 (60.40%), 2: 3164 (29.86%), 3: 859 (8.11%)	{theta1=1, theta2=2, theta3=3, theta4=4}; reference category: theta4	θ the relative importance of education to utility
w_ypfrutppd	number of portions of fresh fruit and vegetables per day	-9=missing,1=5 or more portions,2=3-4 portions;3=1-2 portions,4=none	N: 10597; Groups: 7651; Average group size: 1.39 Category distribution: -9: 253 (2.39%), 1: 8071 (76.16%), 2: 2058 (19.42%), 3: 150 (1.42 %) 4: 65 (0.61%)	{M1=1, M2=2, M3=3, M4=4}; reference category M4	M goods which enhance health. Traditionally interpreted empirically as medical care, here it is strictly restricted to health promoting goods and services
w_ypffdwk	frequency of eating fast food: days in a normal week	-9=missing, 1=every day, or nearly every day,2=about once a week,3=every now and then,4=never or hardly ever	N: 10597; Groups: 7651; Average group size: 1.39 Category distribution: -9: 115 (1.09%), 1: 170 (1.60%), 2: 2120 (20.01%), 3: 5270 (49.73 %) 4: 2992 (27.57%)	{delta1=1, delta2=2, delta3=3, delta4=4}; reference category: delta4	δ the health depreciation rate
w_dvage	Age for whole sample, from birth or age if	10, 11, 12, 13, 14, 15	N: 10597; Groups: 7651; Average group size: 1.39	{t1=10, t2=11, t3=12, t4=13, t5=14, t6=15};	t age or time

			Category distribution: 10: 822 (7.76%); 11: 1810 (17.08%); 12: (20.29%); 13: 2002 (18.89%); 14: 1990 (18.87%); 15: 1994 (18.34%)	reference category: t1	
w_yplvhm	Age you think when you leave home? (-9 indicates missing value)	-9 (missing), all positive values which are larger than w_dvage	N: 10597; Groups: 7651; Average group size: 1.39 Missing (-9): 1394 (13.15%); Mean: 16.35, Standard deviation: 10.49	-	q the age or time when childhood ends and adulthood commences

Unfortunately, not all the variables listed in *Table 4.1* have complete five-year data, containing all five waves. Some variables only have available years 2009, 2012 and 2014 (waves 1, 3 and 5) and therefore I construct an unbalanced panel dataset using these three years only. Since equation (3.14) possesses a complicated form,[88] I assume a linear relationship between health (the dependent variable) and the exogenous variables in order to derive the following econometric model:

$$H^*(t)_{iw} = \alpha_0 + \alpha_i + \phi_1 beta2_{iw} + \phi_2 beta3_{iw} + \phi_3 A2_{iw} + \phi_4 A1_{iw} + \phi_5 theta3_{iw} + \phi_6 theta2_{iw} + \phi_7 theta1_{iw} + \phi_8 M3_{iw} + \phi_9 M2_{iw} + \phi_{10} M1_{iw} + \phi_{11} delta3_{iw} + \phi_{12} delta2_{iw} + \phi_{13} delta1_{iw} + \phi_{14} t2_{iw} + \phi_{15} t3_{iw} + \phi_{16} t4_{iw} + \phi_{17} t5_{iw} + \phi_{18} t6_{iw} + \phi_{19} q + u_{iw} \quad (4.1)$$

Where i and w denote individuals and waves of data, respectively. $H^*(t)_{iw}$, the stock of health capital cannot be observed directly but is reflected by the survey responses captured in the questionnaire variable w_ypsdqc. Equation (4.1) is to be tested using an unbalanced panel dataset, in order to examine whether the coefficients of the variables are of the expected sign, which would function as evidence in support of my theoretical framework. In order to predict the signs of the coefficients in equation (4.1), I obtain the comparative dynamics by partially

[88] The conversion from (3.4) to (4.1) involves expressing health as a function of all the independent variables, separated additively. Since the variables are mostly dummy categories, they are arranged in increasing order where lthe lowest category is listed first in the equation.

differentiating equation (3.14) with respect to the theoretical variables as listed in column four of *Table 4.1*. *Table 4.2* shows these partial derivatives and the expected sign.

Table 4.2. Partial derivatives of equation (3.14) with respect to the exogenous variables in column four of Table 4.1.

Partial derivative	Equation of partial derivative (comparative dynamics)	Theoretical prediction
$\frac{\partial H^*(t)}{\partial \beta}$	$-A\int_0^t \frac{(\omega\theta\beta^2(q-u)^2 - G)e^{-\beta(q-u)}}{\beta^2\omega\theta(q-u)e^{-\beta(q-u)} + G(1-e^{-\beta(q-u)})}du$	Sign of $\frac{\partial H^*(t)}{\partial \beta}$ indeterminate but likely to be negative
$\frac{\partial H^*(t)}{\partial A}$	$M(1-e^{-t}) + t + \int_0^t \ln(A(\beta\omega\theta(q-u) + G(1 - e^{-\beta(q-u)}))\beta^{-1})du$	$\frac{\partial H^*(t)}{\partial A} > 0 \; \forall \; t \in [0, q)$
$\frac{\partial H^*(t)}{\partial \theta}$	$A\beta\omega\int_0^t \frac{q-u}{\omega\theta(q-u) + G(e^{\beta(q-u)} - 1)}du$	$\frac{\partial H^*(t)}{\partial \theta} > 0 \; \forall \; t \in [0, q)$
$\frac{\partial H^*(t)}{\partial M}$	$A(1-e^{-t})$	$\frac{\partial H^*(t)}{\partial M} > 0 \; \forall \; t \in [0, q)$
$\frac{\partial H^*(t)}{\partial \delta}$	$-t$	$\frac{\partial H^*(t)}{\partial \delta} < 0 \; \forall \; t \in [0, q]$
$\frac{dH^*(t)}{dt}$	$A\left(Me^{-t} + \ln\left(A\left(\beta\omega\theta(q-t) + G\left(1-e^{-\beta(q-t)}\right)\right)\beta^{-1}\right)\right) - \delta$	Sign of $\frac{\partial H^*(t)}{\partial t}$ indeterminate but should be positive if δ is small
$\frac{\partial H^*(t)}{\partial q}$	$A\left(ln\left(\frac{\beta\omega\theta q + G(e^{\beta q} - 1)}{\beta\omega\theta(q-t) + G(1-e^{-\beta(q-t)})}\right) - \beta q\right)$	Sign of $\frac{\partial H^*(t)}{\partial q}$ indeterminate but likely to be positive

The comparative dynamics may be illustrated graphically by *Figure 4.2*, which depicts the effect of parametric changes in the exogenous variables, constructed using calibration. The effect of a change in q, the length of childhood is somewhat different to the change in other parameters in that the length of the planning horizon is altered. $\frac{dH^*(t)}{dt}$ is simply the slope of the time path, rather than an alteration to the path itself.

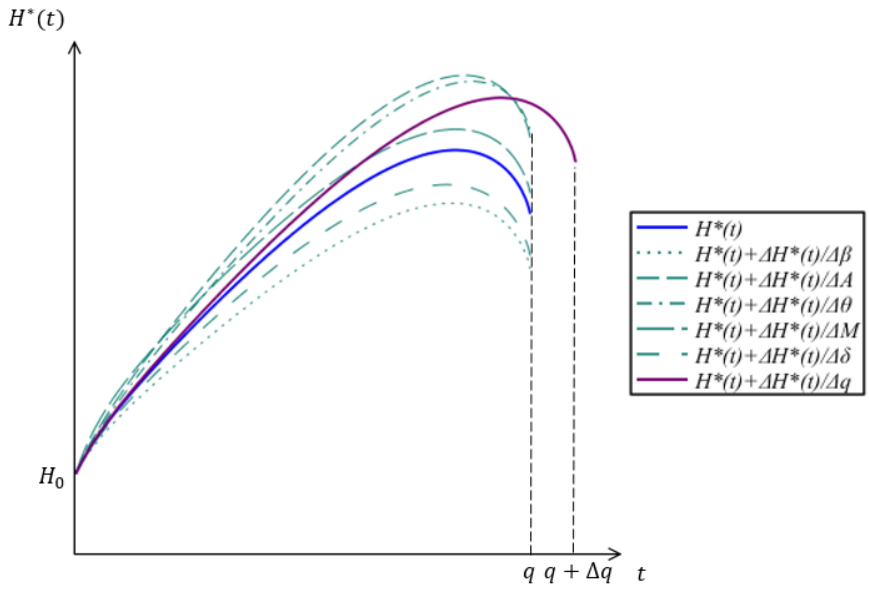


Figure 4.2. Comparative dynamics/partial derivatives of equation (3.14) with respect to changes in exogenous variables in column four Table 4.1.

Using the results in *Table 4.1* and the illustration from *Figure 4.2*, I expect $\phi_{3-10,14-19}$ to be positive and $\phi_{1,2,11-13}$ to be negative. The coefficients of the age dummies ϕ_{15-19} may have some negative signs even though in general they should be positive, since the health time path is not monotonic as seen in *Figure 4.2*.

I employ an ordered logit model to test equation (4.1). This is necessary since the dependent variable is categorical and non-continuous and thus would not be appropriate to apply linear regression models. In addition, as there are multiple categories (three categories) which can be arranged in ascending or descending order to denote increasing or decreasing health capital, it is necessary to extend the binary to an ordered categorical model. An ordered probit model is an alternative option, though the results are very similar to the logit model and so I adopt the logit model as my main model. The probit models are included as a robustness check in that if they differ substantially from the logit models then it would suggest that my results are inconsistent. A slight downside to ordered logit models is that the magnitude of the coefficients generally have no meaning or are at least difficult to interpret. Usually only the signs of the coefficients are of relevance. Nonetheless since I choose to use self-reported measures of health status, the absolute values of any quantitative estimates are of limited utility to begin with in assessing the model. Yet the size of the coefficients can be compared with each other to gain insight into which exogenous variables have the strongest effect on health capital.

A random effects model and the cluster-robust standard errors are used. It is not possible to use fixed effect models in ordered logit/probit panel data regressions, as such estimators have not

yet been develope Cluster-robust standard errors are used to account for group influences such as family, school and neighbourhood which may affect the child's health. I assume that the error term u_{iw} has a zero mean and a negligible correlation with α_i and that α_i is not correlated with any of the exogenous variables. The term α_0 is also eliminated using my model. The regression results are displayed in *Figure 4.3* and *Figure 4.4*, computed using the statistical package Stata. The former is the ordered logit model while the latter is the ordered probit model.

In both cases as shown by *Figure 4.3* and *Figure 4.4*, with the exception of $theta3$, $theta2$ and $theta1$, dummy variables generated by w_ypacvwell and representing the model variable θ, the relative importance of education, the directions of effect are consistent with my theoretical predictions. Furthermore, this result from the variable θ which is contrary to my theoretical prediction is statistically insignificant so is of minor concern.

There are several possible reasons for this contradiction. Firstly, I am unable to measure or account for the variables ω and G, the marginal product of health capital in the accumulation of educational capital and the marginal product of a unit of health capital, respectively. As these variables affect the optimal stock of health capital, subsumed within the error term u_{it}, it is possible that the coefficients for $theta3$, $theta2$ and $theta1$ are statistically biased. This is a form of omitted variable bias and although my theory suggests that the direction of bias is likely to be that the coefficients of θ overestimates rather than underestimates, it is by no means certain. This is because it is also possible that individuals with higher ω and G have an incentive to under-invest in health because less health is required to produce any given stock of a) education and b) flow of healthy time, hence the coefficients of θ will be underestimates of the true value or biased in a downward direction. If the bias is corrected then the coefficients for θ will be positive. Secondly, to the extent that ω and G correlate with θ, though not stated in the theoretical model, biased estimates of $theta3$, $theta2$ and $theta1$ will also be produced. If the correlations are positive then the bias will be in the upward direction and vice versa if the correlations are negative. The third possibility, is that the stock of health capital does not actually enhance the accumulation of educational capital (beyond the generation of healthy time which can be devoted to study), which would be characterized by $\omega = 0$, implying $\frac{\partial H^*(t)}{\partial \theta} = 0$.

It should be noted that the relationship between optimal health and M is positive, contrary to findings reported in most studies. This is most likely the result of selecting consumption of healthy goods (fruit and vegetable portions), rather than medical care/services as the primary goods input to the production of health. This variable is selected as the youth questionnaire in the Understanding Society survey lacks complete data on child medical use. Likewise, the consumption of unhealthy goods (fast food) is very strongly correlated with poor health, representing the rate of health depreciation δ.

Most of the variables associated with t or age are not significant despite being of the expected sign. This can be somewhat expected as the time path for health is non-monotonic though it should be increasing for most t. The same may be said of the variable q. As can be seen from *Figure 4.2* the effects of variations in q are not pronounced for young ages, which may explain the lack of statistical significance, since the data used here is obtained from the youth questionnaire.

The model is unlikely to suffer significantly from missing data bias, since as can be seen in column three of *Table 4.1* missing values constitute a small proportion of all variables, except for w_yplvhm, the age an individual thinks he/she will leave home. When this variable is removed and the regressions re-computed (see *Figure 4.17*) the differences with the original logit and probit models are negligible. This strongly suggests that the missing values of w_yplvhm is not systematic. *Figure 4.18* shows the relationship between the probability of the dependent variable (health status) being missing as a function of the independent variables. There is no correlation for any variable. This again demonstrates that the missingness of data is random. Children which consume more fast food (higher health depreciation rate) for example, are no more likely to not report their health status compared to those who consume less fast food. It would be problematic if there is correlation of missingness across key independent variables, yet this is not the case.

Figure 4.3. Empirical test of equation (4.1): random effects logit model of an unbalanced panel dataset with cluster-robust standard errors

Group variable: **pidp** — Number of groups = **6,574**

Random effects u_i ~ **Gaussian** — Obs per group:

min = **1**
avg = **1.3**
max = **3**

Integration method: **mvaghermite** — Integration pts. = **12**

Wald chi2(**19**) = **309.07**
Log pseudolikelihood = **-7183.3448** — Prob > chi2 = **0.0000**

(Std. Err. adjusted for **6,574** clusters in pidp)

H	Coef.	Robust Std. Err.	z	P>|z|	[95% Conf.	Interval]
beta2	-.1102442	.0748912	-1.47	0.141	-.2570282	.0365398
beta3	-.3813947	.0859437	-4.44	0.000	-.5498412	-.2129482
A2	.5788697	.0801822	7.22	0.000	.4217155	.7360239
A1	1.222343	.0947448	12.90	0.000	1.036646	1.408039
theta3	-.3640698	.5493283	-0.66	0.507	-1.440733	.7125938
theta2	-.8231032	.4965693	-1.66	0.097	-1.796361	.1501547
theta1	-.7004187	.4933072	-1.42	0.156	-1.667283	.2664456
M3	.4196901	.1545306	2.72	0.007	.1168157	.7225645
M2	.3605476	.1550239	2.33	0.020	.0567063	.664389
M1	.4176448	.1707796	2.45	0.014	.082923	.7523665
delta3	-.2490121	.073341	-3.40	0.001	-.3927579	-.1052664
delta2	-.3971899	.0925208	-4.29	0.000	-.5785274	-.2158524
delta1	-.9405401	.2527353	-3.72	0.000	-1.435892	-.445188
t2	.2340248	.1260068	1.86	0.063	-.0129439	.4809935
t3	.1197029	.1171305	1.02	0.307	-.1098686	.3492744
t4	.1535404	.1246034	1.23	0.218	-.0906778	.3977585
t5	.3065445	.123748	2.48	0.013	.0640029	.5490861
t6	.2803288	.1262294	2.22	0.026	.0329237	.5277339
q	.0055573	.0086013	0.65	0.518	-.011301	.0224155
/cut1	-3.111165	.5615488	-5.54	0.000	-4.21178	-2.010549
/cut2	-.4040511	.5556166	-0.73	0.467	-1.49304	.6849375
/sigma2_u	2.385339	.2650772			1.918484	2.965802

Figure 4.4. Empirical test of equation (4.1): random effects probit model of an unbalanced panel dataset with cluster-robust standard errors

```
Random-effects ordered probit regression        Number of obs      =       8,659
Group variable: pidp                            Number of groups   =       6,574

Random effects u_i ~ Gaussian                   Obs per group:
                                                              min =           1
                                                              avg =         1.3
                                                              max =           3

Integration method: mvaghermite                 Integration pts.   =          12

                                                Wald chi2(19)      =      317.75
Log pseudolikelihood  = -7185.6879              Prob > chi2        =      0.0000

                                (Std. Err. adjusted for 6,574 clusters in pidp)
```

H	Coef.	Robust Std. Err.	z	P>|z|	[95% Conf.	Interval]
beta2	-.0643282	.0429005	-1.50	0.134	-.1484117	.0197552
beta3	-.2169486	.0491503	-4.41	0.000	-.3132813	-.1206158
A2	.3394681	.045676	7.43	0.000	.2499448	.4289913
A1	.7042112	.0536285	13.13	0.000	.5991013	.8093212
theta3	-.1786288	.3068239	-0.58	0.560	-.7799925	.422735
theta2	-.4393397	.2761046	-1.59	0.112	-.9804949	.1018154
theta1	-.3670633	.274293	-1.34	0.181	-.9046677	.1705412
M3	.241332	.0880757	2.74	0.006	.0687068	.4139571
M2	.2070294	.088498	2.34	0.019	.0335764	.3804824
M1	.2391717	.0975765	2.45	0.014	.0479253	.4304181
delta3	-.1386457	.0418924	-3.31	0.001	-.2207534	-.056538
delta2	-.2242047	.052862	-4.24	0.000	-.3278123	-.1205971
delta1	-.5328383	.1433514	-3.72	0.000	-.8138019	-.2518748
t2	.1306865	.0722355	1.81	0.070	-.0108924	.2722655
t3	.0628121	.0671753	0.94	0.350	-.068849	.1944732
t4	.0794008	.0714351	1.11	0.266	-.0606094	.2194111
t5	.159728	.0708906	2.25	0.024	.0207851	.298671
t6	.1504019	.0722628	2.08	0.037	.0087694	.2920344
q	.0034789	.0049078	0.71	0.478	-.0061401	.013098
/cut1	-1.730723	.3137582	-5.52	0.000	-2.345678	-1.115768
/cut2	-.1986437	.3102125	-0.64	0.522	-.806649	.4093616
/sigma2_u	.7847275	.0853017			.6341489	.9710609

4.2. Adult Model Empirical Investigation

The adult model's theoretical predictions are presented in section 3.3.2. There are two models relating to adult health which can be tested. The first is the case where the terminal time V is fixed, expressed by equation (3.21). The second case is that of a variable terminal time in which the planning horizon is an endogenous variable to be optimized, V^*, and expressed by equation (3.21''). The first case of fixed terminal time/planning horizon as shown by equation (3.21) is not particularly interesting, as it suggests that the optimal stock of health is independent of many exogenous variables of interest such as income y, and influenced only by G, A and V,

which are difficult to measure. Therefore, my primary interest in empirically verifying the adult model is in testing hypotheses generated by equation (3.21'').

I use data from the British Household Panel Survey (BHPS) to empirically verify the adult model developed by equation (3.21''). The BHPS is the fore-runner to the Understanding Society survey and contains 18 waves of data from 1990 to 2008 before being continued by Understanding Society. Compared to the Understanding Society Survey, the BHPS is more complete spanning a longer time period. Since most of the variables can be found in the BHPS, it is more preferable over Understanding Society. Documentation for the data I use can be accessed online.[89] The data files are accessible via the UK data archive, Study Number 5151.[90]

Just as in the case of the child model, the dependent variable is an ordered categorical non-continuous variable. I use the variable described as 'health over the last 12 months'. The possible responses are as follows: 'Excellent', 'Good', 'Fair', 'Poor', 'Very Poor', 'Missing or wild', 'Don't know', coded by the numbers 1, 2, 3, 4, 5, -9 and -1, respectively. Respondents in the last two categories are removed. 'Excellent' and 'Good' are switched to 5 and 4 while 'Very poor' and 'Poor' are switched to 1 and 2, respectively. The category 3 still represents 'Fair'. In this way the numbers 1-5 denote categories of progressively improving health. The main socio-economic variables which I seek to test in the adult model are income (y), education ($E(q)$) and age (t). Unlike in the child model however, the majority of independent/exogenous variables are continuous rather than categorical. The two exceptions are 'Concentration' and 'Highest academic qualification'. The former is chosen to represent the theoretical variable A, since concentration or mental alertness may be seen as a factor which affects the efficiency of all kinds of input whether time or monetary. The latter is chosen to denote the educational capital which the individual had accumulated upon completion of childhood, $E(q)$. *Table 4.3* below shows the variables selected from the BHPS and the corresponding theoretical variables in equation (3.21''). The final variable shown in the last row is calculated from two variables. Since t in equation (3.21'') denotes years lived *after* childhood, I subtract the reported age (wage12) from school leaving age (wscend) which is used to represent q. As in the child model,

[89] https://www.iser.essex.ac.uk/bhps/documentation/volb/allwaves.html

[90] University of Essex. Institute for Social and Economic Research. (2010). *British Household Panel Survey: Waves 1-18, 1991-2009.* [data collection]. *7th Edition.* UK Data Service. SN: 5151, http://dx.doi.org/10.5255/UKDA-SN-5151-1

not all the theoretical variables have the appropriate data representation and thus their effect on the optimal stock of health capital for an adult cannot be tested at present.

Table 4.3. Questionnaire variables selected from BHPS to empirically test the adult model in equation (3.21'')

Questionnaire variable	Variable description	Possible values	Data summary	Dummy categories	Associating theoretical model variable
whlstat	Health over last 12 months	1=excellent,2=good,3=fair,4=poor, 5=very poor;-9=missing or wild; -7: proxy respondent; -1=don't know; -2 refused	N: 235961; Groups: 31991; Average group size: 7.376 Category distribution: -9: 39 (0.02%), -7: 431 (0.18%); -1: 72 (0.03%); -2: 4 (0.00%); 1: 54457 (23.08%); 2: 106155 (44.99%); 3: 50298 (21.32%); 4: 18887 (8.00%); 5: 5618 (2.38%)	-	$H(t)$ or $GH(t)$ The stock of health capital or flow of healthy time
wghqa	Concentration	1=better than usual;2=same as usual;3=less than usual;4=much less than usual;-9=missing or wild;-7=proxy respondent; -1=don't know; -2=refused	N: 235961; Groups: 31991; Average group size: 7.376 Category distribution: -9: 5526 (2.34%), -7: 11533 (4.89%); -1: 32 (0.01%); -2: 3 (0.00%); 1: 13432 (5.69%); 2: 163643 (69.35%); 3: 36416 (15.43%); 4: 5376 (2.28%)	{A1=1,A2=2,A3=3,A4=4}; reference category: A4	A the efficiency parameter governing the input of goods and time to health
wncigs	Number of cigarettes smoked	All non-negative values possible	N: 235961; Groups: 31991; Average group size: 7.376 -9 denotes missing value, there are 583 missing values (0.025%); range: 0-81. Mean: 15.03; Standard deviation: 8.56	-	δ_0 the exogenous rate of health depreciation
wfimnl	Labour income: last month (range from 0.08-11247.00)	All non-negative values possible	N: 235961; Groups: 31991; Average group size: 7.376	-	y income from work (wage rate)

			-9 denotes missing value, there are 83 missing values, and 5781 negative values (2.49%); range 0-72055.43; Mean: 800.14; Standard deviation: 1176.14		
wqfachi	Highest academic qualification	1=Higher degree; 2=1st degree;3=hnd,hnc,teaching;4=A level,5=O level;6=GCSE;7=none of these;-7=proxy,-9=missing	N: 235961; Groups: 31991; Average group size: 7.376 Category distribution: -9: 2056 (0.89%); -7 5846 (2.54%); 1: 5275 (2.29%); 2: 21323 (9.27%); 3: 14766 (6.42%); 4: 40619 (17.65%); 5: 55826 (24.26%); 6: 11305 (4.91%); 7: 73.081 (31.76)	{E1=1,E2=2,E3=3,E4=4,E5=5,E6=6,E7=7} Reference category: E6	$E(q)$ Stock of educational capital
wage12 minus wscend	Difference between age and school leaving age (range from 0-89)	All non-negative values possible	N: 235961; Groups: 31991; Average group size: 7.376 8 missing or incorrect values (0.00% of data). Range: 0-108; Mean: 50.91; Standard Deviation: 20.80	-	t age or time counted from the beginning of the adulthood phase

Since I specify that income is a function of education, it is not possible to put both income and education in a regression model. The ideal case would be to use education as an instrumental variable to estimate the effect of income on the stock of health capital. However, it is difficult to perform two-staged-least-squares (2SLS) on panel data in which the dependent variable is ordinally categorical. Furthermore, it is possible that education correlates with errors in the equation, or other factors not accounted for which also affect health, and thus making it unsuitable to act as an instrumental variable. I therefore develop two econometric models as shown by equations (4.2) and (4.3).

$$H_{iw}^{*}(t) = \alpha_0 + \alpha_i + \phi_1 A3_{iw} + \phi_2 A2_{iw} + \phi_3 A1_{iw} + \phi_4 delta_{iw} + \phi_5 y_{iw} + \phi_6 t_{iw} + u_{iw}$$

(4.2)

$$H_{iw}^{*}(t) = \alpha_0 + \alpha_i + \phi_1 A3_{iw} + \phi_2 A2_{iw} + \phi_3 A1_{iw} + \phi_4 delta_{iw} + \phi_5 E5_{iw} + \phi_6 E4_{iw} + \phi_7 E3_{iw} + \phi_8 E2_{iw} + \phi_9 E1_{iw} + \phi_{10} E7_{iw} + \phi_{11} t_{iw} + u_{iw} \quad (4.3)$$

Where i and w denote individuals and waves of data, respectively. The terms y_{iw} and $delta_{iw}$ denote income and the exogenous rate of health depreciation, respectively, represented by the questionnaire variables 'Labour income: last month' and 'Number of cigarettes smoked', respectively. t_{iw} represents the variable computed by subtracting 'school leaving age' from 'age'. The dummy categories for A are listed in increasing order from left to right in equations (4.2) and (4.3). For education, I choose $E6$, GCSE, as reference category, being the lowest form of qualification made explicit in the data. Therefore, the education variables in equation (4.3) are also listed in increasing order from left to right, with the exception of $E7$, which represents the category 'none of these'.

In order to determine the sign of the coefficients in equations (4.2) and (4.3), I use comparative dynamic analysis and partially differentiate equation (3.21'') with respect to the exogenous variables. The results of the partial derivatives and the expected signs are shown below in *Table 4.4*. It should be noted that the partial derivatives of optimal health with respect to income and education are exactly the same except that the latter derivative is the scaling of the former by $y'(E(q))$, the change in income with respect to a change in a unit of education, which I expect to be positive. I make no assumption regarding the form of the income and education function.

Table 4.4. Partial derivatives of equation (3.21'') with respect to the exogenous variables in column four Table 4.3.

Partial derivative	Equation of partial derivative	Theoretical prediction
$\frac{\partial H^{*}(t)}{\partial A}$	A complicated function too lengthy to be shown here	Sign of $\frac{\partial H^{*}(t)}{\partial A}$ indeterminate but should be positive for typical parameter values
$\frac{\partial H^{*}(t)}{\partial \delta_0}$	$-(A\delta_0)^{-1}\left(\frac{y^{A+1}}{A\delta_0 P_M P_X^A} e^{A(GH_0-1)-2+\frac{P_M}{y}} - Gt\right)^{-1} t$	$\frac{\partial H^{*}(t)}{\partial \delta_0} < 0 \; \forall \; t \in [0, V]$[91]
$\frac{\partial H^{*}(t)}{\partial y}$	$(Ay)^{-1}\left(\frac{y^{A+1}}{A\delta_0 P_M P_X^A} e^{A(GH_0-1)-2+\frac{P_M}{y}} - Gt\right)^{-1}\left(A+1-\frac{P_M}{y}\right)$	$\frac{\partial H^{*}(t)}{\partial y} > 0 \; \forall \; t \in [0, V]$

[91] I assume that $Gt < \frac{y^{A+1}}{A\delta_0 P_M P_X^A} e^{A(GH_0-1)-2+\frac{P_M}{y}} \; \forall \; t \in [0, V]$

$\frac{\partial H^*(t)}{\partial E(q)}$	$\frac{\partial H^*(t)}{\partial y} y'(E(q))$	$\frac{\partial H^*(t)}{\partial E(q)} < 0 \; \forall \; t \in [0, V]$
$\frac{\partial H^*(t)}{\partial t}$	$-A^{-1}\left(\frac{y^{A+1}}{A\delta_0 P_M P_X^A} e^{A(GH_0-1)-2+\frac{P_M}{y}} - Gt\right)^{-1}$	$\frac{\partial H^*(t)}{\partial t} < 0 \; \forall \; t \in [0, V]$

The effect of parametric changes can also be illustrated graphically using calibration. They are shown below in *Figure 4.5*. The change in education on the optimal stock of health capital is not shown since I do not assume a specific functional form for the education-income equation, yet its general form should be very similar to the effect of a change in income. It should be noted that all changes in exogenous variables affect the length of life or planning horizon either positively or negatively. An increase in the efficiency of health inputs, A and income, y, increases V^*. A rise in the exogenous rate of depreciation δ_0 on the other hand, lowers V^*. Education $E(q)$ has the same effect as income. Using *Table 4.4* and *Figure 4.5*, I can develop hypotheses regarding the expected signs of the coefficients in equations (4.2) and (4.3). For equation (4.2) I expect $\phi_{1-3,5}$ to be positive and $\phi_{4,6}$ to be negative. For equation (4.3) I expect I expect ϕ_{1-3} to be positive and $\phi_{4,11}$ negative. ϕ_{5-10} should also be positive with successively larger coefficients. However, I cannot tell whether ϕ_{10} should be positive or negative as it is not immediately clear how 'None of these' compares to 'GCSE' in relation to the stock of education. It is possible though that this category contains significant numbers of those without any formal qualification at all which may imply a negative ϕ_{10}.

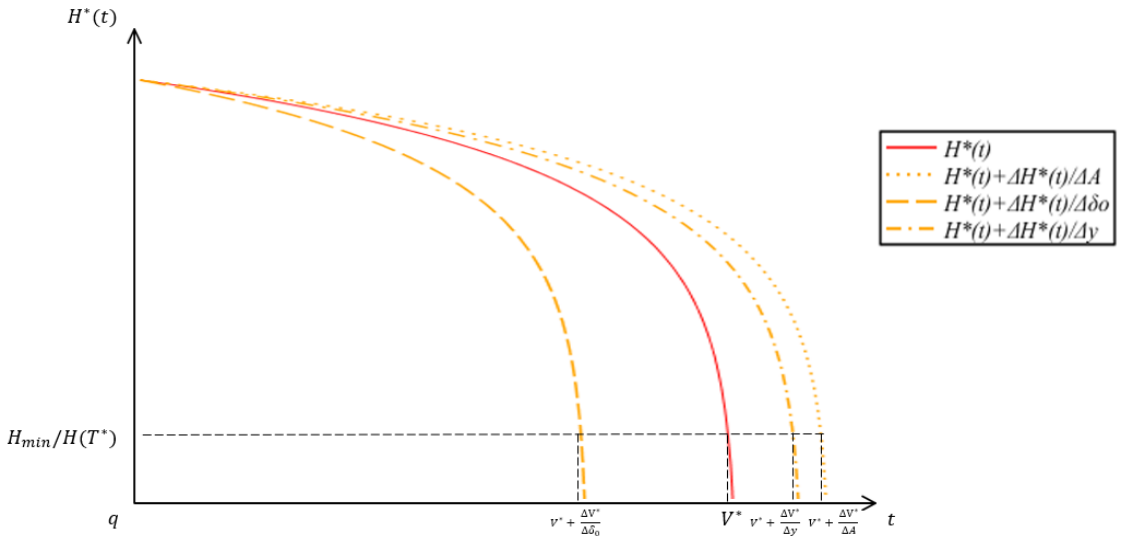


Figure 4.5. Comparative dynamics of the adult model as shown by equation (3.21''), partial derivatives of optimal health capital with respect to the variables in column four Table 4.3

Using data from the BHPS I construct an unbalanced panel dataset of the variables from *Table 4.3*. I apply the random effects logit and probit models to test for equations (4.2) and (4.3). This model is chosen for the same reason as for the empirical testing of the child model expressed by equation (3.14) and its empirical counterpart (4.1), i.e. that a fixed effect estimator has not been developed for ordered logistic/probit models involving panel data. I also use the cluster-robust standard errors in the regression analysis to account for variances due to group effects. The model allows us to eliminate α_0 and α_i from equations (4.2) and (4.3) and I assume that the latter is uncorrelated with the error term u_{iw}. The results of the regression are shown below in *Figure 4.6* to *Figure 4.9*.

As shown in *Figure 4.6* and *Figure 4.7* which test for equation (4.2), the signs of all the variables conform to my theoretical predictions as reported in the partial derivatives in *Table 4.4* and illustrated by *Figure 4.5*. All the variables are highly significant statistically. Efficiency levels (measured by ability to concentrate) and income positively affect optimal health while the exogenous rate of health depreciation (measured by number of cigarettes smoked) and age (difference between age and school leaving age), negatively impact on health. The conclusions are the exact same for both the logit and probit models, which suggests robustness of results.

For *Figure 4.8* and *Figure 4.9* which are the regression results for equation (4.3), all the variables again are of the expected sign as predicted by the partial derivatives shown in *Table 4.4*. The predictions of both the ordered logit and probit models are the same. With the exception of $E5$ and $E6$, all the variables are highly significant. This is somewhat expected since $E5$ denotes O-levels which is essentially equivalent to GCSE prior to education reforms, hence their difference if any remains minimal. Similarly, $E6$ represents professional qualifications rather than higher levels of education per se, although I can perhaps expect those who hold such a qualification to be relatively well educated. $E6$ is therefore somewhat significant, at the 10% level. As shown by *Figure 4.8* and *Figure 4.9*, efficiency levels increase optimal health while health depreciation and age reduce it.

As can be seen from column 3 in *Table 4.3*, the missing values constitute a small proportion of to the total observations. *Figure 4.19* shows that the probability of the dependent variable missing has correlations with A, δ_0 and y – the efficiency parameter, the exogenous rate of health depreciation and income, respectively. However, with the exception for A, the coefficients are small, which suggests that missingness does not vary significantly along the socio-economic lines of δ_0 and y.

Figure 4.6. Empirical test of equation (4.2): random effects ordered logit model of an unbalanced panel dataset with cluster-robust standard errors

```
Random-effects ordered logistic regression      Number of obs      =     58,553
Group variable: pid                             Number of groups   =     11,235

Random effects u_i ~ Gaussian                   Obs per group:
                                                              min =          1
                                                              avg =        5.2
                                                              max =         18

Integration method: mvaghermite                 Integration pts.   =         12

                                                Wald chi2(6)       =    2092.99
Log pseudolikelihood  = -66695.845              Prob > chi2        =     0.0000

                                (Std. Err. adjusted for 11,235 clusters in pid)
```

H	Coef.	Robust Std. Err.	z	P>|z|	[95% Conf.	Interval]
A3	.8350409	.0634155	13.17	0.000	.7107487	.959333
A2	1.67065	.0638947	26.15	0.000	1.545419	1.795882
A1	1.934056	.0745099	25.96	0.000	1.788019	2.080093
delta	-.0132205	.0018001	-7.34	0.000	-.0167487	-.0096923
y	.000308	.0000198	15.52	0.000	.0002691	.0003468
t	-.0166793	.0008236	-20.25	0.000	-.0182935	-.0150651
/cut1	-12.13425	.7301377	-16.62	0.000	-13.56529	-10.7032
/cut2	-4.460129	.085608	-52.10	0.000	-4.627917	-4.29234
/cut3	-2.212976	.0790694	-27.99	0.000	-2.367949	-2.058003
/cut4	.0624335	.0773516	0.81	0.420	-.0891729	.21404
/cut5	3.249196	.0794035	40.92	0.000	3.093568	3.404823
/sigma2_u	3.566124	.0927973			3.388805	3.752721

Figure 4.7. Empirical test of equation (4.2): random effects ordered probit model of an unbalanced panel dataset with cluster-robust standard errors

```
Random-effects ordered probit regression        Number of obs      =     58,553
Group variable: pid                             Number of groups   =     11,235

Random effects u_i ~ Gaussian                   Obs per group:
                                                              min =          1
                                                              avg =        5.2
                                                              max =         18

Integration method: mvaghermite                 Integration pts.   =         12

                                                Wald chi2(6)       =    2106.52
Log pseudolikelihood  = -67076.393              Prob > chi2        =     0.0000

                                (Std. Err. adjusted for 11,235 clusters in pid)
```

H	Coef.	Robust Std. Err.	z	P>|z|	[95% Conf.	Interval]
A3	.4420598	.0354889	12.46	0.000	.3725028	.5116169
A2	.9223993	.0357801	25.78	0.000	.8522717	.9925269
A1	1.065152	.0416118	25.60	0.000	.9835945	1.14671
delta	-.0073502	.0010106	-7.27	0.000	-.0093311	-.0053694
y	.0001708	.0000113	15.11	0.000	.0001486	.0001929
t	-.0093567	.0004595	-20.36	0.000	-.0102572	-.0084561
/cut1	-5.746138	.3055144	-18.81	0.000	-6.344935	-5.147341
/cut2	-2.438974	.047176	-51.70	0.000	-2.531437	-2.346511
/cut3	-1.250261	.0439487	-28.45	0.000	-1.336399	-1.164124
/cut4	.0122463	.0433039	0.28	0.777	-.0726279	.0971204
/cut5	1.80286	.0444393	40.57	0.000	1.715761	1.88996
/sigma2_u	1.068561	.0279431			1.015173	1.124756

Figure 4.8. Empirical test of equation (4.3): random effects ordered logit model of an unbalanced panel dataset with cluster-robust standard errors

Random-effects ordered logistic regression — Number of obs = 57,966
Group variable: **pid** — Number of groups = 11,068

Random effects u_i ~ **Gaussian** — Obs per group:
min = 1
avg = 5.2
max = 18

Integration method: **mvaghermite** — Integration pts. = 12

Wald chi2(**11**) = 2139.03
Log pseudolikelihood = **-66093.224** — Prob > chi2 = 0.0000

(Std. Err. adjusted for **11,068** clusters in pid)

H	Coef.	Robust Std. Err.	z	P>|z|	[95% Conf.	Interval]
A3	.8508894	.064093	13.28	0.000	.7252695	.9765093
A2	1.699332	.0646273	26.29	0.000	1.572664	1.825999
A1	1.9505	.0754338	25.86	0.000	1.802653	2.098348
delta	-.0098561	.0018423	-5.35	0.000	-.0134669	-.0062453
E5	.0956477	.0814462	1.17	0.240	-.0639838	.2552793
E4	.1458571	.0861238	1.69	0.090	-.0229424	.3146567
E3	.4061438	.1135863	3.58	0.000	.1835187	.628769
E2	.4909291	.1092659	4.49	0.000	.2767719	.7050863
E1	.6417495	.1973413	3.25	0.001	.2549678	1.028531
E7	-.5186691	.0840644	-6.17	0.000	-.6834322	-.353906
t	-.013434	.000873	-15.39	0.000	-.015145	-.0117231
/cut1	-12.23396	.7354408	-16.63	0.000	-13.6754	-10.79252
/cut2	-4.55542	.109664	-41.54	0.000	-4.770357	-4.340482
/cut3	-2.311331	.1043376	-22.15	0.000	-2.515829	-2.106833
/cut4	-.0298242	.1027236	-0.29	0.772	-.2311588	.1715104
/cut5	3.162147	.1040342	30.40	0.000	2.958244	3.36605
/sigma2_u	3.714987	.0950328			3.533318	3.905996

Figure 4.9. Empirical test of equation (4.3): random effects ordered probit model of an unbalanced panel dataset with cluster-robust standard errors

Random-effects ordered probit regression — Number of obs = 57,966
Group variable: **pid** — Number of groups = 11,068

Random effects u_i ~ **Gaussian** — Obs per group:
min = 1
avg = 5.2
max = 18

Integration method: **mvaghermite** — Integration pts. = 12

Wald chi2(11) = 2155.38
Log pseudolikelihood = **-66480.955** — Prob > chi2 = 0.0000

(Std. Err. adjusted for **11,068** clusters in pid)

H	Coef.	Robust Std. Err.	z	P>|z|	[95% Conf.	Interval]
A3	.4505155	.0358397	12.57	0.000	.3802711	.52076
A2	.9383113	.0361573	25.95	0.000	.8674443	1.009178
A1	1.073704	.0420903	25.51	0.000	.9912084	1.156199
delta	-.005397	.0010373	-5.20	0.000	-.0074301	-.0033639
E5	.0633337	.0449958	1.41	0.159	-.0248564	.1515238
E4	.0881083	.0473337	1.86	0.063	-.0046641	.1808807
E3	.2398093	.0628281	3.82	0.000	.1166686	.3629501
E2	.2770148	.0604896	4.58	0.000	.1584574	.3955723
E1	.3742786	.1093158	3.42	0.001	.1600235	.5885337
E7	-.2762562	.0463344	-5.96	0.000	-.36707	-.1854424
t	-.007597	.0004873	-15.59	0.000	-.0085522	-.0066419
/cut1	-5.788737	.3111217	-18.61	0.000	-6.398525	-5.17895
/cut2	-2.48108	.0603669	-41.10	0.000	-2.599397	-2.362763
/cut3	-1.295207	.057615	-22.48	0.000	-1.408131	-1.182284
/cut4	-.0306536	.0569495	-0.54	0.590	-.1422726	.0809654
/cut5	1.762493	.0577012	30.55	0.000	1.6494	1.875585
/sigma2_u	1.112764	.0285827			1.05813	1.170219

4.3. Life Expectancy Model Empirical Investigation

As a bi-product of the adult model, an equation of the optimal length of life and exogenous variables is derived as shown by equation (3.23). The optimal length of life may be interpreted as life expectancy. Equation (3.23) cannot be tested using individual data since life expectancy or optimal length of life cannot be observed for any individual and cannot be accurately predicted due to large idiosyncrasies. The actual length of life which are observable in retrospect of the death of the individual are perhaps conceptually closer to optimal length of life than life expectancy yet these values cannot be observed for any individual in the data since they are all still alive. It is difficult to obtain such data since this would require the death certificates and the socio-economic and health behaviour information of those individuals when they were alive. I therefore must use national level average of life expectancy to represent

optimal length of life in an attempt to empirically verify my model. Life expectancy data cannot be obtained for individuals since they are numbers or estimates pertaining to a given population rather than individuals in most cases, hence I must use national level data for this analysis. Since my model predicts life expectancy using solely socio-economic data, there can be large individual factors affecting health which are unaccounted for in my model, such as genetic variations. A main advantage of using national level data is that many of these idiosyncrasies are averaged out at the national level and there is little evidence to believe there are large systematic variations between different national populations which would cause inherent health differentiation at the national level. A potential problem of using national level aggregate data however, is that some of my model theoretical predictions, which are based on individual or microeconomic analysis may not readily apply at the national level. Nonetheless due to data limitation I may only use national level or some other form of aggregate data to empirically test equation (3.23).

To obtain the corresponding socio-economic data I use World Bank DataBank tool which is accessible online.[92] I search for the variables 'Life expectancy at birth, total (years)', 'PM2.5 air pollution, mean annual exposure (micrograms per cubic meter)' and 'GDP per capita (current US$)' for the years 1990 to 2013, for over 180 countries in the world. This covers all the UN countries including the UK. The UK ranks approximately halfway among all the countries in terms of ambient PM2.5 concentration at 15.50 ppm. In terms of life expectancy, the UK is considered high at 81 years. The time period is selected since this period contains the most complete dataset and are the same years used in the Global Burden of Disease (GBD) 2013 study, which is referred to later. Unfortunately, since significant time data points are missing for 'PM2.5 air pollution, mean annual exposure (micrograms per cubic meter)', the data is only available for the most part in five-year intervals between 1990 and 2013. Only the following years are available and used to construct my panel data: 1990, 1995, 2000, 2005, 2010 and 2013.

$PM_{2.5}$ represents the exogenous rate of health depreciation δ_0 while GDP per capita primarily represents income y but is also likely to reflect A, G, H_0 and $E(q)$, which are often highly correlated with a country's GDP per capita – the level of technology, the marginal product of

[92] http://databank.worldbank.org/data/home.aspx

health capital, the stock of health at birth/at beginning of adulthood and education, respectively. I develop an econometric model as shown by equation (4.4) below:

$$V_{jY}^{*} = \alpha_0 + \alpha_j + \phi_1 PM25_{jY} + \phi_2 \ln(GDPperCap_{jY}) + u_{jY} \qquad (4.4)$$

Where j denotes a particular country and Y denotes the year. α_j refers to the country specific factors constant over time which affect optimal length of life/life expectancy.

Instead of using $GDPperCap_{jY}$ as a regressor, I adopt the logarithmic form. From an empirical perspective, the gains in life expectancy as a result of increasing GDP per capita is diminishing. It is perhaps obvious that even despite high material income and advanced medical technology, a country's life expectancy will eventually plateau due to the biological constraints of human lifespan. It is therefore more appropriate to specify that the life expectancy is a function of the logarithm of GDP per capita, which shows that a given *percentage* increase in GDP per capita causes a given unit of increase in life expectancy, thus reflecting this diminishing increase. This is the approach taken by Preston (1975) who observed a logarithmic relationship between national income and life expectancy across countries. On the other hand, the effect of $PM2.5_{jY}$ on life expectancy remains linear, according to the specification in equation (4.4). This is because the effect on life expectancy of air pollution is unlikely to be diminishing. According to epidemiological studies, they are likely to be linear or even exponential, where a threshold level if breached would drastically increase mortality (Pope et al., 2002; Pope et al., 2004).

In order to predict the signs of the coefficients in equation (4.4), I apply comparative dynamic analysis by taking the partial derivatives of the optimal length of life as shown by equation (3.23), with respect to the exogenous variables. *Table 4.5* below shows the partial derivative of the optimal length of life (life expectancy) with respect to the exogenous rate of health depreciation, income and education. The effect of education on the optimal length of life is treated in the same manner as income, with the exception that it is scaled by partial derivative of income with respect to education. From *Figure 4.5*, the effect of changes on the exogenous rate of health depreciation, income and education can be observed graphically.

Table 4.5. Partial derivatives of equation (3.23) or optimal length of life (life expectancy) with respect to the exogenous rate of health depreciation, income and education

Partial derivative	Equation of partial derivative	Theoretical prediction
$\frac{\partial V^{*}}{\partial \delta_0}$	$-(G\delta_0)^{-1}\left(1 - e^{-GA(H_0 - H_{min})}\right)\left(\frac{y^{A+1}}{A\delta_0 P_M P_X^A} e^{A(GH_0 - 1) - 2 + \frac{P_M}{y}}\right)$	$\frac{\partial V^{*}}{\partial \delta_0} < 0$

$\frac{\partial V^*}{\partial y}$	$(Gy)^{-1}\left(1-e^{-GA(H_0-H_{min})}\right)\left(\frac{y^{A+1}}{A\delta_0 P_M P_X^A}e^{A(GH_0-1)-2+\frac{P_M}{y}}\right)\left(A+1-\frac{P_M}{y}\right)$	$\frac{\partial V^*}{\partial y}>0$
$\frac{\partial V^*}{\partial E(q)}$	$\frac{\partial V^*}{\partial y}y'(E(q))$	$\frac{\partial V^*}{\partial y}>0$

I partially differentiate the exogenous variables twice in order to test for whether the effects of such variable changes are increasing or decreasing. The results are shown below in *Table 4.6*. It appears that the negative effect of an increase in the exogenous rate of health depreciation on the optimal length of life (life expectancy) is diminishing while for the positive effect of income on life expectancy tends to be increasing, given the signs of the second order derivatives displayed in the third column of *Table 4.6*.

Table 4.6. Second order partial derivatives of equation (3.23), with respect to the exogenous rate of health depreciation, income and education

Partial derivative	Equation of partial derivative	Theoretical prediction
$\frac{\partial^2 V^*}{\partial \delta_0^2}$	$2G^{-1}\left(1-e^{-GA(H_0-H_{min})}\right)\left(\frac{y^{A+1}}{A\delta_0^3 P_M P_X^A}e^{A(GH_0-1)-2+\frac{P_M}{y}}\right)$	$\frac{\partial^2 V^*}{\partial \delta_0^2}>0$
$\frac{\partial^2 V^*}{\partial y^2}$	$G^{-1}\left(1-e^{-GA(H_0-H_{min})}\right)\left(\frac{y^{A-3}}{A\delta_0 P_M P_X^A}e^{A(GH_0-1)-2+\frac{P_M}{y}}\right)(A(A+1)y^2-2AP_M y+P_M^2))$	Sign of $\frac{\partial^2 V^*}{\partial y^2}$ indeterminate but likely to be positive
$\frac{\partial^2 V^*}{\partial E(q)^2}$	$\frac{\partial^2 V^*}{\partial y^2}y''(E(q))$	Sign of $\frac{\partial^2 V^*}{\partial E(q)^2}$ indeterminate but likely to be positive

Figure 4.10 illustrates the effect of rising income and exogenous health depreciation on life expectancy. The arrows point towards the direction of increase. As can be seen the effect of the exogenous rate of health depreciation has a much larger impact on life expectancy than income, albeit at a diminishing rate. Applying this to the national level, one may argue that using a combination of reducing environmental pollution and economic development which raises average income and education (education has a similar effect on life expectancy as income), the life expectancy of a country can be increased gradually.

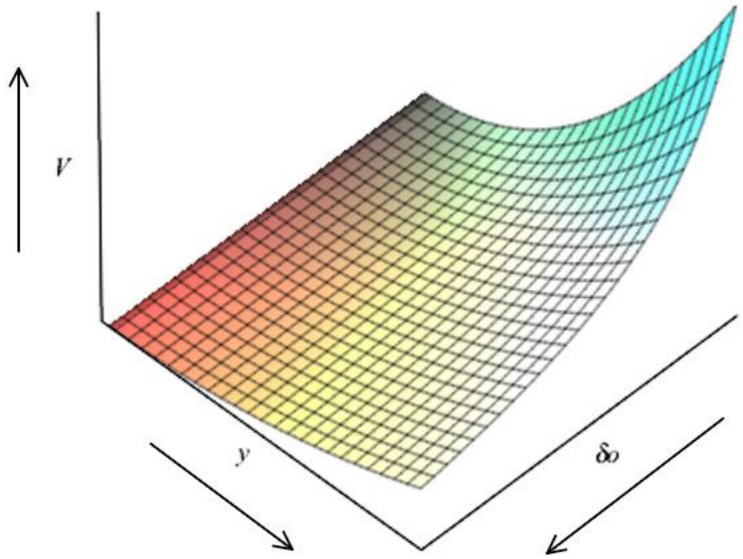


Figure 4.10. Graphical illustration of the effect of exogenous health depreciation (δ_0) and income (y) on life expectancy/optimal length of life (V^*)

Using the comparative dynamic analysis/partial derivatives of *Table 4.5*, I have some theoretical basis to make predictions regarding the sign of the coefficients in equation (4.4). I expect ϕ_1 to be negative and ϕ_2 to be positive. Unlike the previous two cases for estimating the child and adult models in section *4.1* and *4.2*, the dependent variable in this case is continuous and so there are significantly more econometric model options which can be chosen. The most suitable econometric model in this case is the between-effect model, which I apply to the unbalanced panel dataset constructed using World Bank data. This is because the between-effect model emphasises the cross-sectional aspect of the data more and my data contains large cross section cases relative to time periods (180 countries with 6 time points). I also report the more conventional fixed-effect model. The fixed-effect model has the advantage of capturing more time series variations in the data. For both models, α_0 and α_i are eliminated. The results of the regressions are shown below in *Figure 4.11* and *Figure 4.12* for the between-effect and fixed-effect models, respectively.

Figure 4.11. Empirical test of equation (4.4): between-effect model of an unbalanced panel dataset

```
Between regression (regression on group means)   Number of obs     =       935
Group variable: location                         Number of groups  =       164

R-sq:                                            Obs per group:
     within  = 0.4467                                          min =         1
     between = 0.6964                                          avg =       5.7
     overall = 0.6685                                          max =         6

                                                 F(2,161)          =    184.65
sd(u_i + avg(e_i.))=  5.255599                   Prob > F          =    0.0000
```

LEXP	Coef.	Std. Err.	t	P>|t|	[95% Conf.	Interval]
pm25	.020952	.0386099	0.54	0.588	-.0552951	.0971992
ln_GDPperCap	5.1425	.2702259	19.03	0.000	4.608856	5.676144
_cons	26.15827	2.413171	10.84	0.000	21.39272	30.92382

Figure 4.12. Empirical test of equation (4.4): fixed-effect model of an unbalanced panel dataset

```
Fixed-effects (within) regression                Number of obs     =       935
Group variable: location                         Number of groups  =       164

R-sq:                                            Obs per group:
     within  = 0.4474                                          min =         1
     between = 0.6940                                          avg =       5.7
     overall = 0.6665                                          max =         6

                                                 F(2,769)          =    311.25
corr(u_i, Xb)  = 0.3777                          Prob > F          =    0.0000
```

LEXP	Coef.	Std. Err.	t	P>|t|	[95% Conf.	Interval]
pm25	.0460399	.0323239	1.42	0.155	-.0174137	.1094935
ln_GDPperCap	3.674188	.1474157	24.92	0.000	3.384803	3.963573
_cons	37.2972	1.335698	27.92	0.000	34.67516	39.91925
sigma_u	5.7191042					
sigma_e	2.4402004					
rho	.84598662	(fraction of variance due to u_i)				

```
F test that all u_i=0: F(163, 769) = 27.21                     Prob > F = 0.0000
```

The results in *Figure 4.11* are consistent with my prior theoretical predictions. However, only GDP per capita is statistically significant but not $PM_{2.5}$ exposure. This is somewhat expected since measures of ambient pollution at the national level cannot accurately represent true levels of population exposure to the risk factor. Since the dependent variable is continuous, it is meaningful to interpret the magnitude of the coefficients using a linear model. From *Figure 4.11*, it shows that for every 1% increase in a country's GDP per capita, the life expectancy increases by 5.14 years when pollution levels are held constant. The coefficient estimate for $PM_{2.5}$ shows that for every 1 $\mu g/m^3$ increase in annual mean exposure, life expectancy is

increased by 0.210 years, though this estimate is unlikely to be accurate given that the coefficient is statistically insignificant. For *Figure 4.12* the coefficient of $PM_{2.5}$ is also positive, though also non-significant. GDP per capita is of the expected sign. Compared to the model in *Figure 4.11* however, the magnitude is substantially smaller, for every 1% increase in GDP per capita, life expectancy increases by 3.67 years.

Figure 4.13 below employs the fixed effect model which includes year dummy variables, with the year 1990 acting as the reference category. This is equivalent to including a time trend in a time series model, which captures the change in the dependent variable over time not attributed to the independent variables. All the year dummy variables are highly significant statistically and show progressively larger coefficient values as the year increases. This indicates that life expectancy for all countries have increased substantially between 1990 and 2013, irrespective of the increase in GDP per capita and ambient $PM_{2.5}$. As Preston (1975) observed, there is a tendency for life expectancy to increase over time, perhaps due to breakthrough in medical technology, sanitary standards and nutritional intake which permeate all parts of the world, leading to this rise regardless of the degree of economic development in those countries. The coefficient for GDP per capita is considerably reduced when year dummies are included and is no longer significant at the 1% level, though it remains significant at the 5% level. For a 1% increase in GDP per capita, life expectancy only increases by 0.527 years. The coefficient for $PM_{2.5}$ on the other hand actually increases and is now significant at the 5% level. However, it is highly unlikely that higher levels of $PM_{2.5}$ actually increases life expectancy, most likely heavy pollution is correlated with some aspects of economic development, such as the level of industrialisation, which may lead to an increase in life expectancy.

Figure 4.13. Empirical test of equation (4.4): fixed-effect model of an unbalanced panel dataset, including year dummies

```
Fixed-effects (within) regression               Number of obs      =       935
Group variable: location                        Number of groups   =       164

R-sq:                                           Obs per group:
     within  = 0.6003                                         min =         1
     between = 0.2708                                         avg =       5.7
     overall = 0.1892                                         max =         6

                                                F(7,764)           =    163.91
corr(u_i, Xb)  = 0.2019                         Prob > F           =    0.0000
```

LEXP	Coef.	Std. Err.	t	P>|t|	[95% Conf.	Interval]
pm25	.0718107	.0284382	2.53	0.012	.0159844	.127637
ln_GDPperCap	.5274666	.2320108	2.27	0.023	.0720123	.9829209
Y1995	.8097173	.2481079	3.26	0.001	.3226632	1.296771
Y2000	1.969462	.24892	7.91	0.000	1.480814	2.458111
Y2005	3.338259	.277806	12.02	0.000	2.792905	3.883612
Y2010	4.962118	.3358447	14.78	0.000	4.30283	5.621406
Y2013	5.778567	.3627524	15.93	0.000	5.066458	6.490677
_cons	58.88357	1.835313	32.08	0.000	55.28072	62.48643
sigma_u	8.9609697					
sigma_e	2.0820679					
rho	.94877936	(fraction of variance due to u_i)				

```
F test that all u_i=0: F(163, 764) = 38.93                     Prob > F = 0.0000
```

The positive relationship demonstrated between life expectancy and GDP per capita is in fact nothing new and merely confirms much of the previous research on this topic. However, my study provides an additional perspective to view the problem from the microeconomic level. Socio-economic variables at the individual level contribute to health behaviours which lead to differences in the optimal length of life or life expectancy. Although the conclusions from my microeconomic model may not be directly applied to the national level, yet a case can be made that to the extent that GDP per capita reflects systematic cross-country variations of average income, technology and other socio-economic factors, it results in variations in the average life expectancy between countries.

It should be noted that the theoretical model I develop predicts that rising income leads to progressively faster increase in life expectancy, rather than diminishing as predicted by Preston (1975). The econometric model in equation (4.4) reflects the theoretical predictions of Preston (1975) rather than my model. This is chosen since the national level data which are accessible cannot truly account for the variations in individual life expectancies, and conform better to Preston (1975)'s specification that increases in average life expectancies at the national level due to increase in GDP per capita are diminishing. While an increase in individual income

according to my model leads to an increase in the optimal length of life, which is interpreted as *individual* life expectancy, and further increase in income leads to progressively higher optimal length of life/individual life expectancy, the same may not be true if this rationale is applied at the national level. This is because my model assumes that the optimal length of life for individuals in general would be below the biological maximum and therefore have substantial room for increase. In fact in the model, there is no explicit maximum biological constraint imposed and it is assumed that the length of life is determined solely by optimality conditions attributable to resource allocation. It is therefore possible for the increase in optimal length of life or life expectancy due to a rise in income, to be increasing. On the other hand, the average life expectancy data at the national level include the lifespans of individuals who are not properly cared for, and children who die young, with infant mortality being one of the strongest drags on average life expectancy. Once the sufferings of these groups are eradicated, frequently via rising living standards brought about by economic development, it is no longer possible for countries to experience a significant surge in average life expectancies. Therefore, the observed diminishing increase in average life expectancy due to rising national incomes may simply reflect the eradication of extreme poverty and the associated health consequences, rather than that average life expectancy is approaching the biological maximum limit. If this is true then it is possible for life expectancy due to rising incomes, at least at the individual level to be increasing as my model predicts, shown by the second order partial derivatives in *Table 4.6*, or at least that the increase in life expectancy would be non-diminishing. Some commentators suggest that the next generation, millennials born after 1980 may be the first cohort to have an average life expectancy exceeding 100.[93] Certainly it would appear that even developed countries with the highest average life expectancy (in excess of 80 years) can expect to significantly further increase this figure, which has not yet reached the biological maximum.

Whilst it would be ideal to utilise individual data for optimal length of life or life expectancy, it is certainly not possible in my case. This is because the optimal length of life is not actually observable and does not fully translate into 'life expectancy'. The very concept or definition of life expectancy requires compiling aggregate data to estimate how long any given individual would live based on his/her group, sub-group, cohort and the characteristics such as socio-

[93] http://www.telegraph.co.uk/news/politics/11348561/Average-life-expectancy-heading-for-100.html. The article cited as source the Office of National Statistic life expectancy estimate: Statistical bulletin: Past and projected data from the period and cohort life tables, 2016-based, UK: 1981 to 2066

economic status, represented by the group the individual belongs to. The only option to obtain such data would be to acquire death certificates which would allow the investigation in retrospect, *after* the death of the individual thus revealing implicitly the individual's optimal length of life. Socio-economic data of the individual could be employed to examine how the age of death, another interpretation of the optimal length of life, are correlated with factors such as income and education levels whilst the individual was still alive. Whilst this approach would more resemble the theoretical propositions of my model, it would be difficult to obtain good estimates of socio-economic status after death. Furthermore, it would not be possible to apply the panel data method since the age of death is fixed, whilst I interpret the optimal length of life to be variable subject to changes in socio-economic status over time.[94] Therefore, I have no choice but to stick to life expectancy, which is an aggregate measure of optimal length of life.

Our model if applied strictly should generate an econometric model such as equation (4.5) below:

$$\ln(V_{jY}^{*}) = \alpha_0 + \alpha_j + \phi_1 PM25_{jY} + \phi_2 GDPperCap_{jY} + u_{jY} \qquad (4.5)$$

Once again using the partial derivatives of *Table 4.5*, ϕ_1 is expected to be negative and ϕ_2 positive. The model in equation (4.5) predicts that a given increase in GDP per capita raises the life expectancy by a given percentage, thus the increase is progressively larger. On the other hand since the coefficient for $PM25_{jY}$ is negative, an increase in one unit reduces the life expectancy by a given percentage. Since the reduction is by percentage, it is diminishing. The same regression models used to test equation (4.4) are applied to equation (4.5) also. The results are shown below by *Figure 4.14*, *4.15* and *4.16*, which show the between-effect model, fixed-effect model and fixed-effect model with time/year dummies included, respectively. For the between-effect model, the coefficient for $PM_{2.5}$ is in the expected direction, though non-significant. The effect of GDP per capita is highly significant – a $1,000 increase in GDP per capita increases the life expectancy by 0.668%. For the fixed-effect model, the coefficient of $PM_{2.5}$ is in the opposite direction to my expectation but not statistically significant. The coefficient for GDP per capita is in the expected direction and highly significant, though the magnitude is only approximately one third of the estimate generated by the between-effect

[94] Individuals can change their optimal length of life while they are still alive if there is a change in exogenous variables.

model – for a $1,000 increase in GDP per capita, the life expectancy increases by 0.220%. Surprisingly, when time dummy variables are included, the coefficient for $PM_{2.5}$ becomes significant, and the effect of GDP per capita on life expectancy becomes negative.[95] Whilst *Figure 4.14* and *4.15* provide evidence that the increase in life expectancy due to rising income is increasing, the result in *Figure 4.16* is contradictory. Clearly the results in *Figure 4.13* is more believable than that shown in *Figure 4.16*, suggesting that the relationship postulated by Preston (1975) is the empirical reality, at least when using national level data. All the dummy variables are highly significant and are progressively larger meaning that life expectancy has increased from 1990 to 2013. Compared to 1990, life expectancies across all countries are 11.4% higher.

Figure 4.14. Empirical test of equation (4.5): between-effect model of an unbalanced panel dataset

```
Between regression (regression on group means)   Number of obs     =       935
Group variable: location                         Number of groups  =       164

R-sq:                                            Obs per group:
     within  = 0.0687                                         min =         1
     between = 0.3453                                         avg =       5.7
     overall = 0.2912                                         max =         6

                                                 F(2,161)          =     42.45
sd(u_i + avg(e_i.))=  .1232162                   Prob > F          =    0.0000
```

ln_LEXP	Coef.	Std. Err.	t	P>|t|	[95% Conf.	Interval]
pm25	-.0009459	.0008954	-1.06	0.292	-.0027143	.0008224
GDPperCap	6.68e-06	7.40e-07	9.04	0.000	5.22e-06	8.15e-06
_cons	4.154933	.0212845	195.21	0.000	4.112901	4.196966

[95] This suggests that most of the *percentage* increase are accounted for by time trend, and this exponential specification of equation (4.5) is inaccurate compared to (4.4). Alternatively, it may be that the increase in GDP per capita is correlated with other pollution not accounted for by pm2.5 concentration, which reduces life expectancy.

Figure 4.15. Empirical test of equation (4.5): fixed-effect model of an unbalanced panel dataset

```
Fixed-effects (within) regression               Number of obs      =       935
Group variable: location                        Number of groups   =       164

R-sq:                                           Obs per group:
     within  = 0.0718                                         min =         1
     between = 0.2825                                         avg =       5.7
     overall = 0.2436                                         max =         6

                                                F(2,769)           =     29.74
corr(u_i, Xb)  = 0.3346                         Prob > F           =    0.0000
```

ln_LEXP	Coef.	Std. Err.	t	P>|t|	[95% Conf.	Interval]
pm25	.0008786	.0007371	1.19	0.234	-.0005684	.0023256
GDPperCap	2.20e-06	2.88e-07	7.66	0.000	1.64e-06	2.77e-06
_cons	4.160163	.0147408	282.22	0.000	4.131226	4.1891
sigma_u	.13790611					
sigma_e	.05561781					
rho	.86010225	(fraction of variance due to u_i)				

```
F test that all u_i=0: F(163, 769) = 29.47                     Prob > F = 0.0000
```

Figure 4.16. Empirical test of equation (4.5): fixed-effect model of an unbalanced panel dataset, including year dummies

```
Fixed-effects (within) regression               Number of obs      =       935
Group variable: location                        Number of groups   =       164

R-sq:                                           Obs per group:
     within  = 0.5062                                         min =         1
     between = 0.2413                                         avg =       5.7
     overall = 0.0035                                         max =         6

                                                F(7,764)           =    111.90
corr(u_i, Xb)  = -0.3238                        Prob > F           =    0.0000
```

ln_LEXP	Coef.	Std. Err.	t	P>|t|	[95% Conf.	Interval]
pm25	.0011447	.0005561	2.06	0.040	.0000531	.0022363
GDPperCap	-1.36e-06	2.52e-07	-5.39	0.000	-1.85e-06	-8.63e-07
Y1995	.0145425	.004848	3.00	0.003	.0050256	.0240595
Y2000	.0342716	.0048369	7.09	0.000	.0247763	.0437668
Y2005	.0647827	.0049206	13.17	0.000	.0551233	.0744421
Y2010	.0977744	.005115	19.12	0.000	.0877332	.1078155
Y2013	.1138641	.0052968	21.50	0.000	.1034661	.1242621
_cons	4.132192	.012067	342.44	0.000	4.108504	4.15588
sigma_u	.16491753					
sigma_e	.0406972					
rho	.94259864	(fraction of variance due to u_i)				

```
F test that all u_i=0: F(163, 764) = 58.06                     Prob > F = 0.0000
```

4.4. Robustness Check Results

Figure 4.17. Regression of equation (4.1) without the variable q, panel logit model with cluster-robust errors

```
Random-effects ordered logistic regression      Number of obs     =      9,934
Group variable: pidp                            Number of groups  =      7,302

Random effects u_i ~ Gaussian                   Obs per group:
                                                              min =          1
                                                              avg =        1.4
                                                              max =          3

Integration method: mvaghermite                 Integration pts.  =         12

                                                Wald chi2(18)     =     359.96
Log pseudolikelihood  = -8234.3023              Prob > chi2       =     0.0000
```

(Std. Err. adjusted for 7,302 clusters in pidp)

H	Coef.	Robust Std. Err.	z	P>|z|	[95% Conf.	Interval]
beta2	-.1099567	.0694237	-1.58	0.113	-.2460246	.0261111
beta3	-.4103819	.0794936	-5.16	0.000	-.5661865	-.2545773
A2	.5544417	.0742489	7.47	0.000	.4089166	.6999669
A1	1.219407	.0880492	13.85	0.000	1.046834	1.39198
theta3	-.1577281	.4343954	-0.36	0.717	-1.009127	.6936712
theta2	-.6819493	.377354	-1.81	0.071	-1.42155	.057651
theta1	-.5633715	.3739456	-1.51	0.132	-1.296291	.1695483
M3	.3878105	.1428097	2.72	0.007	.1079087	.6677123
M2	.3512186	.1436001	2.45	0.014	.0697675	.6326697
M1	.4056795	.157928	2.57	0.010	.0961462	.7152127
delta3	-.2085212	.0678876	-3.07	0.002	-.3415784	-.075464
delta2	-.3788643	.0856943	-4.42	0.000	-.5468221	-.2109066
delta1	-.6836405	.2321984	-2.94	0.003	-1.138741	-.22854
t2	.2267051	.1158496	1.96	0.050	-.0003559	.4537661
t3	.1496085	.1064399	1.41	0.160	-.0590099	.3582269
t4	.186341	.114211	1.63	0.103	-.0375085	.4101905
t5	.2738616	.1143529	2.39	0.017	.0497341	.4979891
t6	.2551777	.1157255	2.21	0.027	.02836	.4819954
/cut1	-3.048617	.4177412	-7.30	0.000	-3.867375	-2.229859
/cut2	-.3884249	.4114102	-0.94	0.345	-1.194774	.4179244
/sigma2_u	2.298103	.2267073			1.894081	2.788305

It is not possible to deal with missing data using the process of imputation, which involves substituting missing values with for example average values. It is not appropriate here as almost all the independent as well as the dependent variables are categorical and binary, hence the mean cannot be obtained. The only option is to substitute missing values with the modal group,

yet this is likely to cause more bias compared to list-wise deletion since it reinforces the majority groupings which may result in greater skewness.

Figure 4.18. Probability of dependent variable missing (childhood model) as a function of independent variables, logistic regression. Theta3 and delta1 excluded due to zero correlation with dependent variable[96]

Logistic regression — Number of obs = 8,437; Wald chi2(17) = 112.19; Prob > chi2 = 0.0000; Log pseudolikelihood = -157.81046; Pseudo R2 = 0.0744

prob_H_miss~g	Coef.	Robust Std. Err.	z	P>|z|	[95% Conf.	Interval]
beta2	.6541286	.6074786	1.08	0.282	-.5365075	1.844765
beta3	.8210083	.6441034	1.27	0.202	-.4414111	2.083428
A2	-.370912	.5135872	-0.72	0.470	-1.377524	.6357005
A1	.0230228	.5640649	0.04	0.967	-1.082524	1.12857
theta3	0	(omitted)				
theta2	-2.562749	.818197	-3.13	0.002	-4.166386	-.9591125
theta1	-3.1851	.7810721	-4.08	0.000	-4.715973	-1.654226
M3	-.3704245	1.09022	-0.34	0.734	-2.507216	1.766367
M2	.5076953	1.051314	0.48	0.629	-1.552843	2.568234
M1	-.3357388	1.220621	-0.28	0.783	-2.728111	2.056634
delta3	-.2780721	.4407987	-0.63	0.528	-1.142022	.5858775
delta2	-.8761505	.6896952	-1.27	0.204	-2.227928	.4756271
delta1	0	(omitted)				
t2	-.3793521	.7222676	-0.53	0.599	-1.794971	1.036266
t3	-.5635622	.7345079	-0.77	0.443	-2.003171	.8760469
t4	.1876166	.617665	0.30	0.761	-1.022985	1.398218
t5	-.0197771	.6526601	-0.03	0.976	-1.298968	1.259413
t6	-1.822898	1.118538	-1.63	0.103	-4.015193	.3693966
q	.04131	.0174609	2.37	0.018	.0070872	.0755328
_cons	-3.687877	1.389862	-2.65	0.008	-6.411956	-.9637988

[96] All values in these categories have no missing variable in H, in other words they have no explanatory power whatsoever for missingness

Figure 4.19. Probability of dependent variable missing (adulthood model) as a function of independent variables, logistic regression. A3 is excluded due to zero correlation with dependent variable

```
Logistic regression                               Number of obs      =    235,947
                                                  LR chi2(5)         =    2324.53
                                                  Prob > chi2        =     0.0000
Log likelihood = -2678.4292                       Pseudo R2          =     0.3026
```

prob_H_miss~g	Coef.	Std. Err.	z	P>|z|	[95% Conf.	Interval]
A3	0	(omitted)				
A2	4.353294	.1460302	29.81	0.000	4.06708	4.639508
A1	1.303515	.3723128	3.50	0.000	.5737949	2.033234
delta	-.0301836	.0093541	-3.23	0.001	-.0485173	-.0118499
y	-.0003962	.000144	-2.75	0.006	-.0006784	-.000114
t	-.0031511	.0020334	-1.55	0.121	-.0071364	.0008343
_cons	-7.59254	.1797445	-42.24	0.000	-7.944833	-7.240248

4.5. Empirical Results Versus Theoretical Framework

The empirical results as shown in this section are generally supportive of my model theoretical framework and hypotheses. The model developed in Chapter 3 generates a set of testable hypotheses of which only three are chosen for empirical validation. Firstly, the child model, the relevant hypothesis concerns the optimal stock of health capital as a function of age and other exogenous variables. Secondly, the adult model facilitates the same hypothesis but concerning adults. Thirdly, from the adult model develops the equation for optimal length of life, which is expressed as a function of the exogenous variables. Each of the three hypotheses is converted into an econometrics model which assumes linear additively separable forms to be estimated using observable data. The child model, adult model and the optimal length of life model are shown respectively by equations (4.1), (4.2) and (4.4). Equation (4.3) is an alternative formulation of the adult model where the stock of educational capital replaces income as a reduced form equation.[97] Equation (4.5) competes with equation (4.4) as the econometric model representing the optimal length of life. Equation (4.5) is derived based on the strong empirical relationship observed by Preston (1975) at the national level, while equation (4.4) is based on my model proposition of the relationship at the individual level. The expected signs of the coefficients in the econometric models are obtained by applying comparative dynamic analysis or taking the partial derivatives of the original theoretical model with respect to the exogenous variable represented by the variable in the econometric model. For example, equation (4.5) is derived from equation (3.23) the model for optimal length of

[97] It is not possible to specify a structural form equation where income is a function of education, applying 2SLS analysis, since the dependent variable is categorical panel data.

life. The data for ambient $PM2.5_{jY}$ is chosen to represent the theoretical variable δ_0, the exogeneous rate of health depreciation. Therefore, equation (3.23) is partially differentiated with respect to δ_0 and since the result is negative, the coefficient for $PM2.5_{jY}$ in equation (4.5) is negative.

For the child model, equation (4.1) is developed to approximate the model shown by equation (3.14). Though not all the theoretical variables' effect on the optimal length of life could be tested due to a lack of data, the important variables are covered. The results in *Figure 4.3* and *Figure 4.4* shows that with the exception of the variable θ represented by data on 'importance of doing well in gcse or standard grades', the variables are in the expected direction. It should be noted that the variable representing $M(t)$ is positively correlated with the stock of health capital, contrary to the findings of most studies which report negative relationships. This most likely is due to the data used to represent $M(t)$, which unlike other studies represent the degree of medical usage, but rather the consumption of fruit and vegetables, which promotes good health and/or act as a proxy for generally healthy behaviours among children, perhaps instituted by their parents. The fact both the logit and probit models where their results are displayed by *Figure 4.3* and *Figure 4.4* respectively display consistent results gives further support to the theoretical propositions.

For the adult model which is theoretically represented by equation (3.21''), there are two econometric models – equation (4.2) and (4.3). The former allows the test of income variation on the optimal stock of health while the latter does so for variations in the stock of education. The regression results for equation (4.2) are shown in *Figure 4.6* and *Figure 4.7*. All the coefficients are in the expected direction and highly significant. Both the logit and probit models are consistent. The test for equation (4.3) where the results for the logit and probit models are shown in *Figure 4.8* and *Figure 4.9*, also conform to the theoretical predictions, with almost all the variables being statistically significant. The only non-significant variable is $E5$, which denote those with the education level 'O levels'. This statistical non-significance supports rather than undermine my theory since O levels is not particularly different from GCSE, which is the reference category. In other words the data fail to show that those with educational attainment of O level and those with GCSE have different levels of health.

For the life expectancy model, the test for both equation (4.4) and (4.5) consistently report that ambient $PM_{2.5}$ increases life expectancy, with the exception of the result in *Figure 4.14*. They key variable to be investigated is the effect of GDP per capita on life expectancy. GDP per

capita represent income or y in the theoretical model while life expectancy represents V^*, the optimal length of life. Equation (4.4) is specified under the assumption that increases in life expectancy due to rising national income would be diminishing, as is commonly reported since Preston (1975). This is the conventional specification and my results again affirm previous studies in this area. For equation (4.5) however, whilst the results in *Figure 4.14* and *Figure 4.15* show that the coefficient for GDP per capita is significant thus providing evidence in support of my theoretical proposition that increase in health brought about by rising income is increasing, when the year dummy variables are included the results in *Figure 4.16* contradict my theory, showing that an increase in GDP per capita reduces life expectancy. Therefore, there is insufficient empirical evidence to suggest that the relationship between optimal length of life and income is as specified by equation (4.5), and equation (4.4) is preferred.

The strong empirical support for the theoretical model developed in Chapter 3 lays the foundation for the model to be applied as a theoretical basis for modelling the health co-benefits of decarbonisation and air quality policies, and how such policies are likely to interact with socio-economic variables as well as any (in)equality implications.

CHAPTER 5 – Model Application

The theoretical model developed in Chapter 3 and its empirical findings reported in Chapter 4 have a wide range of useful applications for a variety of purposes. This chapter discusses some of the uses, most importantly how it can be applied to modelling the health co-benefit of air quality and decarbonisation policies, incorporating the influences of socio-economic variables. This is outlined in section *5.1*. Section *5.2* describes how my theoretical model can be used to predict the distribution or inequality implications of any health co-benefit arising from air quality or decarbonisation strategies.

The model begins at a theoretical level, where I specify theoretically the relationship between the dependent and independent variables. It is a relatively simple economic model based on the assumption that a representative agent seeks to maximise his or her lifetime utility, subject to certain constraints but operating under conditions of perfect information and certainty. Using optimal control theory and the Pontryagin Maximum principle, a set of solutions are derived for the control, state and co-state variables, which are expressed as functions of exogenous variables.[98] The set of solutions states theoretically the relationship between the variables. Comparative dynamic analysis can be applied to all the optimal solutions to examine the effect of changes in exogenous variables, allowing us to make predictions. This is performed simply by partially differentiating the solution with respect to the exogenous variable of interest.

The econometric models in Chapter 4 help to validate my model predictions. The primary task is to estimate the relationship between the stock of health capital and various exogenous variables, which are expressed by equations (3.14) and (3.21'') for the childhood and adulthood phases, respectively. Since I choose self-reported health status to represent the stock of health capital and the answers are ordered categorical responses, the magnitude of the coefficients relating the exogenous variables to the stock of health capital to the exogenous variables have little theoretical meaning if any at all. It is not possible for example to state that an increase in monthly income by a certain amount leads to a corresponding unit of increase in the stock of health capital as health capital cannot be directly measured but merely proxied using self-reported health categories. Furthermore, the non-linear ordered logit and probit models are not suitable for reading off the magnitude of the coefficients between the dependent and independent variables. All can be stated is that certain exogenous variables affect the stock of

[98] Along with time or age t.

health capital significantly, and whether they contribute to its increase or decrease. If I wish the magnitude of the coefficients to be of meaning, it is necessary to use a continuous measure of the stock of health capital or health status. Examples include QALYs and DALYs, though these data are not often available at the individual level.

For equation (3.23) and the empirical counterpart (4.4), the dependent variable is the optimal length of life, which is interpreted as life expectancy at the national level. Since this variable is continuous, the magnitude of the coefficients obtained using linear models has meaning. I find that for every 1% increase in GDP per capita, optimal length of life or life expectancy increases by 0.0718 years according to the estimate I am most confident with. The alternative econometric model of equation (4.5) whilst more closely aligned to the theory, possesses some empirical inconsistency – when year dummy variables are included, the effect of GDP per capita on life expectancy becomes negative, as shown in *Figure 4.16*. Therefore equation (4.4) and the corresponding empirical estimates are preferred over equation (4.5).

5.1. Applying my Model to Modelling Health Co-benefit of Air Quality and Decarbonisation Policies, Incorporating the Role of Socio-economic Variables

In this section I show how the model of adulthood phase can be applied to epidemiological studies incorporating the role of socio-economic effects. I apply the empirical results to the adulthood phase only, since the data I use and for most data available, the information on children comprise only a small proportion of the epidemiological sample and so are not representative, or are not even recorded.

The child and the adult phases' solutions for optimal stock of health as a function of time or age are illustrated by equations (3.14) and (3.21'') respectively. Both equations consist of an exogenous rate of health depreciation as an independent variable, denoted by δ in the childhood phase but δ_0 in the adulthood phase. This concept of exogenous rate of health depreciation encompasses a spectrum of factors which contribute to the erosion of one's health over time. Examples include unhealthy consumption, which is chosen to represent the health depreciation during the childhood phase. For the adulthood phase of the model, the exogenous health depreciation is represented by the number of cigarettes smoked per day. In the life expectancy model I use ambient PM exposure as the exogenous measure. The risks associated with smoking are similar to and overlap substantially with ambient air pollution, albeit generally believed to be more damaging though affecting a smaller number of the population. I suggest that ambient air pollution can be used to represent the exogenous rate of health

depreciation while the other exogenous variables are collectively defined as socio-economic variables.

It is difficult to quantitatively assess the impact of a change in any environmental variable, such as an air quality indicator, on health status, since health status cannot be precisely measured using a one-dimensional metric. Nonetheless from equations (3.7), (3.7') and (3.7''), it is possible to separate the endogenous rate of health depreciation, $\delta(t)$ from the exogenous rate δ_0 in the adulthood phase. The 'endogenous' depreciation of health is merely the decline in health which is subjected to the factors under the individual's choices, or the control variables. The 'optimal' endogenous rate of health depreciation is the rate of health depreciation which arises if the individual undertakes actions with regards to the input of time, medical care and consumption ($\tau_H^*(t)$, $M^*(t)$ and $X^*(t)$, respectively) which is considered optimal. The optimal endogenous rate of health depreciation can be obtained by substituting equations (3.16) and (3.17), the optimal control paths for the variables time and medical care input respectively, into equation (3.7''):

$$\delta^*(t) = A^{-1}\left(\frac{y^{A+1}}{A\delta_0 P_M P_X^A} e^{A(GH_0-1)-2+\frac{P_M}{y}} - Gt\right) = \frac{d}{dt}H^*(t) \qquad (5.1)$$

Where $\delta^*(t)$ denote the optimal rate of health depreciation. In fact equation (5.1) is but the time derivative of the optimal time path for health capital.

I seek to empirically test equation (5.1). It is difficult to obtain individual data on the measurement of endogenous rate of health depreciation. This is because I am in in search of measures of health depreciation or threats which are directly related to the control variables – time and medical care input. In the adult model of equation (3.21''), and the empirical counterpart of (4.2) and (4.3), the number of cigarettes smoked per day is used to represent the exogenous rate of health capital depreciation δ_0. Such a measure would be unsuitable to represent the endogenous rate of health depreciation since it would be ruling out the effect of consumption ($X(t)$) on health and health capital accumulation, as well as all forms of joint consumption. In order to account for such effects it would be necessary to differentiate between different types of consumption with some that promote health, others damage health and still others are neutral towards health, or specify a vector of goods which constitute $X(t)$. I avoid this approach as it is likely to complicate my analysis and distract me from the main aim of looking at the relationship between socio-economic variables and health. It would therefore be appropriate to measure the endogenous rate of health depreciation using aggregate data

measures similar to the life expectancy model in equation (3.23) and the empirical counterparts in equations (4.4) and (4.5). I propose that the measure Relative Risk (RR) commonly used in epidemiological studies would be particularly suitable to represent the endogenous rate of health depreciation. The RR measure represents the ratio of the incidence[99] between a group exposed to some risk or hazard verse a group which is not exposed (the control group). However, in most epidemiological studies where it is not possible to run controlled laboratory experiments such values are frequently obtained through observation from medical data. The incidence among the control group is often termed the 'background incidence', denoting the theoretical incidence which would prevail in the total absence of a risk or health hazard.

I select the RR associated with PM pollution to represent the endogenous rate of health depreciation. This is an appropriate measure since the RR of a population is dependent on a) the ambient PM exposure, which can be treated as exogenous and b) the control variables of time and medical care input. Both factors would reduce or mitigate the health effects associated with PM pollution at both the individual and aggregate level. The control variables are endogenous but can be expressed as functions of the exogenous variables.

The Global Burden of Disease (GBD) 2013 study[100] estimates the mortality and morbidity cases attributable to particular risk factors for each country, broken down into age groups and sex. It also estimates mortality/morbidity by cause, broken down into the same categories. From the data, I select ambient PM to be the risk factor of interest (since this is the exogenous factor related to the co-benefits calculations at the heart of this dissertation) and the cause(s) to be cardiovascular and chronic respiratory diseases. Acute respiratory disease can also be caused by ambient PM. As described shortly, I regress the endogenous rate of health depreciation with the annual mean exposure, which represents δ_0[101]. Since acute illnesses are unlikely to be sensitive to data on annual mean PM concentration but instead more sensitive to daily or hourly data, I omit such causes when specifying the RR.

The GBD 2013 study does not actually report the RR so I computed the RR associated with ambient PM using the available data from the study. The RR is typically computed by dividing

[99] Number of incidents per unit of time, usually per 1,000.

[100] Data for the GBD 2013 study are available online: http://ghdx.healthdata.org/record/global-burden-disease-study-2013-gbd-2013-age-sex-specific-all-cause-and-cause-specific

[101] The same data variable used to represent δ_0 in equations (3.23), (4.4) and (4.5).

total PM related mortality/morbidity by the background incidents.[102] The background incidents may be calculated by summing the total incidents attributable to cardiovascular and chronic respiratory diseases (two categories which are reported in the GBD 2013 study), minus PM related incidence (which is also reported in the GBD 2013 study). The total PM related mortality/morbidity is simply the cases attributable to cardiovascular and respiratory diseases as already computed. By dividing the PM attributable mortality/morbidity incidents by the background incidents, the RR in my model is obtained.[103] The RR obtained using this method can be considered the RR of PM reflecting the chronic rather than acute risks, since the PM related illnesses used for its computation are considered chronic diseases. I construct RR for countries based on incidence of deaths and DALYs which is a measure combining mortality and morbidity.

For the other variables in equation (5.1), I utilise data from the World Bank database just like for equations (3.23) and (4.4). As mentioned, δ_0 is represented using 'PM2.5 air pollution, mean annual exposure (micrograms per cubic meter)'. 'GDP per capita (current US$)' is used to denote y in equation (5.1). GDP is also likely to correlate strongly with A and G, the efficiency parameter and the marginal product of a unit of health capital, respectively. Up to now the data used for the independent variables are exactly the same as that used in equations (3.23) and (4.4). I however add another variable – the Consumer Price Index (CPI). I use the CPI to represent the variables P_X and P_M, since it is an index capturing the price levels of all goods and services in a country, whether for consumption purposes or medical purposes for improving health. I include the variable in my test of equation (5.1) since the price levels of both consumption goods as well as medical goods is likely to have a strong bearing on people's decisions on how much time and medical care they devote for the purpose of improving health. Table *5.1* below lists the variables I choose to represent the theoretical variables in equation (5.1).

Table 5.1. Data variables and the corresponding theoretical variables represented to empirically test equation (5.1)

Data variable	Associated theoretical model variable
Relative Risk (RR)	$\delta^*(t)$ the endogenous rate of health depreciation

[102] RR could also be computed by dividing the mortality/morbidity incidence over the background incidence, where incidence refers to the rate of mortality/morbidity in a given unit of population whilst incident refers to the absolute number of mortality/morbidity cases.

[103] $RR = \frac{Cardiovascular\ \&\ respiratory\ mortality}{Cardiovascular\ \&\ respiratory\ mortality - mortality\ due\ to\ PM\ pollution} = \frac{Cardiovascular\ \&\ respiratory\ mortality}{Background\ mortality}$

$PM_{2.5}$ concentrations	δ_0 the exogenous rate of health depreciation
GDP per capita	y, G and A
Consumer Price Index	P_X and P_M

All the data shown in Table *5.1* above consist of six years of data – 1990, 1995, 2000, 2005, 2010 and 2013, for over 180 countries. Therefore a panel data set can be constructed from the available data.

Figure 5.1 below shows the relationship between a country's RR due to ambient $PM_{2.5}$ pollution and its GDP per capita, measured in USD. The scatterplot is constructed using six years of data – 1990, 1995, 2000, 2005, 2010 and 2013. From visual inspection, there appears to be a non-linear negative relationship between the two variables. GDP per capita reduces the RR but at a diminishing rate. Therefore, the relationship between RR and GDP per capita may be best represented by an exponential function with a negative coefficient on the variable. A similar general relationship is shown for RR constructed using DALYs compared with GDP per capita, whether outliers are included (*Figure 5.2*) or excluded (*Figure 5.3*).

Given the relationship as shown in *Figures 5.1*, *5.2*, and *5.3*, the theoretical model shown by equation (5.1) can be converted to the following econometric model:

$$\ln(RR_{jY}) = \alpha_j + \alpha_0 + \phi_1 PM2.5_{jY} + \phi_2 GDPperCap_{jY} + \phi_3 CPI_{jY} + u_{jY} \quad (5.2)$$

Where j denotes the country and Y the year.

RR_{jY} is constructed using data from the GBD 2013 study as mentioned above. Two sets of RR are used – one is the RR based on mortality incidents while the other the RR based on DALYs.

Figure 5.1. Relative Risk (RR) of ambient PM related mortality and GDP per Capita ($) for the years 1990, 1995, 2000, 2005, 2010 and 2013

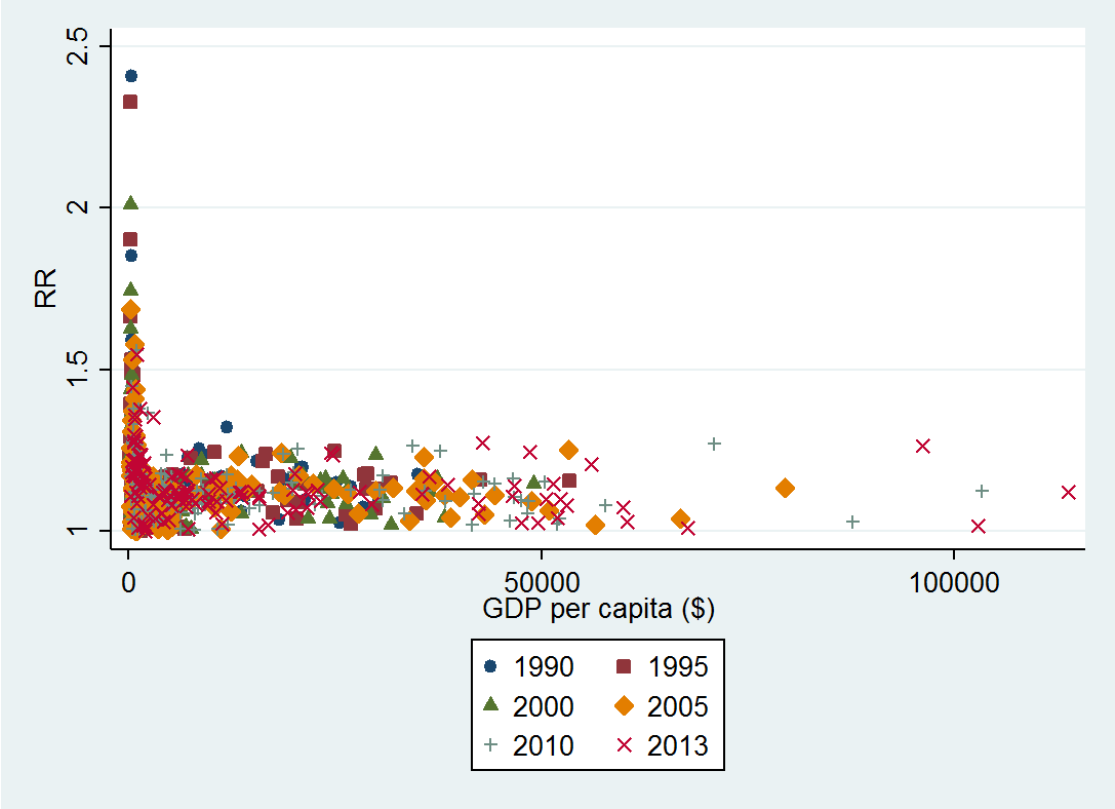
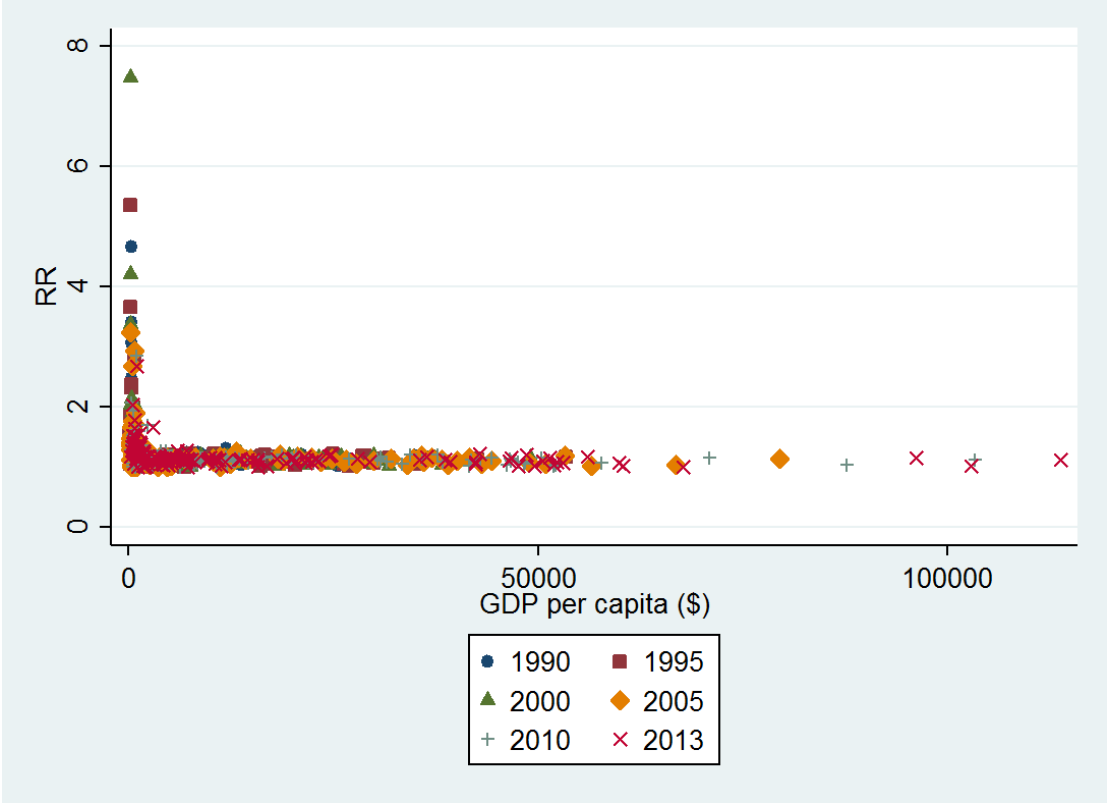


Figure 5.2. Relative Risk (RR) of ambient PM related DALY and GDP per Capita ($) for the years 1990, 1995, 2000, 2005, 2010 and 2013 (The same comment as in the previous figure)

Figure 5.3. Relative Risk (RR) of ambient PM related DALY and GDP per Capita ($) with outliers removed, for the years 1990, 1995, 2000, 2005, 2010 and 2013 (The same comment)



In addition to these two measures of health risks, the GBD 2013 study also contains data on the Years of Life Lost (YLL) and Years Lived with Disability (YLD).[104] The former is an alternative measure for mortality while the latter a measure of morbidity. The other variables in equation (5.2) are obtained from the World Bank databank. The combination of GBD and World Bank data facilitates us to collate a panel data set for 180 countries over 6 year points – 1990, 1995, 2000, 2005, 2010 and 2013. By obtaining the partial derivatives of equation (5.1) with respect to the exogenous variables of interest, I can predict the expected signs of the coefficients in equation (5.2) for ϕ_{1-3}. The partial derivatives are shown below in *Table 5.2*. As can be seen, I expect $\phi_{1,3}$ to be positive and ϕ_2 negative.

Table 5.2. Partial derivatives of equation (5.1) with respect to the exogenous variables in Table 5.1.

Partial derivative	Equation of partial derivative	Theoretical prediction
$\frac{\partial \delta^*(t)}{\partial \delta_0}$	$\frac{y^{A+1}}{A^2\delta_0^2 P_M P_X^A} e^{A(GH_0-1)-2+\frac{P_M}{y}} \left(\frac{y^{A+1}}{A\delta_0 P_M P_X^A} e^{A(GH_0-1)-2+\frac{P_M}{y}} - Gt \right)^{-2}$	$\frac{\partial \delta^*(t)}{\partial \delta_0} > 0 \; \forall \; t \in [0, V]$

[104] The YLL (mortality element) and YLD (morbidity element) are combined to calculate DALY.

$\frac{\partial \delta^*(t)}{\partial y}$	$-\left(A+1-\frac{P_M}{y}\right)\frac{y^A}{A^2\delta_0 P_M P_X^A}e^{A(GH_0-1)-2+\frac{P_M}{y}}\left(\frac{y^{A+1}}{A\delta_0 P_M P_X^A}e^{A(GH_0-1)-2+\frac{P_M}{y}}-Gt\right)^{-2}$	$\frac{\partial \delta^*(t)}{\partial y}<0\ \forall\ t\in[0,V]$
$\frac{\partial \delta^*(t)}{\partial P_X}$	$\frac{y^{A+1}}{A\delta_0 P_M P_X^2}e^{A(GH_0-1)-2+\frac{P_M}{y}}\left(\frac{y^{A+1}}{A\delta_0 P_M P_X^A}e^{A(GH_0-1)-2+\frac{P_M}{y}}-Gt\right)^{-2}$	$\frac{\partial \delta^*(t)}{\partial P_X}>0\ \forall\ t\in[0,V]$
$\frac{\partial \delta^*(t)}{\partial P_M}$	$(y-P_M)\frac{y^A}{A^2\delta_0 P_M^2 P_M}e^{A(GH_0-1)-2+\frac{P_M}{y}}\left(\frac{y^{A+1}}{A\delta_0 P_M P_X^A}e^{A(GH_0-1)-2+\frac{P_M}{y}}-Gt\right)^{-2}$	$\frac{\partial \delta^*(t)}{\partial P_M}>0\ \forall\ t\in[0,V]$

I employ panel data regression models to estimate the coefficients of equation (5.2). As in the case of empirically verifying equation (3.23), (4.4) and (4.5), my preferred model is the between-effect panel estimation. This is because this method gives a greater weight to between or cross sectional (country) variations rather than time series variation, as would be the case for fixed effect model. The time series dimension should not be particularly strong since the variables in my data are relatively fixed over time and I have too few time data points (6 points) to yield precise estimates from the time series perspective alone.[105] Nonetheless I report both the regression results using the between-effect model and the more conventional fixed effect model. *Figure 5.4* and *5.5* show the regression results using the between-effect estimator for RR constructed using mortality cases and DALYs, respectively. *Figure 5.6* and *5.7* show the regression results using the fixed-effect estimator for RR constructed using mortality cases and DALYs, respectively.

As can be seen in *Figure 5.4* and *5.5*, the regression results using between-effect panel estimators, all the variables are of the expected sign. The coefficients for GDP per capita and CPI are statistically significant at the 5% level and approaching 1%. However, the coefficient for $PM_{2.5}$ is not statistically significant. This means that income as measured by GDP per capita has a sizable and negative effect on the rate of health depreciation. Individuals who have higher incomes are likely to be subjected to lower health risks and have a lower depreciation in their health according to my model. Even though the coefficient for CPI is significant, some caution

[105] The temporal change for the same country over time does not possess sufficient variations for inferences to be drawn regarding the relationship between GDP per capital and relative risk, hence it is necessary to give greater weight to the cross-sectional variations as the main source of explanatory power. The between-effect model is therefore selected as the main model over the fixed-effect model.

is necessary in interpreting the regression outcome. This is because CPI data are fixed at 100 for the base year for each country (the base year is 2010 in the World Bank data). Therefore, variations across countries do not reflect differences in the price levels between countries whether for consumption goods or medical care services. Only variations of CPI over time can be interpreted as the effect of prices on the endogenous rate of health depreciation. The coefficient for $PM_{2.5}$ fails to be statistically significant though it is in the expected direction. This is perhaps due to the fact that aggregate measures of ambient PM concentration at the national level over a year are not accurate reflections of true population exposure to the pollutant, since the measurements of pollution tend to be at fixed locations that are easy to access and are in danger of exceeding regulatory limits, rather than being randomly located. At the country level, the spatial resolution for a measure of ambient PM is simply too low. The temporal resolution of annual mean values may also be inadequate yet it is perhaps necessary since GDP per capita and CPI are normally reported annually also. The same general relationship holds true whether the RR is constructed using mortality cases or DALYs.

Figure 5.4. Regression results of equation (5.2), RR constructed using mortality cases associated with ambient PM pollution, between-effect model

```
Between regression (regression on group means)   Number of obs     =       713
Group variable: location                         Number of groups  =       134

R-sq:                                            Obs per group:
     within  = 0.0452                                         min =         1
     between = 0.0728                                         avg =       5.3
     overall = 0.0051                                         max =         6

                                                 F(3,130)          =      3.40
sd(u_i + avg(e_i.))=  .0926398                   Prob > F          =    0.0197
```

ln_RR	Coef.	Std. Err.	t	P>|t|	[95% Conf.	Interval]
pm25	.0005328	.0007988	0.67	0.506	-.0010475	.0021131
GDPperCap	-1.48e-06	6.01e-07	-2.46	0.015	-2.67e-06	-2.88e-07
CPI	.0015857	.0006117	2.59	0.011	.0003756	.0027958
_cons	.0164088	.0498211	0.33	0.742	-.0821563	.1149739

Figure 5.5. Regression results of equation (5.2), RR constructed using DALYs associated with ambient PM pollution, between-effect model

```
Between regression (regression on group means)   Number of obs     =       793
Group variable: location                         Number of groups  =       148

R-sq:                                            Obs per group:
     within  = 0.0343                                          min =         1
     between = 0.0836                                          avg =       5.4
     overall = 0.0173                                          max =         6

                                                 F(3,144)          =      4.38
sd(u_i + avg(e_i.))=  .2203221                   Prob > F          =    0.0055
```

ln_RR	Coef.	Std. Err.	t	P>|t|	[95% Conf.	Interval]
pm25	.0027793	.0018342	1.52	0.132	-.0008461	.0064047
GDPperCap	-4.54e-06	1.41e-06	-3.22	0.002	-7.34e-06	-1.75e-06
CPI	.0021957	.0014082	1.56	0.121	-.0005878	.0049792
_cons	.0113803	.1136363	0.10	0.920	-.2132305	.235991

For estimation of equation (5.2) using the fixed effect model, the results differ substantially. For both *Figure 5.5* and *5.6* ambient concentrations of $PM_{2.5}$ actually reduces the RR. GDP per capita also lowers the RR but the effect is no longer statistically significant. The CPI on the other hand also reduces RR and is highly significant. From the regression results in *Figure 5.6* and *5.7* therefore, it appears that my theoretical predictions are contradicted. However I choose the between effect model as mentioned with the regression results reported in *Figure 5.4* and *5.5*, since it better captures the cross-sectional dimension and is more appropriate in the case of few time data points. According to my best estimate, an increase in the GDP per capita by $1,000 reduces the natural log RR of mortality cases by 0.00148 and the natural log RR of DALYs by 0.00454. The estimates can be used to examine how RR relationships of PM and more generally air pollution are modified by socio-economic variables.

Even though the GBD 2013 study contains data on the mortality and morbidity risk factors divided by age groups, it would not be appropriate to utilise such data and include age as a variable in equation (5.2) to estimate the resulting equation. This is because when my model is tested using aggregate data, it would not be appropriate to equate the age of a person to the average age of a population or its subgroup. In other words, while the age of an individual possesses conceptual value the same cannot be said of the age of a country or its subgroup and there is no way such can be represented by a suitable metric within the models. Specifically, a country does not 'end' its life when the terminal age or time is reached unlike an individual. This immediately nullifies the transversality condition I develop in the theoretical framework.

The best course of action in this circumstance is to exclude age as a variable in my empirical model in equation (5.2). The most appropriate theoretical model for this purpose is that of an overlapping generations model rooted in macroeconomic rather than microeconomic theory, which would substantially complicate my theoretical framework and so should be left for further studies.

Figure 5.6. Regression results of equation (5.2), RR constructed using mortality cases associated with ambient PM pollution, fixed-effect model

```
Fixed-effects (within) regression               Number of obs      =       713
Group variable: location                        Number of groups   =       134

R-sq:                                           Obs per group:
     within  = 0.0637                                         min =         1
     between = 0.0012                                         avg =       5.3
     overall = 0.0006                                         max =         6

                                                F(3,576)           =     13.07
corr(u_i, Xb)  = -0.1073                        Prob > F           =     0.0000
```

ln_RR	Coef.	Std. Err.	t	P>|t|	[95% Conf.	Interval]
pm25	-.0009982	.0004567	-2.19	0.029	-.0018951	-.0001012
GDPperCap	-2.77e-07	1.73e-07	-1.60	0.109	-6.16e-07	6.21e-08
CPI	-.0001941	.0000398	-4.88	0.000	-.0002722	-.000116
_cons	.1705117	.0092559	18.42	0.000	.1523322	.1886911
sigma_u	.0961447					
sigma_e	.03016042					
rho	.91040972	(fraction of variance due to u_i)				

```
F test that all u_i=0: F(133, 576) = 54.31                     Prob > F = 0.0000
```

Figure 5.7. Regression results of equation (5.2), RR constructed using DALYs associated with ambient PM pollution, fixed effect model

```
Fixed-effects (within) regression               Number of obs      =       793
Group variable: location                        Number of groups   =       148

R-sq:                                           Obs per group:
     within  = 0.0392                                         min =         1
     between = 0.0413                                         avg =       5.4
     overall = 0.0003                                         max =         6

                                                F(3,642)           =      8.74
corr(u_i, Xb)  = -0.1334                        Prob > F           =    0.0000
```

ln_RR	Coef.	Std. Err.	t	P>|t|	[95% Conf.	Interval]
pm25	-.0010996	.0019364	-0.57	0.570	-.0049021	.0027028
GDPperCap	3.92e-07	7.52e-07	0.52	0.603	-1.08e-06	1.87e-06
CPI	-.0008118	.0001642	-4.94	0.000	-.0011343	-.0004893
_cons	.2687798	.0387572	6.93	0.000	.1926735	.344886
sigma_u	.23133011					
sigma_e	.13341525					
rho	.7504022	(fraction of variance due to u_i)				

```
F test that all u_i=0: F(147, 642) = 16.09                     Prob > F = 0.0000
```

Using the method of Crawford-Brown et al., (2013) which conducted a meta-analysis of the relationship between GHG emission and ambient PM concentration, it is estimated that a 1% change in CO_2 emission results in a corresponding 1% change in the emission of PM. This is due to the fact that anthropogenic CO_2 and PM often share common sources. For example, the combustion of fossil fuels to meet energy demands generate emissions of both CO_2 which contributes to climate change, and fine PM, which causes cardiovascular and respiratory illnesses via air pollution. This 1% change in the emission of PM, results in a change of ambient concentration by 0.5% during the steady state, i.e. when the particles have settled down and converge to an equilibrium concentration in the atmosphere. On the other hand, a one unit increase ($\mu g/m^3$) in ambient PM concentration results in a 0.1% increase in mortality cases associated with PM (cardiovascular and respiratory illnesses), as measured using RR. For morbidity or non-fatal incidents caused by PM, the increase in RR is 0.3% per one unit increase ($\mu g/m^3$).

Crawford-Brown et al., (2013) found that the coefficient relating PM concentration and RR was consistent across different countries and groups when epidemiological studies were conducted, but the RR varied with socio-economic variables such as national income.[106] This

[106] The coefficient relating RR to ambient PM exposure is termed the β coefficient in Crawford-Brown et al., (2013). Whilst RR is a function of GDP per capita as is hypothesised here, β is consistent across countries and

implies that socio-economic variables such as GDP per capita and ambient PM pollution have no interaction effects. The effect of ambient pollution and GDP per capita on the RR can therefore be considered independent and should be expressed as additive functions of RR such that $\frac{\partial}{\partial GDPperCap}\left(\frac{\partial RR}{\partial PM}\right), \frac{\partial}{\partial PM}\left(\frac{\partial RR}{\partial GDPperCap}\right) = 0$.

I show in my regression that the natural log of RR is reduced by GDP per capita and the effects are statistically significant. In other words, the mortality/morbidity risks of ambient PM exposure are lowered by GDP per capita *for any given unit of ambient pollution*, yet the gradient of the exposure response function relating PM exposure to RR is not affected by GDP per capita or any other socio-economic variables. The results of the regression analysis using the between-effect model as shown in *Figure 5.4* and *5.5* allows us to state the following equations for the relationship between the RR for mortality and morbidity with the exogenous variables.

$$\ln(RR_{jY}) = 0.0164088 + 0.0005328PM2.5_{jY} - 0.00000148GDPperCap_{jY} + 0.0015857CPI \quad (5.3)$$

$$\ln(RRDALY_{jY}) = 0.0113803 + 0.0027793PM2.5_{jY} - 0.00000454GDPperCap_{jY} + 0.0029157CPI \quad (5.4)$$

Where $RRDALY_{jY}$ refers to the Relative Risk constructed using DALYs for country *j* in year Y.

Using the coefficient for GDP per capita shown in *Figure 5.4* and *5.5*, a 1 US dollar increase results in a decrease of the logarithm of RR for deaths and DALYs by 0.00000148 and 0.00000454, respectively. From equations (5.3) and (5.4), a unit change in GDP per capita holding all other variables fixed translates to the following equations:

$$\Delta RR_{jY} = -0.00000148e^{0.0164088+0.0005328PM2.5_{jY}-0.00000148GDPperCap_{jY}+0.0015857CPI}\Delta GDPperCap \quad (5.5)$$

$$\Delta RRDALY_{jY} = -0.00000454e^{0.0113803+0.0027793PM2.5_{jY}-0.00000454GDPperCap_{jY}+0.0029157CPI}\Delta GDPperCap \quad (5.6)$$

appears to be independent of many factors including GDP and other socio-economic variables. Therefore, the sensitivity of different population groups to a unit change in ambient pollution, measured by the percentage increase in associated mortality and morbidity appear to be constant. Socio-economic variables however can alter the baseline mortality and mortality via their impact on the RR.

Equations (5.5) and (5.6) are used to model mortality and morbidity changes attributable to decarbonisation strategies under various scenarios. Assuming that the ambient $PM_{2.5}$ concentration and CPI are at 50µg/m^3 and 100 respectively, the following graph can be plotted to illustrate the relationship between RR and GDP per capita.

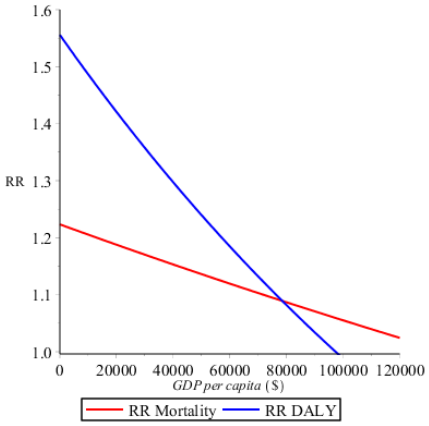


Figure 5.8. Theoretical relationship between RR and GDP per capita based on coefficient estimates shown in Figure 5.4 and 5.5 and equations (5.3) to (5.6)

The red line in *Figure 5.8* shows the relationship between RR and GDP per capita constructed using mortality cases while the blue line shows the RR constructed using DALYs. As can be seen, RR due to DALY starts at a higher level but is more sensitive to changes in GDP per capita, declining rapidly with its increase, compared to RR of mortality cases. However, the relationship and coefficients displayed in equations (5.3) to (5.6) based on regressions of the GDP data may not be the most suitable for modelling the effect of health co-benefits for several reasons.

Firstly, the coefficients for PM are not statistically significant. As mentioned, both the spatial and temporal resolution (especially the former) at the country level over annual mean inadequately captures the true extent of population exposure. Furthermore, in most epidemiological studies, other variables are included to control for 'confounding'. For example, since smoking and other air pollutants including sulphur dioxide and ozone causes similar health risks and symptoms, it would be incorrect to assume that they are solely caused by ambient PM, as is effectively the case in my model. The coefficient estimates for the effect of ambient PM on RR would be biased in such a case even if statistically significant. I do not include the control variables in my model since good quality and relatively complete national level data on smoking and other pollutants are not available. I therefore should not use this coefficient to model changes in RR in response to changes in PM emissions or ambient

concentration, or to model the health co-benefits arising from air quality or decarbonisation policies.

Secondly, as also alluded to, the coefficient for CPI does not capture cross-sectional variations, which is the main variations in my data given the large cross-sectional sample size relative to the short time points. This is because the CPI is fixed at 100 in the base year of 2010, for all countries in my data. As an example, the United States a highly developed country in which medical services are extremely expensive (high P_M) has the same price level as Cuba, an economically underdeveloped country where the state provides for healthcare and so in effect have very low or even zero P_M. Any change in the price level can only be compared with the country itself and hence only the time series dimension of the effect of CPI on RR are captured. Therefore, the coefficient for CPI just like that for $PM_{2.5}$ should not be used in the modelling of health co-benefit. I include CPI in my regression equation since it acts as a control variable and may improve the estimated coefficient of GDP per capita, much like including data on smoking and other air pollutants help to reduce confounding for the estimate coefficient of ambient PM. Thirdly, epidemiological evidence strongly suggests that the relationship between RR and ambient $PM_{2.5}$ is linear. Moreover, the exposure-response relationship is not distorted by GDP per capita and other socio-economic variables. However, my logarithmic model as shown by equation (5.2) is non-linear and thus the marginal effect of a unit change in GDP per capita and ambient PM are dependent on each other due to this functional form. I specify the logarithmic functional form since *Figure 5.1*, *5.2* and *5.3* strongly suggests that the relationship between RR and GDP per capita is one of negative exponentiation. The effect of GDP per capita increase on RR should be diminishing since it is impossible to eliminate all PM associated risks simply by economic growth, without correspondingly reducing air pollution. The logarithmic specification is most appropriate for estimating the coefficient between RR and GDP per capita but once I obtained the estimation, I should model the RR using different functional forms which satisfy the assumption of linearity between RR and ambient PM exposure, as well as its functional independence with GDP per capita.

Based on the above concerns, I adopt the following modifications to equations (5.3) and (5.4). Firstly, instead of using the estimated exposure response coefficient for $PM2.5$ from the GDP data I adopt the RR coefficients[107] reported by Crawford-Brown et al., (2013). According to

[107] The β coefficients

the meta-analysis, a 1 μg/m^3 increase in ambient PM concentration increases the RR by 0.001 for mortality cases and 0.003 for morbidity cases. Note however that Crawford-Brown et al., (2013)'s reported coefficients are for ambient PM pollution in general and includes PM_{10}, which refers to particulate matters less than 10 micrometres or less in diameter. $PM_{2.5}$ is a subset of PM_{10}, often known as fine particulate matter which has diameters less than 2.5 micrometres. $PM_{2.5}$ is considerably more hazardous than PM_{10} per unit of ambient exposure (the coefficient on RR is up to twice as large) and constitutes roughly half of the total mass of PM_{10} (Chow et al., 1994). I should therefore adjust the corresponding exposure-response coefficients of Crawford-Brown et al., (2013) to 0.002 and 0.006 for mortality and morbidity respectively. I choose the RR computed using mortality incidence to match 0.002 per unit increase in $PM_{2.5}$ concentration while the RR computed using DALY to correspond to 0.006. Secondly, I exclude CPI from equations (5.3) and (5.4). The CPI variable is included in the regression analysis purely to improve the estimate of the coefficient for GDP per capita. Once it has served its purpose, CPI is no longer needed for modelling co-benefit. Thirdly, I should alter the functional form so that the change in ambient concentration alters the RR linearly while the change in GDP per capita reduces the RR non-linearly. This combines evidence from epidemiological studies with my observation that the RR values of countries are generally related to the GDP per capita in an negative exponential relationship.

Combining the above three considerations, I modify equation (5.3) and (5.4) to (5.7) and (5.8) below:

$$RR_{jY} = 0.002PM2.5_{jY} + e^{0.0164088-0.00000148GDPperCap_{jY}} \quad (5.7)$$

$$RRDALY_{jY} = 0.006PM2.5_{jY} + e^{0.0113803-0.00000454GDPperCap_{jY}} \quad (5.8)$$

$e^{0.0164088}$ and $e^{0.0113803}$ can be regarded as my estimate of the background mortality and morbidity risk associated with ambient PM, respectively. They are the level of health risks in the absence of any effect from either ambient PM or GDP per capita. Equations (5.5) and (5.6) are modified to equations (5.9) and (5.10) below:

$$\Delta RR_{jY} = 0.002PM2.5_{jY} - 0.00000148e^{0.0164088-0.00000148GDPperCap_{jY}}\Delta GDPperCap_{jY} \quad (5.9)$$

$$\Delta RRDALY_{jY} = 0.006PM2.5_{jY} - 0.00000454e^{0.0113803-0.00000454GDPperCap_{jY}}\Delta GDPperCap_{jY} \quad (5.10)$$

Assuming that the ambient $PM_{2.5}$ is at 50 μg/m^3, I replot the relationship between RR and GDP per capita in *Figure 5.8* using my modified equations from (5.7) to (5.10).

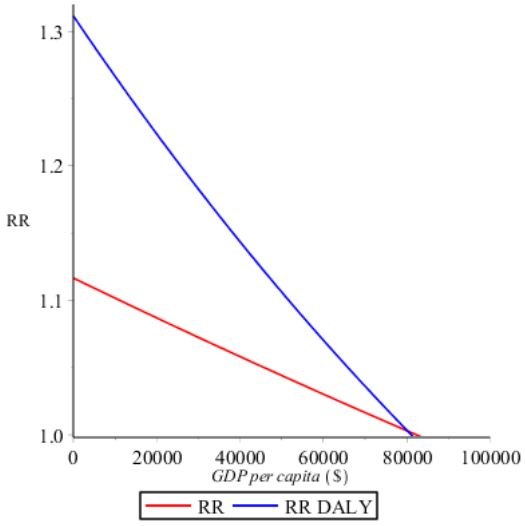


Figure 5.9. Theoretical relationship between RR and GDP per capita based on coefficient estimates shown in Figure 5.4 and 5.5 and the updated equations (5.7) to (5.10)

Decarbonisation policies have the potential to generate significant health co-benefit via the reduction in ambient PM pollution, yet most health co-benefit studies do not account for the role of economic development and other changes in socio-economic factors. I consider several scenarios with different combinations of CO_2 emission, GDP change and population growth between 2010 and 2050:

- Scenario 0 (baseline): Global population is brought smoothly to an equilibrium value of 10.5 billion, with a reduction in the rate of population growth of 3% per year in all nations (i.e. the reduction during a given year is 3% of the growth rate at the start of the year). Growth rate in *per capita* emissions in the Annex I[108] and non-Annex I nations continues unabated into the future. This would produce cumulative global emissions of 1,330 GtC or 4,870 $GtCO_2$ between 2000 and 2100 and therefore fails to meet any of the global climate policy targets by a factor of more than 2. Inflation-adjusted *per capita* GDP increases by 1% per year in Annex I nations and 5% per year in non-Annex I nations.
- Scenario 1: Global population is brought smoothly to an equilibrium value of 10.5 billion, with a reduction in the rate of population growth of 3% per year in all nations. The Annex I nations reduce carbon intensity of the economy at 3% per year beginning

[108] A list of Annex I countries can be found here: http://www.oecd.org/env/cc/listofannexicountries.htm. In general Annex I countries are developed counties which are required to take on climate change mitigation responsibilities while non-Annex I countries refer to developing countries which are permitted to take on relatively less burden for economic reasons.

in 2015. The non-Annex I nations reduce carbon intensity of the economy at 5.2% per year beginning in 2020. The rate of growth of energy demand for Annex I nations is slowed by 4% per year beginning in 2020 (4% of the current rate of growth, not a 4% reduction in energy demand). The rate of growth of energy demand for non-Annex I nations is slowed by 2% per year beginning in 2050 (2% of the current rate of growth, not a 2% reduction in energy demand). Inflation-adjusted *per capita* GDP increases by 1% per year in Annex I nations and 5% per year in non-Annex I nations.

- Scenario 2: Global population is brought smoothly to an equilibrium value of 10.5 billion, with a reduction in the rate of population growth of 3% per year in all nations. The Annex I nations reduce carbon intensity of the economy at 5% per year beginning in 2015. The non-Annex I nations reduce carbon intensity of the economy at 2% per year beginning in 2050. The rate of growth of energy demand for Annex I nations is slowed by 10% per year beginning in 2020 (10% of the current rate of growth, not a 10% reduction in energy demand). The rate of growth of energy demand for non-Annex I nations is slowed by 1% per year beginning in 2050 (1% of the current rate of growth, not a 1% reduction in energy demand). Inflation-adjusted *per capita* GDP increases by 1% per year in Annex I nations and 5% per year in non-Annex I nations.
- Scenario 3: This is a scenario based on Scenario 1 but with more dramatic declines in *per capita* emissions from the non-Annex I nations after 2020 as shown in Figure 2. Inflation-adjusted *per capita* GDP increases by 1% per year in Annex I nations and 5% per year in non-Annex I nations.

The various scenarios with regards to the emission of CO_2 are shown below in *Figure 5.10*, *5.11* and *5.12*.[109] The blue lines denote Annex I countries while the red lines denote non-Annex I countries.

[109] This part of the modelling, namely from *Figure 5.10* to *Figure 5.15* was computed by Professor Douglas Crawford-Brown, who kindly agreed to assist me in the process, since he had the data and spreadsheet already setup for the purpose of the modelling.

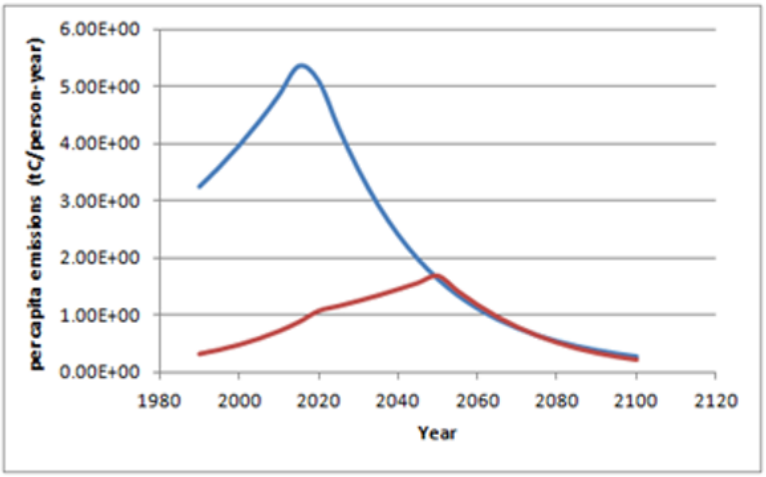


Figure 5.10. Per capita CO2 emission under scenario 1, blue line represents Annex I countries while red line represents non-Annex I countries

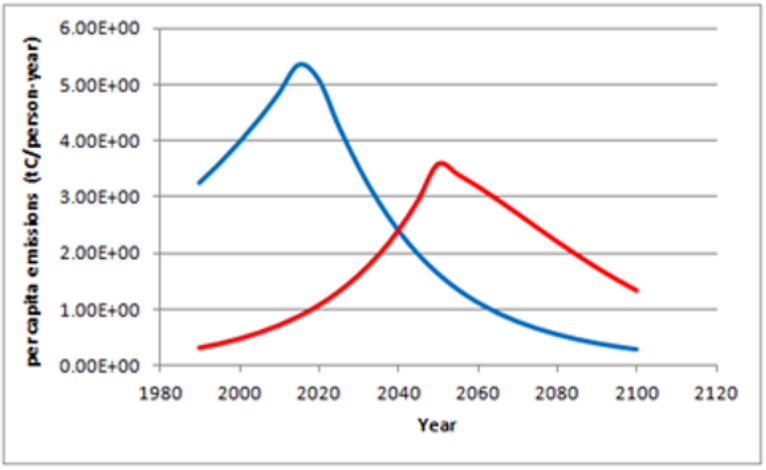


Figure 5.11. Per capita CO2 emission under scenario 2, blue line represents Annex I countries while red line represents non-Annex I countries

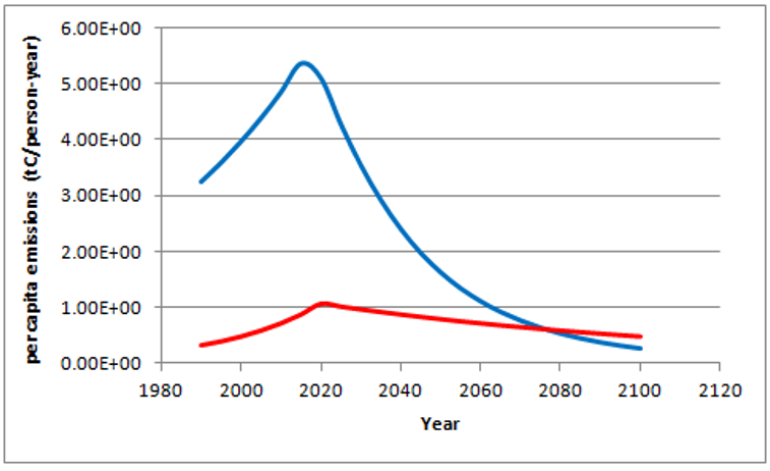


Figure 5.12. Per capita CO2 emission under scenario 3, blue line represents Annex I countries while red line represents non-Annex I countries

Under each scenario I estimate the corresponding co-reduction in PM emission and the associated effect on the steady state of ambient PM concentration based on the meta-analysis

reported by Crawford-Brown et al. (2013). I then map the resulting health effect or exposure-response using the methodology developed in this section as specified by equations (5.7) to (5.10), especially (5.9) and (5.10) which directly calculates the change in health risks. I compare scenarios 1, 2 and 3 with the baseline scenario (scenario 0), which assumes minimal efforts to reduce CO_2 and other GHG emissions. The differences in mortality between each scenario and the baseline scenario represents the health co-benefit of reduced mortality for the decarbonisation strategies pursued under each scenario. I focus my analysis here solely on mortality and do not explore morbidity. Morbidity estimates may be computed using the same process except the use of equations (5.8) and (5.10) where (5.7) and (5.9) are used for mortality.

In most studies of health co-benefits whether via reducing the emission of CO_2 or policies designed to improve air quality, the effects of socio-economic variables are neglected. Since higher incomes or stronger economic performance reduces health risks associated with ambient PM pollution, and given that most countries grow economically under the various climate policy scenarios, especially the 'developing economies' or non-Annex I countries, it is likely that such health co-benefit studies that do not reflect economic growth over-estimate the health impact of reducing CO_2 emissions and the emissions of other pollutants. This finding may represent a paradox for developing economies concerned about public health. By diverting resources to improve air quality and/or reduce the emissions of CO_2 the public directly benefit from the health co-benefits, yet if such benefits are achieved at the expense of forgoing economic growth opportunities which limits the income growth particularly of its poorest citizens, the health co-benefits will be reduced and may even be negative in the net.

Figure 5.13 below illustrates the estimated cumulative global health co-benefit from 2010 to 2050 for scenarios 1 to 3, under the assumption that the RR of mortality is unaffected by changes in GDP per capita over time or across countries. In other words the change in RR_{jY} is simply the change in ambient $PM_{2.5}$ concentration multiplied by the coefficient 0.002, the same approach as in Crawford-Brown et al., (2013). On the other hand *Figure 5.14* illustrates the estimated cumulative global health co-benefit from 2010 to 2050 for scenarios 1 to 3, assuming that increases in GDP per capita reduces the RR at a negative exponent rate as shown in equation (5.9), accounting at the same time for changes in ambient PM.

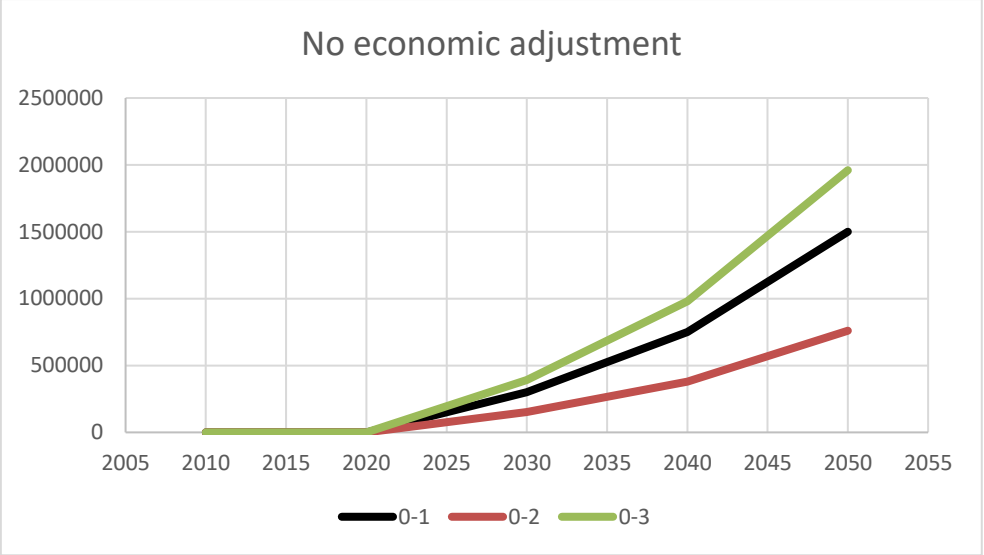


Figure 5.13. Cumulative health co-benefit between 2010 and 2050, for scenarios 1,2 and 3, excluding the effect of GDP per capita change on RR. The y-axis denotes the annual incidents of averted mortality related to ambient PM pollution and the x-axis denote the yea

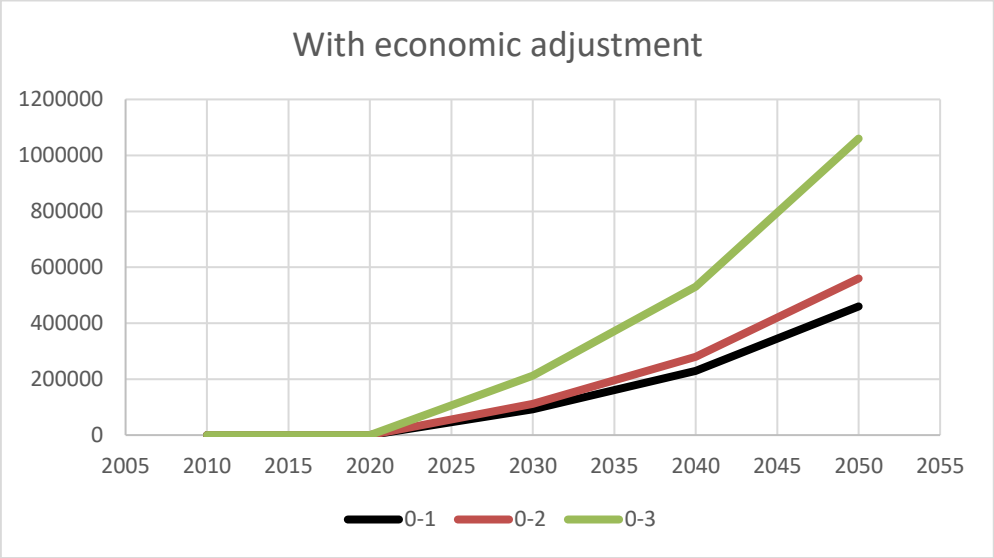


Figure 5.14. Cumulative health co-benefit between 2010 and 2050, for scenarios 1,2 and 3, including the effect of GDP per capita change on RR.

The y-axis denotes the annual incidents of averted mortality related to ambient PM pollution and the x-axis denote the year

The final cumulative health co-benefit for the different scenarios whether including the effect of GDP per capita or not, can perhaps be better represented by displaying the numbers as shown in *Figure 5.15* below.

	No GDP change		GDP change		Fraction difference	
	Total cumulative	Annual average	Total cumulative	Annual average	Total cumulative	Annual average
0	1.20E+08	3.00E+06	9.00E+07	2.25E+06	2.50E-01	2.50E-01
1	9.00E+07	2.25E+06	8.10E+07	2.03E+06	1.00E-01	1.00E-01
2	1.05E+08	2.63E+06	7.90E+07	1.98E+06	2.48E-01	2.48E-01
3	8.10E+07	2.03E+06	6.90E+07	1.73E+06	1.48E-01	1.48E-01
0-1	3.00E+07	7.50E+05	9.00E+06	2.25E+05	7.00E-01	7.00E-01
0-2	1.50E+07	3.75E+05	1.10E+07	2.75E+05	2.67E-01	2.67E-01
0-3	3.90E+07	9.75E+05	2.10E+07	5.25E+05	4.62E-01	4.62E-01

Figure 5.15. Cumulative global health co-benefit up to 2050 for various scenarios, with and without considerations of GDP per capita, as well as the fractional differences when such considerations are factored.

Rows one to four show mortality attributable to ambient PM pollution up to 2050 for the baseline scenario, scenarios 1, 2 and 3, respectively. Rows five to seven show the difference between the baseline scenario and scenarios 1,2 and 3, respectively. Columns 1 and 2 show the cumulative mortality up to 2050 and the annual average respectively, without considering the effect of GDP change. Columns 3 and 4 show the same information considering GDP change. Columns 5 and 6 show the fractional difference when GDP is no considered versus the inclusion of GDP change.

Out of the three scenarios 1, 2 and 3 in reference to the baseline scenario 0, scenario 3 pursues the most stringent decarbonisation strategy followed by scenario 1, with scenario 2 the least. This is shown by *Figure 5.10*, *5.11* and *5.12*. From *Figure 5.13*, it is apparent that greater stringency in pursuing global decarbonisation leads to higher health co-benefit, and the differences between the three scenarios and the baseline scenario are directly proportional to the degree of decarbonisation ambition. This is the case when one completely ignores the role of economic growth over time as well as different levels of economic development between countries. When GDP per capita is factored in however, *Figure 5.14* shows that the health co-benefits decrease significantly for all three scenarios. What is interesting however, is that the decrease is not proportional for the three scenarios. By around 2030 the annual global health co-benefit under scenario 2 begins to overtake that of scenario 1. All this can be summarised in *Figure 5.15*. In columns 5 and 6 I see that all the fractional differences between the same scenarios with the exception of whether GDP change is included, are positive. This means that whenever GDP change is included in health co-benefit calculations, the magnitude of the health co-benefit inevitably decreases (although the health co-benefit remains a positive value).

5.2. Applying my Model to Explore the Inequality Implications of Air Quality and Decarbonisation Policies

Our theoretical model which I reformulate to obtain equation (5.1) can be used to analyse the inequality implications of air quality/environmental policies and decarbonisation strategies. If ambient PM pollution is interpreted as the exogenous rate of health depreciation δ_0 or at least a component of it, then the rate of health depreciation may be stated as a function of the stringency of an environmental policy, decarbonization strategy, or a vector of scenarios which ultimately influences δ_0. This is shown by equation (5.11) below:

$$\delta_0 = \delta_0(\rho) \qquad \delta_0' < 0 \; \forall \; \rho \tag{5.11}$$

Where ρ denotes the stringency of air quality/decarbonisation policies or a vector of scenarios which are expected to reduce ambient air pollution and health capital depreciation.

The health co-benefit of air quality policies and decarbonisation strategies can be obtained by the following equation:

$$\frac{\partial H^*(t)}{\partial \delta_0}\delta_0' = -(A\delta_0)^{-1}\left(\frac{y(E)^{A+1}}{A\delta_0 P_M P_X^A} e^{A(GH_0-1)-2+\frac{P_M}{y(E)}} - Gt\right)^{-1} t\delta_0' \tag{5.12}$$

Given that $\delta_0' < 0$, health co-benefits are positive.

The health co-benefit equation shown above by (5.12) is expressed as a function of exogenous variables. Most of these exogenous variables can be considered socio-economic variables. The most important socio-economic variables of interest are income (y), education (E) and age (t). If equation (5.12) is differentiated partially with respect to these variables, then I obtain a picture of how the health co-benefit of air quality/decarbonisation policies varies along these socio-economic lines. A positive value of the partial derivative with respect to income for example, indicates that health co-benefits accrue more to those who are of higher income and the converse is true in the case of a negative partial derivative. *Table 5.3* below shows the partial derivatives of the health co-benefit function (5.12) with respect to the three socio-economic variables. I assume that $y'(E)$ is always positive i.e. that education always increases the income, but I do not specify the exact functional form.

Table 5.3. Partial derivatives of health co-benefit from air quality/decarbonisation policies as shown by equation (5.12), with respect to income (*y*), education (*E*) and age (*t*).

Partial derivative	Equation of partial derivative	Theoretical prediction
$\frac{\partial}{\partial y}\left(\frac{\partial H^*(t)}{\partial \delta_0}\delta_0'\right)$	$-(A\delta_0)^{-1}\left(A+1-\frac{P_M}{y}\right)\frac{y^A}{A\delta_0 P_M P_X^A}e^{A(GH_0-1)-2+\frac{P_M}{y}t}\left(\frac{y^{A+1}}{A\delta_0 P_M P_X^A}e^{A(GH_0-1)-2+\frac{P_M}{y}}-Gt\right)^{-2}$	$\frac{\partial}{\partial y}\left(-\frac{dH^*}{d\delta_0}\right)<0\ \forall\ t \in [0,V]$
$\frac{\partial}{\partial E}\left(\frac{\partial H^*(t)}{\partial \delta_0}\delta_0'\right)$	$-y'(A\delta_0)^{-1}\left(A+1-\frac{P_M}{y}\right)\frac{y^A}{A\delta_0 P_M P_X^A}e^{A(GH_0-1)-2+\frac{P_M}{y}t}\left(\frac{y^{A+1}}{A\delta_0 P_M P_X^A}e^{A(GH_0-1)-2+\frac{P_M}{y}}-Gt\right)^{-2}$	$\frac{\partial}{\partial E(q)}\left(-\frac{dH^*}{d\delta_0}\right)<0\ \forall\ t \in [0,V]$
$\frac{\partial}{\partial t}\left(\frac{\partial H^*(t)}{\partial \delta_0}\delta_0'\right)$	$(A\delta_0)^{-1}\frac{y^{A+1}}{A\delta_0 P_M P_X^A}e^{A(GH_0-1)-2+\frac{P_M}{y}}\left(\frac{y^{A+1}}{A\delta_0 P_M P_X^A}e^{A(GH_0-1)-2+\frac{P_M}{y}}-Gt\right)^{-2}$	$\frac{\partial}{\partial t}\left(-\frac{dH^*}{d\delta_0}\right)>0\ \forall\ t \in [0,V]$

The results in *Table 5.3* above predicts that health co-benefits are lower for groups which have higher incomes and those who are more educated, yet are larger for those who are older. Air quality and decarbonization policies therefore have the potential to generate health co-benefits which promote equality, since according to my model, these groups which are often considered less privileged groups, are the greater beneficiaries of such policies.

It is possible to empirically test the predictions in *Table 5.3*, though I do not conduct such test as part of this thesis. Since it is not possible to obtain individual data relating to estimated health co-benefit, such a test perhaps requires aggregate data also. It is necessary to obtain data on different income, education and age groups, monitoring their change in health following alterations in the ambient PM exposure. The next Chapter discusses the policy implications of the results of the theory developed in Chapter 3, the empirical results of Chapter 4 and the application to health co-benefit studies from decarbonisation presented in this Chapter.

CHAPTER 6 – Policy Implication

Having developed the main thrust of my theoretical framework in Chapter 3, empirically verifying the main testable hypotheses derived from my theory in Chapter 4, and explored some of the main application of my model in Chapter 5, I move on to discuss the policy implications of my findings in this chapter. The implications are divided into three main types. First, the health capital model developed brings forth several hypotheses, which are validated and therefore can be used as a basis for proposing general health policies concerning socio-economic factors. This is discussed in section 6.1. Second, the reformulation of the health capital model by making the endogenous or optimal time path of health depreciation the dependent variable has implications for air quality, decarbonisation and environmental policies. This is discussed in section 6.2. Thirdly, through manipulating the health depreciation function, it is possible to predict the inequality implications associated with air quality and decarbonisation policies. In fact, it can be shown that a general toolkit can be developed which predicts the inequality implications of any policy related to the endogenous variables in my model. These are discussed in section 6.3.

6.1. Policies Implications Concerning Health and Socio-economic Variables

In Chapter 3, a theoretical model is developed based on Grossman (1972)'s health capital model. One of the main novelties of the model is the division of the life cycle analysis into the dual phases of childhood and adulthood. During the childhood phase, there are two state variables, which are the stock of health and education. However, during the adulthood phase the stock of education becomes fixed or exogenous and the sole state variable is the stock of health capital. The main model output from my theory is the derivation of the optimal time path for the stock of health capital, for both the childhood and adulthood phase shown by equations (3.14) and (3.21''), respectively. The main testable hypotheses are shown by equations (4.1), (4.2) and (4.3). The effect of a change in various exogenous variables on health, are derived using comparative dynamic analysis and shown in *Table 4.2* and *Table 4.4* for the childhood and adulthood phases, respectively. They are illustrated graphically by *Figure 4.2* and *Figure 4.5* for the childhood and adulthood phases, respectively. Since the two phases are separated both theoretically and empirically, it is helpful to discuss them separately. Section *6.1.1* discusses the issues raised regarding the childhood phase model and the policy implications, while section *6.1.2* discusses the same for the adulthood phase. The life expectancy or optimal length of life model developed from the adulthood phase model is described in section *6.1.3*.

It should be noted that since my research is theoretically focused, the empirical and policy implications are in most cases generic. Many of the policy suggestions simply reinforce what is already known and suggested by medical professionals, though more from a behavioural perspective. It is not really possible to draw very specific policy implications unless the insights of the theory is applied to a very specific context. However, the main strength of the theory in my opinion is not in direct policy application but that it will highlight and emphasise certain principles and issues which regulators should bear in mind when designing specific policies.

6.1.1. Policy Implications of Findings for the Childhood Phase Model

For the childhood phase of the model, the main equation which shows the relationship between the dependent and independent variables, which are of interest to us is shown by equation (3.14). Equation (3.14) produces the econometrics model/equation to be estimated shown by (4.1). The important exogenous variables are β, A, θ, M, δ, t and q. These represent in the following order the subjective rate of time discount, the parameter governing the efficiency of inputs into the production of health capital and education, the relative importance of education to the child compared to childhood utility, the use of medical care or services,[110] health depreciation, time/age of the child and time/age when childhood ends, respectively. These variables are in turn represented by questionnaire response variables to the following questions 'gets head-aches, stomach-aches or sickness', 'restless and cannot stay still for long', 'is easily distracted. is difficult to concentrate', 'importance of doing well in GCSEs or standard grades', 'number of portions of fresh fruit and vegetables per day', 'frequency of eating fast food: days in a normal week', 'age for whole sample, from birth or age if', and 'age you think when you leave home?', respectively. These are shown in *Table 4.1*. I do not consider the effect of the variables G and ω, which represent the marginal product of health capital in the production of health time and the marginal product of health capital in assisting the accumulation of education respectively, on the optimal stock of health capital, since no suitable data are available.

It should be noted that none of the above exogenous variables can be considered socio-economic variables.[111] Important socio-economic variables such as income are omitted since by definition, most children do not participate in full-time employment and earn income from

[110] Or any goods and materials, which are believed to contribute positively towards health.

[111] t, which represents time/age of the child, can perhaps be considered more of a socio-economic variable for adults but for the childhood stage it is primarily a biological variable.

work. This presents a slight challenge to my study since I am primarily interested in knowing the socio-economic determinants of health. However, I expect most of the exogenous variables to be strongly correlated with the socio-economic variables of the child's parents or household circumstances. The most obvious example is θ, the relative importance of education to the child. Empirical studies consistently report strong correlation between the education of parents and their children (Black et al., 2003; Chevalier et al., 2013)**.** Therefore, it is likely that children with highly educated parents are instilled with the values of the importance of education due to family upbringing and thus have a high θ value. This may also be the case for wealthier families. On the other hand however, θ may correlate with factors, which are not necessarily socio-economic in nature. For example, θ is very likely to correlate with culture, ethnicity and family background. Certain ethnic minorities are known to place heavy emphasis on the education of their children while other groups pay less attention to the education of the future generation. θ may simply be a reflection of a child's personality concerned with determination to succeed in life. If so, θ cannot be explained by socio-economic variables of either the child or his/her parents. It cannot be explained by any form of data available with the exception of perhaps genetic information of the child, which completely falls outside the scope of my analysis. Nonetheless, there is growing research exploring the linkage between certain genetic components and health problems.

The variables A, M and δ are also likely to correlate with the socio-economic status of the child's parents. Wealthier parents may provide greater resources to the child in the form of better learning and sporting equipment, tutors and instructors, and perhaps some form of tacit knowledge, which increases the efficiency of the child's input in the production of both health and educational capital. Grossman (1972) models education as the efficiency parameter governing the production of health capital, which gives rise to the 'health-education' gradient whereby strong correlations of health and education are often observed. The alternative case is presented here that some third factor(s) encompassed in the efficiency parameter A, which correlates with socio-economic variables, contributes to the efficiency in the production of *both* health and education, and thereby resulting in the health-education gradient. Wealthier families are also likely to spend more on the medical care for their children and purchase health insurance plans, hence M should correlate positively with parental income. However, in countries where government programmes provide for the medical care of children or substantially subsidise it, M may display smaller variation and its correlation with parental

income reduced. Likewise, wealthier families may invest more resources to protect the health of their children from harmful effects hence δ would correlate negatively with parental income.

β on the other hand is unlikely to be correlated with any socio-economic variables of either the child or his/her parents. It is probably a variable reflecting child personality and therefore can be treated as a random exogenous variable. Likewise, little or no correlation exists between t and parental socio-economic status. There may be some relationship between q and socio-economic status of parents. This is because the variable q represents three phenomena, which can be considered biological, legal and social, connected with the arrival of adulthood. Firstly, the arrival of q in a child's life represents the arrival of adulthood from a biological perspective in that the child's bodily development phase is completed. I model this by altering the specification of the health production function for $t > q$. The health investment function for $t < q$ is represented by equation (3.4'') while for $t > q$ it is switched to equation (3.7''). This is the case since as a child, he or she can build up the stock of health capital using time and input of health producing goods and services, but as an adult whose body has taken shape such inputs can serve only to reduce the rate of health decline. Secondly, the age q marks the age a child becomes an adult legally. Therefore q may denote some landmark age a child has reached, such as 16, 18 or 21. Coupled with this is often a time when the child faces some major examination such as A levels and other university/college entrance exams. Therefore, the child seeks to maximise the accumulated stock of educational capital up to q. The higher the stock of educational capital, the better the performance in the exams. Thirdly, q marks the end of childhood from a social perspective in that the child leaves home and must support himself or herself financially. The model in Chapter 3 makes the implicit assumption that the three phenomena, biological, legal and social termination of childhood and the commencement of adulthood completely coincide, and are represented by a single number q. In reality this is unlikely to be the case, yet for simplicity this assumption is adopted.

The absolute and relative influence of the biological, legal and social aspects of q determine the degree to which q is affected by the socio-economic status of the child's parents. The biological influences on q would not correlate with any socio-economic variables. However, there may be substantial variations between individual children since the biology of each child and when the body stops growing are governed by genetic factors. The legal influences on q likewise would not correlate with the socio-economic variables. Moreover, there would be little or no variation between individual children. The social influences on q display both the

greatest variation between individual child and correlation with the socio-economic status of the child's parents. Every family differs in the age up to which they support their child financially. Some stops support at the age of 18, others after the completion of university and gaining employment. Some families continue to support the child long into their adult life. The age at which the child stops receiving support from their parents is likely to correlate strongly with the family's socio-economic status. Wealthy families are likely to continue their support for longer than less affluent families.

The potential for the exogenous variables to correlate with socio-economic variables of the child may result in the problem of endogeneity. This is because the latter becomes subsumed under the error term, which should not correlate with the exogenous variables in a regression. The ideal econometric model in this case would be a two-stage-least-squared regression, yet this is not possible using panel data where the dependent variable is non-continuous. Furthermore, my data for the socio-economic variables of the child's parents or household displays relatively less variation compared to these exogenous variables. Moreover, I aim to test variables directly concerned with the child according to my model and testable hypotheses, rather than secondary variables. The bias resulting from the endogeneity, however, is unlikely to be very large as there are other independent factors, which influence these exogenous variables. Moreover, since I apply the ordered logit regression, the actual magnitudes of the coefficient effects are unimportant and only the signs may be used for interpretation. Unless the degree of bias is extremely large it should not affect my conclusion by altering the signs of the conclusions. For these reasons, using this set of exogenous variables to explain the effects on child health is most appropriate.

As can be seen from *Figures 4.3* and *4.4* that the direction of the effect on the health of children, with the exception of the variable θ, are all in line with my expectations. The subjective rate of discount, reflecting the degree of the child's time preference, reduces the optimal stock of health capital. Fuchs (1980) conducts a thorough though somewhat exploratory experiment on the relationship between time preference and health (as well as schooling). They report that the implicit discount rate (interest rate) of individuals is weakly correlated with health negatively. This is consistent with my result, which shows that a small increase in the rate of discount[112] is associated with a statistically insignificant fall in health capital. However, a substantial

[112] When the response to the survey question 'restless and cannot stay still for long' changes from 'not true' to 'somewhat true'.

increase in the rate of discount[113] displays a significant negative relationship. As Fuchs (1980) argues there may be two possible scenarios regarding the rate of discount. First, time preference may be established early in life and remain stable throughout life. Alternatively, time preference may be influenced by education so that more educated individuals have lower rates of discount. My model assumptions in Chapter 3 are similar to the first scenario, where I define a constant subjective rate of discount β. However, it is certainly plausible that β is non-constant, influenced by education, family values, culture and religion.

According to my model, children with a higher rate of discount value current levels of utility relatively more. As a result, they devote less time to acquiring health capital, $\tau_H(t)$ whilst valuing free time, $\tau(t)$ more highly. I therefore observe the negative relationship in my model consistent with Fuchs (1980). The same can be said regarding devotion to educational capital – children with higher discount rates would substitute greater $\tau_E(t)$ for $\tau(t)$, leading to lower stock of educational capital ceteris paribus. This is also confirmed by the reported negative relationship between time preference and schooling in Fuchs (1980).

From a policy perspective, I can therefore make the case that reducing β would increase the health of children (as well as their education). The government can perhaps utilise the education system to instil in children the value of patience, not exhausting all their resources at once, and encourage them to save for the future. Since the scopes for children to save and invest financial resources are rather limited, health and education would function as their main opportunity to save and invest. Programmes and research can be conducted to convey the idea that there is clear link between exercise, a form of time input into health, and the level of health. Furthermore, clearer distinctions should be made to the children regarding activities which constitute $\tau_H(t)$ and those which merely constitute $\tau(t)$. The message may be conveyed that whilst time is devoted to $\tau_H(t)$ away from $\tau(t)$, it will increase future free time since a higher stock of health capital reduces sick days, so that $\tau(s) > \tau(t)\ \forall\ s > t$.

Figures 4.3 and *4.4* also show that there is a positive relationship between health and the efficiency parameter A, represented by the responses to the question 'is easily distracted. Is difficult to concentrate'. I know that concentration affects productive efficiency at least in time input, and likely also the factor input. Therefore, the response to the above question is a suitable

[113] When the response to the survey question 'restless and cannot stay still for long' changes from 'not true' to 'certainly true'.

representation of A. The policy implications of this finding are that if children can be made to concentrate, to focus or provided with the necessary equipment and knowledge to work and exercise effectively, their health would improve. This is because a higher efficiency parameter raises incentives for the child to invest more time into either the production of health or educational capital.

The variable θ is the only exogenous variable whose effect on health capital is opposite to my expectation. *Figures 4.3* and *4.4* both show that an increase in θ is negatively associated with the stock of health. θ is represented by the response to the question 'importance of doing well in GCSEs or standard grades'. The reference dummy category is 'not at all important'. Compared to this category, the other three categories 'not very important', 'important' and 'very important', which denote progressively higher values of θ, are associated with lower health. None of the categories however, show statistically significant correlation. The model in Chapter 3 assumes that health capital raises the rate of educational capital accumulation. Therefore, children who wish to reach a high stock of educational capital by $t = q$, due to a preference for education (high value of θ), have incentives to invest in health, thus should possess higher stocks of health capital ceteris paribus. It appears from the results of *Figures 4.3* and *4.4* that this assumption and the reasoning process is unlikely to be true. Perhaps health does not play a substantial role in learning beyond generating more healthy time, which may or may not be devoted to learning. If this is the case, children who wish to succeed academically may be forced to sacrifice time devoted to health investment, such as exercise, to study in order to increase education. The results suggest that health and education are conflicting or substitutionary goals for the child, rather than complementary. Note this finding does not imply that health and schooling are negatively correlated. It is often the case that important variables connected with both health and education, such as A the efficiency parameter, induces the child to obtain higher levels of both health and education. θ does not represent the stock of education but merely the relative importance of educational attainment to the child. A potential policy implication of this finding is that education policies should be designed in a way to minimise the conflict between health and education. The education curriculum should be set in a way, which prevents excessive substitution of $\tau_H(t)$ for $\tau_W(t)$. Perhaps mandatory physical education lessons of minimum hours per week should be imposed to ensure that $\tau_H(t)$ does not fall below a level, which would be considered unhealthy. Alternatively, the importance in health can be incorporated into the education curriculum via certain means. For example, in China there are concerns over the physical health of children in recent years due to heavy

examination pressures. As a result, certain athletic achievements can be used to supplement examination scores. Such a policy would be akin to increasing ω, the marginal product of a unit of health capital in the production of educational capital. Raising ω via educational policy would incentivise children and their parents to invest in the health of their children for the purposes of increasing the stock of educational capital. Moreover, since the payback of health capital in enhancing educational capital would last $q - x$ years, where x is the year the child decides to invest heavily in health, such an investment is likely to be taken early so that x is likely to be small, implying that children have the incentive to make such investment decisions early rather than later in his/her life.

Figures 4.3 and *4.4* also show that increases in M is positively associated with health capital. M is represented empirically using data from the question 'number of portions of fresh fruit and vegetables per day', where by the minimum category 'none' is selected as the reference category. All categories – '1-2 portions', '3-4 portions' and '5 or more portions' show large and statistically significant improvement in health compared to the reference category. M is primarily used to denote medical care and services employed by the child, but may also include any goods or services used to enhance or to protect health. Since no data on medical care is available in the youth self-completed questionnaire in Understanding Society, data on fruit and vegetable consumption are used.

Most empirical studies examine the relationship between medical care/services employed and health at the micro level report negative relationships (Wagstaff, 1986). Intuitively, this is obvious since medical care usage often is an indicator of poor health and used only by unhealthy individuals in the hope of returning back to health. It is difficult to control for the problem of endogeneity, which is argued by Grossman (2000) for the observed negative relationship reported by other studies. For the childhood phases of my model, endogeneity is unlikely to be a problem since medical care for children are exogenously determined by parents and fully paid for by the NHS. It is unfortunate that suitable data for medical care usage does not exist for the youth questionnaire. At the aggregate or national level, the opposite is true and empirical studies tend to report positive association between medical spending and measures of health. This disparity in relationship between the micro and macro level is not fully understood. However, this may be due to the difference in which health is measured at the micro compared to the macro level. At the macro level indicators of health usually take the form of life expectancy or infant mortality, which are highly sensitive to national health

spending, particularly in developing countries (May and Smith, 2011). On the other hand at the micro-level indicators of health relate more to morbidity rather mortality measures. Long-term chronic illnesses for example, can be extremely difficult and expensive to cure. Moreover the high medical spending exhibits strong self-selection bias where those who spend most on medical care tend to be those with severe health issues, which would be averaged out if the data is taken at the macro-economic level. For these reasons I propose that empirically at the micro level, medical care usage should not be seen as an input to improve health, but as an indicator of poor health or high rate of health depreciation. Only certain medical input such as preventative medicine, vaccination and regular health check-ups which are *independent* of the user's health condition should be considered as direct health inputs, rather than all forms of medical care at large.

The results in *Figures 4.3* and *4.4* demonstrate that consumption of health food, fruit and vegetables contribute positively to health. However, the difference between consuming above 1-2 portions does not differ significantly in its effect on health, suggesting diminishing returns to M. Empirically, if it is decided that the consumption of health promoting goods and services rather than traditional medical care constitute M, the challenge is to differentiate M from other consumption goods, $X(t)$. Perhaps $X(t)$ can be defined as the consumption of only health neutral goods, which enhances utility. There is, however, also the issue of consumption goods, which negatively impact health, such as cigarettes, (excessive) alcohol and non-prescribed drugs, which are not modelled and accounted for. The next variable to be discussed δ, the rate of health depreciation is represented empirically using fast food consumption frequency, which may be considered as a form of $X(t)$, which negatively impacts health. The policy implications of the findings here is that the government should require parents to provide sufficient quantities of healthy food to all children, subsidising them whenever necessary. There is no need to provide excessive amount but only ensure a minimum standard. The government may directly provide such goods to children during school times. There are now attempts in many schools to create healthier meals for children (Story et al., 2009; Belot and James, 2011).

The time path of childhood health capital is non-monotonic in that it reaches a maximum at a certain age t before declining, as illustrated by *Figure 4.2*. The data for age in the Understanding Society questionnaire is used to represent t. The reference category is age 10. Compared to this category, the results in *Figure 4.3* and *4.4* show that all other age categories listed, 11, 12, 13, 14 and 15 are associated with better health, though many categories are not

statistically significant. It is not possible to predict via the theory alone the age at which change in health becomes negative. Ideally two terms for age should be included in the regression of equation (4.1), t and t^2, which facilitate the capturing of the turning point. If the theoretical prediction is correct, the coefficient for t^2 should be negative. However, it is not appropriate to include such variables since the data available via the Understanding Society youth self-completed questionnaire does not list age as a continuous variable but instead provides the discontinuous categories from 10-15 years of age. It is perhaps the case that $t = 15$ falls left of the turning point since the decline generally begins only when $t \to q$. As described earlier q encompasses three events in the child's life – biologically coming of age, legally becoming an adult and face major examinations, and leaving home whilst becoming responsible for one's own life. It appears that even at age 15, which are the oldest children in my sample, there is a significant gap until any of the landmark age is reached, which should be until at least 18. Nonetheless the child is likely to face some interim examination pressures such as GCSE exams, generally taken at age 15/16, so some effects of the approach of q may be expected. According to the results in *Figures 4.3* and *4.4*, the improvement in health via increase in age is not apparent until age 14 and 15. The finding that health increases with age has limited policy application, since becoming older is the natural progression of the population. Perhaps similar but contrary to the fact that aging population requires greater medical attention, a population which has a significant number of people coming of age, may reduce such medical burdens for the government.

The response to the question 'age you think when you leave home?' is used to represent the variable q. The results in *Figures 4.3* and *4.4* that increasing the age a child leaves home is associated with better health is relevant, although the results are not statistically significant. The increase in health via an increase in q is illustrated in *Figure 4.2* by comparing the main blue line ($H^*(t)$) with the extended purple line (q). As can be seen, the effects are not very pronounced when t is small. This is also reflected by the partial derivative $\frac{\partial H^*(t)}{\partial q}$ shown in the last row of *Table 4.2*. From that equation, it can easily be seen that the partial derivative increases as t increases. Therefore, the statistical insignificance of the results in *Figures 4.3* and *4.4* is somewhat expected, since an increase in q would not in and of itself result in an increase in health, especially when age is small, which is the case with the sample used. The statistical insignificance in the expected direction testify to the strength rather than the

weakness of the model, and it is likely that an increase in q will result in significant health improvement of children who are older.

The policy implication of this finding is that q should be extended for as long as reasonably feasible. I expect a dip in health as children approach age q. The effect may be caused by the approach of major examinations. Therefore, it may be beneficial for children if such assessments are delayed for as long as possible. The 11-plus for example, an examination to determine high school entry has already been cancelled in the United Kingdom with the exception of Northern Ireland. If children know that they will leave home at a later age and/or know that their parents can support them, this negative health effect may also be diminished. Governments should therefore encourage parents to save and support their children for as long as feasibly possible. The theoretical reason for this dip is due to the assumption of disjoint childhood and adulthood phases, whereby children have an incentive to exhaust their resources, including health capital as q approaches, seeing that it has no further use beyond q. In reality, childhood and adulthood are connected and most children realise this to an extent. Government policies and education should therefore be designed in a way to facilitate children to plan for their entire lives and not merely certain phases of their lives alone. With important examinations and children's mindsets detached from the adult world, an atmosphere of 'all or nothing' may be created subconsciously, which encourage children to focus only on short-term goals related to their childhood phase, such as education. Perhaps continuous development, the reduction in the significance of certain landmark age both legally and culturally, as well as more awareness of life beyond childhood including the opportunities and possibilities, would significantly reduce a sudden decline in health.

6.1.2. Policy Implications of Findings for the Adulthood Phase Model

For the adulthood phase model, the theoretical relationship between health, the dependent variable and the other exogenous variables are shown by equation (3.21''). Equation (3.21'') is translated into equations (4.2) and (4.3). The main exogenous variables are A, δ_0, y, $E(q)$, and t. These represent in the following order the subjective rate of time discount, the parameter governing the efficiency of inputs into the production of health capital, the exogenous rate of health depreciation, income/wage, stock of educational capital (accumulated as a child), and age/time (after childhood). These variables are in turn represented by the following questionnaire variables from the BHPS – 'Concentration', 'Number of cigarettes smoked', 'Labour income: last month', 'Highest qualification' and 'Difference between age and school

leaving age', which are shown in *Table 4.3*. The variables not considered in the empirical analysis include β, G, H_0, P_X and P_M. They represent the subjective rate of discount, the marginal product of a unit of health capital in generating healthy time, the initial stock of health capital,[114] price of a unit of goods consumption and price of a unit of medical goods/services, respectively. These variables are not considered empirically due to lack of suitable data to represent them.

Two equations rather than one are derived from equation (3.21'') since the variables y and $E(q)$, income/wage and education, respectively, should not both be included in a single econometric model. This is because income y is a function of education $E(q)$, and it is not possible to account for this composite function in the econometric models due to the dependent variable data being non-continuous and categorical in a panel data framework. Equation (4.2) contains y and the other exogenous variables listed in *Table 4.3*, with the exception of education $E(q)$, while equation (4.3) is the same as (4.2) but replaces income with education. Since y and $E(q)$ are correlated, equation (4.2) may have the problem of endogeneity, since $E(q)$ being subsumed under the error term correlates with one of the independent variables. It is possible therefore that the coefficient estimate of y on the dependent variable health is biased. However, the bias is unlikely to be large and significant enough to lead to the conclusion that health and income are actually correlated negatively if the bias is removed. Moreover, the magnitude of the coefficient estimate is immaterial given that the dependent variable is non-continuous and categorical. Only the direction of effect and statistical significance matter. It would not be appropriate to include both income and education in a single equation since the high correlation between the two variables is likely to result in the problem of multi-collinearity.

It should be pointed out that with the exception of one or two variables, all the variables in both equations (4.2) and (4.3) are highly statistically significant. All the variables are of the expected sign. This can be seen from *Figures 4.6, 4.7, 4.8* and *4.9*. The predicted effects of a change in the exogenous variables on the stock of adulthood health capital are shown in *Table 4.4* and illustrated graphically by *Figure 4.5*. Since the length of life is endogenous in the adulthood phase of the model, all changes in exogenous variables not only do they affect the stock of

[114] Initial health in reference to the commencement of adulthood is equivalent to the health capital stock attained at the end of childhood.

health at any age t but also the optimal length of the adult life, which I interpret as life expectancy.

The first exogenous variable considered in the models is A, the efficiency parameter. This is represented empirically by the response to the question 'concentration', which is similar to that used to represent the variable A in the childhood model. The category 'much less than usual' is selected as the reference category so that all other categories represent increasing A relative to this reference category. The other categories in order of rising A are 'less than usual', 'same as usual' and 'better than usual'. The results of the regression analysis for the wage model of equation (4.2), shown in *Figures 4.6* and *Figure 4.7* for the logit and probit models respectively, both reveal that the other reference categories are associated with better health. Moreover, the coefficient sizes demonstrate that progressively higher A as implied by the categories, are associated with progressively better health. The positive coefficients for the category 'better than usual' are the largest, followed by 'same as usual' and then 'less than usual'. For all categories, the coefficients are highly statistically significant, well above the 1% confidence level, with p-values approaching zero. The regression results associated with equation (4.3), where education is included instead of wage/income, are shown in *Figures 4.8* and *4.9*. *Figure 4.8* and *Figure 4.9* show the exact same message as *Figure 4.6* and *Figure 4.7*, whereby all three non-reference categories associated with the variable A are positive and highly significant with p-values approaching zero. Likewise, the coefficients for the category 'better than usual' are the largest, followed by 'same as usual' and in turn by 'less than usual'.

The policy implication of this finding is that factors, which affect the efficiency of inputs to health preservation[115] have the potential to lead to health improvement in adults. It is unclear, however, what factors would affect such a kind of efficiency. The model here utilises self-reported ability to concentrate to represent A, since being able to concentrate or focus clearly contributes to the efficiency of getting things done, whether that includes work or exercise and health preservation. A potential problem, however, of using this as the data is that the ability to concentrate might itself be an indication of health status, rather than the efficiency parameter, which is hypothesised to lead to better health via its effect on health behaviour and resource allocation. According to the concept of the 'Health-education gradient' alluded to earlier, those

[115] I replace the term 'health production' with 'health preservation' in the adulthood model since I assume that at this stage actions taken by the individual can no longer increase the stock of health capital but merely reduce the rate at which it declines.

who are more educated possesses greater knowledge of how to utilise their time and medical goods/services ($M(t)$). Whilst strong correlations between health and education are consistently reported in many empirical studies (Cutler and Lleras-Muney, 2006), including the results of this thesis in *Figure 4.8* and *Figure 4.9*, which shall be discussed shortly, there is very little empirical evidence to suggest that education significantly affects the input-output relationship in health production/preservation. This is even less likely to be the case in the United Kingdom and other developed countries where methods, information and knowledge are made widely available to the public who require only a minimum level of education (being able to read and write) in order to take advantage of them. In many developing countries, perhaps education is a means to obtain private information regarding health production/preservation and therefore such effect may be stronger. The government may increase A by providing greater public knowledge and awareness of health related issues so that the citizens become better informed. For example, the benefits of screening for cancer and sexual diseases can be emphasised so that detection and early treatment become the norm, which are far less costly and more resource efficient compared to treatment at later stages of the disease. The informative policies are likely to raise the A of the entire population at large and is unlikely to overly benefit certain portions of society more than others, at least in a developed country such as the United Kingdom. As can be seen from *Figure 4.5*, an increase in A leads to a higher stock of health for all t. The positive effects are more pronounced for larger t. An increase in A also leads to an increase in the optimal length of life (life expectancy).

The second variable to be discussed is δ_0, the exogenous rate of health depreciation. δ_0 is represented empirically using the variable 'number of cigarettes smoked'. It is now commonly accepted by many medical professionals that smoking possesses a major risk to health. Therefore, cigarette consumption is used to represent the exogenous rate of health depreciation, assuming that it is unrelated to the other exogenous variables. The results shown in *Figure 4.6* and *Figure 4.7*, corresponding to the model in equation (4.2), and the results of *Figure 4.8* and *Figure 4.9* corresponding to the model equation (4.3), all show the same results – smoking is significantly associated with lower health. The level of statistical significance is extremely high and certainly within 1% confidence interval given that the p-values are approaching zero.

The policy implication of this finding is that smoking and other health damaging consumption should be discouraged and limited to a minimum. There is also medical evidence suggesting

that passive smoking poses a significant threat to health, hence banning smoking in certain public areas may reduce δ_0. Cigarette consumption is chosen to represent the exogenous rate of depreciation as some of the health effects associated with smoking whether active or passive, are very similar to that of ambient air pollution from PM, which will be discussed shortly (Pope et al., 2009). Since it is very difficult to obtain accurate data on individual exposure to ambient PM pollution, cigarette consumption gives a good indication of the likely health effects. As expected, the effect on health is negative. This is consistent with my predictions, which can be seen graphically in *Figure 4.5*. An increase in the exogenous rate of health depreciation δ_0 leads to lower stock of health capital for all t, with the negative effects more pronounced for larger values of t. An increase in δ_0 also reduces the length of life/life expectancy. Therefore, if the government pursues environmental policies, which improve air quality by reducing ambient PM pollution, the health and life expectancy of its citizens are likely to improve substantially.

The third variable to discuss is age or t. I define the adulthood age as the actual age minus the age at which the individual left school. This corresponds with the t in adulthood model, which is not the age from birth but the age after childhood, and it is implicitly assumed here that leaving school is equated with q, the age at which childhood ends and adulthood begins. *Figure 4.6*, *Figure 4.7*, *Figure 4.8* and *Figure 4.9*, the empirical results for equations (4.2) and (4.3), all unanimously show that higher adulthood age is associated with lower health. The coefficients are highly significant, being close to zero.

There is very little policy implication for this finding, since it is not possible to stop a person getting old, and in general I can expect that health declines with age. However, in an ageing society such as the United Kingdom where the population on average is getting older, the entire society may become less healthy. The government must plan for more sick days since a lower stock of health capital translates into more sick days per unit of time. In addition, medical expenses may rise since those who are of lower health tend to be heavy users of medical care. This is because medical care and services as argued earlier in the childhood model should no longer be regarded as $M(t)$, the material goods/services, which contribute to health,[116] but instead as a signal that the individual faces a higher rate of depreciation δ_0, experiencing some

[116] It is immaterial whether $M(t)$ raises the stock of health capital as in the childhood phase, or lowers the rate of health depreciation, as in the case of adult, since whatever the case $M(t)$ and health would be positively associated, which is contrary to most empirical studies reporting negative correlations.

health issues. From *Figure 4.5* it is observable that the time paths for health capital face a sudden drop to H_{min} at some stage in t whereby the rate of health decline accelerates exponentially. The critical age values whereby health deterioration is particular fast should be identified. The retirement age should be set at an age well before such a turning point in health.

The variable income or wage y is included only in equation (4.2) and not (4.3). Income/wage is a positive function of education in the model, which is certainly supported by many empirical studies since the development of the human (educational) capital model by Becker (1962). It is assumed here in the regression analysis of equation (4.2) that this relationship between income/wage and education does not lead to significant endogeneity effect, which causes major biases and alter the conclusion. In other words, for the regression analysis of (4.2), the income variable y is treated as exogenous. y is represented empirically by the variable 'Labour income: last month' from the BHPS. The regression results of equation (4.2) are shown in *Figure 4.6* and *Figure 4.7* for the logit and probit models, respectively. The results in both figures are the same – income/wage significantly increases the stock of health. The coefficients are highly significant, with p-values close to zero.

The positive relationship between health and income is also supported by most empirical studies (Gravelle et al., 2002). There is clearly a relationship between 'health, wealth and prosperity'. The policy implication of this finding is that incomes and wages need to be risen as a means to improve health. By raising the income of the population, health would 'naturally' improve since with more income this would allow the purchase of more $M(t)$, which benefits health. A higher income would also facilitate the purchase of more material goods $X(t)$, and as a result less $\tau(t)$ would be necessary to achieve a given level of utility at any particular t, which allows more time to be invested in maintaining health $\tau_H(t)$. Without raising the economic or financial status of a person, it is thus difficult to achieve significant improvement in health since such individuals are likely to substitute their income and time to $X(t)$ and $\tau(t)$, respectively. With the case of $X(t)$, since a logarithmic function is assumed[117] if the value of $X(t)$ is too low, it would result in very low or even negative utility, and therefore encourage most of the income to be devoted to $X(t)$ in order to avoid a sharp decline in utility, therefore leaving little if any for $M(t)$. Furthermore, if a subset of $X(t)$ consists of unhealthy consumption and/or that the entire bundle of goods consumption is generally unhealthy, as may

[117] See equation (3.1'') for example.

more likely be the case for those on lower incomes, health is likely to suffer more as a result of low income. Policies such as minimum wage legislations can increase the income of the poorest, which would be beneficial for health by encouraging greater devotion of resources to $M(t)$ and $\tau_H(t)$. However, if minimum wage actually increases unemployment, as some economists claim, it would have the opposite intended effect on health.

The empirical model of equation (4.2) omitted the variables P_X and P_M due to the inability to observe the price levels faced at an individual level,[118] yet these are likely to have a substantial effect on health. An increase in P_X and or P_M is tantamount to a reduction in income y, since for any given level of income the individual can purchase less quantities of $X(t)$ and $M(t)$. A rise in P_X has an ambiguous effect on health since although it encourages substitution of $X(t)$ for $M(t)$ due to an alteration in price ratio, since less $X(t)$ can be purchased using the original budget allocated to $X(t)$, those on lower incomes may experience a sharp decline in utility at t and must therefore allocate greater resources to $X(t)$, which ends up reducing $M(t)$. This is unlikely to be the case for rich individuals who already consume a high quantity of $X(t)$ where a rise in P_X would induce a fall in $X(t)$ and a rise in $M(t)$. With an increase in P_M the effect is less ambiguous – individuals would substitute $M(t)$ for $X(t)$ leading to lower health. The implication, therefore, is that a rise in income must be real income in order to affect health positively. The government should pay attention to inflation and the general price level of commodities, especially $M(t)$. For the case of the UK where consumers do not directly face P_M since health services are largely provided freely to the public, the central focus should be on the cost of running the NHS, since a higher cost implies that as a nation or society, less units of medical care would be available. Nonetheless it is argued earlier that medical care does not in general reflect $M(t)$, the goods and services used to augment health, but rather is a reflection of poor health or health depreciation. Therefore, the priority in this case would be to reduce or subsidise products which are beneficial for health, such as fruit and vegetables, so that P_M is reduced.

The equation (4.3) contains only the variables representing the stock of education $E(q)$. Just like income, education is generally positively related to health. Since income and education can be highly correlated it is not appropriate to include both variables in a single equation model. The data variable chosen to represent $E(q)$ from the BHPS is 'Highest academic

[118] It is possible however to observe the aggregate price level, and the most appropriate measure would probably be the Consumer Price Index (CPI).

qualification'. The reference category is 'GCSE', which is considered the lowest ascertainable degree in the dataset. 'O level' is similar to 'GCSE' while 'A level', '1st degree' and 'Higher degree' represent progressive increase in the level of educational capital. On the other hand, it is not entirely clear where the categories 'hnd, hnc, teaching' and 'none of these' fit in relation to the reference category. However, I can expect in general that 'hnd, hnc, teaching' being professional qualifications would represent a higher stock of $E(q)$, while 'none of these' may possess a significant portion without any form of formal qualification recognised in the United Kingdom and therefore are associated with expected $E(q)$ lower than GCSE.

The empirical results shown in *Figure 4.8* and *Figure 4.9,* which are for the logit and probit models, respectively, both support the theoretical prediction that higher education is associated with better health. Compared to the reference category 'GCSE', 'O level' does not show a significant improvement in health, which is expected since O level cannot be radically distinguished from GCSE. Those with an A-level degree are associated with better health compared to the reference group, though the statistical significance is only at the 10%, not the typical 5% level. Those with a 1st degree, higher degree and teaching qualification are all associated with better health compared to the reference category. The positive association is highly significant even at the 1% level, and the p-values close to zero. For the category 'none of these', the regression results show a very strong statistically negative relationship compared to the reference category.

The policy implication of this finding is that improving education will result in improvement in health. The mechanism by which health will be improved however is different from the 'health-education gradient' of Grossman (1972), where it is argued that higher education increases the efficiency of input in both the market and non-market sectors, which would be akin to the effect of an increase in A in this model. The mechanism of my model via which improvement in education leads to an improvement in health stems entirely on the effect of education on the wage rate or income. It has already been demonstrated that higher income/wage is associated with better health, and this effect is simply amplified by the increase in education, which is an important (though not exclusive) determinant in income/wage. Since investment in education or 'human capital' is often regarded as a means by which national incomes and productivity, which are themselves strongly correlated with individual incomes, are raised; thereby investing in education at the national level will lead to health improvement nationally.

6.1.3. Policy Implications of Findings for the Life Expectancy/Optimal Length of Life Model

The life expectancy or optimal length of life model is a bi-product of the adulthood phase model. The main model is shown by equation (3.23), which is derived in view of the length of the planning horizon in the adulthood phase, since the variable V is endogenous rather than exogenously determined, and its optimal value can be expressed as a function of the other exogenous variables. In general as can be seen in *Figure 4.5*, a change in any exogenous variable changes the optimal planning horizon V^*, either increasing it or decreasing it.

The empirical counterpart to equation (3.23) is equation (4.4). Since it is not possible to observe life expectancy at the individual level, which is my interpretation to the theoretical notion of 'optimal length of life', I use aggregate data at the national level to empirically test equation (4.4). I use GDP per capita to represent income/wage y and the annual mean ambient concentration of $PM_{2.5}$ to represent the exogenous rate of health depreciation δ_0. The other variables are not included though GDP per capita is often a reflection of many of the exogenous variables such as A, the level of technology, and $E(q)$, the stock of education, being highly correlated with them. The theoretical predictions of the effects of a change in y and δ_0 on the optimal length of life/life expectancy are shown in *Table 4.5*, and can also be seen by the changes on the length of the planning horizon in *Figure 4.5*. These are used to predict the signs of the coefficients in equation (4.4). ϕ_1 in equation (4.4) is expected to be negative while ϕ_2 is expected to be positive. *Figure 4.10* illustrates the predicted combined effects of changes in y and δ_0, which suggests potential conflict in a country's development policies with a trade-off between economic and environmental agendas, if they are represented by y and δ_0 respectively.

The regression results of equation (4.4) are shown in *Figure 4.11* and *Figure 4.12*. *Figure 4.11* shows the analysis using a panel data between-effect model while *Figure 4.12* shows the results if the more traditional fixed effect model is employed. From *Figure 4.11*, which shows the regression results using the between-effect model, it can be seen that the coefficient for ambient $PM_{2.5}$ is not statistically significant, and in the opposite direction to that predicted by the theory. For the coefficient of GDP per capita, it is highly significant at the 1% level having a p-value close to zero. For *Figure 4.12*, which shows the results if the more traditional fixed-effect model is used, the coefficient for $PM_{2.5}$ is also in the opposite direction to the expectation, though again not statistically significant. The coefficient for GDP per capita on the other hand

remains highly significant and of the expected sign, just as the result in *Figure 4.11*. The magnitude of the coefficient, however, is considerably smaller than that reported in *Figure 4.11*. A 1% increase in GDP per capita according to estimates from the between-effects model leads to an increase in life expectancy by 5.14 years, while the equivalent estimate from the fixed-effect model is 3.67 years. The statistical insignificance as well as the unexpected sign for the coefficients of the variable $PM_{2.5}$ may be attributed to the inability of national mean annual measures of ambient concentration to accurately reflect cross-national exposure.

If year dummy variables are included in the fixed regression, the effect of GDP per capita on life expectancy becomes considerably smaller. As reported in *Figure 4.13*, a 1% increase in GDP per capita only raises life expectancy by a little more than 0.5 years, which is around a 10^{th} of the highest estimate using the between-effect model with results shown in *Figure 4.11*. This lower figure is more realistic and in line with what I have observed for life expectancies as GDP per capita increased.

The coefficient for GDP per capita ceases to be statistically significant at the 1% level but remains significant at the 5% level. Surprisingly the coefficient for $PM_{2.5}$ becomes significant at the 5% level also, though at the opposite to the theoretical prediction. The time dummy variables become the crucial variables in explaining the rise in life expectancy. All the time dummy variable coefficients are significant at the 1% level (with p-values close to zero) and show progressive increase over time. Compared to 1990, life expectancy on average in 2013 was 5.78 years higher.

The policy implication of the life expectancy/optimal length of life model is that socio-economic changes, represented by increases in GDP per capita, contribute significantly to increases in life expectancy, though at a diminishing rate, since human lifespan has an upper limit. Environmental damage on the other hand can be a drag to life expectancy, though my results fail to provide conclusive evidence on this matter. This may be a result of a lack of accurate data to measure environmental quality, particularly the aspect of environmental quality, which is concerned with health such as air pollution, at the national level. Alternatively, it is probable that environmental variables are negatively associated with GDP per capita for many countries, since the process of industrialisation the main method of economic development often results in environmental degradation. A strong positive correlation between pollution and GDP per capita may result in the regression analysis showing a positive correlation between ambient pollution and life expectancy, unable to entangle the negative

effect of pollution from the positive association with GDP, which improves life expectancy. For very high income countries such as the OECD, the relationship between environmental pollution and GDP per capita may reverse to negative, as claimed by the environmental Kuznet curve.

Raising incomes or GDP per capita and reducing environmental pollution are therefore means by which life expectancy can be increased according to the finding. This theoretical prediction, however, is derived from micro-economic analysis whilst the empirical analysis is conducted using aggregate national level data, hence it is necessary to make an implicit assumption that national variations in these variables translate into systematic variations of these variables at the individual level. Income distribution and inequality may distort the applicability of this result though I assume the effects would not lead to particularly large distortion in the results. The results imply that economic growth is a crucial factor in raising life expectancy and must remain a key priority for most countries, especially low to medium income countries, which are not experiencing significant diminishing returns in life expectancy gains from GDP growth.

The results of *Figure 4.13* suggest that besides GDP per capita, there are other factors that increase life expectancy. This is reported by Preston (1975) that the life expectancy and GDP per capita relationship did indeed change over time, generally increasing. It is postulated that revolutions in public health independent of economic growth during these periods were responsible for the upward shift in the life expectancy and GDP per capita curve. The continued rise in life expectancy over time as shown by *Figure 4.13* suggests such factors may still be in play despite the analysis being conducted in a period after the medical revolution. A rise in nutritional intake across the world for example may have played a similar role between 1990 and 2013.

6.2. Policy Implications Concerning Air Quality and Decarbonisation

Many co-benefit studies have argued that decarbonisation strategies have the potential to generate significant health co-benefits. Crawford-Brown et al. (2013) estimated total annual global health co-benefit, which can be achieved via demand management alone, under various decarbonisation scenarios. This thesis seeks to supplement the work of Crawford-Brown et al. (2013) by providing information regarding how socio-economic variables may affect health co-benefit from decarbonisation and other environmental policies. The application of my finding to the health co-benefit modelling process is shown in Chapter 5. The methodology is applied only to ambient PM pollution in my case but in principle may also be applied to other

pollutants and hazard. The theoretical variable δ_0 denoting the exogenous rate of health depreciation is interpreted as the pollutant or hazard, which in this case is annual mean ambient concentrations of $PM_{2.5}$ at the national level, but may be replaced or augmented by pollutants such as SO_2 and ozone. On the other hand y, income/wage, is interpreted as a measure of socio-economic status, which in this case is national GDP per capita. Other measures of income such as Gross National Income (GNI) and Gross Value Added (GVA) would also be appropriate. The (optimal) endogenous rate of health depreciation $\delta^*(t)$ is the dependent variable and is interpreted as the Relative Risk (RR) or Excess Relative Risk (ERR), which is commonly used in epidemiology. The Consumer Price Index (CPI) is included to represent P_X and P_M, acting as a control variable in the regression.

The health economic model developed in Chapter 3 is used to derive equation (5.1), which is converted to the econometric model of equation (5.2). The predictions for the expected signs of the coefficients in (5.2) are shown in *Table 5.2*. ϕ_1 and ϕ_3 in equation (5.2) are expected to be positive since the partial derivatives of $\delta^*(t)$ with respect to δ_0, P_X and P_M are all positive. ϕ_2 is expected to be negative as $\frac{\partial \delta^*(t)}{\partial y} < 0$. The regression results are shown in *Figure 5.4*, *Figure 5.5*, *Figure 5.6* and *Figure 5.7*, which all convey a consistent message. *Figure 5.4* and *Figure 5.5* employ the between-effect model while *Figure 5.6* and *Figure 5.7* employ the fixed effect model. The dependent variables RR in *Figure 5.4* and *Figure 5.6* are computed using PM related mortality cases, while in *Figure 5.5* and *Figure 5.7* they are computed using DALYs.

The coefficients for $PM_{2.5}$ in all four cases are not statistically significant. This can be expected to an extent since annual mean concentration, measured at the national level, does not provide an accurate reflection of exposure. For the coefficients of GDP per capita, the fixed effect models of *Figure 5.6* and *Figure 5.7* show that they are not statistically significant. In *Figure 5.7*, the direction of effect is even opposite to that predicted. The between-effect models are chosen over the fixed effect models since they are more suitable at measuring the cross-sectional variations, which are substantially larger compared to the time series variations in the dataset.

The coefficients reported in *Figure 5.4* and *Figure 5.5* for GDP per capita are used to augment the modelling procedure of Crawford-Brown et al., (2013). The change in RR due to mortality and morbidity are shown by equations (5.9) and (5.10), respectively. Three scenarios are specified in Chapter 5 regarding changes in population, emission of carbon dioxide and

economic growth, which are compared to a baseline scenario. The cumulative health co-benefit of the three scenarios compared to the baseline from 2010 to 2050, when the effects of economic growth are not considered, are shown in *Figure 5.13* while *Figure 5.14* shows the results if changes in GDP per capita are factored in. The total cumulative health co-benefits under various scenarios are shown in *Figure 5.15*, along with annual mean health co-benefit.

Under all scenarios, the magnitude of health co-benefit diminishes substantially if GDP per capita is factored into the modelling process. This is due to the assumption that economic growth and rising GDP per capita reduces the RR associated with ambient PM, based on the findings reported in equations (5.7) and (5.8), so that a reduction in ambient PM pollution from decarbonisation, which reduces mortality and morbidity incidents by a given percentage, leads to less absolute reduction.

In terms of public policy, this finding leads to an important dilemma. Many rapidly industrialising countries such as China and Thailand are also among those experiencing the most severe pollution, including ambient air pollution in major cities. In the pursuit of rapid economic development, public health is often sacrificed yet this very process of economic development proves to be a consistently reliable method for improving public health. If a country forgoes economic development and diverts its resources to reducing air pollution, this may not result in net health improvement and would thus be a lose-lose solution in terms of both health and the economy. Countries, which already initiated the process of industrialisation and are in the middle-income bracket, are likely to be particularly vulnerable to the challenges presented by this dilemma. On the one hand the economy is developing rapidly but on the other hand air pollution reaches serious levels, which not only have health consequences but also impede economic progress by increasing the number of sick days, reducing labour productivity. For such countries, the solution to air pollution related health is further economic growth rather than forgoing it. A real danger would be if a country is consigned in the 'middle-income' trap, where they are unable to continue economic development and experience high levels of air pollution. Furthermore, these middle-income countries may have reached the turning point in the environmental Kuznets curve. Economies frequently develop initially due to manufacturing and industrialisation up to a point, where further growth is usually the product of services and the knowledge economy. Therefore, countries which have matured in terms of industrialisation and manufacturing, should seek alternative drivers of growth that are less energy intensive.

This has been the pursuit of China's recent Five Year Plan.[119] Economic growth rather than air pollution should therefore remain the top priority for developing countries, though the type of economic activities should be constantly evaluated at different phases of the country's economic development.

Gupta and Barman (2010)'s model supports my findings and suggests that in the long run there is no conflict between the solution for social welfare maximisation and growth rate maximisation, demonstrated by the long-run equilibrium. The divergence between social welfare and economic growth is short term and non-persistent since the unique steady state equilibrium point does not satisfy 'saddle-point' stability. The finding is in contrast to some economic models, which suggest that divergence between some form of welfare maximisation and growth maximisation policies, can persist due to environmental externalities. Gupta and Barman (2010)'s model is more conclusive on this matter since it is the only study to simultaneously model the effects of health, environment and economy.

Another interesting dilemma implied in my results may create potential conflict in decarbonisation and air quality policies. It is suggested that as GDP per capita increases, health co-benefit from decarbonisation and air quality policies decline. This implies that wealthier countries generally have less incentives to engage in decarbonisation and air quality policies from a public health perspective and as countries grow economically, this incentive diminishes progressively. Indeed for developed countries, the potential health co-benefits are often too small to justify the cost of decarbonisation and air quality strategy implementation. In any case they are substantially smaller than developing countries as the series of Lancet report on climate change reveal. This is problematic since decarbonisation and air quality policies arguably are the most necessary for low and middle income countries, but the incentives to engage in such policies fall as these countries become developed.

The findings and the policy implications therefore place decarbonisation and air quality policies in a tricky position, since they suggest that there is no point during the economic development phase where they should become a priority. This would appear as a counter argument to environmentalism whereby conservation and environmental protection, rather than economic development, are the priorities of society. It may even be seen as a challenge to the concept of sustainable development, promoting a 'do-nothing', hoping optimistically that

[119] http://www.china-un.org/eng/zt/China123456/

economic growth will sort out all the problems in time. However, the findings and policy implications should not be interpreted as such for several reasons. Firstly, for a long time the environment-economy trade-off debate have neglected a consideration crucial to human welfare, namely health. The arguments are often concerned with aspects of the environment, which are not directly connected to health, whereas this research places health and a major health hazard from economic activity, air pollution, at the centre of the analysis. My results focus on health and conclude in many instances that decarbonisation and air quality policies may not contribute to net health improvement if the beneficial effects of economic growth to health are also considered. Secondly, economic growth and the associated activities are often regarded as detrimental to the environment without distinguishing between the types. It may be claimed that all forms of economic activity damage the environment since it is inevitable that some form of environmental impact will be made as a result. Nevertheless, if the effect on public health is the criterion used to assess the environmental impact, then in general economic growth, which stems from industrial and manufacturing activities, tends to be negative, while services are neutral and even positive (Shafik, 1994). This study is not just proposing that economic growth should be the solution to public health issues associated with air pollution, but equally importantly, for countries to frequently alter the economic structures at the appropriate time in order to ensure sustained economic growth and reduction in air pollution simultaneously, rather than being locked into a single economic pattern continuously. Nonetheless, it is indeed accurate to claim that the basis of this study is anthropocentric, since it is primarily concerned with only the aspect of environment, which pertains to human welfare, rather than the preservation of the environment as an intrinsic duty.

The stage at which mass decarbonisation and air quality policies are most suitable is perhaps before and during, rather than after industrialisation. Prior to industrialisation most countries have low levels of pollution and low GDP per capita. A rise in pollution level at this stage when GDP per capita is low can be highly detrimental to health. Low GDP is often associated with low levels of nutritional intake, lack of medical services provision, and low education so that the public may not be aware of the risk of air pollution, all of which result in a high level of health impact from air pollution, for a given level of ambient concentration. A sudden rise in ambient air pollution in this case would result in dramatic negative health impact, since the percentage increase in particulate matter related illness would be built upon a high level of baseline mortality/morbidity due to low GDP per capita. Therefore, during the initial industrialisation stage, if the rise in air pollution can be curbed, substantial harm to public

health may be preventable. Action should be taken immediately following industrialisation since if it is delayed, the rising GDP per capita would reduce the incentives of such policies.

So far I have discussed air quality and decarbonisation policies jointly, making little distinction between them. In light of the findings, it may be even more difficult for countries to implement decarbonisation policies since they are often less cost effective than air quality policies at reducing health impact. For developing countries especially, it would not be economically practical to focus on decarbonisation, and the high costs and investment necessary may divert resources away from economic growth. Instead, the priorities should be on reducing ambient air pollution using air quality policy, and accepting the corresponding reduction in carbon emission as co-benefit. In other words, decarbonisation and climate change agendas are to become subservient to health and air quality. This may mean that decarbonisation initiatives are not ambitious enough and that the 2˚C target is unlikely to be achieved, yet in the current economic and political climate, this is perhaps the only practical approach. Currently only a number of countries in the world are seriously committed to reducing carbon emissions, which may be derailed by poor economic performance. In addition, the recent election of Donald Trump in the United States signals the reversal of climate change policies initiated during the Obama era. Even though Trump stated that he does not believe in the notion of humanly induced climate change, he nonetheless vowed to improve air and water quality. Placing health and air quality issues at the centre of policy is therefore the only way decarbonisation goals can survive this age of climate change scepticism and economic turmoil.

6.3. Inequality Implications of Air Quality and Decarbonisation Policies

The solutions developed via the theoretical framework outlined in Chapter 3 produces the equation (3.21’’), the optimal time path of adult health capital, which can be used to predict the (in)equality implications of decarbonisation and air quality policies as described in section 5.3 of Chapter 5. The basic premise is that the exogenous rate of health depreciation δ_0 can be empirically represented using aggregate data of a region’s ambient air pollution or other environmental indices connected to health, while the other exogenous variables most notably y or income are empirically represented by socio-economic variables. δ_0 rather than being a variable is treated in this case as a function of a particular policy or a vector of policy variables $\delta_0(\rho)$, such that the health co-benefits can be represented theoretically by the equation (5.12). The health co-benefit equation of (5.12) would be a function of the exogenous variables, many of which are socio-economic. Therefore it is possible to assess how the health co-benefits alter

along socio-economic lines, represented by the exogenous variables. The assessment involves taking the partial derivative of equation (5.12) with respect to the socio-economic or exogenous variable of interest. The partial derivatives are shown in *Table 5.3*. They remain untested theoretical propositions regarding how health co-benefit from environmental and decarbonisation policies are likely to vary along socio-economic lines.

As can be seen in *Table 5.3*, the partial derivatives of health co-benefits with respect to income, education and age are negative, negative and positive, respectively. This study does not intend to test these propositions empirically. Nonetheless, the propositions conform to the predictions of most empirical studies concerning air pollution and socio-economic variables. The policy implication therefore is that decarbonisation and air quality policies are beneficial from a social equity perspective. However, the proposition as well as most of the empirical studies cited do not consider the cost of policy implementation. Such policies may be financed by taxation, or the regulatory burden may fall on the public. It is possible that the implementation of the policies cause more disruption to those who are of the disadvantage groups, outweighing the equality bonus of the policies.

Aside from predicting the inequality implication, the model in Chapter 3 facilitates a general tool to predict the theoretical effect of any relevant policy on an endogenous variable, as well as how the effects may vary along socio-economic lines. In order to carry out this process, the first step involves selecting the endogenous variable of interest, which a particular policy is likely to affect. The list of endogenous variables are shown by equations (3.10) to (3.15) for the child model and equations (3.16)-(3.18), (3.23) and (3.21'') for the adulthood model.

As an example, I may wish to assess how policies designed to improve the health of children affect their education. Equation (3.15) denoting the optimal stock of educational capital for a representative child is selected. A single policy or a vector of policy ρ is assumed to affect the exogenous rate of child health depreciation δ, which becomes a function of ρ and can be written as $\delta(\rho)$. ρ may include measures to introduce healthier food, reducing unhealthy food consumed by children, and/or increase exercise through physical education[120] as alluded to earlier. The effect of policy ρ on the educational capital of children may be written as $\frac{\partial E^*(t)}{\partial \delta}\delta'(\rho)$. The result consists of two components – how a given policy alters the rate of child

[120] Although increasing exercise may be interpreted as a matter relating to $\tau_H(t)$, the time children devote to exercise, rather than δ.

health capital depreciation, and how the change in child health capital depreciation affects the stock of educational capital. $\frac{\partial E^*(t)}{\partial \delta}\delta'(\rho)$ and many equations obtained via this manner, i.e. analysing how a given policy will affect an endogenous variable of interest, will in general be a function of some or all of the exogenous variables in the original equation, which in this case is (3.15). The exogenous variables shown describe child characteristics, though as argued earlier in section *6.1*, they are likely to be strongly correlated with the child's upbringing and/or the socio-economic status of his or her parents. $\frac{\partial E^*(t)}{\partial \delta}\delta'(\rho)$ can be further differentiated partially with respect to the exogenous variable of interest in order to predict how the policy is likely to vary along the exogenous variables.

Assuming I seek to investigate are those policies which are beneficial for child health, i.e. those which will reduce the exogenous rate of health depreciation δ, the impact of this policy on the optimal stock of educational capital can be written as $-\frac{\partial E^*(t)}{\partial \delta}\delta'(\rho)$.[121] The partial derivative is shown by equation (6.1) below:

$$-\frac{\partial E^*(t)}{\partial \delta}\delta'(\rho) = \frac{1}{2}\omega t^2\delta'(\rho) \tag{6.1}$$

Equation (6.1) is positive for all $t > 0$, meaning that the policy at reducing the exogenous rate of health depreciation is positive for the child's educational stock of capital as long as the child is older than zero years of age. The effectiveness of the policy depends on two variables only – ω and t, the marginal product of health on the accumulation of educational capital, and age respectively (multiplied by the effectiveness of the policy at reducing depreciation). The partial derivatives of (6.1) with respect to the two exogenous variables are both positive. Therefore, the positive effect on child educational capital increases with higher ω, the marginal product of a unit of health capital, and t, age.[122] Since ω cannot easily be measured or even conceptualised, my attention focuses on t, which is an important socio-economic variable.[123]

[121] I assume that the partial derivative of an exogenous variable with respect to policy is always positive, i.e. $\delta'(\rho) > 0$. The reduction in child health depreciation as a result of the implementation of the policy is expressed by multiplying the entire equation by -1.

[122] Bear in mind, however, my empirical results shown in *Figure 4.3* and *Figure 4.4, which* imply that $\omega = 0$, meaning that health does not affect the accumulation of educational capital. If this is the case then the policy of reducing the health depreciation of children would also be ineffective at increasing education, as equation (6.1) would be zero.

[123] Although compared to adult, age is not often regarded as a child socio-economic variable, but often regarded to be a biological variable.

Assuming that $\omega > 0$, the increase in educational capital as a result of policy ρ would be more pronounced for older children than for younger children. The policy implication of this finding is that health promoting policies would benefit the educational attainment of older children compared to younger children. This suggests that the policy can be beneficial and equitable simultaneously, since it is arguably more important for older children closer to entering the workforce to receive a boost in education than for younger children. According to equation (6.1), the positive effect of the policy is not a function of θ, the importance a child places on his or her education. Therefore the policy is unlikely to result in inequality in educational gain where those who are more interested in education gain relatively more than those who do not, but instead, the policy will result in an uniform increase in education for all children of the same age.

Using an example from the adulthood model, suppose one is interested in the effect of a price increase (increase in P_M) in medical goods and services $M(t)$ on their consumption.[124] As mentioned earlier it may be better to interpret $M(t)$ as health promoting goods and services, such as good food and nutrition, and the increase in P_M, the increase in the prices of these goods and services. Suppose a policy is aimed at reducing the price of health promoting goods and services so that P_M may be expressed as a function of the policy, $P_M(\rho)$. The effect of the policy on the use of health promoting goods and services can be expressed by equation (6.2) below.

$$-\frac{\partial M^*(t)}{\partial P_M} P_M{}'(\rho) = yA^{-1}P_M^{-2}P_M{}'(\rho) \tag{6.2}$$

Equation (6.2) is always positive so long as the parameters are positive and that $P_M{}'(\rho)$ is positive. This means that a policy, which reduces the price of $M(t)$ for example via subsidy, will increase its use. The important socio-economic variable in the partial derivative of equation (6.2) is income, y. This means that the benefit of increasing the use of health goods and services via their price reduction due to the policy varies along the line of income. If equation (6.2) is further differentiated partially with respect to income y, the result is clearly positive. This means that the benefit of a policy which reduces the price of health promoting

[124] Although it had been argued earlier that $M(t)$ should not represent medical goods and services but rather health goods and services, which are conducive to health. The use of medical goods and services often reflect poor health, and that the individual has some underlying health issues. Medical care and services can do little to fundamentally improve the health of those who use it, and instead are focused on treating symptoms, preventing them from deteriorating.

goods and services (and perhaps also medical care) accrue more to those who are of high income and less to those of low income. Such a policy therefore may increase inequality.

The above two examples demonstrate the flexibility of the theoretical model described in Chapter 3. It is possible to use the model to analyse the impact of any policy, which will affect the exogenous variables, and the ultimate effect on the endogenous variables. How the effect of the policy is likely to vary along the exogenous variables is also predictable, which can be used to predict the equality or inequality implications of the policy. This tool is therefore useful in generating theoretical predictions regarding relationships between key variables pertaining to policy interest, which can be used to drive future empirical studies.

CHAPTER 7 – Discussion and Conclusion

The thesis began with the motivation of building a framework whereby socio-economic factors may be incorporated into the modelling of health co-benefit from decarbonisation. In order to achieve this goal, a health capital model was developed inspired by Grossman (1972) and others, using the dynamic optimisation technique of optimal control theory applying the Pontryagin Maximum Principle. The development of this model formed the underlying theoretical framework of the thesis. It was also an opportunity to address some of the concerns and short-comings in the existing literature of health capital models. One of the main contributions of the model developed in this thesis was the specification of a dual phase life-cycle analysis, separated into childhood and adulthood phases each with some unique characteristics. For each phase, there were different emphases which were incorporated into the model. For the childhood phase, there were two state variables – health and education. As well as seeking to maximise the childhood utility function, the child had the alternative aim of achieving a high stock of educational capital at the end of his or her childhood years. During the adulthood phase it was assumed that the stock of education to be fixed and the adult would be concerned only with health. The stock of health capital and how it declines over time determines how long the person can live.[125] Certain endogenous and exogenous variables were interchanged between the childhood and adulthood phases of the model. For example, consumption and time devoted to work were exogenous in the childhood and adulthood phases respectively, since it was assumed that they would be determined by guardians and employers respectively, and thus the individual would have little or no control over them. The most important distinction however between the childhood and adulthood phases of the model, was the manner via which the health investment function operated. During the childhood phase it was possible to increase the stock of health capital through investment in health, in a manner similar to the mechanisms employed by other health capital models. However, when the child reached adulthood, it was no longer possible to increase the stock of health via health investment. Health investment may only limit the decline of health capital at this stage. This reflects the notion that the bodily and mental development of children are processes or opportunities to build up health capital as an asset for the benefit of later life. It is well known that decisions to promote or damage health are not equally potent for all stages of life. Certain actions such as balanced nutritional diet are more effective during young ages than old, while

[125] Life ends when the stock of health falls to H_{min}.

other actions such as high intake of cholesterol would be more damaging to an older individual than to a young. This is a feature which has not been adequately reflected in existing health capital models and which my model has attempted to address in a limited degree.

The health capital model developed in this thesis generated a number of testable hypotheses in the form of model solutions, which were the optimal time paths for the endogenous variables derived using dynamic optimisation. Due to the scope and focus of the thesis, only the endogenous variable health or health capital was tested empirically. The optimal stock of health capital was expressed as a function of time t, which could be interpreted as age, an important socio-economic variable, as well as other exogenous variables most of which could be considered socio-economic, such as income and education. Comparative dynamic analysis was applied to predict the effect of a change in certain exogenous variables on health capital, the dependent or endogenous variable. This was performed by taking the partial derivative of the equation for optimal stock of health capital with respect to the exogenous variable in question. For example, by partially differentiating the optimal stock of health with respect to income, the effect of an increase in income on health over the course of life could be predicted theoretically. Comparative dynamic analysis differs from comparative static analysis in that the former is capable of examining how a change in exogenous variable alters the entire (optimal) time path of the endogenous variable while the later reveals only the change in equilibrium value of the endogenous variable without reference to time. Grossman (1972)'s original model employed comparative static analysis while later studies utilised comparative dynamic analysis based on Oniki (1973).[126] The studies which applied comparative dynamic analysis were forced to implicitly assume that the rate of health depreciation was constant or independent of age. This was potentially problematic as pointed out by Dalgaard and Strulik (2014) in that in the event that the length of life was assumed to be an endogenous process, there would be no guarantee that the optimal length chosen would be finite. The model developed in this thesis addressed this issue and offered a simplified solution by assuming that the depreciation of health was independent of the stock of health. This also greatly simplified the modelling process in that it was no longer necessary to assume that the marginal product of a unit of health capital was diminishing. The dual assumptions of health depreciation as a function of existing stock of

[126] The method developed by Oniki (1973) uses phase diagram analysis which allows the simultaneous modeling of two state variables.

health[127] and diminishing marginal product of health capital, often made in most health capital models equate to or can be simplified to the alternative set of assumptions where both the depreciation and marginal product of health capital were constant or independent of existing stock. Other more complicated features of existing health capital models are also simplified by the model developed in this thesis. For example, rather than specifying an endogenous variable known as 'commodity' or 'consumption' which formed an argument in the utility function, the factors which contributed or 'produced' commodity/consumption directly entered the utility function in the model I developed.

In order to empirically verify the testable hypotheses which described how the optimal stock of health in children and adults, which were equations (3.14) and (3.21'') respectively, panel data econometric models were used. For all empirical tests in this thesis including for these two equations, panel data were used with the assumptions of linearity and the exogenous/independent variables being additively separable. For the childhood model of equation (3.14), the data for Understanding Society's youth self-completed questionnaire was used as the data source. This is a relatively new set of longitudinal data with large samples though the observations are relatively short, with only five years of data from 2009. The adulthood model of equation (3.21'') used the data from the British Household Panel Survey which is the forerunner to the newer Understanding Society. Although compared to Understanding Society, the BHPS contain smaller samples of cross-section, it has been running for longer and hence the time series profile is much longer with 18 years ('waves) of data. Comparative dynamic analyses were employed as the basis for predicting the signs of each exogenous variable in equations (3.14) and (3.21''). For both equations, the regressions report results which strongly support my theoretical framework and testable hypotheses, with a few minor exceptions.

As a bi-product of the adult model, since the assumption of endogenous rather than fixed planning horizon was selected, equation (3.23) was tested empirically, which showed the optimal length of life as a function of various exogenous variables. The optimal length of life was interpreted empirically as life expectancy. This meant that only aggregate data could be used to empirically test equation (3.23), since life expectancy data do not exist at an individual level. Moreover, the concept of life expectancy may not even make sense at the individual level

[127] Meaning that individuals with higher stock of health capital or healthier individuals lose more health capital in absolute terms per unit of time for any given rate of health depreciation.

since how long an individual can expect to live is based on the average lifespan of a specified population or cohort and hence by definition life expectancy must be at the aggregate level. Equation (3.23) was tested using national level data from the World Bank. A panel data set with six years of repeated observation was constructed where the dependent variable was life expectancy at birth. The independent variables consisted of the ambient mean concentrations of $PM_{2.5}$ recorded for each country and the GDP per capita. $PM_{2.5}$ was used to denote δ_0, the exogenous rate of health depreciation, while GDP per capita was used to denote a range of exogenous variables of socio-economic nature of which income y was chief. The regression results revealed strong positive correlations between life expectancy and GDP per capita, as would be expected. This was the case even when time dummy variables were included to account for the trend described by Preston (1975) where it was observed that the life expectancy of all countries increase over time irrespective of economic growth, with a few minor exceptions. The coefficients for $PM_{2.5}$ were not statistically significant and in many cases showed positive associations with life expectancy, which was opposite to my expectation. This was most likely the result of low resolution data for $PM_{2.5}$ both spatially and temporally meaning that they are not good quality indicators of the true level of ambient air pollution exposure and the exogenous rate of health depreciation.

The econometric model applied to test equation (3.23) however was somewhat different from the theoretical proposition in that it was assumed that the positive effect of GDP per capita on life expectancy was progressively diminishing. This was to reflect the common methodology applied in estimating the relationship between life expectancy and national income following Preston (1975). However, the model developed in this thesis actually predicted that the increase in optimal length of life due to an increase in income would not be diminishing but instead subject to increase. Therefore if this was strictly applied, it would be necessary to specify an econometric model where GDP per capita causes positive non-linear and increasing rises in life expectancy. I did specify such a model in equation (4.5), but it appeared to be contradicted by the empirical evidence. A possible explanation could be due to the fact that my theoretical model analysed individual or microeconomic behaviour, while the data used to validate it was at the national or macroeconomic level. As such there would be a subtle conceptual difference between the dependent variable in the theoretical model which was the optimal length of life, and the empirical representation using actual data, which was life expectancy measured at the national level. The optimal length of life strictly speaking would refer to how long a given individual 'chooses' to live, given the exogenous variables, and not how long he or she can

expect to live based on inferences drawn from national averages. This conceptual difference may mean that it would not be possible to obtain the exact same empirical validation of my theoretical model using aggregate data. Ideally it would have been better to use individual data. The most appropriate would be death certificates denoting the time and age of death, since the actual rather than expected years lived would correspond more closely to the concept of optimal length of life. Nonetheless it would be difficult to obtain comprehensive data for death certificates along with socio-economic variables associated with the person when he or she was alive.

Having empirically validated the theoretical model, it was used to augment the modelling of health co-benefit from decarbonisation strategies. Only the adulthood model was used for this task. The equation for optimal health capital was rearranged so that an optimal rate of endogenous health depreciation became the dependent variable. The optimal rate of health depreciation referred to the rate of health depreciation which would prevail if the control variables were at optimum. The resulting equation of (5.1) showed that the optimal endogenous health depreciation was a function of the exogenous health depreciation and other exogenous variables. Empirically testing such a model reformulation, it was argued that the endogenous rate of health depreciation could be represented by (Excess) Relative Risk (RR), which is an important metric frequently used in epidemiology to quantify the health risk exposure of a given population to a particular hazard. The exogenous rate of health depreciation on the other hand should be represented by the measure of hazard which in my case was the ambient $PM_{2.5}$ concentration. The exogenous variables, most of which can be interpreted as socio-economic variables were represented using GDP per capita while the price for consumption and medical[128] goods were represented by the Consumer Price Index (CPI). Taken together, I developed a theoretical basis in which the RR may be expressed as a function of a) the hazard, b) socio-economic variables and c) price levels. For part c) I concluded that it was not a good measure of the true price levels experienced at the nation level, since CPI would always be standardized at 100 for all countries at the base year and therefore it was not possible to compare cross-country variations in price levels.[129] The independent variables of ambient

[128] Health promoting

[129] Using GDP per capita Purchasing Power Parity (PPP) instead of nominal GDP per capita in the regression was another alternative. This is avoided since it would confound the effect of income and price levels. The CPI as an index should capture both the consumer and medical goods component being a weighted index. However, in many countries medical care prices inflate faster than general goods and this will not be very accurately reflected in the CPI. Index of medical care index are available for the UK produced by the Personal Social Services Research

$PM_{2.5}$, GDP per capita and CPI were obtained from the World Bank, the exact same panel data set as those used to validate the life expectancy model of (3.23). The dependent variable RR however, was obtained from the Global Burden of Disease 2013 study (GBD, 2013), by computing the all-cause mortality and morbidity associated with the risk factor ambient PM exposure for all the countries in the year 1990, 1995, 2000, 2005, 2010 and 2013. The panel data for $PM_{2.5}$, GDP per capita and CPI were also available for these six years and hence were combined with the data for RR. I employed a number of panel data models but chose the between-effects model as my main estimates, since such a model bust reflects the cross-country dimension, which was the main source of variation given that the time series points were relatively few and far apart.

The main variable of interest to us was the effect of GDP per capita on RR associated with ambient PM. According to the best estimates, a $1,000 dollar increase in a country's GDP per capita would reduce the RR by 0.148% and 0.454% for RR constructed using mortality cases and DALYs, respectively. It is interesting to note that the coefficient of GDP per capita on RR is three times higher when applied to a measure of mortality compared to one of morbidity. This was also the finding of Crawford-Brown et al., (2013) for the coefficient of ambient PM on the RR. The coefficients for $PM_{2.5}$ reported in my models were not statistically significant and even in the opposite direction to what was expected. Therefore it was not appropriate to use the coefficients obtained for the purpose of modelling health co-benefit from decarbonisation. In fact, Crawford-Brown et al., (2013)'s meta-analysis study had been very comprehensive covering studies performed in many countries, which displayed strong consistency in the reported coefficients of ambient PM and RR. Therefore their coefficient estimates for PM were retained in my modelling of health co-benefit with some minor adjustments, rather than using the coefficients shown in my regression models. Crawford-Brown et al., (2013) reported that for every $1\mu/m^3$ increase in PM_{10} exposure, the relative risk for mortality and morbidity cases increase by 0.1% and 0.3%, respectively. Since Crawford-Brown et al., (2013)'s study estimated the RR's response to ambient PM_{10} level, the reported coefficients needed to be converted to values appropriate for $PM_{2.5}$, which was what my empirical model used. Since in general there is the consensus that $PM_{2.5}$ as a class of pollutant

Unit which publishes annual 'Unit Costs of Health and Social Care' statistics. I cannot use this data in my regression since comparable data at the international level does not exist. More importantly as has been argued on numerous occasions that $M(t)$ should not be seen as medical care usage but as health promoting goods and services, P_M should reflect these goods and services. Therefore CPI is a more appropriate measure than the indices of medical care.

is at least twice as hazardous as PM_{10} per unit of exposure, the coefficients of Crawford-Brown et al., (2013) were doubled when applied to the modelling of health co-benefits in this study. Therefore for every $1\mu/m^3$ increase in ambient $PM_{2.5}$, the RR for mortality and morbidity increases by 0.2% and 0.6%, respectively.

For the purpose of modelling the effect of health co-benefit from decarbonisation and air quality policies, four scenarios were devised. Only mortality was modelled. The baseline scenario, known as scenario 0 outlined the annual mortality from 2010 to 2050 attributable to CO_2 and PM emissions in the case of business-as-usual, i.e. very little effort devoted to decarbonisation and/or air quality improvement. The estimation of mortality was based on Crawford-Brown et al., (2013). The other scenarios – 1, 2 and 3, each showed the annual mortality which may arise due a different level of stringency in decarbonisation initiatives, which were all greater than that of scenario 0, from 2010 to 2050. The annual difference in mortality between each of the scenarios from 1 to 3 compared to scenario 0 were interpreted as the annual health co-benefit of a given decarbonisation policy depending on which scenario was compared with scenario 0, the baseline scenario.

The main contribution of this thesis is the modification of this health co-benefit process via the inclusion of the effect of socio-economic variable. After all the theoretical examination and empirical analyses shown in this thesis, it was evident that GDP per capita would be the most appropriate variable to represent the socio-economic variables which affect the RR and in turn the health co-benefit from decarbonisation, at least at the national level. The inclusion of GDP per capita at the national level as a function of RR provides a simple and convenient approach to including the effect of socio-economic variable to the analysis of health risks at this level. I found that GDP per capita reduces the RR associated with ambient PM at a diminishing rate but do not alter the coefficient of ambient PM on RR. (i.e. no interaction effect of PM and GDP per capita on RR) The effect of GDP per capita increase is essentially to lower the background health risk associated with ambient PM. Given that the effect of ambient PM increases the RR linearly meaning that an increase in ambient PM raises the background health risk by a constant percentage, the effect of increasing GDP per capita lowers the population's sensitivity to PM pollution in absolute terms (though not in percentage terms, which is the common measure in epidemiology).

The policy implication is that the increase in GDP per capita or national income will reduce the effectiveness of decarbonisation and air quality policies in generating health co-benefit.

The best time for a country to engage in decarbonisation is when the country is low income and not after it has developed economically. In fact, the incentive to decarbonise or improve air quality would quickly diminishing as the country develops economically. This will lead to a 'procrastination' whereby a country is never seriously committed to decarbonisation and air quality improvement efforts, especially the former which are far less cost effective than the latter in generating health improvement. This is not even considering the potentially negative consequences on health if decarbonisation diverts resources away from economic growth which would only further lower the incentives. This study therefore reveals a policy conundrum. Decarbonisation and air quality improvement initiatives should therefore be taken early rather than later in a country's development process, particularly if the measures do not compete significantly with economic development goals. This contrasts with notions such as 'pollute first clean up later' and the environmental Kuznets curve where environmental amelioration would occur after and not before economic development – there are clearly forces at work which will hinder environmental amelioration.

Another useful tool developed as part of the thesis is the theoretical prediction of whether a given policy is likely to result in health impacts which vary along socio-economic lines and thus have implications for (in)equality. This was achieved by first taking the partial derivative of the equation for optimal stock of health with respect to the variable which would be affected by a chosen policy, before taking the second order partial derivative with respect to the socio-economic variable in question. This tool was applied in the thesis to analyse how the distribution of health co-benefit from decarbonisation and air quality policies is likely to vary along the socio-economic variables of income, education and age. It was revealed that the health co-benefits accrue less to those who are high income, highly educated and the young, and thereby benefit the low income, lowly educated and older population proportionally more. Thus decarbonisation and air quality policies have the potential to be equitable. The tool developed here is more flexible and can be used to theoretically assess the (in)equality implications of any policy on any of the model's endogenous variables, along the lines of any exogenous variables in the model.

The methodology of this study suffered from several theoretical and empirical limitations. Firstly regarding the theoretical limitations, the study's theoretical framework was rooted in the health capital model and its various predictions. The economic model was one of the simplest forms – a rational representative agent operating under conditions of perfect information and certainty. Therefore it could not account for the various influences which may affect the

outcome of the optimal control, state and co-state variables. The separation of the childhood and adulthood phases of the model whilst an important contribution of the thesis aimed at identifying important differences at different stages of life was by means no perfect. Specifically the decision making processes between childhood and adulthood were completely disjoint and unconnected except via exogenous variables such as (theta), the relative importance of education to the child, which at best only implicitly revealed how a child would plan for his or her adult life.

In terms of the empirical limitations, the major weakness resided in the data used. The main health models of the childhood and adulthood phases were verified using micro level or individual data, but this was not the case for the life expectancy model, which could only be verified using aggregate data at the national level. Aggregate data was also used for assessing the socio-economic influences on health co-benefit from decarbonisation. This part of the task applied a variation of the health capital model in which the endogenous rate of health depreciation acted as the dependent variable in question, which was represented empirically using the Relative Risk (RR) associated with ambient particulate matter pollution. Another empirical weakness was that the main dependent variable of health capital was represented using data on self-reported health. Such data were non-continuous, categorical and as a result I could not apply linear econometric models but instead had to use ordered logit and probit models. The range of econometric tools at my disposal was substantially reduced by the dependent variable being in this format and interpretation of the regression results and coefficients became less straightforward.

In addition, some of the data used are not perfect in that they do not fully represent the theoretical variables in question. For example, the rate of subjective discount β is represented using the response to the questionnaire variable 'Restless and cannot stay still for long'. Even though this representation is clearly plausible given that 'Restless and cannot stay still for long' reflects an individual's patience, which no doubt affects the person's discount of future cost and benefit, this remains somewhat of a stretch in conceptual definition. It may also be that being restless is an inherent medical issue which will certainly correlate with health but not in the manner suggested by the model. Nonetheless these are the variables I can find which corresponds closest to the theoretical variables. Since I used secondary questionnaire resources, I was unable to design questionnaires which seek responses with perfect match. Future research in this area if resource permitting should design tailored questionnaires to better measure the variables. In addition, some variables are missing in the regression equation which may cause

bias, and this may be especially problematic given that a random effects model was the only option. These variables are often conceptual such as the marginal product of health on educational capital ω hence difficult to measure. Future research should begin probing into ways these variables can be measured, even if imperfectly.

In order to address the theoretical short-coming, the ideal case would be the development of an economic model with multiple heterogenous agents which interact strategically, operating under uncertainty and perhaps also risk aversion. This would require the mathematical technique of differential/stochastic games which can be extremely complex, with no guarantee that an optimal solution or equilibrium exists. Another potential theoretical development would be the construction of a macroeconomic general equilibrium model. Although I was able to quantify how decarbonisation and air quality policies may improve health and how these improvements termed health co-benefits interact with socio-economic variables, there was not an overall theory to present a case of an optimal decarbonisation policy incorporating the concerns raised in this thesis. I could not therefore advise policy makers whether the extent of decarbonisation of a country is adequate given the circumstances. On the empirical side given that the major limiting factor was the data available, there was perhaps little which could be done. It would be preferable if micro level data were available for the life expectancy model and the model for endogenous rate of health depreciation. For the health capital model, although alternative metrics of health were available which would be continuous, they tended to be highly specialised indicators developed from the medical profession which do not reflect overall health status as well as self-reported health. These options from the theoretical and empirical side should be explored in future research. New data at both micro and macro level can be used to improve the quantitative estimates of how socio-economic variables alter the health co-benefit from decarbonisation and air quality policies while more sophisticated economic theoretical models continue to be developed to explain the observed empirical relationships.

The area most fruitful for future research is perhaps on the (in)equality implications of decarbonisation policies as well as other public policy, since vastly inequitable policies are unlikely to gain political support and implementation. This thesis sets out the basic framework for analysing a range of policies pertaining to health but stops short of empirical examination. Future research should examine the empirical validity of their proposition.

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APPENDIX

The Appendix here describes how the optimal solutions in my theoretical framework of Chapter 3 are derived. The optimal solutions for the childhood phase are shown by equations (3.10) to (3.15) while those for the adulthood phase are shown by equations (3.16) to (3.23).

The child's inter-temporal utility function which is assumed to be additively separable in its arguments of goods consumption and free time is shown by equation (3.1''):

$$U(t) = (\log\big(X(t)\big) + GH(t) - \tau_W(t) - \tau_H(t)))e^{-\beta t} \tag{3.1''}$$

The equations of motion for the state variables are given by equations (3.4'') for health and (3.5') for education, respectively.

$$\dot{H} = A(\ln(\tau_H(t) + 1) + Me^{-t}) - \delta \tag{3.4''}$$

$$\dot{E} = A\ln(\tau_W(t) + 1)) + \omega H(t) \tag{3.5'}$$

Since the representative child seeks to maximise the weighted sum of the inter-temporal utility function and the stock of educational capital at $t = q$ when childhood ends, the objective function to be maximised is shown by equation (3.6).

$$\int_0^q U(t)e^{-\beta t}\,dt + \theta E(q)e^{-\beta q} \tag{3.6}$$

The above optimal control problem known as the problem of Bolza whereby not only must the integral functional be maximised, but also another term. This makes the application of the Pontryagin Maximum Principle to obtain the optimal solutions somewhat difficult. However, such a type of problem can be easily converted into a standard problem whereby all the terms of the objective function are within the integral. To do this, I assume the following:

$$E(q)e^{-\beta q} = \int_0^q \frac{d}{dt}E(t)e^{-\beta t}\,dt = \int_0^q (\frac{d}{dt}E(t) - \beta E(t))e^{-\beta t}dt \tag{A1}$$

I assume that $E(0) = 0$, or that the initial level of educational capital is zero.

I can substitute equation (3.5'), the time derivative of a stock of educational capital into equation (A1), replacing $\frac{d}{dt}E(t)$ I obtain the following equation:

$$E(q)e^{-\beta q} = \int_0^q (A\ln(\tau_H(t) + 1)) + \omega H(t) - \beta E(t))e^{-\beta q}dt \tag{A2}$$

By substituting equation (3.1'') and (A2) into (3.6), I obtain the objective function to be maximised:

$$\int_0^q [\log\big(X(t)\big) + GH(t) - \tau_W(t) - \tau_H(t)) + \theta(A\ln(\tau_H(t) + 1)) + \omega H(t) - \beta E(t))e^{-\beta q}]e^{-\beta t}dt \quad \text{(A3)}$$

The general function form of equation (A3) is shown by equation (3.6'') in Chapter 3 of the main text. Using equation (A3) I form the Hamiltonian function for the childhood optimal control problem:

$$J = \log\big(X(t)\big) + GH(t) - \tau_W(t) - \tau_H(t)) + \theta(A\ln(\tau_W(t) + 1)) + \omega H(t) - \beta E(t))e^{-\beta q} + \lambda_H(t)(A(\ln(\tau_H(t) + 1) + Me^{-t}) - \delta) + \lambda_E(t)(A\ln(\tau_W(t) + 1)) + \omega H(t)) \quad \text{(A4)}$$

Equation J is often termed the present value Hamiltonian function, since the exponential discounting term $e^{-\beta t}$ becomes dis-applied. Solving the optimal control theory using the present value Hamiltonian function or a standard Hamiltonian function does not influence the results and the choice is purely for the sake of convenience and mathematical succinctness.

The Pontryagin Maximum Principle requires that the Hamiltonian function J be maximised for all $t \in [0, q]$ under the optimal control variables $\tau_H^*(t)$ and $\tau_W^*(t)$. The Hamiltonian function J is strictly concave in its arguments as shown by the following second order derivatives:

$$\frac{\partial^2 J}{\partial \tau_H(t)^2} = -A\lambda_H(t)(\tau_H(t) + 1)^{-2} < 0 \quad \text{(A5)}$$

$$\frac{\partial^2 J}{\partial \tau_W(t)^2} = -A(\theta + \lambda_E(t))(\tau_W(t) + 1)^{-2} < 0 \quad \text{(A6)}$$

Therefore the first optimality condition can be used to locate the extrema, or the optimal value of the control variables in each time period t which maximises the Hamiltonian function. By applying the first order optimality condition $\frac{\partial J}{\partial \tau_H(t)} = \frac{\partial J}{\partial \tau_W(t)} = 0$, I derive the optimal time paths for the two control variables as shown:

$$\tau_H^*(t) = A\lambda_H(t) - 1 \quad \text{(A7)}$$

$$\tau_W^*(t) = A(\theta + \lambda_E(t)) - 1 \quad \text{(A8)}$$

According to the Pontryagin Maximum Principle, the time derivatives of the co-state paths can be calculated as shown below:

$$\dot{\lambda}_H = -\frac{\partial J}{\partial H} + \beta\lambda_H(t) \quad \text{(A9)}$$

$$\dot{\lambda}_E = -\frac{\partial J}{\partial E} + \beta\lambda_E(t) \quad \text{(A10)}$$

I proceed to solving the optimal co-state path for education first. Since $\dot{\lambda}_E = \beta((\theta + \lambda_E(t))$, I can solve the above first order differential equation to derive the optimal co-state path for education:

$$\lambda_E^*(t) = k_1 e^{\beta t} - \theta \quad \text{(A11)}$$

Where k_1 is an arbitrary constant. Given that I assume that the terminal value of the stock of education at $t = q$ is free to vary and take on any value, I may apply the transverality condition that $\lambda_E^*(q) = 0$. By this condition, the constant k_1 can be definitized:

$$k_1 = \theta e^{-\beta q} \quad \text{(A12)}$$

Substitute equation (A12) into (A11) replacing k_1 I obtain equation (3.13) as shown in Chapter 3.

$$\lambda_E^*(t) = -\theta\left(1 - e^{-\beta(q-t)}\right) \quad \text{(3.13)}$$

Equation (A9) can be shown to be:

$$\dot{\lambda}_H = -\omega(\theta + \lambda_E(t)) - G + \beta\lambda_H(t) \quad \text{(A9')}$$

Notice from equation (A9') that the time derivative of the co-state path for health capital is a function of the co-state path for educational capital. This is because health capital enhances the accumulation of educational capital. Therefore the co-state variable of health, which represents the 'shadow price' or value of a unit of health capital is dependent on the co-state variable of education, or the value of a unit of educational capital. Note that the reverse is not true since I assume that the accumulation of health capital is independent of the stock of educational capital. It is for this reason I proceed to solve the optimal co-state path for educational capital first. By substituting equation (3.13) into (A9') and replacing $\lambda_E(t)$ with $\lambda_E^*(t)$, I obtain the following first order differential equation:

$$\dot{\lambda}_H = -\omega\theta e^{-\beta(q-t)} - G + \beta\lambda_H(t) \quad \text{(A9'')}$$

By integrating equation (A9'') with respect to t I obtain equation (A13) below:

$$\lambda_H^*(t) = k_2 e^{\beta t} + G\beta^{-1} - \omega\theta e^{-\beta(q-t)}t \quad \text{(A13)}$$

I apply the transversality condition $\lambda_H^*(q) = 0$ since the stock of health capital when childhood ends is free to vary. This transversality condition allows us to definitise the arbitrary constant k_2:

$$k_2 = \left(\omega\theta q - \frac{G}{\beta}\right) e^{-\beta q} \quad \text{(A14)}$$

Substitute (A14) back into equation (A13) I obtain equation (3.12) of Chapter 3, which shows the optimal time path of the co-state variable for health capital:

$$\lambda_H^*(t) = (\omega\theta(q-t) - G)e^{-\beta(q-t)} + G\beta^{-1} \quad \text{(3.12)}$$

Having solved the optimal co-state paths for the two state variables, the remaining problems are relatively easy to solve. I begin by substituting the optimal co-state paths of equation (3.12) and (3.13) into the optimal control paths of (A7) and (A8), replacing $\lambda_H(t)$ and $\lambda_E(t)$ with $\lambda_H^*(t)$ and $\lambda_E(t)$, respectively. The corresponding optimal control time paths are shown by equations (3.10) and (3.11) in Chapter 3. I assume that the parameter variables are large enough so that the optimal control variables are interior solutions for all t, i.e. greater than zero.

$$\tau_H^*(t) = \max\left\{A\left((\omega\theta(q-t) - G)e^{-\beta(q-t)} + G\beta^{-1}\right) - 1{,}0\right\} \quad \text{(3.10)}$$

$$\tau_W^*(t) = \max\left\{A\theta e^{-\beta(q-t)} - 1{,}0\right\} \quad \text{(3.11)}$$

The optimal control paths (3.10) and (3.11) are then substituted into the equations of motion (3.4'') and (3.5'), respectively. I should solve the optimal state path for health capital before educational capital since the later is a function of the former. The substitution of equation (3.10) into (3.4'') yields the following equation:

$$\frac{d}{dt}H^*(t) = A\left(Me^{-t} + lnA + \ln\left(\omega\theta(q-t)e^{-\beta(q-t)} + G\beta^{-1}\left(1 - e^{-\beta(q-t)}\right)\right)\right) - \delta \quad \text{(A15)}$$

The * denote that the optimal control and co-state variables have been substituted into the equation of motion.

The optimal time path for the stock of health capital can be obtained by integrating (A15) with respect to t and using the initial condition that $H^*(0) = H_0$[130], definitise the arbitrary constant which arises as a result of the integration. However, A15 is a rather complicated function to integrate due to the logarithmic term. It is therefore necessary to state the integral in the following form:

$$H^*(t) = \int_0^t \frac{d}{dt} H^*(u)du + H_0 \tag{A16}$$

Equation (A16) produces the optimal solution for the time path of health capital, as shown by equation (3.14) in Chapter 3.

$$H^*(t) = A\left(M(1 - e^{-t}) + \int_0^t \ln\left(A\left((\omega\theta(q-u) - G)e^{-\beta(q-u)} + G\beta^{-1}\right)\right)du\right) - \delta t + H_0 \tag{3.14}$$

Now I substitute equations (3.10), (3.11) and (3.14) into equation (3.5'), replacing $\tau_H(t)$, $\tau_W(t)$ and $H(t)$ with $\tau_H^*(t)$, $\tau_W^*(t)$ and $H^*(t)$, respectively. This yields equation (A17) below:

$$\frac{d}{dt}E^*(t) = A(\ln(A\theta - \beta(q-t)) + \omega\left(A\left(M(1-e^{-t}) + \int_0^t \ln\left(A\left((\omega\theta(q-u) - G)e^{-\beta(q-u)} + G\beta^{-1}\right)\right)du\right) - \delta t + H_0\right) \tag{A17}$$

Integrating (A17) with respect to t yields:

$$E^*(t) = \omega\left(H_0 - \frac{1}{2}\delta t\right) + A\omega M(t + e^{-t}) + A\beta^{-1}(\ln(A\theta) - \beta(q-t)) + \frac{1}{2}A\omega(\ln(A) + \ln(\omega\theta(q-t)e^{-\beta(q-t)} + G\beta^{-1}(1 - e^{-\beta(q-t)}) + 2\ln(t)) + k_3 \tag{A18}$$

Using the initial condition that $E^*(0) = 0$, I can definitise the arbitrary constant k_3 in equation (A18) above.

$$k_3 = -A(\omega M + \beta^{-1}\ln(A\theta) - q) \tag{A19}$$

If equation (A19) is substituted into (A18) and simplified, I obtain equation (3.15) as in Chapter 3, the optimal stock of educational capital.

[130] The initial stock of health capital at $t = 0$ is exogenously given.

$$E^*(t) = \frac{1}{2}At\left(\ln\left((\omega\theta(q-t)-G)e^{-\beta(q-t)} + G\beta^{-1}\right)\omega t + 1\right) + \omega\left(AM\left(t-(1-e^{-t})\right) - \frac{1}{2}t(\delta t - H_0)\right) \quad (3.15)$$

I have now obtained all the solutions to the childhood phase model, and shall proceed to solve the adulthood phase. I assume that for adults the subjective rate of discount is zero i.e. $\beta = 0$. This greatly simplifies my analysis with little or no cost to the analytical power. Moreover, I do not have adult data on the value of β, hence I cannot test for its effect on the optimal solution empirically, even if I include it in my model. The adult's inter-temporal utility function can be written as:

$$U(t) = (\log\big(X(t)\big) + GH(t) - \tau_W(t) - \tau_H(t))) \quad (A20)$$

I assume that the consumption and use of medical services of an adult must be subject to the budget constraint (3.8') and that there are no savings.

$$P_X X(t) - P_M M(t) = y\tau_W(t) \quad (3.8')$$

I may substitute equation (3.8') into the adult's inter-temporal utility function (A20). One approach is to make $\tau_W(t)$ the subject in equation (3.8'):

$$U(t) = (\log\big(X(t)\big) + GH(t) - \frac{P_M(t)M(t)+P_X X(t)}{y} - \tau_H(t))) \quad (A21)$$

The equation of motion for the adulthood phase optimal control problem is shown by (3.7''). There is only a single state variable for the adulthood phase since the adult is no longer attempting to amass educational capital, which is taken as exogenous in my model. The only state variable is the stock of health capital.

$$\dot{H} = -\delta\big(I(t)\big) = -\frac{\delta_0 e^{-A\tau_H(t)}}{AM(t)+1} \quad (3.7'')$$

Using equation (A21) and (3.7'') I form the Hamiltonian function below:

$$Z = (\log\big(X(t)\big) + GH(t) - \frac{P_M(t)M(t)+P_X X(t)}{y} - \tau_H(t))) - \lambda_H(t)(\frac{\delta_0 e^{-A\tau_H(t)}}{AM(t)+1}) \quad (A22)$$

The Hamiltonian function Z is concave in its control variable arguments $\tau_H(t)$, $M(t)$ and $X(t)$, since the second order partial derivatives of the Hamiltonian function with respect to these variables are all negative:

$$\frac{\partial^2 Z}{\partial \tau_H(t)^2} = -\delta_0 A^2 e^{-A\tau_H(t)}(AM(t)+1)^{-1}\lambda_H(t) < 0 \quad \text{(A23)}$$

$$\frac{\partial^2 Z}{\partial M(t)^2} = -2\delta_0 A^2 e^{-A\tau_H(t)}(AM(t)+1)^{-3}\lambda_H(t) < 0 \quad \text{(A24)}$$

$$\frac{\partial^2 Z}{\partial X(t)^2} = -X(t)^{-2} < 0 \quad \text{(A25)}$$

The first order optimality conditions can therefore be used to locate the optimal control values which maximise the Hamiltonian function for all t. Via the condition $\frac{\partial Z}{\partial \tau_H^*(t)} = \frac{\partial Z}{\partial M^*(t)} = \frac{\partial Z}{\partial X^*(t)} = 0$, the optimal paths for the control variables can be solved. However, due to the functional form I choose to represent investment in health, the two factors which contribute to health investment $\tau_H(t)$ and $M(t)$ are not independent of each other. The optimal amount of $\tau_H^*(t)$, time investment in health, depends on the optimal quantity of medical services $M^*(t)$, which is used, at any time period t, such that $\frac{\partial \tau_H^*(t)}{\partial M(t)}, \frac{\partial M^*(t)}{\partial \tau_H(t)} \neq 0$. It is therefore necessary to solve $\tau_H^*(t)$ and $M^*(t)$ simultaneously. This yields equations (A26) and (A27) below:

$$\tau_H^*(t) = \ln(A\delta_0 P_M \lambda_H(t) y^{-1})^{A^{-1}} \quad \text{(A26)}$$

$$M^*(t) = (y - P_M)(AP_M)^{-1} \quad \text{(A27)}$$

I assume that $y > P_M$, hence the optimal quantity of medical to be used is always positive.

The optimal control path for consumption can also be obtained easily:

$$X^*(t) = \frac{y}{P_X} \quad \text{(A28)}$$

The optimal consumption is a constant across time and is denoted by the ratio of income and the price per unit of consumption.

I now seek to find the optimal time path of the state variable $\lambda_H^*(t)$. I begin by employing the Maximum Principle condition $\dot{\lambda}_H = -\frac{\partial Z}{\partial H}$. This yields the following first order differential equation, which is but a constant represented by the marginal product of a unit of health capital:

$$\dot{\lambda}_H = -G \quad \text{(A29)}$$

$\lambda_H^*(t)$ is solved very simply by integrating A29 with respect to t, which yields equation (3.19) in Chapter 3.

$$\lambda_H^*(t) = c_1 - Gt \tag{3.19}$$

The definitisation or the arbitrary constant c_1 depends on the transversality condition I specify. If I state that T the length of life or V the remaining adult years are fixed, and that the terminal value of health capital is free to take on any value, then I can apply the transversality condition $\lambda_H^*(V) = 0$. This would allow us to immediately definitise c_1.

$$c_1 = GV \tag{3.20}$$

Equation (3.20) is shown in Chapter 3.

Substitute (3.20) into (3.19);

$$\lambda_H^*(t) = G(V - t) \tag{A30}$$

Equation (A30) can be substituted back into the optimal control equations. In fact only the optimal control variable $\tau_H^*(t)$ is dependent on the value of a unit of health capital. (A309) is to be substituted into equation (A26), replacing $\lambda_H(t)$:

$$\tau_H^*(t) = \ln(A\delta_0 P_M G(V - t) y^{-1})^{A^{-1}} \tag{A26'}$$

Equations (A26') and (A27), which are the optimal control functions can be substituted into the equation of motion (3.7''):

$$\frac{d}{dt} H^*(t) = -y\big(AP_M G(V - t)\big)^{-1}\big((y - P_M)P_M^{-1} + 1\big)^{-1} \tag{A31}$$

Integrate (A32) with respect to t and using the condition that $H^*(0) = H_0$[131], I derive the optimal stock of health capital as shown in equation (3.21) in Chapter 3:

$$H^*(t) = (GA)^{-1} \ln\left(\frac{V-t}{V}\right) + H_0 \tag{3.21}$$

However, if the adulthood problem's end time specification is stated as a variable terminal time problem, the solution process is somewhat more onerous. It is not possible to apply the transversality condition $\lambda_H^*(V) = 0$ and have a clear path to deriving the solution. I must bring

[131] The initial state of health capital is fixed. However the initial state of health capital at the beginning of the adulthood phase is the same stock of health at the end of the childhood phase at $t = q$. I set the initial time for the adulthood phase model at $t = 0$ rather than $t = q$ purely for the sake of convenience, yet it should be borne in mind that the later is conceptually the true t value at the beginning of the adulthood phase.

the co-state variable with the arbitrary constant c_1 undefinitised into the control and state variables, and solve using other sets of conditions. I bring equation (3.19) into (A26), replacing $\lambda_H(t)$ with $\lambda_H^*(t)$:

$$\tau_H^*(t) = \ln(A\delta_0 P_M(c_1 - Gt)y^{-1})^{A^{-1}} \quad \text{(A26'')}$$

Bring (A26'') and (A27) into equation (3.7''):

$$\frac{d}{dt}H^*(t) = -y\left(AP_M(c_1 - Gt)\big((y - P_M)P_M^{-1} - 1\big)\right)^{-1} \quad \text{(A32)}$$

If I integrate (A32) with respect to t, I obtain equation (3.21') of Chapter 3.

$$H^*(t) = (GA)^{-1}\ln(Gt - c_1) + c_2 \quad \text{(3.21')}$$

There are now two arbitrary constants c_1 and c_2 to be definitized. Moreover, I specify that the terminal time T or the remaining adulthood time V is an endogenous variable dependent on the values of the control variables and can be expressed as a function of the other exogenous variables. I also assume that $H^*(V) = H_{min}$, meaning that at life's end, the stock of health capital retained by the adult would fall to the minimum level required to sustain life. The initial condition of health capital as already mentioned above is fixed by the health accumulated at the end of the childhood phase and I use the equation $H^*(0) = H_0$ to express.

The transversality condition applied to variable terminal optimal control problems such as the current specification that the adulthood life time is endogenous, is given by $Z^*(V) = 0$. In other words, the Hamiltonian function, evaluated when the optimal control variables are substituted in, must equal to zero at $t = V$. This condition essential requires that all the opportunities to maximise or further increase the Hamiltonian function are exhausted fully. If I locate a value of V which satisfies the state conditions, then it is an optimal value V^* such that $F(V^*) = \int_0^{V^*} Z(t)dt$ is maximised. An increase or decrease in V reduces the value of the function F. It can be shown that $Z(V^* + \epsilon) < 0$ and $Z(V^* - \epsilon) > 0$, where ϵ is an arbitrarily small positive number.

Using the initial condition for the state variable $H^*(0) = H_0$, the terminal condition $H^*(V^*) = H_{min}$ as well as the transversality condition $Z^*(V^*) = 0$, I form a set of three simultaneous equations. The initial and terminal conditions are applied first to the state variable in equation (3.21'). The optimal control functions (A26''), (A27) and (A28) are substituted into the

Hamiltonian function in equation (A22) along with (3.19) and (3.21'), replacing $\lambda_H(t)$ and $H(t)$ with $\lambda_H^*(t)$ and $H^*(t)$, respectively. The t variable in the Hamiltonian function is then set to V^*. The set of simultaneous equations are shown below:

$$(AG)^{-1}\ln(c_1) + c_2 = H_0 \tag{A33}$$

$$(AG)^{-1}\ln(c_1 - GV^*) + c_2 = H_{min} \tag{A34}$$

$$GC_2 - 1 - 2A^{-1} + P_M(Ay)^{-1} + \ln(y(A\delta_0 P_M)^{-1}(GV^* - c_1)^{-2})^{A^{-1}} = 0 \tag{A35}$$

The above set of three simultaneous equations contains three unknown endogenous variables – c_1, c_2 and V^*. If (A33), (A34) and (A35) are solved simultaneously in search of the three known variables then I arrive at equations (3.20'), (3.22) and (3.23) as shown in Chapter 3.

$$c_1 = \frac{y}{A\delta_0 P_M}\left(\frac{y}{P_X}\right)^A e^{A(GH_0-1)-2+\frac{P_X}{y}} \tag{3.20'}$$

$$c_2 = (GA)^{-1}\left(A + 2 - \frac{P_M}{y} - \ln\left(\frac{A\delta_0 P_M}{y}\left(\frac{P_M}{y}\right)^A\right)\right) \tag{3.22}$$

$$V^* = (GA\delta_0 P_M)^{-1}\left(1 - e^{-GA(H_0 - H_{min})}\right)\left(\frac{y}{P_X}\right)^A e^{A(GH_0-1)-2+\frac{P_M}{y}} y \tag{3.23}$$

Knowing the value of c_1, I substitute equation (3.20') into (A26''), to solve the optimal time path for adult time input for health investment.

$$\tau_H^*(t) = \ln\left(y^{-1}A\delta_0 P_M\left(\frac{y^{A+1}}{A\delta_0 P_M P_X^A}\right)e^{A(GH_0-1)-2+\frac{P_M}{y}}\right)^{A^{-1}} \tag{A26'''}$$

Equation (3.20') and (3.22) can both be substituted into (3.21') to obtain the optimal time path for health capital, which is shown by equation (3.21'') in Chapter 3.

$$H^*(t) = (GA)^{-1}\left(\ln\left(\frac{A\delta_0 P_M}{y}\left(\frac{P_X}{y}\right)^A\left(e^{A(GH_0-1)-2+\frac{P_M}{y}} - Gt\right)\right) + A + 2 - \frac{P_M}{y}\right) \tag{3.21''}$$