ESSAYS ON HEALTH AND ECONOMIC GROWTH

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Intan Zanariah Zakaria

B. Econ (IIUM, Kuala Lumpur), M.A. Econ (York, Toronto)

Department of Economics

University of Leicester

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To my dearest mother

Essays on Health and Economic Growth

Abstract

Intan Zanariah Zakaria

This thesis consists of three chapters. The first chapter is 'Endogenous Fertility in a Growth Model with Public and Private Health Expenditures'. In this chapter, we build an overlapping generations model that incorporates endogenous fertility choices, in addition to public and private expenditures on health. Following the seminal analysis of Bhattacharya and Qiao (JEDC, 2007) we assume that the effect of public health investment is complementary to private health expenditures. We find that this effect reinforces the positive impact of the capital stock on aggregate saving. Furthermore, this complementarity can provide an additional explanation behind the salient features of demographic transition; that is the fertility decline along the process of economic growth.

The second chapter is 'Growth and Demographic Change: Do Environmental Factors Matter?'. In this chapter, we incorporate health-damaging pollution into a three-period overlapping generations model in which life expectancy, fertility and economic growth are all endogenous. We show that environmental factors can cause significant changes to the economy's demographics. In particular, the entrepreneurial choice of less polluting production processes, induced by environmental policy, can account for such demographic changes as higher longevity and lower fertility rates. Thus, we provide a novel environment channel of demographic transition.

The third chapter is 'The Effects of Foreign Aid on Growth: Health Aid versus Untied Aid'. In this chapter, we build an overlapping generations model with foreign aid and private health expenditures. The effect of an increase in foreign aid on growth is ambiguous as it depends on the proportion of health aid and the proportion of untied aid allocation to individuals. We also introduce health aid in the production function and we find that the growth impact is non-monotonic. There are thresholds of aid for which the growth impact of aid is negative (positive) if aid lies within (outside) these thresholds.

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In the name of Allah, the most Gracious, the most Compassionate

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Introduction

Health is a fundamental aspect of the development process. It can be considered as an outcome of the growth process; where people with higher income are healthier because they have more resources to spend on goods and services for health improvements. Additionally, health is also a major cause of the development process because good health yields positive outcomes such as productive labour force and poverty reduction (WHO 2008). In light of the above facts, this thesis presents three chapters in health and theory of growth.

In most countries, the provision of public health services is undertaken by their respective governments in view of providing at least the basic health care to the population in an economy. Additionally, individuals also spend part of their resources not only for the maintenance but also for the improvements of their own health status. The provision of the former could enhance the effectiveness of the latter in promoting the population's health status in the economy (Bhattacharya and Qiao 2007). This characteristic between the two types of spending can be seen in many instances. For example, in order to combat heart diseases effectively, patients may combine healthier diet and medication with an appropriate medical advice from general practitioners.

Chapter 1, entitled "Endogenous Fertility in a Growth Model with Public and Private Health Expenditures" presents a framework that links the complementarity between public and private health expenditures (an idea that is introduced by a seminal work of Bhattacharya and Qiao (2007)) with fertility in the endogenous growth framework. The complementarity between both expenditures means that the effectiveness of private health expenditures is reinforced by public health services. This chapter shows that an increase in public health spending by the government could encourage individuals to spend more of their resources towards health improvements during their old age. Therefore, they will save more during young adulthood for this purpose and adjust their labour supply by reducing the time spent on raising children so as to earn more resources. This amplifies the capital stock and economic growth. Furthermore, it could also provide an additional explanation as to why fertility rates decline along the process of economic development.

Another issue that has attracted significant interest over the years is the issue of environmental impacts on health status. For an instance is pollution- a by-product of economic activities. Due to its health-damaging character, pollution could risk the quality of life of population. This adverse effect of environmental factor on life expectancy could change the reproduction behaviour of individuals. Therefore, changes in the environment quality may actually cause demographic changes as well.

Chapter 2, entitled "Growth and Demographic Change: Do Environmental Factors Matter?" analyses the scenario mentioned above in an overlapping generations model. Particularly, it demonstrates how environmental factors could actually cause demographic changes; namely higher life expectancy and lower fertility rates. In this chapter, we assume that producers of intermediate products have a choice between two different types of technology; one with higher emission rate and another one with lower emission rate. The introduction of the environmental tax could induce the firms to use a cleaner technology of production as long as the economy exceeds a level of income. The switch from dirtier technologies to less polluting ones causes life expectancy to increase and consequently, fertility rates to fall.

Another critical issue in health is the lack of better public healthcare services in the least developed countries. Poor economic conditions have hindered the ability of these countries to provide good quality of public health services. Therefore, the proponents of foreign aid suggest that health aid should be given to these countries so as to achieve better health status for their respective populations. Nevertheless, the provision of aid that is tied to a particular project, such as health, has led to long-standing debates among policy makers and researchers, particularly in terms of its repercussions on the economic growth rates of the recipient countries. In fact, there are mixed views as to whether foreign aid should be tied to specific projects or not.

In Chapter 3, entitled "The Effects of Foreign Aid on Economic Growth: Health Aid versus Untied Aid", we attempt to analyse the growth-aid nexus in an OLG framework with private health spending. In the first model of this chapter, we consider two types of aid i.e. aid that is tied to health improvement activities and untied aid that takes the form of income transfers to young and old. We model the framework such that tied aid is given to co-finance the existing private health expenditures of old individuals. The implication of foreign aid on economic growth of the recipient country is ambiguous as it critically depends on the part of aid distributed to health improvement activities and also the part of aid that is transferred to the young.

In the second model, we introduce health aid as an input in the production function of the recipient economy. We find that the growth impact of overall aid is non-monotonic. That is, the repercussion of aid on growth can only be positive (negative) if it lies outside (within) a range of aid thresholds. These results facilitate us to explain the reason why existing studies of aid-growth nexus show mixed results.[1]

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[1] The calculations of individual's utility maximisation and profit maximisation of firms for all three chapters are relegated to the Appendix.

Chapter 1

Endogenous Fertility in a Growth Model with Public and Private Health Expenditures [2]

1.1 Introduction

One of the salient features of demographic transition is the decline of the fertility rate along the process of economic development (e.g., Kirk 1996; Ehrlich and Lui 1997; Galor 2012). Among the various explanations that have emerged while trying to explain this outcome, a prominent place belongs to those that attribute it to improvements in the population's health status at more advanced stages of an economy's development process. A common feature to these analyses is that such improvements are either exogenous or they are driven by an aggregate externality according to which public health expenditures

[2] A paper version of this chapter has been published. The publication details are:
Varvarigos, D., and Zakaria, I.Z. 2013. "Endogenous fertility in a growth model with public and private health expenditures", *Journal of Population Economics*, 26, 67-85.

improve the population's health characteristics. In Blackburn and Cipriani (2002) the provision of public health services improves life expectancy and, thus, raises the effective return on education. As a result, households respond by reducing their expenditures during the earlier stages of their lifetime – among them, the expenditures for child rearing. A similar mechanism pervades the analysis of Zhang and Zhang (2005), the difference being that life expectancy is an exogenously given parameter. Kalemli-Ozcan (2003) assumes that child survival follows an exogenous stochastic process and shows that a reduction in child mortality induces a reduction in the number of children reared and a corresponding increase in the parental investment to each child's human capital. In Soares (2005) parents derive utility, not only from their surviving offspring, but also from the length of each surviving child's lifespan. He shows that improvements in these health characteristics can generate a demographic transition as the economy develops.[3]

None of the aforementioned analyses, however, considers private health spending as an additional factor determining an agent's health status despite the fact that, in reality, private expenditure appears to represent a significant part of total health spending in many economies. For example, a recent publication by the World Health Organisation (2010) reports that private health expenditures amount to roughly 50% of the total in the United States, they range from 20%-40% of total health expenditures in some countries of the European Union, while in many less-developed countries they contribute to more-than-half of total health expenditures.

[3] For empirical support on the negative relation between improved health status and fertility rates, see Finlay (2007).

The more recent papers by Strulik (2008) and Manuelli and Seshadri (2009) are notable exceptions since they take account of the importance of private health expenditure in their analyses of fertility choices. Strulik (2008) builds a two-period overlapping generations model with subsistence consumption and infant survival. The latter includes an exogenous component – positively related to average income per capita among others – and an endogenous one, capturing the resources that parents devote towards the health care of their offspring. He finds that the relationship between fertility and income is inverted-U-shaped due to two opposing effects. On the one hand, higher income relaxes the strain imposed by subsistence consumption and leaves more resources available for child rearing. On the other hand, an increase in income induces parents to devote more resources towards their children's health care in order to take advantage of the increased public health spending and, thus, reduce the infant mortality rate even further. They do this at the expense of their fertility rate. Manuelli and Seshadri (2009) calibrate a general, multi period model in which both fertility decisions and agents' lifetimes are endogenous – the latter being determined by health capital that is supported by private spending. They find that their model fits well to the data and can explain major cross country differences in fertility rates and life expectancy at birth. Their model allows them to attribute these differences to changes in either Total Factor Productivity or taxes on labour income.

In this chapter, we offer an alternative and novel mechanism via which the presence of private health spending can account for the incidence of fertility decline at higher stages of economic development. In particular, we consider both private and public health expenditures in the manner suggested by the seminal analysis of Bhattacharya and Qiao (2007). In their paper, they analyse a growth model in which public health investment is supportive to the effectiveness of the expenditures that individuals incur for the improvement of their health status. They find that an increase in the public provision of health services induces individuals to reduce their saving and, correspondingly, increase the resources they devote towards health improvements. As a result, the dynamics of capital intensity become non-monotonic and may admit periodic (endogenous limit cycles) or even aperiodic (i.e., chaotic) equilibria. Nevertheless, the focus of their paper is not related to demographic change, that is why they do not consider endogenous fertility.

Our model utilises the main idea of Bhattacharya and Qiao (2007) – that is, public health investment being complementary to (optimally chosen) private health spending – but modifies their set-up in the following respects: (i) we assume that individuals are reproductive during their young adulthood and they choose the number of their offspring in an optimal fashion; (ii) we allow individuals to consume during both periods of their adulthood, and not only during their old age;[4] (iii) rather than considering private health expenses as incurred during young adulthood, with the purpose of improving life expectancy, we assume that individuals incur their health expenses during the final period of their lifetime in order to improve their utility-enhancing health status.

[4] This particular extension is not critical for the subsequent results. These remain similar even if individuals care only about old-age consumption.

Our results can be summarised as follows. We find that, rather than generating a trade-off between saving and private health spending, the provision of public health services induces an increase in *both* private health spending *and* saving. This is due to the fact that agents wish to have more resources when old in order to take advantage of the higher productivity of public health services and, consequently, improve their quality of life during the final stage of their lifetime. In addition to the motive for increased saving, this effect has ramifications for the fertility choices of reproductive agents. Particularly, they will try to mitigate the strain of higher saving on their first period consumption by increasing the time they devote to earning labour income in order to increase the resources available for consumption. As a result, they are induced to bear and raise fewer children during their young adulthood as means of consumption smoothing over their lifetime. As young individuals take advantage of the higher productivity of public health services by providing more labour supply to increase their resources during old, this implies that their labour market participation has a positive correlation with public health spending. In this case, we can consider households as couples (i.e. husbands and wives) who make decisions together in terms of allocating time for working and also for rearing children. A positive correlation is found between labour participation rate and public health expenditures (as percentage of total health expenditures) for the World Bank data of OECD countries in 2011 (see Figure 1.1).

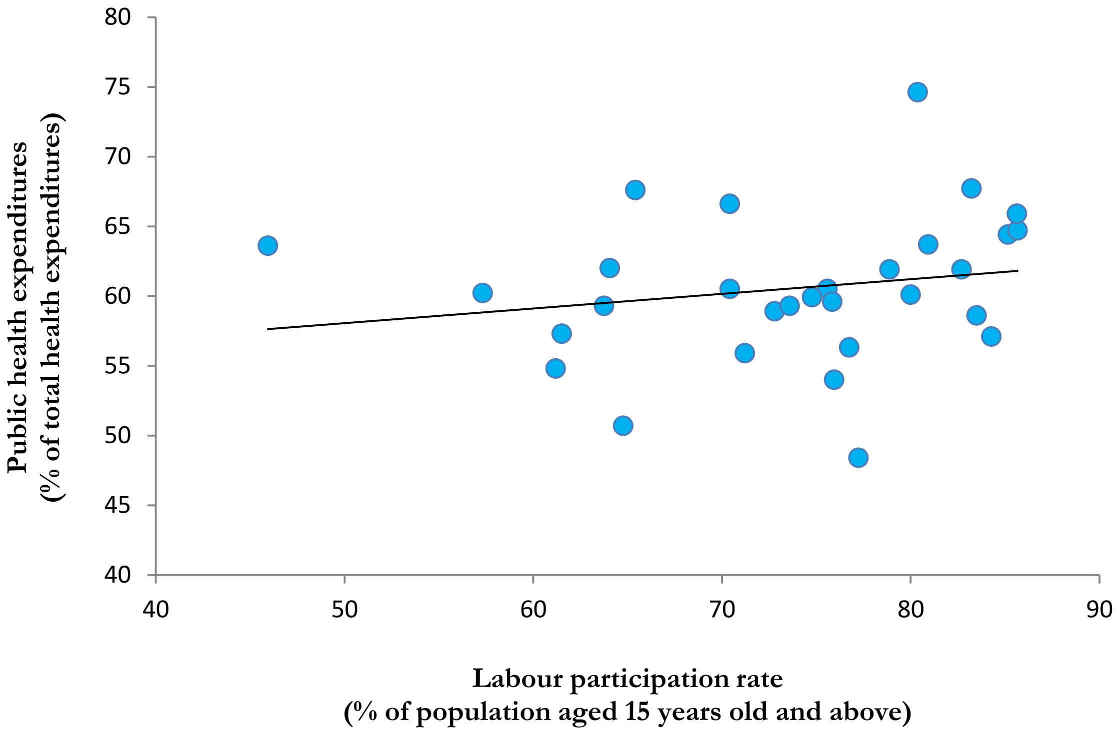

Figure 1.1

Labour participation rate and public health expenditures in OECD countries (2011)

The assumption according to which individuals incur health expenses when old is not critical for the result according to which fertility declines are attributed to the interaction between private and public health expenditure. We show this formally by solving a similar model, but assuming that health status takes the form of capital, given that private health spending is incurred by young agents.[5] The only result that changes is the one concerning the marginal propensity to save which, similarly to Bhattacharya and Qiao (2007) in which the young devote resources to health improvements, falls as a result of an increase

[5] Of course, the intuition differs as it will transpire in Section 1.5 where we provide the formal analysis.

in public health services provision. Furthermore, we show that our main results are robust to an alternative health production technology with similar characteristics, that is a technology whereby the efficiency of private health spending is supported by the provision of public health services.

Given the above, our analysis joins the strand of literature that attributes the decline in the fertility rate to improvements in the health status of reproductive agents. Nevertheless, there is an important difference between our analysis and the aforementioned body of literature. In particular, we demonstrate *the importance of the interplay between public and private health spending in generating the decline in fertility rates* – an idea that has eluded the attention of existing theories on the nexus between economic growth and demographic transition. This importance has actually been already identified by Cigno (1998) in his analysis of endogenous fertility in a static model with endogenous infant mortality. Assuming that infant mortality falls as a result of public health spending, he argues that there is no *a priori* clear mechanism between the latter and the resources that parents devote towards their children's health. In particular, public and private health expenditures may be either complements or substitutes depending on how parents form their decisions. In the former case, the optimal fertility rate increases whereas in the latter case it declines.

Concerning the link between private and public health expenditure, our assumption is admittedly not as general as the one utilised by Cigno (1998). Our focus is on the particular case whereby the effectiveness of private health expenditure is enhanced in an environment where the productivity of the sector that offers health services is supported by higher public spending. Nevertheless, we are able to offer a different dimension in comparison to Cigno (1998) since the complementary effect of public health expenditure on its private counterpart is responsible for declining rather that increasing fertility. In addition, another difference of our model is that it is a dynamic one with particular emphasis on the issues of capital accumulation and economic growth.

There is another important difference of our setting compared to some of the aforementioned literature (Cigno 1998; Kalemli-Ozcan 2003; Soares 2005; Strulik 2008). That is why we need to stress that our intention is not to provide another link of health and fertility based on the issue of infant mortality. While the latter is indubitably a very important aspect of demographic transition, our purpose is to offer an alternative, intuitive, but yet unexploited mechanism that attributes variations of fertility choice on health issues that, while not necessarily fatal, they could have significant repercussions for an adult's quality of life and well-being (e.g., chronic conditions and illnesses; various forms of physical injuries and disabilities; depression and anxiety disorders etc.). We believe that this mechanism is potentially important and, thus, worthy of consideration. Therefore we have focused on it by following other analyses (e.g., Becker et al. 1990; Tamura 1996; Galor and Weil 1996, 2000; de la Croix and Doepke 2003) that abscond from infant mortality when considering the issue of endogenous fertility.

One may argue that the existence of public health services in supporting the effectiveness of private health spending for health of old adults can be a form of old age insurance. Note that, the focus in this chapter is not to analyse the role of children as providers of old age security for their parents. Despite the fact that this is an indubitably important issue, there are a large number of analyses that already explore the significance role of children as old age insurance in relation to other alternative such as social security and etc. (e.g., Zhang and Zhang, 2005; van Groezan et al., 2003; Wigger, 1999).

The rest of the chapter is organised as follows. In Section 1.2 we outline the basic set-up of the economy. Section 1.3 derives the model's equilibrium and Section 1.4 analyses the dynamics of capital accumulation as well as the demographic transition. In Section 1.5 we show that our results on the link between fertility choice and the interactions between private and public health spending can remain robust under some modified set-ups. Section 1.6 concludes.

1.2 The Economy

Time is discrete and indexed by $t = 0,1,...\infty$. We consider an economy which produces a homogeneous commodity and is inhabited by reproductive individuals who live for three periods and belong to overlapping generations. The three periods of an individual's lifetime are *childhood*, *young adulthood* and *old adulthood*. Agents make decisions only after they reach their adulthood. These decisions are dictated by the desire to maximise their lifetime utility function

$$u^t = \ln(c_t) + \gamma \ln(n_t) + \beta \ln(h_{t+1} c_{t+1}), \quad \gamma > 0\,,\ \beta \in (0,1),$$

or, equivalently,

$$u^t = \ln(c_t) + \gamma \ln(n_t) + \beta [\ln(c_{t+1}) + \ln(h_{t+1})], \tag{1.1}$$

subject to the constraints

$$c_t = (1-\tau)(1-qn_t)\omega_t - s_t, \quad q > 0, \; \tau \in (0,1), \tag{1.2}$$

$$c_{t+1} = r_{t+1}s_t - x_{t+1}, \tag{1.3}$$

$$h_{t+1} = Hx_{t+1}^{\delta\varepsilon_{t+1}}, \quad H > 0, \; \delta \in (0,1). \tag{1.4}$$

In the previous expressions, c_t denotes consumption during young adulthood, c_{t+1} denotes consumption during old adulthood, n_t is the number of children raised by a young adult, ω_t is the market wage per unit of labour, s_t denotes saving, r_{t+1} is the gross interest on saving, h_{t+1} is the old adult's health status and x_{t+1} is the old adult's spending towards health improvements. When young, each person is endowed with a unit of time which she allocates between raising children and providing labour services. Raising each child requires q units of time. Therefore, the young adult will use her remaining time to earn labour income – an income that is subject to a flat tax rate τ. She divides her disposable income between consumption and saving. The latter is deposited to a financial intermediary with the purpose of providing the agent with retirement income when she becomes an old adult. When old, the agent can potentially face some health problems which she can tackle by using part of her retirement income for the improvement of her health status. The remaining part of retirement income is used so as to satisfy her consumption needs. Note that by augmenting the utility from old age consumption by health status – that is, writing the last term of lifetime utility as $\ln(h_{t+1}c_{t+1})$ – we assume that improved health increases an old agent's quality of life. The higher the health status is, the greater the utility enjoyed for given levels of consumption (see Pitt et al. 1990).[6]

With respect to the link between public and private health expenditures, we follow Bhattacharya and Qiao (2007) in using the expression in (1.4) for which it is assumed that

$$\varepsilon_{t+1} = Z(p_{t+1}), \tag{1.5}$$

Where

$$p_{t+1} = \frac{g_{t+1}}{N_t}. \tag{1.6}$$

The function $Z(p_{t+1})$ in (1.5) satisfies $Z(0) = 1$, $Z(\infty) = \overline{\varepsilon} > 1$, $Z' > 0$, $Z'' < 0$, $Z'(0) = \varphi > 0$ and $Z'(\infty) = 0$.[7] Given these, it is straightforward to establish that $Z(p_{t+1}) > Z'(p_{t+1})p_{t+1}$ holds. In equation (1.6), the variable g_{t+1} is the stock of public capital devoted to health services and N_t is the population of those agents who are young in period t and therefore will be old during $t+1$.[8] The presence of the variable N_t is meant to capture a congestion-type effect. In particular, those who are young in period t will access public health services when they become old, i.e., in period $t+1$. We assume that, for given g_{t+1}, a larger population of agents mitigates the benefit accrued to each agent individually. Thus, public health is non-excludable but rival.

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[6] For a similar assumption on health expenses being incurred during old adulthood, see Gutiérrez (2008).

[7] We assume that $\delta\overline{\varepsilon} < 1$ in order to ensure the concavity of h_{t+1} with respect to x_{t+1}. A functional form that satisfies all these conditions is $Z(p_{t+1}) = 1 + \frac{\varphi p_{t+1}}{1 + p_{t+1}}$ with $\varphi = \overline{\varepsilon} - 1$.

[8] The idea that public health spending contributes to some type of capital formation is intuitive once we think of spending on hospitals, medical equipment, support for medical research and training etc.

Given the above, the assumptions illustrated through (1.4), (1.5) and (1.6) provide a mechanism through which the public investment in health services is complementary and supportive to private health expenditures. Particularly, the former promotes the effectiveness of the latter in improving the agent's health status during old adulthood.[9] This can be formally expressed through

$$\frac{dh_{t+1}}{dx_{t+1}}\frac{x_{t+1}}{h_{t+1}}=\delta\varepsilon_{t+1}=\delta Z\left(\frac{g_{t+1}}{N_t}\right), \tag{1.7}$$

i.e, the elasticity of health status with respect to private health spending is increasing to the stock of public capital that the government devotes towards health services.

Of course, there is a notable difference between our setting and that of Bhattacharya and Qiao (2007) when it comes to the modelling of health improvements. In their model, an agent's health spending occurs during her youth because the main motive is to increase her life expectancy. In our model, the old individuals are those that devote resources towards health improvements as they try to enhance the quality of life during the final period of their existence. Certainly, this is not an alien assumption. Life expectancy, albeit hugely important, is by no means the only factor that determines the health status of a person. There is a variety of nonfatal medical conditions that can cause great discomfort and mitigate the quality of life unless treated effectively.

[9] We can think of many examples that justify this assumption. The presence of qualified professionals – in the national health system – that offer support and advice on various difficulties that may emerge while people are trying to quit smoking (e.g., cravings etc.) may provide an incentive for smokers to seek and buy treatments that support Nicotine Replacement Therapy (patches, gums etc.). Clinical depression can be combated more effectively if sufferers combine antidepressant medication with appropriate counselling by qualified psychiatrists – counselling that is sometimes offered by professionals employed in the national health system. See Bhattacharya and Qiao (2007) for further examples in support of this conjecture.

Examples include chronic conditions and illnesses such as bronchitis; osteoporosis; prostatitis; periodontitis; dermatitis; diabetes; and various forms of physical injuries and disabilities. Furthermore, there are mental illnesses that have significant implications for a person's emotional well-being – for example, depression and anxiety disorders.

Another reason why we opt for this specification is related to an interesting outcome concerning the dynamics of capital accumulation. Although in Bhattacharya and Qiao (2007) the public provision of health services reduces the marginal propensity to save, the different timing of private health spending in our model results in a positive relation between public spending and the marginal propensity to save. As we shall see later, this effect reinforces the monotonicity of the economy's dynamics thus ruling out the emergence of periodic equilibria and endogenous fluctuations. Instead, in our case we may have multiple path-dependent equilibria. Given our focus on demographic aspects, this is actually a welcomed aspect in our analysis. In Section 1.5, we show that our results on the link between fertility choice and the interactions between private and public health spending can remain robust even under the assumption that individuals devote resources for health improvements when they are young. What changes in this case is the result concerning the saving rate which becomes an inverse function of the economy's capital stock. As we want to abscond from limit cycles, we have opted for the formulation discussed in the preceding part of the analysis.

In any time period t, there is a large number (normalised to one) of competitive firms who combine labour from young adults, L_t, and capital from financial intermediaries, K_t, so as to produce Y_t units of output according to

$$Y_t = AK_t^a L_t^{1-a}, \quad A > 0, \; 0 < a < 1. \tag{1.8}$$

In equilibrium, labour demand will be equal to labour supply. The latter is given by the total labour units devoted by the economy's young adults. Thus,

$$L_t = (1 - qn_t)N_t. \tag{1.9}$$

Firms who maximise profits will equate the marginal product of each input with the respective marginal cost. Taking account of (1.9), profit maximisation leads to

$$\omega_t = (1-a)AK_t^a L_t^{-a} = (1-a)Ak_t^a (1-qn_t)^{-a}, \tag{1.10}$$

and

$$r_t = aAK_t^{a-1} L_t^{1-a} = aAk_t^{a-1}(1-qn_t)^{1-a}, \tag{1.11}$$

where $k_t = K_t / N_t$ is the stock of capital per worker.

1.3 Equilibrium

A young adult will choose quantities for c_t, n_t, s_t, c_{t+1} and x_{t+1} to maximise (1.1) subject to (1.2)-(1.4), taking ω_t, r_{t+1} and ε_{t+1} as given. After some straightforward algebra, the first order conditions associated with an agent's optimal problem allow us to derive the solutions for saving, fertility and private health expenditures. These are

$$s_t = \frac{\beta(1+\delta\varepsilon_{t+1})}{1+\beta(1+\delta\varepsilon_{t+1})}(1-\tau)\omega_t(1-qn_t), \tag{1.12}$$

$$n_t = \frac{\gamma / q}{1+\beta(1+\delta\varepsilon_{t+1})+\gamma}, \tag{1.13}$$

and

$$x_{t+1} = \frac{\beta\delta\varepsilon_{t+1}}{1+\beta(1+\delta\varepsilon_{t+1})} r_{t+1}s_t. \tag{1.14}$$

Now, we shall assume that the government uses its collected revenues in period t to finance the formation of public capital that will be available next period, i.e., during $t+1$, according to a balanced budget rule. Given (1.9), we have

$$g_{t+1} = \tau L_t\omega_t = \tau(1-qn_t)N_t\omega_t. \tag{1.15}$$

Combining (1.15) with (1.5), (1.6) and (1.10) leads to

$$\varepsilon_{t+1} = Z\left(\tau(1-a)Ak_t^a(1-qn_t)^{1-a}\right). \tag{1.16}$$

Substituting (1.16) and (1.10) in (1.12) and (1.13) yields

$$s_t = \frac{\beta[1+\delta Z\left(\tau(1-a)Ak_t^a(1-qn_t)^{1-a}\right)]}{1+\beta[1+\delta Z\left(\tau(1-a)Ak_t^a(1-qn_t)^{1-a}\right)]}(1-\tau)(1-a)Ak_t^a(1-qn_t)^{1-a}, \tag{1.17}$$

and

$$n_t = \frac{\gamma / q}{1+\beta[1+\delta Z\left(\tau(1-a)Ak_t^a(1-qn_t)^{1-a}\right)]+\gamma}. \tag{1.18}$$

These solutions allow us to derive[10]

Proposition 1. *The economy's saving is increasing in the stock of capital per worker while the fertility rate is decreasing in the stock of capital per worker.*

Proof. See Appendix A. □.

The intuition behind the result of Proposition 1 is the following. A higher capital stock increases the government's revenues and allows a greater provision of public capital towards health services. As a result, the effectiveness of private health expenditures increases and old adults will find optimal to devote more resources towards them. Naturally, this implies that young adults will find desirable to have more resources available at the beginning of their old adulthood. Indeed, they can achieve this by saving a larger fraction of the disposable income they earn when young. Therefore, agents decide to limit the resources they keep during their reproductive period. This is an outcome to which they respond by reducing the number of children they rear and, correspondingly, increasing their effort to earn labour income. They do so

[10] Depending on different parameter values, n_t can become less than one in the steady state. However, this possibility can be ruled out by an appropriate parameter restriction – particularly, if the parameter q is sufficiently low. Alternatively, rather than normalising the total endowed time to unity, we could have endowed young individuals with a larger time endowment – so large, to guarantee that fertility is always above one. Qualitatively, none of our results would be affected.

because they try to counteract the negative effect on their consumption during youth and smooth out their consumption profile over their lifetime.

Notice that the complementarity between public and private health expenditures strengthens the positive link between saving and capital per worker of the standard overlapping generations model. In a standard overlapping generations model, the positive link between saving and capital per worker arises from the fact that saving increases with the wage rate, and the latter increases with the capital per worker. In our model, this mechanism is reinforced by the increase of public health expenditure arising from the increase in tax revenues due to the increase in income per capita, which strengthens the positive externality on old adults' private health expenditure and hence, young adults' incentive to save.[11]

1.4 Economic Growth, Health and Endogenous Fertility

With Proposition 1 we have established that

$$s_t = s(k_t), \quad s'(k_t) > 0. \tag{1.19}$$

and

$$n_t = n(k_t), \quad n'(k_t) < 0. \tag{1.20}$$

---

[11] When this complementarity is absent, the saving function will be $s_t = \frac{\beta(1+\delta)}{1+\beta(1+\delta)}(1-\tau)(1-a)Ak_t^a(1-q\hat{n})^{1-a}$ where $\hat{n} = \frac{\gamma / q}{1+\beta(1+\delta)+\gamma}$. In comparison with equation (1.17), s_t is still increasing with the level of capital per worker, but the increase will induce neither the decline of the fertility rate at higher levels of income nor the possibility of multiple equilibria. Instead, the dynamics of capital accumulation will resemble the dynamics of canonical OLG model. Therefore, as we shall see later, the complementarity between public and private health expenditures is pertinent for the type of demographic transition that will be analysed in Section 1.4.

Now, let us use the financial market equilibrium, $K_{t+1} = s_t N_t$, together with the growth rate of the population, $N_{t+1} = n_t N_t$, to get

$$k_{t+1} = \frac{s_t}{n_t} = \frac{s(k_t)}{n(k_t)}. \tag{1.21}$$

Substituting (1.17), (1.19) and (1.20) in (1.21) we get

$$k_{t+1} = \frac{\beta[1+\delta Z\left(\tau(1-a)Ak_t^a(1-qn(k_t))^{1-a}\right)]}{1+\beta[1+\delta Z\left(\tau(1-a)Ak_t^a(1-qn(k_t))^{1-a}\right)]} \frac{\eta k_t^a(1-qn(k_t))^{1-a}}{n(k_t)} = \psi(k_t), \tag{1.22}$$

where $\eta = A(1-a)(1-\tau)$. Equation (1.22) describes the dynamics of capital accumulation. We can use this to derive the economy's long-run equilibrium and to trace its transitional dynamics towards it. Formally, we analyse these issues in

Proposition 2. *For $k_0 > 0$, the economy will asymptotically converge to at least one long-run equilibrium $\hat{k}$ such that $\hat{k} = \psi(\hat{k})$ and $0 < \psi'(\hat{k}) < 1$. In the transition towards this steady-state equilibrium, the economy will grow at a positive, but declining, rate over time as long as $k_0 < \hat{k}$.*

Proof. See Appendix A. □ .

The transition graph, manifested in (1.22), rises monotonically because saving is increasing and fertility is decreasing in the stock of capital per worker. To understand the importance of this outcome in this particular setting, we re-emphasize the fact that, despite public health investment being complementary to private health expenditures, the economy will not admit periodic equilibria (endogenous cycles). The possibility of endogenous cycles may not emerge in our case. Endogenous cycles require $\psi'(k_t) < 0$ (see Figure 1.2); whereas in our model, $0 < \psi'(k_t) < 1 \;\; \forall \;\; k_t > 0$. If anything, this complementarity actually enhances the monotonicity of capital dynamics in our model because it increases the marginal propensity to save as the economy develops. For this reason, and despite the complex nature of equation (1.22), the model's dynamics may be qualitatively similar to those of the canonical OLG model (see Figures 1.3 and 1.4). As we explained earlier, this stark contrast to the previously established result of Bhattacharya and Qiao (2007) rests on our assumption that the old, rather than the young, are actually those who devote resources towards health improvements that enhance their overall quality of life.

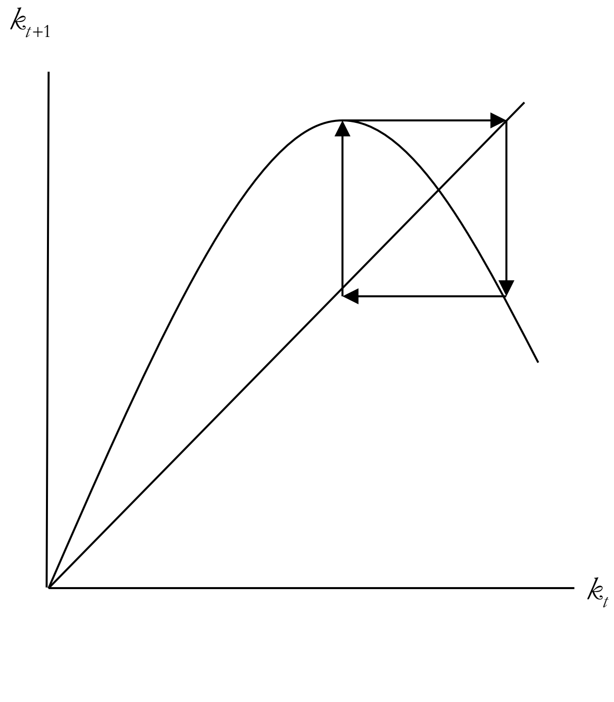

Figure 1.2

Endogenous cycles

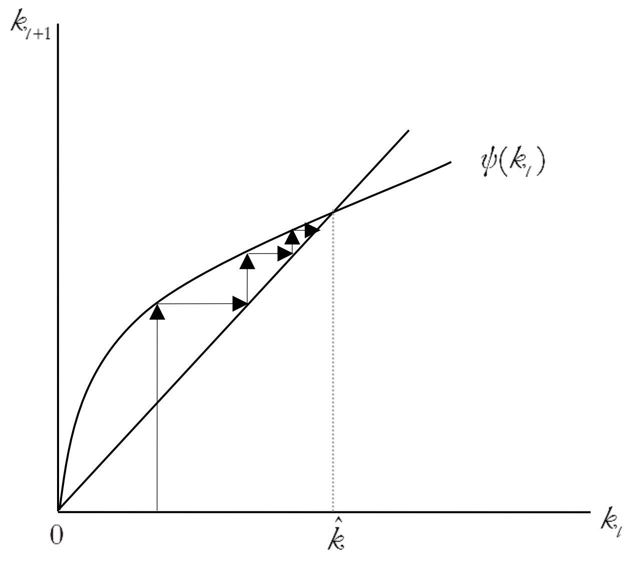

Figure 1.3

The dynamics of capital accumulation (unique steady-state).

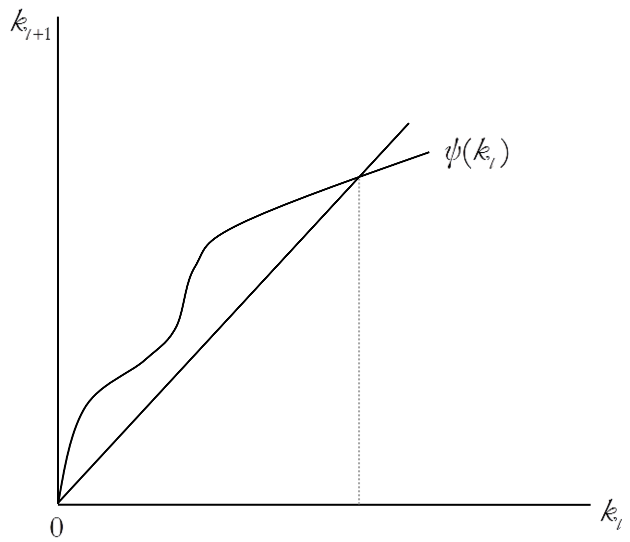

Figure 1.4

A unique steady-state (with points of inflexion on the transition graph).

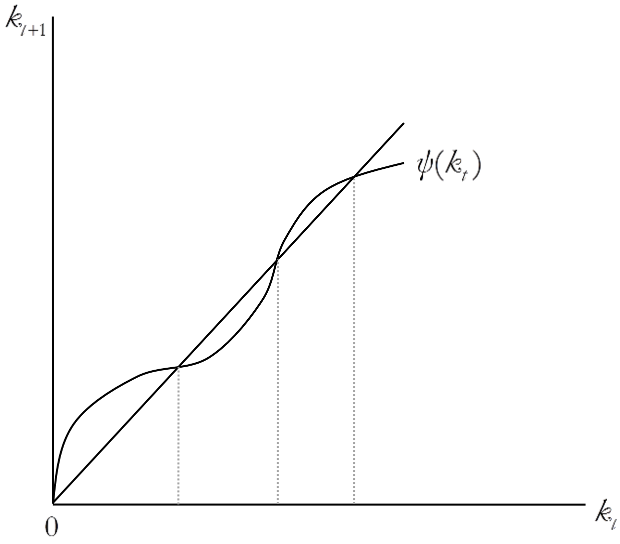

Figure 1.5

The possibility of multiple equilibria.

In our model, however, it is also possible that the dynamics in (1.22) admit multiple, path-dependent, steady state equilibria – as illustrated in Figure 1.5. The reason for this outcome is the bi-directional effects between saving, fertility, and the capital stock. On the one hand, higher saving and lower fertility increase the rate of capital formation; on the other hand, the higher capital stock increases the marginal propensity to save and reduces the fertility rate due to the complementary effect of public health investment on its private counterpart. Correspondingly, these bi-directional effects (which, by the way, do not permeate the canonical OLG model with a unitary elasticity of intertemporal substitution) could generate points of inflexion on the transition graph that may be responsible for the multiplicity of equilibria. Note that, in this case, at least one of these interior equilibria will be unstable. For example, in the case where three interior equilibria exist (see Figure 1.5) the middle one will be unstable. Effectively, it will emerge as an endogenous threshold that determines whether (depending on initial conditions) the economy will converge either to the low- or the high-income equilibrium in the long-run.

Unfortunately, the complexity of the dynamics in (1.22), coupled with the implicit nature of the functions $Z(\cdot)$ and (unavoidably) $n(\cdot)$, do not allow us to derive analytical conditions under which a situation similar to the one depicted in Figure 1.5 may emerge. The only outcomes that can be proven analytically is that the equilibrium at the origin is unstable and that at least one stable steady-state equilibrium exists (if multiple equilibria exist, this is the highest one in value).

Nevertheless, the main point of our analysis is not the possibility of path-dependent equilibria. Instead, our purpose is to use this framework so as to provide a novel explanation behind the fertility decline observed in economies that reach more advanced stages of their development process. This is formally shown in

Proposition 3. *Suppose that $k_0 < \hat{k}$ and that there is no $\tilde{k} \in (k_0, \hat{k})$ such that $\tilde{k} = \psi(\tilde{k})$. Then for $t = 0,1,2,...$ it is $n_0 > n_1 > n_2 > \cdots$. This demographic transition is solely associated with the complementarity between public and private health expenditures.*

Proof. It follows as a corollary of the results established in Propositions 1 and 2. Note that if $\varepsilon_{t+1} = 1\ \forall t$, i.e., if public health spending is not complementary to its private counterpart, then $n_t = \frac{\gamma / q}{1+\beta(1+\delta)+\gamma} = \hat{n} \; \forall t$ and demographic change does not occur. □ .

Our analysis shares some similarities with existing theories that reproduce fertility declines in the process of economic development. However, it also has important differences in comparison to them. The similar aspect is that we attribute the decline in the fertility rate to improvements in the health status of reproductive agents. The difference emerges from the fact that we exemplify *the importance of the interplay between public and private health spending* – an idea that has eluded the attention of most existing theories. Hence, the current set-up improves our understanding on the underlying forces behind some striking facts of demographic transition.

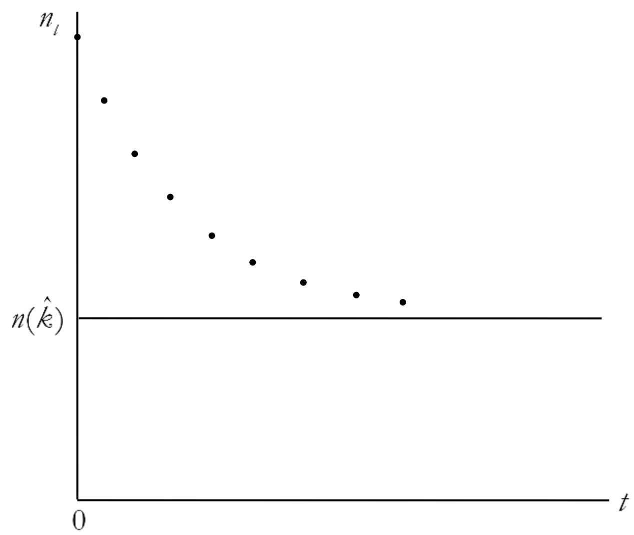

Figure 1.6

Demographic transition

1.5 Alternative Approaches

1.5.1 Private Health Spending During Youth

Consider the model of Section 1.2, with the only difference being that the young adults are those who devote resources with the purpose of forming their health capital. Thus, equations (1.2), (1.3), and (1.4) change to

$$c_t = (1-\tau)(1-qn_t)\omega_t - s_t - x_t, \tag{1.23}$$

$$c_{t+1} = r_{t+1}s_t, \tag{1.24}$$

$$h_{t+1} = Hx_t^{\delta\varepsilon_{t+1}}, \tag{1.25}$$

respectively.

It is straightforward to establish that the solution of the model leads to the following equilibria for fertility, saving, and health spending:

$$n_t = \frac{\gamma / q}{1+\beta[1+\delta Z\left(\tau(1-a)Ak_t^a(1-qn_t)^{1-a}\right)]+\gamma}, \tag{1.26}$$

$$s_t = \frac{\beta}{1+\beta[1+\delta Z\left(\tau(1-a)Ak_t^a(1-qn_t)^{1-a}\right)]+\gamma}(1-\tau)\omega_t, \tag{1.27}$$

$$x_t = \frac{\beta\delta Z\left(\tau(1-a)Ak_t^a(1-qn_t)^{1-a}\right)}{1+\beta[1+\delta Z\left(\tau(1-a)Ak_t^a(1-qn_t)^{1-a}\right)]+\gamma}(1-\tau)\omega_t. \tag{1.28}$$

As it is evident, the solution for fertility in (1.26) is identical to the corresponding expression in Section 1.2 (i.e., equation 1.18). Therefore, the model's result concerning the negative effect of k_t on fertility and the fact that this effect exists only due to the interaction between public and private health spending remain intact. The intuition however is different. Once more, an increase in k_t will allow the provision of more publicly provided health services for a given tax rate. Individuals respond by increasing their health expenditure at the expense of their current consumption. Now, however, agents can retain a more uniform pattern of consumption over time, by reducing both their saving rate and the number of children they decide to give birth to. The latter effect is the outcome of their decision to provide more labour and, thus, increase the available resources during young adulthood.

A notable difference in this scenario is the fact that, as in Bhattacharya and Qiao (2007), the marginal propensity to save is decreasing in the stock of physical capital. Note that this effect will actually be reinforced by the increase in labour supply, due to lower fertility, as the capital stock increases.

1.5.2 An Alternative Form for the Health Generation Function

Once more, let us consider the model of Section 1.2. Now, however, we consider a different functional form for health status. In particular, we replace (1.4) by

$$h_{t+1} = (1-\delta)p_{t+1} + f(\delta p_{t+1})x_{t+1}, \tag{1.29}$$

where $\delta \in [0,1)$, $f'(\cdot) > 0$ and $f(0) = \underline{f} > 0$. The variable p_{t+1} is still given by (1.6) as it captures the benefit from public health services. The idea behind the health generation function in (1.29) is that the benefit from public health spending has two components. On the one hand, some public health provision is beneficial to health status irrespective of whether individuals contribute resources towards their health improvements or not. On the other hand, part of the economy's public services support the effectiveness of private health expenditure by increasing the productivity of the health sector – an effect that is captured through the presence of the increasing function $f(\cdot)$. Naturally, $\delta \in [0,1)$ provides a flexible parameterisation of the relative strength of these two effects.

Another change in comparison to the model of Section 1.2, is related to the production technology which now takes the form of

$$Y_t = K_t^a (A_t L_t)^{1-a}. \tag{1.30}$$

The variable A_t indicates some type of labour-augmenting technological progress. Following Frankel (1962) and Romer (1986), we assume that this is related to the average capital-labour ratio according to a learning-by-doing externality. That is

$$A_t = \Psi \frac{K_t}{L_t}, \quad \Psi > 0. \tag{1.31}$$

The reason why we opt for this specification here is because, despite the fact that we assume logarithmic preferences, the presence of the term $(1-\delta)p_{t+1}$ in (1.29) means that optimal decisions will depend on the interest rate. To maintain analytical tractability we use a production technology, commonly employed in the endogenous growth literature, whose main property is that the marginal product of capital is not inversely related to its stock. In this case, the marginal product of capital is constant at $r_t = a\Psi^{1-a} = \hat{r}$. Furthermore, given (1.9), it is straightforward to establish that the equilibrium wage is

$$\omega_t = \frac{(1-a)\Psi^{1-a}k_t}{1-qn_t}. \tag{1.32}$$

Solving this model and using (1.6), (1.9), (1.15) and (1.32) in the solutions, it is straightforward to establish the following results for saving, fertility, and private health spending:

$$s_t = \frac{\left[2\beta(1-\tau) - \dfrac{\hat{r}^{-1}(1-\delta)\tau}{f\left(\delta\tau(1-a)\Psi^{1-a}k_t\right)}\right]}{1+2\beta}(1-a)\Psi^{1-a}k_t, \tag{1.33}$$

$$n_t = \frac{1}{q}\times\frac{\dfrac{\gamma}{1+2\beta+\gamma} + \dfrac{\gamma[\hat{r}(1+2\beta+\gamma)(1-\tau)]^{-1}(1-\delta)\tau}{f\left(\delta\tau(1-a)\Psi^{1-a}k_t\right)}}{1+\dfrac{\gamma[\hat{r}(1+2\beta+\gamma)(1-\tau)]^{-1}(1-\delta)\tau}{f\left(\delta\tau(1-a)\Psi^{1-a}k_t\right)}} = n(k_t), \tag{1.34}$$

$$x_{t+1} = \frac{\left[\beta\hat{r}(1-\tau) - \dfrac{(1+\beta)(1-\delta)\tau}{f\left(\delta\tau(1-a)\Psi^{1-a}k_t\right)}\right]}{1+2\beta}(1-a)\Psi^{1-a}k_t. \tag{1.35}$$

In these results, we assume that $f(0) = \underline{f}$ is sufficiently high to guarantee a strictly positive solution for health spending. Given this, we can use (1.34) to verify that $n'(k_t) < 0$ if and only if $\delta > 0$. In the limiting scenario where $\delta = 0$, we have $n'(k_t) = 0$ as well. In other words, the decline of the fertility rate as the economy grows depends crucially on the idea that (part of) public health expenditures enhance the productivity of the health sector and, consequently, boost the efficiency of private health spending. This is exactly the result we established in our original model (see Proposition 3).

1.6 Conclusion

We have established a new mechanism in explaining a salient feature of demographic transition. In particular, the decline of fertility during the process of growth is attributed to the complementary effect of public health investment on private health expenditures – an effect that has been introduced in the manner of the seminal analysis of Bhattacharya and Qiao (2007) (see equations (1.4)-(1.7)). As the economy grows, the public capital available for health services increases and improves the effectiveness of private health expenditures. Old adults will find that it is optimal for them to increase the resources they devote towards the improvement of their health status. In order to ensure the availability of these resources, they reduce their expenditures when young in order to save more. Given that childrearing costs are among these expenditures, reproductive young adults will reduce the number of children they give birth to. Consequently, the fertility rate declines.

Furthermore, our model shows that as long as some health expenses are incurred by the old, the complementary impact of public health spending on its private counterpart need not be a source of economic instability. Actually, it reinforces the monotonicity of the economy's dynamics by motivating individuals to save a larger part of their labour income – thus, supporting capital accumulation.

Chapter 2

Growth and Demographic Change: Do Environmental Factors Matter?[12]

2.1 Introduction

The question on whether demographic changes are inherently linked to environmental issues is by no means a new one. In the past, many analysts have presented evidence and argued that population growth contributes to the decay of the natural environment as it has been associated with such problems as deforestation; air and water pollution; global warming; increased waste etc. (see Cropper and Griffiths 1994; Dietz and Rosa 1997).

The scatterplot in Figure 2.1 provides a snapshot of cross-country differences on the link between environmental quality and fertility rates in

[12] Joint work with Dr. Dimitrios Varvarigos. The details of the first version of this chapter are: Varvarigos, D., and Zakaria, I.Z. 2011. "Growth and Demographic Change: Do Environmental Factors Matter?", Department of Economics of University of Leicester working paper no. 11/46.

2012.[13] The data includes low-, middle-, and high-income countries for which there is an Environmental Performance Index (EPI) available. The EPI constitutes a broad measure of environmental quality, including "performance indicators tracked across policy categories that cover both environmental public health and ecosystem vitality."[14] The scatterplot seems to corroborate with the aforementioned arguments as it suggests a negative relation between environmental quality and fertility rates.

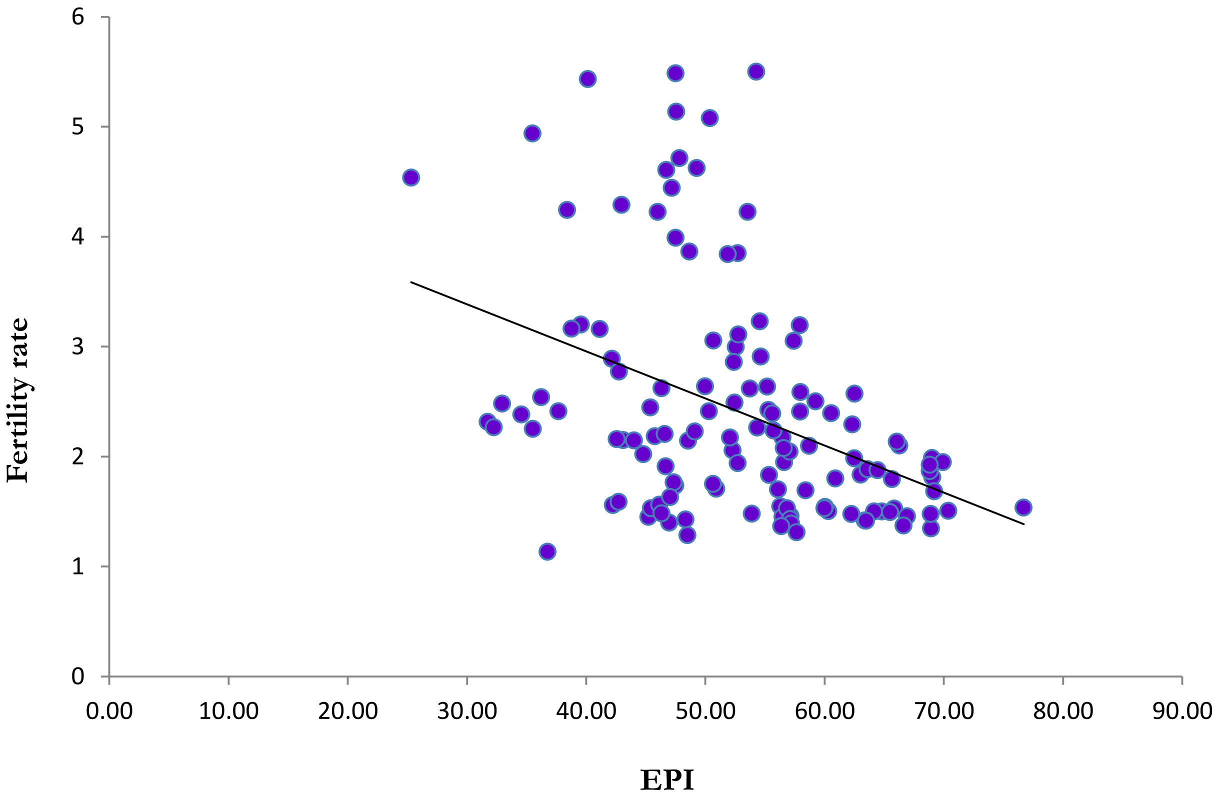

Figure 2.1

EPI and fertility rate

[13] Data on fertility are taken from the World Bank (http://data.worldbank.org/indicator/SP.DYN.TFRT.IN).

[14] Quote taken from http://epi.yale.edu/. Note that higher scores for the EPI correspond to better environmental performance.

In the next figure (Figure 2.2), we utilise the component of the EPI that captures the negative health repercussions of pollution. Now the plot depicts an ever more pronounced negative link between environmental quality and fertility. In other words, there seems to be a stronger relation between fertility rates and the part of the natural environment that promotes the health characteristics of the population.

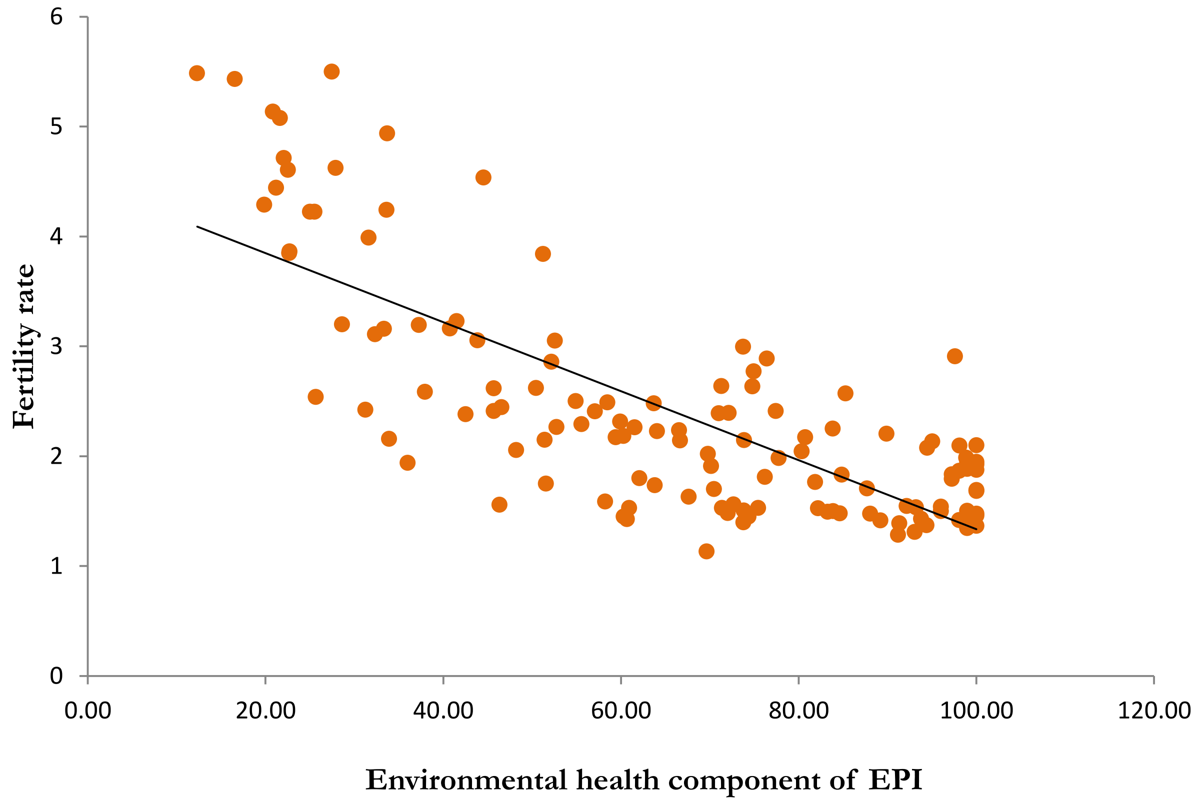

Figure 2.2

Environmental health component of EPI and fertility rate

This is perhaps an indication that the public health repercussions of environmental degradation may be important on the determination of a link between population growth and environmental quality. These repercussions are of course well-documented. Water pollution is a major cause of gastrointestinal conditions (such as diarrhoea and cholera) and it has been linked to neurological disorders.[15] Air pollution is responsible for chronic respiratory diseases (such as asthma and bronchitis) and for various forms of cancer. The soil can be polluted by various carcinogenic chemicals which can affect humans both directly (through contact) and indirectly (through the food chain).[16] General waste, in addition to being a major contributor to the previously mentioned forms of pollution, attracts insects and rodents that can be carriers of disease organisms – some of them, such as malaria; yellow fever; plague; E-coli; and leptospirosis, being potentially fatal.[17] Given examples such as these, it is perhaps little surprise that according to Pimentel *et al.* (1998), the direct and indirect effects of environmental degradation can account for almost 40% of deaths worldwide.

There can be little doubt on the fact that changes in the size of the population can have significant effects in the quality of the natural environment. Nevertheless, one can cast some doubt on the conventional wisdom that views the causality underlying this relation as working solely through one direction – from population growth to environmental degradation. There is no *a priori* reason to preclude the possibility that changes in environmental quality may actually cause variations in the rate of population growth. Indeed, in her review of the literature on demographic change and the environment, Pebley (1998) argues that "environmental change may also have important effects on demographic outcomes" (Pebley 1998, p. 384). Our purpose in this paper is to

[15] See the Environmental Protection Agency's webpage at water.epa.gov/drink/contaminants/#List.
[16] See Pimentel *et al.* (1998).
[17] See Pinnock (1998).

consider the negative health effects of pollution in order to develop an economic theory that illustrates how and why environmental factors may actually cause changes in the rate of population growth. By doing so, we provide a formal argument in support of this Pebley's (1998) conjecture. Particularly, the strong evidence on the negative relation between population growth and indicators of environmental quality may also embed a causal link *from* environmental change *to* population change.

Existing theories that have sought to explain the joint determination of economic growth, fertility, longevity and mortality have absconded from issues pertaining to environmental quality (e.g., Blackburn and Cipriani 2002; Lagerlöf 2003). Other theoretical analyses have incorporated environmental quality in models of growth and (endogenous) life expectancy but have neglected the issue of fertility choices (e.g., Pautrel 2009; Varvarigos 2010; Jouvet *et al.* 2010). A recent strand of literature that examine the interactions between pollution and optimal fertility choices, employ models where mortality and life expectancy are exogenous (Lehmijoki and Palokangas 2010; Bretschger 2013; Constant *et al.* 2013). To the best of our knowledge, our study is the first to explicitly consider production-induced environmental degradation within a growth model where *both* fertility *and* life expectancy are endogenous, thus suggesting that some well-documented demographic facts, as well as changes to economic outcomes such as economic growth, may be (partially) attributed to factors associated with pollutant emissions. While the analysis of Galor and Moav (2005) has tackled these demographic issues and their relation to the environment, there are significant differences in their set-up when compared to ours. Firstly, the negative health repercussions of environmental strain in their model derive from higher population density. In our model, the environmental strain derives from the emission of pollutants that are by-products of economic activity. Moreover, the engine of growth in their model is technological progress that is increasing in the population size, whereas in our model economic growth is driven by capital accumulation. This is also an important difference because the optimal saving behaviour that fuels the accumulation of physical capital is central to the emergence of our main results on the link between demography and the environment.

Our analysis is theoretical in nature. We build a discrete-time overlapping generations model where reproductive households face the probability of passing away early during their maturity and intermediate good producers/entrepreneurs face a tax on the pollutants emitted during their productive activity. Existing studies carried by Requate and Unold (2003), Requate (2005), and the OECD (2007), support the idea that environmentally related taxes encourage changes in production processes that are based on cleaner production techniques and environmental R&D. In a different empirical investigation, Komen *et al.* (1997) find that higher GDP growth is positively associated with the promotion of new technologies that are directed towards environmental improvements. Both these facts represent mechanisms that are central to our model's equilibrium outcomes. We show that the process of economic growth will generate sufficient resources so that entrepreneurs who face an emission tax may opt for a less polluting production method. When this happens, the reduction in emissions per unit of output causes an increase in longevity. Consequently, households will find optimal to increase their saving in order to carry more resources towards future consumption. In addition to a higher saving rate, the latter effect is also associated with a reduction in fertility. This is because households will try to smooth their consumption profile by providing more labour when young, with the purpose of counteracting the adverse effect of a higher saving rate on their current consumption. This can only be achieved by a reduction in the time/effort they devote towards child rearing; hence both the fertility rate and the growth rate of the population fall.

All in all, our analysis proposes a positive relation between pollution and fertility rates, driven by the health improvements associated with better environmental conditions. This idea provides an alternative explanation for the strong empirical evidence on the positive relation between population growth and environmental degradation. The main causal effect in our framework works from environmental factors to changes in the population size of the economy. On the outset, these results may seem to be at odds with the circumstances surrounding the striking demographic changes that occurred around the mid-19th century onward – particularly, the observation that, during that period, changes such as reduced mortality and lower fertility occurred at a time of growing pollution during the process of industrialisation. Our arguments against such views are the following. Firstly, the demographic transition that occurred in the 19th century is indeed a major but by no means the only episode of changes in fertility and life expectancy in history. At least in terms of descriptive data, such trends have been observed more recently and there is nothing to preclude the possibility that the underlying forces behind them may be different.

Secondly, even if we are inclined to focus solely on that early period, the adoption of a broader concept of pollution would provide greater support to our result. For example, economic activity adds to general waste which can be also considered as a major contributor to environmental degradation. Bearing this in mind, we can allude to historical analyses, such as those by Szreter (1994, 2004a, 2004b) and Szreter and Mooney (1998) who strongly argue that, as a response to the disease outbreaks and deteriorating health conditions that resulted from urbanisation and pollution, the authorities in the United Kingdom and Sweden implemented policies of such environmental improvements as urban sanitation, improvements in food quality and the availability of cleaner water. These policies lead to a significant improvement in health conditions and life expectancy during the late part of the 19th century.[18]

The structure of our analysis is as follows. Section 2.2 describes the economy's main characteristics. Section 2.3 analyses the model's equilibrium and explains the mechanism through which the emission rate falls endogenously in the process of economic development. In Section 2.4 we present the main results concerning the joint determination of pollution per unit of output, economic growth, fertility and longevity. In Section 2.5 we conclude.

[18] Based on reconstructed demographic data from that period, Szreter (2004b) questions the whole idea that the increase in incomes associated with industrialisation was responsible for any health improvements at all. In fact, he points out that "overall health failed to improve between 1811 and 1871, despite enhanced purchasing power" (p. 80) and that "the principal reason for the failure of the national average life expectancy to register any further gains between 1811 and 1871 was due mainly to deteriorating health conditions in Britain's industrializing towns and cities." (p. 80)

2.2 The Economy

We construct an overlapping generations model where time takes the form of discrete periods that are indexed by $t = 0,1,2,\ldots$. In addition to a government, every period there are two groups of agents active in the economy. Henceforth, we shall be referring to these distinct groups as *households* or *workers* and *entrepreneurs* or *intermediate good producers*.

At the beginning of each period, a unit mass of entrepreneurs comes into existence. Each of them lives for only one period and enjoys utility by consuming units of the economy's final good.[19] She is endowed with a technology that allows her to combine labour units from households, denoted L_{it}, and capital from financial intermediaries, denoted K_{it}, to produce a specific variety i of an intermediate product according to

$$y_{it} = BK_{it}^{\beta}(A_t L_{it})^{1-\beta}, \tag{2.1}$$

where $B > 0$ and $0 < \beta < 1$. The variable A_t indicates some type of labour-augmenting technological progress for which we assume that it is related to the average capital per worker ratio, according to a learning-by-doing externality (Romer 1986).[20] That is

$$A_t = \Theta \int_0^1 \frac{K_{it}}{N_t} di, \quad \Theta > 0, \tag{2.2}$$

where N_t is the total population of young households/workers. The entrepreneur sells her product to perfectly competitive firms who combine all

[19] Thus, profit maximisation corresponds to utility maximisation.

[20] This assumption allows the existence of equilibrium with positive growth in the long-run. This is because it generates (aggregate) constant returns to physical capital, as the production can be reduced to a form resembling the 'AK' variety of technologies.

the available varieties of intermediate products to produce units of the economy's final consumption good according to

$$Y_t = \left(\int_0^1 y_{it}^{\frac{\sigma-1}{\sigma}} di \right)^{\frac{\sigma}{\sigma-1}}, \tag{2.3}$$

where $\sigma > 1$ is the elasticity of substitution between different varieties of intermediate inputs. We shall assume that the final good is the numéraire. The price of each intermediate good is denoted P_{it}, whereas the average price level is $P_t = \left(\int_0^1 P_{it}^{1-\sigma} di \right)^{1/(1-\sigma)}$.

As a result of her activity, each entrepreneur is responsible for the emission of $\mu_{it} > 0$ units of pollution per unit of intermediate good produced. Therefore, the total pollutants emitted by each entrepreneur are $\mu_{it} y_{it}$. We assume that the government follows an environmental policy characterised by an *ad valorem* emission tax $\tau > 0$ imposed to each entrepreneur. Net revenue is therefore $\frac{P_{it}}{P_t}(1-\tau\mu_{it}) y_{it}$, where $\tau\mu_{it} < 1$ is assumed to be satisfied.

Denoting the marginal cost of production by m_t, we can write the entrepreneur's profits as

$$\pi_{it} = \left[\frac{P_{it}}{P_t}(1-\tau\mu_{it}) - m_t \right] y_{it}. \tag{2.4}$$

Entrepreneurs have the choice of reducing their emissions, and therefore their tax obligation, by incurring a fixed cost, denoted $\varepsilon > 0$, for a clean-up operation that decreases the emission rate of their technology. Without loss of generality, we shall assume that this fixed cost is measured in units of entrepreneurial effort, i.e., it is not pecuniary. The entrepreneurial technology will either emit $\mu_{it} = \overline{\mu}$ pollutants per unit of production if no such fixed cost is incurred, or $\mu_{it} = \underline{\mu}$ units of pollution per unit of production if the entrepreneur decides to incur this effort cost. Naturally, we assume that $\overline{\mu} > \underline{\mu}$.[21] Thus, an entrepreneur's utility is given by[22]

$$u_{it}^{\text{entrepreneur}} = \begin{cases} \pi_{it}, \text{ if } \mu_{it} = \overline{\mu} \\ \\ \pi_{it} - \varepsilon, \text{ if } \mu_{it} = \underline{\mu} \end{cases}. \tag{2.5}$$

The economy is also inhabited by reproductive households who face a potential lifetime of three periods and belong to overlapping generations. The three periods of a household's lifetime are *childhood*, *young adulthood* and *old adulthood*. Decisions are made only after agents reach their adulthood. At the beginning of their young adulthood, they are endowed with a unit of time which they decide to allocate between labour and child rearing. For each unit of labour supplied to entrepreneurial firms, households receive the competitive salary w_t. Rearing each child caries a time/effort cost of $q > 0$. Denoting the total number of children raised in each household by n_t, the previous assumptions imply that household members will supply $1 - qn_t$ units of labour.

Each young household also receives a transfer, H_t^{young}, from the government – a transfer that is proportional to labour income according to

[21] We do not necessarily need to associate this scenario with a technology choice. We can equivalently interpret this choice as one where, by incurring the fixed cost, entrepreneurs can eliminate a fraction $\zeta \in (0,1)$ of their total emissions. In this case, $\underline{\mu} = (1-\zeta)\overline{\mu}$.

[22] Recall that the entrepreneur is risk neutral. Nothing would change qualitatively had we assumed that an entrepreneur's utility is non-linear.

$H_t^{young} = h_t^{young} w_t (1 - qn_t)$ ($h_t^{young} > 0$). Households decide how much to consume and how much to save for retirement. Nature does not bestow to them a labour endowment when old. Therefore, they do not have any alternative source of income from which they could finance their future consumption needs, other than saving. With the purpose of introducing endogenous lifetime, we follow Chakraborty (2004) by assuming that households may die prematurely and not reach their old adulthood. In particular, they will survive to old adulthood with probability $\psi_t \in [0,1)$. We also assume that retirement income (i.e., the income accrued from saving) is augmented by a proportional subsidy H_{t+1}^{old}. Denoting saving by s_t and the gross rate of interest on deposits by r_{t+1}, we have $H_{t+1}^{old} = h_{t+1}^{old} r_{t+1} s_t$ ($h_{t+1}^{old} > 0$).[23] Consequently, a household's lifetime utility is given by[24]

$$U^t = \ln c_t^{t-1} + \gamma \ln n_t + \psi_t \ln c_{t+1}^{t-1}, \quad \gamma > 0, \tag{2.6}$$

where c_t^{t-1} denotes consumption during young adulthood and c_{t+1}^{t-1} denotes consumption during old adulthood. Notice that we follow the standard approach of assuming that households have preferences over the number of children they raise.[25] Note that if parents cared equally about the number of offspring when old, i.e., if lifetime utility was written $U^t = \ln c_t^{t-1} + \gamma \ln n_t + \psi_t (\ln c_{t+1}^{t-1} + \gamma \ln n_t)$, then the equality of income and

[23] The fact that transfers are assumed to be proportional to income may appear at odds with conventional wisdom. Nevertheless, the reader may recall that the members of each age cohort are identical in terms of income. Consequently, there are no issues or implications for income inequality in our model. The role of these subsidies is not to redistribute income *per se*. As it will transpire later, their actual role is to eradicate the distortive effect of emission taxes on labour and capital income (see Section 2.3.1).

[24] In the utility function, a superscript indicates the period where the agent is born while the subscript indicates the period in which the actual activity takes place.

[25] See Galor and Weil (1996); Palivos (2001); and Liao (2011) among others.

substitution effects would imply that life expectancy would not impinge on fertility decisions. This is just an artefact of the presence of logarithmic preferences. Therefore, to eliminate this possibility while retaining the analytical convenience associated with logarithmic preferences, we use the specification in (2.6). Note, however, that we could generalise this scenario without any loss in the qualitative results of our model, by assuming that the utility benefit from rearing children is weighted differently at different stages of a parent's lifetime. In terms of our lifetime utility, our equilibrium results would be qualitatively identical had we assumed that $U^t = \ln c_t^{t-1} + \gamma \ln n_t + \psi_t (\ln c_{t+1}^{t-1} + \zeta \ln n_t)$, as long as $0 < \zeta < \gamma$. To save on notation, we use the specification in (2.6) which, as we will show later, results in the same qualitative implications.[26]

Earlier we indicated that the government imposes a tax $\tau \in (0, 1/\mu_{it})$ on total emissions by each entrepreneur. With a unit mass of entrepreneurs, this action results in total revenues of $\tau \int_0^1 \frac{P_{it}}{P_t} \mu_{it} y_{it} di$. The government uses its revenues to finance the income transfer to all young households, $H_t^{young} N_t = h_t^{young} w_t (1 - qn_t) N_t$, the subsidy to the retirement income of all *surviving* old households, $H_t^{old} \psi_{t-1} N_{t-1} = h_t^{old} r_t s_{t-1} \psi_{t-1} N_{t-1}$, and government

[26] The distinction between households and entrepreneurs follows a similar assumption in Chakraborty and Lahiri (2007). The reason why we make a distinction between them, rather than assuming that profits from the entrepreneurial activity accrue to old agents, is solely related to analytical tractability and has no bearing to the main implications of our model. We could have assumed that the old own the intermediate goods firms and that the profits accrue to the old generation, without any change in the qualitative nature of our results. Nevertheless, the cost (in terms of intractability) of such an approach would be very high given that the prevailing emission rate impinges on life expectancy, fertility choices and saving decisions. Under such circumstances, the optimal choice of the emission technology would become very complicated technically, without adding any important implication to our main message.

consumption which is denoted g_t. The government has to abide by a balanced budget rule. Hence,

$$\tau\int_0^1 \frac{P_{it}}{P_t}\mu_{it}y_{it}di = h_t^{young}w_t(1-qn_t)N_t + h_t^{old}r_t s_{t-1}\psi_{t-1}N_{t-1} + g_t. \tag{2.7}$$

As we noted earlier, the presence of varying longevity is crucial for the interactions between saving and fertility choices. Following others (Chakraborty 2004; Varvarigos 2010) we assume that a household's lifetime is endogenous. Particularly, we assume that ψ_t is given by

$$\psi_t = \Psi(x_t), \tag{2.8}$$

where x_t is a variable that describes the health profile of the household.[27] The function in (2.8) satisfies $\Psi' > 0$, $\Psi'' < 0$, $\Psi(\infty) \in (0,1)$, $\infty > \Psi'(0) > 0$ and $\Psi'(\infty) = 0$.

Existing empirical evidence shows that as economies develop and people become more educated, they are more prone to adopt a lifestyle that contributes to an improvement of their overall health status (e.g. Smith 1999). A further argument in favour of GDP as a promoting factor to health is that it increases the pool of tax revenues that the government may use to provide essential public health services. Another crucial factor that seems to have a profound effect on health is environmental quality. For instance, various by-products of economic activity, such as toxins; smoke; chemicals; and general waste, erode the quality of air as well as the quality of natural resources such as water, soil etc. Consequently, they result in significant adverse effects on the

[27] Notice that the expected lifespan of a household is $2+\psi_t$. For this reason, we will be making use of such terms as 'life expectancy' and 'longevity' interchangeably.

health status of people who are exposed to such environments. Various empirical studies appear to confirm this conjecture (e.g. Murray and Lopez 1996; Pimentel *et al.* 1998; Brunekreef and Holgate 2002).

We try to capture the aforementioned ideas by assuming that the variable x_t is related to average income, denoted $\bar{Y}_t$, and pollution, denoted M_t, according to $x_t = X(\bar{Y}_t, M_t)$. In general, this function satisfies $X_{\bar{Y}_t} > 0$ and $X_{M_t} < 0$, but for analytical purposes, we shall be focusing our attention to the specific functional form

$$x_t = \frac{\bar{Y}_t}{M_t}. \tag{2.9}$$

Other analyses that introduce the negative effect of pollution on longevity are those of Varvarigos (2010) and Jouvet *et al.* (2010). There are two reasons why we use this specific functional form. Firstly, it displays the desirable qualitative properties (in terms of health effects) while being very tractable in technical terms. In addition, it allows us to abscond from the health effects of population density and capital accumulation, which have already been examined in analyses such as those by Galor and Moav (2005) and Palivos and Varvarigos (2010), and focus on the impact of the emissions' generator on the population's health status – the actual issue of interest in this analysis. It should also be noted that this exact functional form has been employed extensively in the literature that examines the interactions between economic growth and environmental quality (e.g., Pautrel 2009; Clemens and Pittel 2011).

Recall that, in our setting, pollution is a by-product of entrepreneurial activities in the production of intermediate goods. To maintain analytical tractability without altering the strength of the mechanisms that govern our subsequent results, we assume that pollution is generated by

$$M_t = \int_0^1 \mu_{it} y_{it} di . \tag{2.10}$$

As it is evident from Equation (2.10), we follow others (e.g., Jones and Manuelli 2001) in focusing our attention to the flow of pollution. This is because we want to focus on the change of environmental technology along the process of economic development. Thus, we retain physical capital as the only stock variable in our model. If we consider the stock of pollution, the transitional dynamics will be severely complicated without adding any additional insight into the main mechanisms that permeate our analysis. For an explicit analysis of dynamics in a similar model with two stock variables (pollution and physical capital) but without fertility choices, see Varvarigos (2013). There it becomes evident that the manner through which the level of development impinges on the environmental technology choice is similar to our analysis.

The preceding discussion completes the description of our theoretical framework. In the following section, we derive and characterise the equilibrium of our model.

2.3 Equilibrium

We shall begin the derivation of the model's equilibrium by solving the profit maximisation problem of an intermediate good producer. As we indicated in Section 2.2, the entrepreneur's choice on the cleanliness of the technology she will employ is discrete; hence it can be separated from her other choices. For this reason, we shall solve the problem using two distinct stages. In the first stage, the entrepreneur chooses the technology she will implement by comparing her utility associated with either the low- or the high-emission technology. In the second stage, she chooses the amount of capital and labour she will employ, as well as the price of her product, for any technology described by μ_{it}.

First of all, we can use (2.3) to find that profit maximisation by the (perfectly competitive) producers of final goods will lead to a demand relation according to which the share of an intermediate product in total demand is an inverse function of its relative price.[28] That is

$$y_{it} = p_{it}^{-\sigma} Y_t, \tag{2.11}$$

where $p_{it} = \frac{P_{it}}{P_t}$ is the relative price of the intermediate product. Next, we substitute (2.11) in (2.4) and maximise with respect to the relative price p_{it} to get

$$p_{it} = \frac{\sigma}{(\sigma - 1)(1 - \tau\mu_{it})} m_t. \tag{2.12}$$

The result in (2.12) is the standard condition according to which the relative price is set as a mark up over the marginal cost of production m_t.

Concerning the choice of capital and labour employed in production, cost minimisation leads to[29]

$$w_t = m_t (1 - \beta) B K_{it}^{\beta} A_t^{1-\beta} L_{it}^{-\beta}, \tag{2.13}$$

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[28] See Appendix B for a formal derivation.

[29] The cost minimisation problem is $\min_{K_{it}, L_{it}} w_t L_{it} + R_t K_{it}$ subject to Equation (2.1). It is solved by using the Lagrangean $\Lambda_t = w_t L_{it} + R_t K_{it} + m_t \left[y_{it} - B K_{it}^{\beta} (A_t L_{it})^{1-\beta} \right]$.

and

$$R_t = m_t \beta B K_{it}^{\beta-1}(A_t L_{it})^{1-\beta}, \tag{2.14}$$

where R_t is the rental cost of capital while the marginal cost m_t is associated with the Lagrange multiplier of the cost minimisation problem.

From now on we will be focusing our attention to an equilibrium that is symmetric across entrepreneurs. That is, $P_{it} = P_t$, $K_{it} = K_t$, $L_{it} = L_t$, $\mu_{it} = \mu_t$ and $y_{it} = y_t$ for every i. For this reason, we drop the subscript i from the subsequent analysis. Naturally, a symmetric equilibrium implies that the relative price satisfies $p_{it} = p_t = 1$. We can substitute this result in (2.12) to derive

$$m_t = \frac{\sigma-1}{\sigma}(1-\tau\mu_t). \tag{2.15}$$

Substituting (2.15) in (2.13) and (2.14) yields

$$w_t = (1-\tau\mu_t)\frac{\sigma-1}{\sigma}(1-\beta)BK_t^{\beta}A_t^{1-\beta}L_t^{-\beta}, \tag{2.16}$$

and

$$R_t = (1-\tau\mu_t)\frac{\sigma-1}{\sigma}\beta B K_t^{\beta-1}(A_t L_t)^{1-\beta}, \tag{2.17}$$

respectively. By virtue of (2.3) and (2.1), the symmetric equilibrium implies that

$$Y_t = y_t = BK_t^{\beta}(A_t L_t)^{1-\beta}, \tag{2.18}$$

while (2.4) and (2.15) imply that each entrepreneur's variable profits are equal to

$$\pi_t = (1-\tau\mu_t)\frac{1}{\sigma}y_t. \tag{2.19}$$

We now turn our attention to the optimal decisions made by households. The budget constraints faced by households during the two periods of their adulthood are $c_t^{t-1} = (1 + h_t^{young})w_t(1 - qn_t) - s_t$ and $c_{t+1}^{t-1} = (1 + h_{t+1}^{old})r_{t+1}s_t$. Their objective is to choose c_t^{t-1}, n_t, s_t and c_{t+1}^{t-1} to maximise their lifetime utility in (2.6), taking ψ_t, w_t and r_{t+1} as given. It is straightforward to establish that the solutions to this problem are given by

$$s_t = \frac{\psi_t}{1 + \psi_t}(1 + h_t^{young})w_t(1 - qn_t), \tag{2.20}$$

and

$$n_t = \frac{\gamma}{q(1 + \gamma + \psi_t)}. \tag{2.21}$$

The intuition behind these results is straightforward. Equation (2.20) reveals that households will save a fraction of their total earnings (that is, their labour income augmented by the government subsidy). Their propensity to save is increasing in the variable that determines their life expectancy. In particular, a higher ψ_t increases the utility benefit of consuming when old; hence, it motivates agents to substitute future for current consumption. In Equation (2.21), we can see that the fertility rate is inversely related to ψ_t because, as the utility from consuming when old increases, households will optimally want to carry more resources towards saving. Nevertheless, they will also try to smooth their consumption profile. They can do this by working more during their young adulthood in order to increase their available resources – an action that, nevertheless, leaves them with less time available to rear children.[30]

Next, we can combine (2.8), (2.9) and (2.10) together with $y_t = \bar{Y}_t$ to get $\psi_t = \Psi(1/\mu_t)$, where $\Psi_{\mu_t} < 0$. Note that the functional form in (2.9) allows us to eliminate the *direct* effect of $y_t = \bar{Y}_t$ on ψ_t due to the counterbalancing effects of economic development and pollution. This is actually a welcome aspect because it permits us to focus on the demographic implications of different emission rates. In any case, later it will become clear that income still has a positive, albeit *indirect*, effect through the contribution of the growth process on the choice of a lower μ_t. Substituting $\psi_t = \Psi(1/\mu_t)$ in (2.21) yields

$$n_t = \frac{\gamma}{q[1+\gamma+\Psi(1/\mu_t)]} = n(\mu_t). \tag{2.22}$$

The result in Equation (2.22) allows us to derive

Proposition 1. *The optimal fertility rate is positively related to the amount of emissions per unit on output. That is* $n'(\mu_t) > 0$.

Proof. It is $n'(\mu_t) = \frac{\partial n_t}{\partial \Psi(1/\mu_t)} \Psi_{\mu_t}$. Since $\frac{\partial n_t}{\partial \Psi(1/\mu_t)} < 0$ and $\Psi_{\mu_t} < 0$, we get $n'(\mu_t) > 0$. $\square$.

[30] With regard to our previous comment on the way through which fertility choices are incorporated in a household's preferences, it can be easily established that a lifetime utility that takes the form $U^t = \ln c_t^{t-1} + \gamma \ln n_t + \psi_t (\ln c_{t+1}^{t-1} + \zeta \ln n_t)$ would generate an optimal fertility rate of $n_t = (\gamma + \zeta\psi_t)/q[1+\gamma+(1+\zeta)\psi_t]$. The qualitative effects of life expectancy on fertility would be identical to the ones in our current set-up, as long as $0 < \zeta < \gamma$. Effectively, we just require the 'substitution effect' associated with variations in ψ_t to dominate the corresponding 'income effect'.

In terms of intuition, a higher μ_t reduces longevity because of the adverse health effect from the emission of harmful pollutants. As this reduces the relative importance attached to old age consumption, the equilibrium can only be restored by a reallocation of resources that favours the rearing of more children.

2.3.1 Capital Accumulation and the Emission Rate

The engine of output growth in our economy is the accumulation of physical capital. Furthermore, growth can be sustained in the long-run due to the presence of a learning-by-doing externality in the determination of labour productivity. As we shall see later, complex transitional dynamics are avoided by virtue of the AK-type technology employed in our model.

Capital is accumulated by perfectly competitive financial intermediaries who accept deposits by young workers in exchange for the gross rate of return r_{t+1} per unit of deposited income. They subsequently transform these saving deposits into capital by accessing a technology that transforms one unit of time-t output into one unit of time-$t+1$ capital. The capital is supplied to intermediate good producers at a rental cost of R_{t+1} per unit.

Evidently, the zero profit condition for financial intermediaries implies that[31]

$$r_{t+1} = \frac{R_{t+1}}{\psi_t}. \tag{2.23}$$

[31] We assume full depreciation of capital.

Furthermore, we have

$$K_{t+1} = N_t s_t \,, \tag{2.24}$$

which indicates that the collective savings by all young workers/households (whose population is N_t) are the inputs in the investment process that leads to the formation of physical capital. Of course, the demographics of our economy imply that the population size of young households evolves according to

$$\frac{N_{t+1}}{N_t} = n_t \,. \tag{2.25}$$

Substituting (2.25) in (2.24) and using the notational standard $k_{t+j} = K_{t+j} / N_{t+j}$ ($j = 0,1,...$) to denote capital per worker, we can write (2.24) as

$$k_{t+1} = \frac{s_t}{n_t} \,. \tag{2.26}$$

Using (2.2), (2.26) and $L_t = N_t(1 - qn_t)$ in (2.16) and (2.17) we get

$$w_t = (1 - \tau\mu_t)\frac{\sigma - 1}{\sigma}(1 - \beta)B\Theta^{1-\beta}k_t(1 - qn_t)^{-\beta} \,, \tag{2.27}$$

and

$$R_t = (1 - \tau\mu_t)\frac{\sigma - 1}{\sigma}\beta B\Theta^{1-\beta}(1 - qn_t)^{1-\beta} \,, \tag{2.28}$$

respectively.

Earlier, we indicated that the government imposes a proportional tax on emissions and uses the proceeds to finance a programme of transfers/subsidies to (young and old) households, as well as government consumption expenses. Now, we shall assume that this programme of transfers/subsidies is designed to eradicate the cost accrued to households, as a result of the taxation of pollutant emissions. We justify this assumption by appealing to the idea that workers/savers do not have any control or responsibility for the emission of pollutants. This responsibility rests with the entrepreneurs. For this reason, it may be desirable to 'correct' any negative repercussions that accrue to households for choices over which they have no control whatsoever.[32] In fact, such a policy design is by no means a mere theoretical construction. In their letter to *The Guardian*, Annick Hansen and James Hansen (a scientist in the field of climatology) provide arguments in support of emission (carbon) taxes. Their suggestion is that "the public will support the tax if it is returned to them, equal shares on a per capita basis…deposited monthly in bank accounts."[33]

Given these arguments, we postulate that the programme of transfers/subsidies is designed so that

$$(1+h_t^{young})w_t(1-qn_t)=\frac{\sigma-1}{\sigma}(1-\beta)B\Theta^{1-\beta}k_t(1-qn_t)^{1-\beta}, \tag{2.29}$$

and

$$(1+h_t^{old})\frac{R_t}{\psi_{t-1}}s_{t-1}=\frac{\frac{\sigma-1}{\sigma}\beta B\Theta^{1-\beta}(1-qn_t)^{1-\beta}}{\psi_{t-1}}s_{t-1}. \tag{2.30}$$

Effectively, the scheme is designed in a manner that eliminates the term $(1-\tau\mu_t)$ from the returns to labour and capital (which is also the return to saving according to Equation 2.23). Using Equations (2.27)-(2.30), it is straightforward to establish that

$$h_t^{young}=h_t^{old}=\frac{\tau\mu_t}{1-\tau\mu_t}. \tag{2.31}$$

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[32] As in Varvarigos (2013), it should be noted that this type of allocation of government revenues is Pareto improving because households are strictly better off (their consumption increases in both periods) whereas the utility of entrepreneurs remains unaffected. Thus, the government can achieve its environmental policy objective with no cost in terms of household welfare.

[33] http://www.guardian.co.uk/world/2009/jan/01/letter-to-barack-obama.

Substituting (2.31) back to the government's budget constraint, we can eventually obtain government consumption as[34]

$$g_t = \tau \mu_t \frac{1}{\sigma} y_t . \tag{2.32}$$

We are now ready to obtain the economy's growth rate. First, we substitute (2.20), (2.22), (2.27) and (2.31) in (2.26). Subsequently, some straightforward algebra allows us to derive

$$\frac{k_{t+1}}{k_t} - 1 = \Omega(\mu_t) = \frac{(\sigma-1)(1-\beta)B\Theta^{1-\beta}q}{\sigma\gamma}\Psi(1/\mu_t)\left[\frac{1+\gamma+\Psi(1/\mu_t)}{1+\Psi(1/\mu_t)}\right]^{\beta} - 1 . \tag{2.33}$$

As we can see, the growth rate of capital per worker is a function of the emission rate μ_t. There are two ways through which the latter impinges on the economy's growth rate, both of them working through the emission rate's effect on life expectancy. On the one hand, the emission rate determines the marginal propensity to save – thus, the funds available for investment. On the other hand, it also affects fertility decisions and correspondingly, the rate of population growth as well as the amount of labour that households offer. As it turns out, all these effects work in the same direction, thus leading to the result in

Proposition 2. *The growth rate of capital per worker is negatively related to the amount of emissions per unit of output. That is* $\Omega'(\mu_t) < 0$.

[34] In Appendix B, we show formally that these results are consistent with the equilibrium in the goods market.

Proof. Using (2.33), it is straightforward to establish that

$$\Omega'(\mu_t) = \frac{(\sigma-1)(1-\beta)B\Theta^{1-\beta}q\Psi_{\mu_t}}{\sigma\gamma}\left[\frac{1+\gamma+\Psi(1/\mu_t)}{1+\Psi(1/\mu_t)}\right]^{\beta}\left[1-\beta\frac{\Psi(1/\mu_t)}{1+\Psi(1/\mu_t)}\frac{\gamma}{1+\gamma+\Psi(1/\mu_t)}\right]<0$$

because $\Psi_{\mu_t} < 0$. □.

Earlier we established that a higher μ_t reduces longevity. This effect causes a reduction in the marginal propensity to save, thus reducing the amount of saving for a given amount of labour income. Furthermore, by leading to an increase in the fertility rate, the reduction in labour supply reduces disposable income available for saving. Finally, the higher rate of population growth implies a direct reduction in the amount of investment per household. All these effects result in a lower rate of growth. In what follows, and given the result in Proposition 2, we shall be assuming that parameter values are such that $\Omega(\overline{\mu}) > 0$; that is, the growth rate of capital per worker is still positive even with the highest possible emission rate.

Recall that entrepreneurs will choose their emission per unit of production to maximise utility through the expression in (2.5), taking the supply of labour as given. Using (2.1), (2.2) and (2.19), we can rewrite this expression as

$$u_t^{\text{entrepreneur}} = \begin{cases} (1-\tau\overline{\mu})\dfrac{1}{\sigma}B\Theta^{1-\beta}(1-qn_t)^{1-\beta}K_t, \text{ if } \mu_t = \overline{\mu} \\ \\ (1-\tau\underline{\mu})\dfrac{1}{\sigma}B\Theta^{1-\beta}(1-qn_t)^{1-\beta}K_t - \varepsilon, \text{ if } \mu_t = \underline{\mu} \end{cases}. \tag{2.34}$$

Of course, (2.34) reveals that the emission rate will be endogenously determined from

$$\mu_t = \begin{cases} \overline{\mu}, \text{ if } K_t < \hat{K}_t \\ \\ \underline{\mu}, \text{ if } K_t \geq \hat{K}_t \end{cases}, \tag{2.35}$$

where

$$\hat{K}_t = \frac{\varepsilon\sigma}{B\Theta^{1-\beta} Z_t}, \tag{2.36}$$

and $Z_t = \tau(\overline{\mu} - \underline{\mu})(1 - qn_t)^{1-\beta} > 0$. Intuitively, a choice of lower emissions per unit of production is beneficial in terms of variable profits because it reduces the fraction of revenues lost in the form of taxes. Nevertheless, given the fixed effort cost associated with a cleaner production process, this benefit will dominate only after the economy's resources (in terms of capital) exceed the endogenous threshold given by $\hat{K}_t$.[35]

The results in (2.22), (2.35) and (2.36) raise the possibility that there are complementarities in the joint determination of optimal fertility rates (by households) and the cleanliness of production technology (by entrepreneurs). The intuition is the following. On the one hand, the fertility rate is increasing in the emission rate for the reasons to which we alluded earlier. On the other hand, a higher fertility rate will make the choice of a relatively cleaner production process less desirable, simply because more time spent on child-rearing reduces

[35] For any value of τ, there will always be a level of threshold. Therefore, an increase in τ cannot eliminate the threshold but can only reduce it. Moreover, given the initial stock of capital a possible policy is to set the tax high enough so that the threshold is below the initial level of capital. However, given that the tax reduces the disposable income of the threshold, it is unclear whether the threshold will be optimal or not.

labour supply, and therefore output – an effect that entices entrepreneurs to postpone the adoption of a cleaner production method.

Let us denote the composite parameter terms $\lambda \equiv \frac{\varepsilon\sigma}{B\Theta^{1-\beta}\underline{Z}}$ and $\xi \equiv \frac{\varepsilon\sigma}{B\Theta^{1-\beta}\overline{Z}}$, where $\underline{Z} = \tau(\overline{\mu} - \underline{\mu})(1 - qn(\overline{\mu}))^{1-\beta}$ and $\overline{Z} = \tau(\overline{\mu} - \underline{\mu})(1 - qn(\underline{\mu}))^{1-\beta}$. We can use previous results to infer

Lemma 1. *It is* $\lambda > \xi$.

Proof. It follows from Proposition 1, $\frac{\partial Z_t}{\partial n_t} < 0$ and $\frac{\partial \hat{K}_t}{\partial Z_t} < 0$.

□ .

Now let us assume that, given Equation (2.22), the model's parameters allow $n(\underline{\mu}) - 1 > 0$. For instance, this can happen with a sufficiently low value for q. In this case, taking account of Proposition 1 and Equation (2.25), we can see that the growth rate of the population is always positive. Recalling that $\Omega(\overline{\mu}) > 0$, it is true that the growth rate of the aggregate capital stock (per entrepreneur) is positive as well, i.e., $\frac{K_{t+1}}{K_t} = \frac{k_{t+1}}{k_t}\frac{N_{t+1}}{N_t} > 1$; alternatively, $K_{t+1} > K_t$.[36] Now, let us consider an economy for which $K_0 < \xi$. Naturally, there must be some periods $\tilde{T} > \underset{\sim}{T} \geq 1$ such that $K_{\underset{\sim}{T}-1} < \xi < K_{\underset{\sim}{T}} < K_{\tilde{T}-1} < \lambda < K_{\tilde{T}}$.

[36] Given that we have assumed a unit mass of entrepreneurs, the aggregate capital stock and the capital stock used by each entrepreneur are indistinguishable.

Hence, the determination of the emission rate can be formally described through

Lemma 2. *There are time periods* $\tilde{T} > \underset{\sim}{T} \geq 1$ *such that*

$$\mu_t = \begin{cases} \bar{\mu} & \textit{for} \quad t = 0, \ldots, \underset{\sim}{T} - 1 \\ \textit{either } \bar{\mu} \textit{ or } \underline{\mu} & \textit{for} \quad t = \underset{\sim}{T}, \ldots \tilde{T} - 1 \\ \underline{\mu} & \textit{for} \quad t = \tilde{T}, \tilde{T} + 1, \ldots \end{cases} . \tag{2.37}$$

Proof. It follows directly from Lemma 1, $K_0 < \xi$ and $K_{t+1} > K_t$. $\square$.

Evidently, the economy will undergo a transition characterised by a move from the more polluting production technology to the less polluting one. The intuition is that the process of economic growth will, at some point, entice entrepreneurs to incur the fixed cost of adopting a production method that entails a lower emission rate. Doing so becomes optimal at relatively advanced stages of economic development, simply because the opportunity cost of not switching to a cleaner production method, i.e., the emission tax, becomes greater compared to the fixed cost of adoption. Note however that, for intermediate stages of the development process, we observe the presence of multiple equilibria. During that stage (from $t = \underset{\sim}{T}$ to $t = \tilde{T}$), the choice of technology is characterised by indeterminacy, as the use of either the relatively dirty or the relatively clean technology is optimal. This indeterminacy is associated with the complementarities that are involved in the joint formation of optimal fertility choices by households and optimal technology choices by entrepreneurs. In any case, the result in Lemma 1 will have significant implications for issues pertaining to demographic changes in our economy. This is an issue to which we turn in the following section of our analysis.

2.4 Growth, Fertility, and Longevity

The results of the previous section indicate that, at some point of its development process, the economy will experience a reduction in the pollutant emission rate. As we shall see, this outcome has significant implications for both demographic and economic outcomes. Concerning the former, one major result comes in the form of

Proposition 3. *The economy will undergo a demographic transition in the sense that it will experience an increase in life expectancy and a reduction in the rate of population growth. Formally,*

$$\psi_t = \begin{cases} \Psi(1/\overline{\mu}) & for \quad t = 0,\ldots,\underset{\sim}{T}-1 \\ either\ \Psi(1/\overline{\mu})\ or\ \Psi(1/\underline{\mu}) & for \quad t = \underset{\sim}{T},\ldots\tilde{T}-1 \\ \Psi(1/\underline{\mu}) & for \quad t = \tilde{T},\tilde{T}+1,\ldots \end{cases}, \tag{2.38}$$

such that $\Psi\left(1/\overline{\mu}\right) < \Psi\left(1/\underline{\mu}\right)$, *and*

$$n_t = \begin{cases} n(\overline{\mu}) & \textit{for} \quad t = 0,...,\underset{\sim}{T} - 1 \\ \textit{either } n(\overline{\mu}) \textit{ or } n(\underline{\mu}) & \textit{for} \quad t = \underset{\sim}{T},...\tilde{T} - 1 \\ n(\underline{\mu}) & \textit{for} \quad t = \tilde{T}, \tilde{T} + 1,... \end{cases}, \tag{2.39}$$

such that $n(\overline{\mu}) > n(\underline{\mu})$.

Proof. It follows from Proposition 1, Lemma 1, and $\Psi_{\mu_t} < 0$.

□.

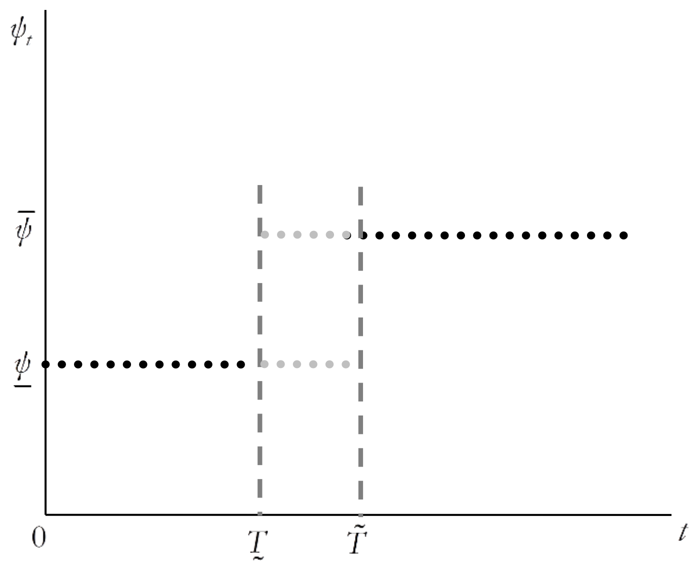

Figure 2.3

Life expectancy

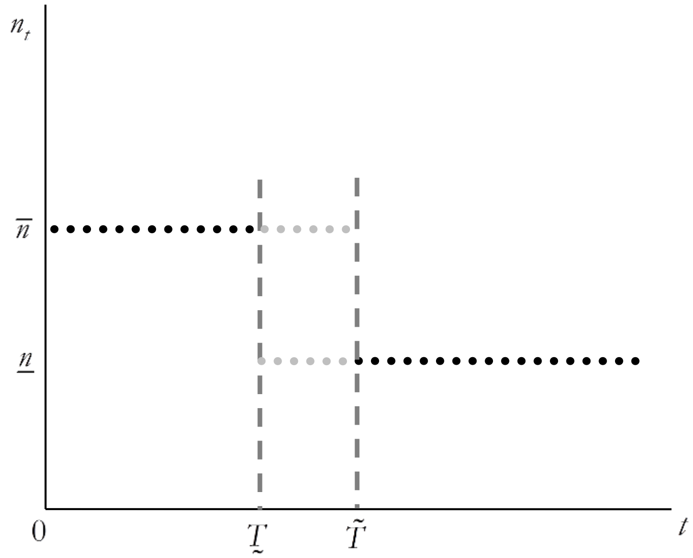

Figure 2.4

Fertility rate

A similar distinct change can be observed in relation to the economy's growth rate. This becomes evident in

Proposition 4. *There are time periods* $\tilde{T} > \underset{\sim}{T} \geq 1$ *such that*

$$\Omega(\mu_t) = \begin{cases} \Omega(\overline{\mu}) & \textit{for} \quad t = 0,\ldots,\underset{\sim}{T}-1 \\ \textit{either } \Omega(\overline{\mu}) \textit{ or } \Omega(\underline{\mu}) & \textit{for} \quad t = \underset{\sim}{T},\ldots\tilde{T}-1 \\ \Omega(\underline{\mu}) & \textit{for} \quad t = \tilde{T},\tilde{T}+1,\ldots \end{cases}, \quad \Omega(\overline{\mu}) < \Omega(\underline{\mu}). \quad (2.40)$$

Proof. It follows from Proposition 2 and Lemma 1. □.

The two previous propositions reveal that the economy will undergo a distinct change in both its economic (i.e., output growth) and demographic (i.e., fertility and longevity) outcomes. In particular, the process of development will allow the economy to accumulate the resources – in terms of capital stock – which are necessary to entice entrepreneurs to incur the fixed effort cost of implementing a less polluting production method. The resulting reduction in emissions will improve the health status of the population and increase life expectancy (see Figure 2.3), thus enhancing the households' savings motive. Subsequently, this outcome will eventually result in lower fertility rates (see Figure 2.4) that cause a reduction in the rate of population growth, as households try to smooth their consumption profile by increasing their labour supply at the expense of the time that is available for rearing children. Hence, the novelty of our analysis rests on the idea that environmental factors – that is the choice of less polluting production processes induced by environmental policy – are crucial in the joint determination of economic growth and various aspects of demographic change.

As expected, the intermediate stages of the development process result in multiple equilibria and indeterminacy with respect to both economic and demographic outcomes. This is of course a by-product of the similar result in Lemma 2. Multiple equilibria in the choice of technology are associated with multiplicity in life expectancy and consequently fertility rates. Furthermore, these demographic changes have implications for output growth. Indeed, the growth rate also entails multiplicity and indeterminacy during the intermediate stages of the economy's transitional dynamics.

2.5 Conclusion

In this paper, we have sought to fill a gap in the literature by analysing a model showing that the interactions between economic growth and environmental factors can account for observed variations in some important demographic characteristics. Specifically, we offer a novel mechanism according to which the endogenous change of the emission rate that occurs in the presence of environmental taxation, brings forth a joint change in both life expectancy and fertility. What is particular interesting with our framework and results is that we have provided a scenario according to which the causality on the nexus between environmental characteristics and population growth may actually work from the former to the latter. This is a conjecture that, despite being in contrast to current conventional wisdom, is intuitive and may provide further understanding on the negative relation between population growth and the quality of the natural environment.

When placed within the vast literature on the interrelation between economic growth and demographic change, our analysis aims at providing a new explanation on the conscious choice of some households to have fewer children. Neither do we make any claim that our mechanism is the only one in explaining the fertility reduction, nor do we suggest that the conscious decision to remain childless or raise fewer children is the only determinant behind the demographic change. An important aspect of the latter is that the falling fertility in industrialized countries may also be associated with population aging, meaning that there are relatively fewer people in the reproductive cohorts. This is an indubitably important issue; nevertheless, it goes beyond the scope of our paper.

Our model is constructed in a manner that allows analytical solutions. Thus it benefits from the clear-cut and detailed description of all the mechanisms involved whereas the absence of unnecessary complication allows us to avoid aspects that could blur the intuition. As always, the model can be enriched with elements that would allow us to study additional effects whose analysis do not comprise a part of this paper's objective. For example, we could have used a more general form for the production function so that we could analyse the more complicated transitional dynamics that are unavoidably absent within an AK-type technology. Furthermore, we could enrich the characteristics of population changes by allowing infant (in addition to adult) mortality. Additionally, we could also extend the study by having welfare analysis such as analysis of optimal tax rate that maximises economic growth. However, given the current set up of the model, in which we have a scenario where tax reduces disposable income; it is hard to apply a policy that sets the tax rate high enough so that we can achieve earlier and higher growth rate with favourable demographic transition. Therefore, a different framework is necessary for this objective. As stated earlier, these issues go beyond the purpose of our current study which seeks to focus on the causal effects of pollution on the economy's demography. Nevertheless, they are definitely important; hence, they represent a potentially rewarding avenue for future research work.

Chapter 3

The Growth Effects of Foreign Aid: Health Aid versus Untied Aid

3.1 Introduction

Since post-World War II, aid has been transferred by donors to recipient countries in view to assist with their socio economic needs. The main objectives of the transfers are to alleviate poverty and to promote economic growth (Sachs et. al 2004). However, whether it is true that aid could promote the above mentioned objectives is still under long-standing debates among policy makers and researchers. The present chapter analyses the growth impact of two different types of aid; i) aid that is tied to activities that can promote health improvements (or productive aid) and ii) untied (or pure) aid that takes the form of income transfers.

There are rich empirical studies that examining the aid-growth nexus in different contexts. At the empirical level, the answer has been mixed. While some studies claim that aid works well in amplifying growth of the recipient countries (e.g., Papanek 1973; Dalgaard et. al 2004; Minoiu and Reddy 2007), others contend that it is ineffective and does not offer any positive impacts on growth (e.g., Easterly 2007a, 2007b; Rajan and Subramanian 2008, Nowak-Lehmann et. al, 2012). Meanwhile, other analyses have attempted to delve into the issue further by disentangling the effects of different types of aid such as tied and untied aid.[37] The proponents of tied aid argue that this form of aid could lead to improved economic outcomes of the recipient countries by relaxing resource constraints and directly enhancing services such as education and health (e.g., Sachs et. al 2004). In addition, Minoiu and Reddy (2010) claim that aid that is tied to a particular project for development purposes can become effective in increasing the growth rate in comparison to pure aid. Furthermore, Neanidis and Varvarigos (2009) argue that in the case of volatile aid, foreign aid has a positive impact on growth if it is allocated for productive or development activities. However, the most recent analysis by Doucouliagos and Paldam (2011) indicates that the effectiveness of this particular type of aid on growth performance is actually insignificant.[38]

In response to the mixed results of the existing studies, Kalyvitis et. al (2012) raise a question as to whether aid flows are excessive or insufficient. While a number of studies are sceptical about the growth impact of aid, the

[37] For more extensive empirical literatures on different types of aid and growth relationship, see Hansen and Tarp (2000) and Doucouliagos and Paldam (2009).
[38] For more empirical findings on tied and untied aid, see for example Miquel-Florensa (2007).

United Nation (2006) reports that there are several cases that show aid flows can actually promote investment and growth of the recipient countries. Republic of Korea and Taiwan are the examples of Asian Tigers that are now enjoying positive economic growth after receiving aid during their initial stage of development between 1946 and 1978. Therefore, based on differences in views on aid-growth nexus, Kalyvitis et, al (2012) and Alvi et. al (2008) suggest that the relationship between aid and growth is non-monotonic. They further argue that there is a threshold level of aid for which above (below) this threshold, aid positively (negatively) affect growth.

At the theoretical level, there is a growing number of studies that analyse the aid-growth nexus (e.g., Chatterjee et. al 2003; Hodler 2004; Economides et. al 2008; Kalaitzidakis and Kalyvitis 2008; Kitaura 2009). Chatterjee et. al (2003) are among the first who examine this relationship theoretically in the context of tied aid versus untied aid. They show that aid can stimulate growth if it is tied to productive projects such as health or education. Chatterjee and Turnovsky (2007) incorporate a labour-leisure choice in Chatterjee et. al (2003) and find that untied aid negatively affects economic growth of the recipient countries as it increases leisure at the expense of work effort. On the other hand, Chao et. al (2012) show that aid that is tied to activities that abate pollution fails to promote growth as it crowds out public investment and reduces productivity of public input in the production process.

Another group of theoretical studies attempts to analyse the impact of health-promoting aid (either tied or in-kind aid) in comparison to pure aid. For instance, Azarnet (2008) studies how foreign aid that is distributed to children in order to meet their nutritional needs (and hence, their health improvements) can affect fertility, education and growth. He finds that this aid does not promote growth- the reason being is that an increase in aid reduces parents' cost per child prompts parents to substitute child quality for quantity. Consequently, whereas the number of children increases, parents' spending on the education of each child decreases, resulting in the human capital and economic growth.

Vasilakis (2011) develops a two-period overlapping generations model to analyse the effectiveness of a specific type of aid; that is the World Food Programmes (WFP)- designed to improve children's nutrition intake. He shows that the relationship between this type of health-related aid and economic growth depends on the methods of implementation in the recipient countries. In particular, if WFP takes the form of a school feeding programme, it reduces the quantity cost of raising each child. Moreover, the provision of WFP in terms of school meals also locks the recipient country in poverty because it leads to reduction in the parents' spending on their children's nutrition that consequently increases fertility and slows down human capital accumulation and economic growth. Therefore, in order for aid to positively affect growth, WFP has to be sufficiently high so that human capital exceeds a certain threshold. As human capital exceeds the threshold, an increase in aid enables parents' resources to be diverted from spending on children's health improvements to children's education expenditures. Nevertheless, if WFP takes the form of fixed amount of food per child transferred to households, an increase in aid decreases economic growth as both parents' contribution towards the children's health as well as the children's length of time spent for education decrease too. However, if the aid is distributed for the improvement of the health sector infrastructure, an increase in aid will reduce infant mortality and will increase the development of human capital and economic growth of the recipient country.

Neanidis (2012) employs a two-period OLG model to study the link between two types of humanitarian aid (i.e. in-kind aid and monetary aid), fertility and growth, using the assumption that health status of adults is linearly dependent on their health status during childhood. In addition, he also assumes that childhood health depends not only on parents' health but also on the provision of in-kind aid (i.e. per child food aid for improvement of nutrition and thus, the health status of children) and on the time spent on rearing children. He finds that the effect of humanitarian aid on fertility and growth is ambiguous because of the opposite effects of in-kind aid and monetary aid. Particularly, the former promotes growth and reduces fertility rates by improving the health status of children and their productivity during adulthood. On the contrary, the latter impedes growth as the reduction in cost of rearing children encourages parents to reproduce more children at the expense of children's health status and their productivity during adulthood. In order to verify the ambiguity in his theoretical results, Neanidis (2012) further investigates this issue empirically and discovers that aid has no effects on both fertility and growth. Agénor and Yilmaz (2012) examine the growth impacts of aid that is tied to public health services in a representative agent framework of an open economy. In their model, health-related aid determines health capital. The latter enters production function as input. Furthermore, in their model, health status of individuals depends solely on foreign aid. Their result demonstrates that foreign aid tied to health services stimulates growth. Similar to the empirical findings, all of the theoretical studies stated above have also shown differing results.

The study of aid-growth nexus in Chapter 3 adds to the existing theoretical studies of aid tied to the health improvements activities and its impact on growth. Instead of focusing on aid that is tied to the health spending for children, we direct our attention to the aid that is tied to the health spending for old adults. As the results of tying aid to children's health related activities show different consensus, it is therefore, worth an attempt to examine whether tying aid to old adults' health improvement activities will also yield the same results i.e. whether aid will support or discourage the economic growth of the recipient economy. In contrast with the study by Agénor and Yilmaz (2012), we examine this issue in the presence of private health expenditures. Particularly, we employ a two-period overlapping generations model where donors transfer two different types of foreign aid, namely aid that is tied to the health improvements of old adults and untied aid which takes the form of income transfers. Both cohorts of agents (i.e. young and old) receive the income transfers offered through untied aid.

The results of our study indicate that the growth effect of aid is ambiguous as it depends critically on the distribution between tied and untied aid, as well as the distribution of untied across the two different cohorts of individuals- young and old. Particularly, when all aid is allocated to the co-financing of private health expenditures, the impact of foreign aid on growth is negative as it undermines saving behaviour due to health aid and private health spending being substitute. When aid takes the form of pure income transfer, the only part that is offered to young individuals is growth promoting. Otherwise, if people expect to receive income transfers when old, they will reduce their saving when young, exactly because they will try to increase consumption during young adulthood given the prospect of higher lifetime income.

Additionally, we also examine a scenario where health aid is an input in the production function. By doing this, we extend our analysis of the aid-growth nexus on the supply side of the economy. We find that the impact of aid on growth is non-monotonic. Particularly, there are thresholds of aid for which the growth impact of aid is negative (positive) if aid lies within (outside) these thresholds.

The structure of this chapter is as follows. Section 3.2 elaborates the characteristics of the economic framework, while the Section 3.3 analyses the general equilibrium. Section 3.4 examines the impact of foreign aid on economic growth, followed by Section 3.5 that analyses the growth impact foreign aid when health aid is considered as an input in the production function. Section 3.6 examines the growth impact of health aid and its allocation on demand and supply side, as well as the growth impact of untied aid given to the young cohort. Section 3.7 concludes.

3.2 The Economy

We consider an overlapping generations economy in which time is discrete and indicated by $t = 0,\ 1,\ 2, ...$ The economy is populated by an infinite sequence of individuals who have perfect foresight and face a two-period lifetime; *young* *adulthood* and *old adulthood*. There are also firms that produce homogeneous goods for consumption. Additionally, the economy receives foreign aid from donors in the following manner. Foreign donors transfer aid to the economy as a proportion of the recipient's output per worker, y_t (e.g., Neanidis and Varvarigos 2009; Economides et al. 2008; Hodler 2007; Chaterjee 2003).[39] That is

$$F_t = fy_t \ . \tag{3.1}$$

In equation (3.1), f will be the measure of foreign aid in our growing economy. This assumption can also be interpreted as an indication that donors should reward recipient countries that show good economic performance and promote economic growth successfully (Hodler 2007; Blackburn and Forgues-Puccio 2011). A proportion of $\gamma \in [0,1]$ of foreign aid, F_t is allocated for the purpose of co-financing private health expenditures (tied aid), while the remaining $(1-\gamma)$ is transferred as untied aid to both young and old individuals to supplement their existing income. Specifically, a fraction of $\varrho \in [0,1]$ of pure aid, $(1-\gamma)F_t$, is given to current young individuals in the economy.

Individuals make decisions in both periods. Each of them derives lifetime utility according to

$$U_t = \ln c_t + \beta \ln h_{t+1}c_{t+1} \ , \quad \beta \in (0,1),$$

or, equivalently,

$$U_t = \ln c_t + \beta[\ln h_{t+1} + \ln c_{t+1}] \tag{3.2}$$

[39] We adopt this technical assumption to allow analytical tractability in our analysis. Particularly, this assumption enables us to reach an equilibrium with long-run output growth.

subject to constraints

$$c_t = w_t + \varrho(1-\gamma)F_t - s_t\ , \quad \varrho,\ \gamma \in [0,1], \tag{3.3}$$

$$c_{t+1} = r_{t+1}s_t + (1-\varrho)(1-\gamma)F_{t+1} - x_{t+1}, \tag{3.4}$$

$$h_{t+1} = H(x_{t+1} + \gamma F_{t+1})^{\varepsilon}, \qquad H > 0,\ \varepsilon \in (0,1). \tag{3.5}$$

In the previous expressions, c_t denotes consumption during young adulthood and c_{t+1} denotes consumption during old adulthood. Every young individual supplies a unit of labour effort inelastically in the labour market and receives the market wage rate per unit of labour, w_t. The variable s_t denotes saving, r_{t+1} is the gross interest rate on saving, h_{t+1} is the health status during old adulthood, x_{t+1} is the old adult's spending towards health improvements. In equation (3.2), the utility of old age consumption is augmented by health status i.e. $\ln h_{t+1}c_{t+1}$. In other words, improved health status increases the life quality of old individuals, and that is why they have the incentive to spend resources for its improvement. Nevertheless, we can see from equation (3.5) that a part of foreign aid at period $t+1$, i.e. γF_{t+1} is used to co-finance private health expenditure for the improvement of health status during old adulthood. The inclusion of aid that is tied to the provision of resources towards health improvements will lead to significant repercussions on aggregate saving and capital accumulation in the economy.

In each period, there is a unit mass of competitive firms that combine labour from young individuals, L_t and capital from financial intermediaries, K_t, to produce Y_t unit of output according to

$$Y_t = AK_t^a (Z_t L_t)^{1-a}, \quad A > 0, \quad a \in (0,1). \tag{3.6}$$

The variable Z_t is an indicator of labour productivity. Following Frankel (1962) and Romer (1986), we assume that labour productivity is proportional to the average stock of capital in the economy, $\overline{K}_t$, according to

$$Z_t = \theta \frac{\overline{K}_t}{L_t}, \quad \theta > 0. \tag{3.7}$$

This assumption captures the idea that workers gain knowledge and become more productive by handling more capital goods (i.e. learning by doing externalities).[40] The preceding discussion completes the description of our theoretical framework. We show the derivation and characteristics of the equilibrium of our model in the next section.

3.3 Equilibrium

The objective of young adult is to choose quantity for c_t, s_t, c_{t+1} and x_{t+1} so as to maximise equation (3.2) subject to equations (3.3-3.5), taking w_t, r_{t+1}, F_t and F_{t+1} as given. The solutions to this problem are

$$s_t = \frac{\beta(1+\varepsilon)\left[w_t + \varrho(1-\gamma)F_t\right]}{1+\beta(1+\varepsilon)} - \frac{\left[1-\varrho(1-\gamma)\right]F_{t+1}}{\left[1+\beta(1+\varepsilon)\right]r_{t+1}} \tag{3.8}$$

and

$$x_{t+1} = \frac{\beta\varepsilon r_{t+1}\left[w_t + \varrho(1-\gamma)F_t\right]}{1+\beta(1+\varepsilon)} - \frac{\left[\gamma(1+\beta) - \beta\varepsilon(1-\varrho)(1-\gamma)\right]}{1+\beta(1+\varepsilon)} F_{t+1}. \tag{3.9}$$

[40] This assumption is made to guarantee the existence of equilibrium with positive long run growth.

Equations (3.8) and (3.9) show that both saving and private health expenditures are affected by foreign aid.[41] Other things being equal, an increase in the part of aid distributed to young individuals, F_t, increases both saving and private health expenditures. Nevertheless, the part of foreign aid distributed to the old, either through pure transfers or tied health spending, has a negative effect on both saving and private health spending. The intuition is as follows. It is very clear that an increase in F_t helps to augment young individuals' income. With more income, they will be able to consume more during young adulthood. In order to smooth out their consumption profile, they will try to increase their (effective) consumption during old age. Therefore, they will save more in order to have more resources to spend for the consumption of goods and health services when old.

Now, consider an increase in F_{t+1} - particularly in the part offered in the form of lump-sum transfers. This will enhance disposable income during old adulthood. As c_{t+1} increases, the marginal utility of c_{t+1} falls. Individuals will try to smooth consumption by increasing consumption during young adulthood. Therefore, they will find it optimal to allocate less of their first period income towards saving. Qualitatively similar is the effect of an increase in the part of F_{t+1} that is dispensed for health services. As health aid has the potential to crowd out private health expenditures, when F_{t+1} increases, individuals will find it optimal for them to reduce private health spending and divert their old age

[41] In order to enable us to analyse the repercussions of aid that is tied to health and untied (pure) aid in our model, we assume that $x_{t+1} > 0$ is always applied. See Appendix C for the sufficient (but not necessary) condition of positive x_{t+1}.

income towards consumption. Once more, consumption smoothing will entail an increase in c_t and a corresponding reduction in s_t.

We now turn our attention to the optimal decision made by firms. Profit maximisation by firms requires the marginal product of each input equals to their respective marginal costs. Taking into account equilibrium condition in the labour market (i.e. labour demand equals labour supply) in which $L_t = 1$, along with $\bar{K}_t = K_t$, profit maximisation of firms leads to

$$w_t = (1-a)A\theta^{1-a}k_t, \tag{3.10}$$

and

$$r_t = aA\theta^{1-a}. \tag{3.11}$$

where $k_t = \dfrac{K_t}{L_t}$ is the stock of capital per worker. [42]

The equilibrium in financial markets requires saving equals investment. That is

$$s_t = k_{t+1}. \tag{3.12}$$

Using equation (3.8) to substitute out for s_t into (3.12), and then combining with (3.1), (3.10) and (3.11), equilibrium implies

$$k_{t+1} = \frac{1}{\left(1+\beta(1+\varepsilon)\right)} \times [.],$$

where $[.]$ is

$$\beta A\theta^{1-a}(1+\varepsilon)\left[(1-a)+\varrho(1-\gamma)f\right]k_t - \frac{\left[1-\varrho(1-\gamma)\right]f}{a}k_{t+1}. \tag{3.13}$$

---

[42] The gross interest rate is constant for all time periods because we employ AK-type production function.

Equation (3.13) is the first order difference equation that describes the dynamics of physical capital accumulation in the economy. It shows that saving and investment vary with f, indicating how foreign aid affects capital accumulation. Given that the latter is the engine of growth in the economy, the next section presents the formal analysis on how and why economic growth is affected by foreign aid.

3.4 Foreign Aid and Growth

In this section, we demonstrate how foreign aid can affect growth of the recipient country. To begin with, let us obtain the equilibrium growth rate. Solving (3.13) for k_{t+1}, dividing both sides by k_t and subtracting a unit from both sides yields

$$\frac{k_{t+1}}{k_t}-1=\frac{a\beta(1+\varepsilon)A\theta^{1-a}\left[(1-a)+\varrho(1-\gamma)f\right]}{a\left[1+\beta(1+\varepsilon)\right]+\left[1-\varrho(1-\gamma)\right]f}-1=\Psi(f). \tag{3.14}$$

As we can see in expression (3.14), economic growth is a function of foreign aid per unit of output, f.[43] Now, we can examine the growth implications of f using the first derivative of $\Psi(f)$ with respect to f. That is

$$\frac{\partial\Psi(f)}{\partial f}=\frac{a\beta(1+\varepsilon)A\theta^{1-a}}{\left(a\left[1+\beta(1+\varepsilon)\right]+\left[1-\varrho(1-\gamma)\right]f\right)^2}\times[.], \tag{3.15}$$

where $[.]=$

$$\left\{a\left[1+\beta(1+\varepsilon)\right]+\left[1-\varrho(1-\gamma)\right]f\right\}\varrho(1-\gamma)-\left[(1-a)+\varrho(1-\gamma)f\right]\left[1-\varrho(1-\gamma)\right],$$

[43] We restrict our attention to parameter values that guarantee the non-negativity of the growth rate. That is $\Psi(f)>0$.

or (after simplification), $[.]$ can be written as

$$a\left[1+\beta(1+\varepsilon)\right]\varrho(1-\gamma)-(1-a)\left[1-\varrho(1-\gamma)\right].$$

Obviously, the sign of (3.15) depends on the sign of $[.]$. The latter critically depends on the different settings of values of parameters γ and ϱ, i.e. the proportion of total aid allocated to health expenditures co-financing (tied aid) and also the proportion of untied (pure) aid allocated to the young. Therefore, the manner through which foreign aid, f, affects economic growth is not straightforward.

Nevertheless, our previous result can allow us to identify the different conditions under which foreign aid affects economic growth either positively or negatively. We will firstly present these results formally and then discuss their economic interpretation. We begin by defining $\delta = \dfrac{1-a}{1-a+a\left(1+\beta(1+\varepsilon)\right)}$.

Proposition 1. *Foreign aid has a positive (negative) effect when* $\gamma < \hat{\gamma}$ $(\gamma \geq \hat{\gamma})$.

Proof. We can use the expression that determines the sign of $\Psi(f)$ so that $\Psi'(f) > 0$ iff $\varrho(1-\gamma) > \dfrac{1-a}{1-a+a\left(1+\beta(1+\varepsilon)\right)} \equiv \delta$. From this, we can have $\varrho > \dfrac{\delta}{1-\gamma}$. Given that $\varrho, \gamma \in [0,1]$, thus, we can find an upper bound for γ, for which an increase in f can lead to a positive impact on growth. That is

$\frac{\delta}{1-\gamma} < 1 \;\Rightarrow\; \gamma < 1-\delta = \hat{\gamma} \;\Rightarrow\; \frac{\partial\Psi(.)}{\partial f} > 0$. Therefore, this implies that we have

$\frac{\delta}{1-\gamma} \geq 1 \;\Rightarrow\; \gamma \geq \hat{\gamma} \;\Rightarrow\; \frac{\partial\Psi(.)}{\partial f} \leq 0$. □.

Proposition 2. *Foreign aid has a positive (negative) effect as long as* $\varrho > \hat{\varrho}$ $(\varrho \leq \hat{\varrho})$.

Proof. We follow the same method as we did for Proposition 1. Given that $\Psi'(f) > 0$ iff $\varrho(1-\gamma) > \delta$, we can rewrite this condition as $\gamma < 1 - \frac{\delta}{\varrho}$. Given that $\gamma, \varrho \in [0,1]$, we can find the lower bound of ϱ for which an increase in f can increase growth. That is $1 - \frac{\delta}{\varrho} > 0 \;\Rightarrow\; \varrho - \delta > 0 \;\Rightarrow \varrho > \delta = \hat{\varrho} \;\Rightarrow\; \frac{\partial\Psi(.)}{\partial f} > 0$. Therefore, we can also have $1 - \frac{\delta}{\varrho} \leq 0 \Rightarrow \varrho - \delta \leq 0 \Rightarrow \varrho \leq \delta = \hat{\varrho} \Rightarrow \frac{\partial\Psi(.)}{\partial f} \leq 0$.

□.

The results presented in Proposition 1 and 2 reveal that the overall effect of aid on economic growth is ambiguous and depends on the allocation of foreign aid between tied (health-oriented) aid and untied or pure (transfer) aid, as well as the allocation of the untied part of aid among the young and the old. The interpretation is clear because we have previously seen the various effects of aid on saving behaviour.

According to Proposition 1, for a given allocation of untied aid between the young and the old, the positive effect is only possible to achieve when the fraction of aid that is tied to health improvement activities is sufficiently small.

This is due to the fact that this kind of aid can diminish the saving incentive as people reduce their own health spending and consume more in both periods (consumption smoothing). If the allocation of pure transfers is unfavourable to the young, then this effect is dominant and growth is reduced.

Proposition 2 has a similar explanation. For a given allocation of foreign aid between tied and untied, the effect on growth will only be positive if the young receive a relatively high fraction of aid transfers. This is because the young will save some part of this transfer while they will reduce saving if they expect to receive aid when old. If the part allocated to the tied aid is sufficiently high, then the results are different and the aid impact is always negative. If a substantial amount of foreign aid is devoted to the support of health spending, then the disincentive to save when young is so strong (because individuals will reduce health spending and increase consumption in both periods), that the overall effect of foreign aid is negative.

3.5 Extending the Health Aid on the Supply Side of the Recipient's Economy

To complement the previous analysis and for the policy making purposes, it is instructive if we also consider the supply-side effects of health aid in the existing framework. Therefore, in this section we attempt to achieve this objective by introducing a proportion of health aid that is allocated as an input in the production of the recipient economy. The extension is as follows. Once more, let us consider the model in Section 3.2. Now, however, the total health aid, γF_t, is further allocated to two different categories. That is, a proportion of $\varphi \in [0,1]$ of health aid is transferred to old adults to co-finance their private health expenditures and the remaining $(1-\varphi)$ of this aid will go to the production function as one of the inputs (i.e. as health capital). Taking this into account, we can rewrite equation (3.5) as

$$h_{t+1} = H(x_{t+1} + \varphi\gamma F_{t+1})^{\varepsilon}. \tag{3.16}$$

Furthermore, the production function in equation (3.6) now becomes

$$Y_t = A_t K_t^a (Z_t L_t)^{1-a}, \tag{3.17}$$

where

$$A_t = \left[(1-\varphi)\gamma \frac{F_t}{\overline{Y}_t}\right]^{\lambda}, \qquad \lambda \in (0,1), \tag{3.18}$$

and $\overline{Y_t}$ is the average income in the economy.

As we can see, A_t in (3.17) now depends on the provision of health services from donors. However, following Barro and Sala-i-Martin (2004) there is a congestion effect of aid as shown in equation (3.18). This means, for a given proportion of health aid, $(1-\varphi)\gamma$, an increase in income per worker, $\overline{Y}_t$ lowers the availability of aid in terms of health services to each producer and thus, reduces total output produced in the economy.

It is straightforward to establish that the solution of the model leads to the following equilibria for saving and private health spending respectively.

$$s_t = \frac{\beta(1+\varepsilon)[w_t + (1-\gamma)\varrho F_t]}{1+\beta(1+\varepsilon)} - \frac{\beta[(1-\gamma)(1-\varrho)+\varphi\gamma]F_{t+1}}{1+\beta(1+\varepsilon)r_{t+1}}, \tag{3.19}$$

and

$$x_{t+1} = \frac{\beta\varepsilon r_{t+1}[w_t + (1-\gamma)\varrho F_t]}{[1+\beta(1+\varepsilon)]} - \frac{[\varphi\gamma(1+\beta) - \varepsilon(1-\varrho)(1-\gamma)]F_{t+1}}{[1+\beta(1+\varepsilon)]}. \tag{3.20}$$

Similar to the analysis in Section 3.4, both saving and private health expenditures are affected by foreign aid. Aid given in the first period of individuals' lifetime, F_t, augments young adults' income. Therefore, an increase in the part of aid distributed to the young adults, F_t, enables individuals to have more disposable income and to consume more during youth. Due to consumption smoothing profile, they will save more in their first period to permit more consumption in the next period of their lifetime. Not only that, their resources for their health improvement activities will also increase. As a result, they will increase their private health expenditures.

On the other hand, an increase in the part of aid allocated to the old adults decreases saving and private health expenditures. In order to analyse this, let us consider the part of aid in the form of lump-sum or in-kind transfers. This augments disposable income of the old adults. Therefore, an increase in this part of aid will encourage old adults to increase their consumption and to reduce private health spending in the second period of their lifetime. Furthermore, they will also find that it optimal to reduce saving and increase consumption during youth. Similarly, an increase in part of aid, F_{t+1}, that is allocated for the purpose of co-financing private health spending also mitigates saving. This is because health aid has a possibility to crowd out private health spending. As F_{t+1} increases, individuals will respond optimally by reducing their spending on health and channelling their disposable income towards consumption in both periods. Due to consumption smoothing activities, saving will decrease.

Now we turn to the optimal decision of firms. Given equilibrium in the labour market (i.e. $L_t = 1$), profit maximisation of the firms leads to the following.

$$w_t = (1-a)[(1-\varphi)\gamma]^{\lambda} f^{\lambda}\theta^{1-a}k_t, \tag{3.21}$$

and

$$r_t = a[(1-\varphi)\gamma]^{\lambda} f^{\lambda}\theta^{1-a}. \tag{3.22}$$

Both equations (3.21) and (3.22) show that a fraction of $(1-\varphi)$ of health aid augments the returns to labour and capital (i.e. wage rate, w_t and interest rate, r_t). In other words, health aid that enters production function as input allows labour and capital to earn more pay off for their contributions in the output production. Using (3.21) and (3.22) in (3.19) together with equilibrium condition in the financial market (i.e. $s_t = k_{t+1}$), rearranging terms accordingly and then subtracting a unit from both sides yields economic growth expression as follows.[44, 45]

$$\frac{k_{t+1}}{k_t} - 1 = \frac{a\beta(1+\varepsilon)[(1-\varphi)\gamma]^{\lambda} f^{\lambda}\theta^{1-a}}{a(1+\beta(1+\varepsilon)) + [(1-\gamma)(1-\varrho)+\varphi\gamma]f} \times [(1-a)+(1-\gamma)\varrho f] - 1 = \Phi(f). \tag{3.23}$$

[44] If we let $\lambda = 0$ and $\varphi = 0$, the growth rate expression in (3.23) will be identical to the original version in expression (3.14).
[45] Similar to the expression (3.14), we restrict the parameter values that guarantee positive growth rate. That is $\Phi(f) > 0$.

Equation (3.23) shows that the proportion of health aid that is used as input in the production, as well as proportion of tied aid for young adults positively affect growth of the recipient country. However, the proportion of health aid that co-finances private health spending during old and the proportion of in-kind/tied aid transferred to the old individuals adversely affect the growth. In order for us to pedantically analyse the overall effect of foreign aid on growth, we do the same procedure as in Section 3.4. That is, we take the first derivative of $\Phi(f)$ with respect to foreign aid, f. Therefore, we have

$$\frac{\partial \Phi(f)}{\partial f} = \frac{a(\beta+\varepsilon)\theta^{1-a}[(1-\varphi)\gamma]^{\lambda} f^{\lambda-1}}{(a(1+\beta(1+\varepsilon))+f[(1-\gamma)(1-\varrho)+\varphi\gamma])^{2}} \times V(f), \tag{3.24}$$

where

$$\begin{aligned} V(f) = \quad & \lambda(1-\gamma)\varrho[(1-\gamma)(1-\varrho)+\varphi\gamma]f^{2} \\ & +\{(1+\lambda)a(1+\beta(1+\varepsilon))(1-\gamma)\varrho-(1-\lambda)(1-a)[(1-\gamma)(1-\varrho)+\varphi\gamma]\}f \\ & +\lambda a(1-a)(1+\beta(1+\varepsilon)). \end{aligned} \tag{3.25}$$

The sign of $\frac{\partial \Phi(f)}{\partial f}$ depends on the sign of $V(f)$. Given that $V(f)$ is a quadratic expression, the determination of its sign is not straightforward. Nevertheless, $V(f)$ can allow us to study the different conditions under which foreign aid, f can affect growth. We present the formal results in the following propositions.

Proposition 3. *Suppose* $(1+\lambda)a(1+\beta(1+\varepsilon))(1-\gamma)\varrho > (1-\lambda)(1-a)[(1-\gamma)(1-\varrho)+\varphi\gamma]$ *holds. An increase in foreign aid will increase the economic growth of the recipient countries.*

Proof. See Appendix C. □ .

Proposition 4. *Suppose* $(1+\lambda)a(1+\beta(1+\varepsilon))(1-\gamma)\varrho < (1-\lambda)(1-a)[(1-\gamma)(1-\varrho)+\varphi\gamma]$ *holds.*

i) *For any* $\lambda < \bar{\lambda}$ *, an increase in foreign aid positively affect economic growth only when* $f < f_1$ *and* $f > f_2$ *. However, for any* $f_1 \leq f \leq f_2$ *, an increase in foreign aid only dampens the growth.*

ii) *For any* $\lambda \geq \bar{\lambda}$*, an increase in foreign aid positively affects growth.*

Proof. See Appendix C. □.

According to Proposition 3, for a given allocation of foreign aid between tied and untied aid and under certain parameter condition, the effect of aid on growth can be positive.

On the other hand, the first part of results in Proposition 4 shows that whether aid can positively or negatively affect growth depends critically on the value of the elasticity of health aid as input in the production function (i.e. λ). Particularly, if the elasticity of health aid in the production function is less than a certain threshold (i.e. $\lambda < \bar{\lambda}$), then the aid, f can amplify growth only if it is sufficiently low or sufficiently high (i.e. $f < f_1$ or $f > f_2$). However, aid may dampen growth if it is between the range of $f_1 \leq f \leq f_2$. The second part of

Proposition 4 reveals that it is possible to have positive effect of aid on growth when the elasticity of health aid is sufficiently high (i.e. $\lambda \geq \bar{\lambda}$). The findings that are presented here may shed some light in the explanation of the reasons why existing evidence produce mixed results on the growth repercussions of foreign aid. Moreover, as pointed out by Kalyvitis et. al (2012), the identification of aid threshold allows us to determine differences in its impact on the growth of the recipient country.

3.6 The Relationship Between Health Aid, In-kind Aid and Growth

We further extend the analysis by examining the growth effect of each types of aid proportion allocated by the donor to the recipient country. This is further studied in the following sub-sections.

3.6.1 The Growth Impact of the Overall Health Aid (γ).

In order to examine how the overall health aid, γ affect growth of the recipient country, we do the similar procedure as in previous sections i.e. taking the first derivative of $\Psi(f)$ with respect to γ. Thus, it is

$$\frac{\partial \Psi(f)}{\partial \gamma} = \frac{a(\beta+\varepsilon)\theta^{1-a}[(1-\varphi)\gamma f]^{\lambda}}{(a(1+\beta(1+\varepsilon))+f[(1-\gamma)(1-\varrho)+\varphi\gamma])^{2}} \times M(\gamma), \qquad (3.26)$$

where

$$M(\gamma) = -\lambda\varrho[\varphi-(1-\varrho)]\gamma^2 f^2$$

$$-\left\{(1+\lambda)[a(1+\beta(1+\varepsilon)+(1-\varrho)f]\varrho+(1-\lambda)[(1-a)+\varrho f][\varphi-(1-\varrho)\right\}\gamma f$$

$$+\lambda[(1-a)+\varrho f][a(1+\beta(1+\varepsilon)+(1-\varrho)f].$$

(3.27)

Depending on the conditions, the impact of γ on growth rate can be positive or negative. This can be formalised in the following propositions.

Proposition 5. *Suppose* $1-\varrho<\varphi$ *holds. Then, the impact of overall health aid,* γ *on growth rate is positive when* $\gamma<\gamma^g$ *and negative when* $\gamma\geq\gamma^g$ *; where* γ^g *is a composite parameter terms.*

Proof. See Appendix C. $\square$.

Proposition 6. *Suppose*

i) $1-\varphi\geq\varrho$ *and;*

ii) $(1-\lambda)[(1-a)+\varrho f][(1-\varrho)-\varphi]>(1+\lambda)\varrho[1+\beta(1+\varepsilon)+(1-\varrho)f]$ *hold.*

Then, the impact of overall health aid, γ *on growth rate is positive.*

Proof. See Appendix C. $\square$.

Proposition 5 shows that given $1-\varrho<\varphi$ holds, an increase in the proportion of health aid, γ can only improve growth of the recipient country if it is below a threshold, γ^g . Otherwise the impact will be negative. Meanwhile, the condition $1-\varrho<\varphi$ means that the proportion of untied aid given to the old individuals is less than the proportion of health aid for co-financing private health spending of this cohort, φ . The intuition of the results in Proposition 5 is as follows. We know that the proportion of tied aid allocated to the old individuals, $(1-\varrho)$ can be utilised by this cohort for consumption, c_{t+1} and spending for their health status improvement, x_{t+1}. We also know that health aid allocated for the purpose of co-financing x_{t+1} has a possibility to crowd out x_{t+1}. The condition $1-\varrho<\varphi$ presents the possibility that the proportion of health aid for co-financing old adults' health spending to crowd out x_{t+1} is relatively high than when this condition is reversed. However, when $\gamma<\gamma^{g}$, an increase in γ can still positively affect growth because the crowding out effect of health aid allocated to the old individuals is not strong enough to diminish the magnitude increase in x_{t+1}. As a result, incentive of old adults to spend on x_{t+1} is still strong enough to reassure positive saving during youth, which accordingly can increase total capital accumulation that generates more economic growth. Nevertheless, as $\gamma\geq\gamma^{g}$, the crowding out effect of health aid for old adults on x_{t+1} becomes more apparent and relatively stronger. Therefore, if there is an increase in γ, it will only mitigate the growth of the recipient country.

On the other hand, Proposition 6 demonstrates that when $1-\varphi\geq\varrho$ holds, an increase in γ will always stimulate growth. This is because as $1-\varphi\geq\varrho$ holds, the crowding out effect now is relatively weaker.

3.6.2 The Growth Impact of Health Aid for Old Adults (φ).

Additionally, we attempt to analyse the specific type of health aid allocation i.e. health aid for old adults, φ . We find that the growth rate of the recipient country will be positive if φ is sufficiently low. However, if φ exceeds its threshold, that is $\bar{\varphi}$, the repercussion of health aid for old adults to the growth rate will be negative. We present the summary of these results in the following proposition.

Proposition 7. *An increase in the health aid for old adults, φ increases (decreases) growth if $\varphi < \bar{\varphi}$ ($\varphi \geq \bar{\varphi}$); where $\bar{\varphi}$ is a composite parameter terms.*

Proof. See Appendix C □ .

Proposition 7 establishes that, for given proportion of health/tied aid, γ and untied aid to the young adults, ϱ, an increase in the proportion of health aid to co-finance old private health expenditures of old adults may give an inverted U-shaped impact on growth of the recipient country. Particularly, there is a threshold, $\bar{\varphi}$, for which if φ is sufficiently low, an increase in φ may promote growth. The intuition can be clarified as follows. As mentioned in Section 3.4, health aid allocated to the old adults and private health spending are substitute. Therefore, when φ increases at a very low proportion (i.e. when $\varphi < \bar{\varphi}$), the increase in φ is not dominant enough to allow health aid for old individuals to crowd out the private health spending undertaken by this cohort in the economy. In this case, the net effect of this type of health aid will be positive because old individuals will still want to spend more for their health improvement activities (even though they also receive health aid from donors). In order to pursue this, they will save more during young adulthood. This act will increase the total saving and thus, will also improve capital accumulation and growth.

Additionally, on the supply side, $\varphi < \overline{\varphi}$ implies that $(1-\varphi)$ is sufficiently high. Recall back that $(1-\varphi)$ is the proportion of health aid that enters the production function as input. Even though an increase in φ will reduce the proportion of health aid in the production function, the decline in output will be very marginal. Therefore, after taking into account the impact of φ on both the demand and supply sides of the recipient country, the overall impact on growth will still be positive.

Nevertheless, when $\varphi \geq \overline{\varphi}$, an increase in φ will be sufficient enough to allow crowding out effect of health aid on private health spending. Furthermore, the proportion of health aid on the supply side, $(1-\varphi)$ is now relatively lower than before. Now, the decline in output on the supply side will be critical. Hence, the overall effect of an increase in φ on growth will be negative.

3.6.3 The Growth Impact of In-Kind Aid Transferred to Young Adults (ϱ).

In contrast with health aid, the effect of untied aid for young adults on growth of the recipient country is very straightforward. That is

Proposition 8. *An increase in untied aid for young adults, ϱ leads to an increase in growth.*

Proof. See Appendix C. $\square$.

The intuition of Proposition 8 is also very straightforward. Given f , γ and φ, an increase in ϱ allows young adults to have more income (at their disposal) than before. This enables them to not only consume but also to save more when they are young. Moreover, an increase in ϱ means a decrease in $(1-\varrho)$. Given other things constant, this indicates a decline in the individuals' income when old. As individuals expect to receive less donation/untied aid during their second period of lifetime, they will respond to this by saving more during young. Therefore, the total savings and capital stock will increase and consequently promote growth of the recipient country.

3.7 Conclusion

In this chapter, we have modelled and analysed the impacts of foreign aid on the growth of the recipient country when it is categorised into two distinct types of aid i.e. tied (health) aid and also untied (pure) aid. We have found that the analysis yields ambiguous results for the fact that these results depend on the proportion of aid allocated to tied aid as well as untied aid. On the one hand, the part of aid transferred to young individuals increases saving. On the other hand, the part of aid allocated to health improvement activities discourages saving. These conflicting effects of foreign aid on savings lead to ambiguous effect on economic growth. For a given allocation of foreign aid between tied and untied aid, the positive growth effect of aid can only occur if the part of untied aid that takes the form of income transfers to young individuals is relatively high and the part of aid allocated to the health improvement activities is sufficiently low. Otherwise the effect of aid on growth will be negative due to the existence of strong saving disincentive of young individuals caused by high allocation of tied aid. When all foreign aid is tied to health improvement activities, the effect of aid on growth rate will definitely be negative. In this case, our result shares the same findings as the study of Vasilakis (2011), which identifies that health-related aid in his analysis is distributed in fixed amounts of food per child to households. However, Vasilakis (2011) examines the issue in the context of health-related aid for children's health status while our analysis studies the issue in the context of aid that is tied to health improvement activities of old adults.

Moreover, the ambiguity of the results arises from the fact that some types of tied aid (in our case –aid that is tied to the health improvements) may provide disincentives for growth promoting activities. However, the first part of the analysis does not imply that all types of tied aid are detrimental to economic growth. Therefore, we have also shown in Section 3.5 that when health aid is further extended to the supply side of the economy, the growth impact of aid due to health (tied) aid could be non-monotonic. In other words, there are thresholds of foreign aid for which it can either positively or negatively affect growth of the recipient country. In this case, our results echo with the empirical findings of Alvi et. al (2008) and Kalyvitis et. al (2012); in which they argue that the relationship of aid and growth is non-linear and that the existence of foreign aid thresholds are possible. Our results also show that the large-scale aid flows can have a significant impact on growth of the recipient country in the long-run. This confirms with the report of United Nation (2006) in which it states that aid flows have resulted in enhancing investment and growth over the last years for countries like Republic of Korea and Taiwan. Moreover, the non-monotonic results in our analysis originate from the impact of specific type of tied aid, that is health aid and its allocation to either demand side or to supply side. This shows that tied aid (in this case health aid) can actually improve growth of the recipient country if its allocation on the supply side of the economy can be increased.

Conclusion

The role of health in promoting economic growth has attracted researchers to delve further into the study of the link between these two issues in different contexts. This thesis presented three chapters in health and economic growth theory.

Health systems of any country consist of two types, a public and a private one. Chapter 1 have shown that the complementarity between public and private health expenditures can stimulate positive rather than negative impact of capital stock on aggregate saving. Therefore, the results obtained in this chapter contradict the findings presented in Bhattacharya and Qiao (2007) mainly because we assumed that private health expenditures occur during old period instead of during young period of an individual's lifetime. Furthermore, this complementarity can elucidate the stylised facts of one aspect of demographic transitions, that is, the decline in fertility rates. Therefore, in terms of policy implication, the provision of public health spending by the government could facilitate an economy to reduce the overpopulation problem while attempting to achieve positive economic development.

Future work extension may consider the inclusion of investment in education of children and/or adults in the existing framework. This may allow us to analyse how education and health investments (public and private) interact in an environment of complementarity between both spending when endogenous fertility is taken into consideration. All of these issues are certainly fruitful avenues for future research.

The next chapter which is Chapter 2 have provided an analytical framework of the joint determination of economic and demographic changes. That is as the environmental quality improves, life expectancy increases and fertility rates falls when an economy grows. We have incorporated pollution and the choice of 'going green' into a three-period overlapping generations growth model with endogenous longevity and fertility. In particular, our model has shown that an endogenous change of emission rates induced by environmental tax may lead to changes in demography i.e. higher longevity and lower fertility rates.

Therefore, policy makers that intend to achieve higher economic growth and life expectancy along with reduction in population's birth rates could use environmental policy to induce firms to adopt cleaner technology so as to encourage enhanced health status and longer life expectancy. In addition to the benefits in terms of environmental quality, this may also change the behaviour of reproduction by reducing fertility rates.

In the last chapter, which is Chapter 3 we have attempted to explore the foreign aid-growth relationship. We have categorised foreign aid into aid that is tied to the adults' health improvement activities, and untied (pure) aid that takes the form of income transfers to young and old. We have analysed this relationship in the presence of private health expenditures. The result is equivocal as it critically depends on the part of aid allocated to the health improvement activities and also the part of aid allocated to income transfers to the young. For a given tied and untied aid, a sufficiently high proportion of aid to health improvement activities may dampen economic growth. We believe that the results of the first model in Chapter 3 have significant policy implications, because it is vital that the donors allocate foreign aid in such a way that it could bring benefits rather than harmful ramifications to the recipient countries. In order to promote economic growth in the recipient countries, it is advisable for the donors to distribute more of untied aid that is transferred specifically to the young rather than the tied aid. According to the existing framework, the best aid allocation decision is achieved when all foreign aid is donated as income transfers to the young, because this motivates more saving and capital accumulation, and thereby improves economic growth of the recipient countries. Nevertheless, our study does not imply that all types of tied aid may be harmful or damaging to the economic growth. There are some other types of health aid such as health aid for children, young workers and etc. that may offer different outcomes if they are introduced in the existing framework. However, this is not the focus of our analysis in Chapter 3, though they are definitely important. Therefore, this can be another research area that could be explored in the future.

Additionally, we introduce health aid on the supply side of the economy of the recipient country. The results reveal that there are threshold for which aid could amplify growth if it lies outside the thresholds of aid. Nevertheless, if aid allocated by the donor lies within the thresholds, it could mitigate growth. The results in this second model of the chapter assist us in elucidating the reason behind mixed results of the existing studies.

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Appendix

Appendix A

Appendix for Chapter 1

1. First Order Conditions (FOCs) for profit maximisation of firms in Section 1.2

Using equation (1.8) and cost function of firm i.e. $\omega_t L_t + r_t K_t$, we can rewrite profit function as follows

$$\max_{L_t, K_t} \pi_t = AK_t^a L_t^{1-a} - (\omega_t L_t + r_t K_t). \tag{A1}$$

The optimisation problem of a firm is to maximise (A1). So, we can obtain the FOCs of L_t and K_t by taking the first derivative of (A1) with respect to L_t and K_t separately and equate each of them equal to zero. Therefore, we have

$$L_t : (1-a)AK_t^a L_t^{-a} - \omega_t = 0, \tag{A2}$$

and

$$K_t : aAK_t^{a-1} L_t^{1-a} - r_t = 0, \tag{A3}$$

respectively. Profit maximisation of firms requires marginal products of inputs equal their marginal costs. Therefore, we can rewrite (A2) and (A3) as

$$\omega_t = (1-a)AK_t^a L_t^{-a} \tag{A2a}$$

$$r_t = aAK_t^{a-1}L_t^{1-a} \tag{A3a}$$

Given (1.9) and $k_t = \dfrac{K_t}{N_t}$, it is straightforward to establish (1.10) and (1.11) from (A2a) and (A3a) respectively.

2. FOCs and work of solutions for utility maximisation and equilibrium in Section 1.3.

Substitute (1.2)-(1.4) in (1.1) to obtain unconstrained utility function as follows

$$\max_{s_t, n_t, x_{t+1}} \quad u^t = \ln\left[(1-\tau)(1-qn_t)\omega_t - s_t\right] + \gamma \ln(n_t) + \beta \ln\left[Hx_{t+1}^{\delta\varepsilon_{t+1}}\right] + \beta \ln(r_{t+1}s_t - x_{t+1}). \tag{A4}$$

The optimisation problem of an individual is to maximise (A4). Taking ω_t, r_{t+1} and ε_{t+1} as given, we take partial derivatives of (A4) with respect to s_t, x_{t+1} and n_t separately and set each of them equal to zero. Thus, we get the following FOCs

$$s_t : \frac{\beta r_{t+1}}{r_{t+1}s_t - x_{t+1}} - \frac{1}{(1-\tau)(1-qn_t)\omega_t - s_t} = 0. \tag{A5}$$

$$n_t : \frac{\gamma}{n_t} - \frac{q\omega_t(1-\tau)}{(1-\tau)(1-qn_t)\omega_t - s_t} = 0. \tag{A6}$$

$$x_{t+1} : \frac{\delta\varepsilon_{t+1}}{x_{t+1}} - \frac{1}{r_{t+1}s_t - x_{t+1}} = 0. \tag{A7}$$

Rearrange (A7) so that

$$x_{t+1} = \frac{\delta\varepsilon_{t+1}}{1+\delta\varepsilon_{t+1}} r_{t+1} s_t. \tag{A8}$$

Next, substitute (A8) in (A5) so as to obtain

$$\frac{\beta}{s_t\left(1/(1+\delta\varepsilon_{t+1})\right)} = \frac{1}{(1-\tau)(1-qn_t)\omega_t - s_t}. \tag{A9}$$

Rearrange (A9) so as to get equation (1.12). That is

$$s_t = \frac{\beta(1+\delta\varepsilon_{t+1})}{1+\beta(1+\delta\varepsilon_{t+1})}(1-\tau)\omega_t(1-qn_t)$$

Now, we can substitute (1.12) in (A6) to get equation (1.13) for n_t. That is

$$n_t = \frac{\gamma/q}{1+\beta(1+\delta\varepsilon_{t+1})+\gamma}.$$

Using (1.13) in (1.12) and then substituting (1.12) into (A8), we can therefore obtain (1.14) as follows

$$x_{t+1} = \frac{\beta\delta\varepsilon_{t+1}}{1+\beta(1+\delta\varepsilon_{t+1})} r_{t+1} s_t.$$

3. Proof to Proposition 1

Using equation (1.18), we can define

$$J(qn_t, k_t) = qn_t - \frac{\gamma}{1+\beta[1+\delta Z\left(\tau(1-a)Ak_t^a(1-qn_t)^{1-a}\right)]+\gamma}. \tag{A10}$$

We can also use (1.6), (1.10) and (1.15) to write

$$\tau(1-a)Ak_t^a(1-qn_t)^{1-a} = p_{t+1}. \tag{A11}$$

From (A10) we have

$$J_{k_t}(\cdot,\cdot)=\frac{\gamma\beta\delta Z'\left(\tau(1-a)Ak_t^a(1-qn_t)^{1-a}\right)\tau(1-a)Aak_t^{a-1}(1-qn_t)^{1-a}}{\{1+\beta[1+\delta Z\left(\tau(1-a)Ak_t^a(1-qn_t)^{1-a}\right)]+\gamma\}^2}>0. \quad \text{(A12)}$$

Alternatively, we can substitute (A11) in (A12) to write

$$J_{k_t}(\cdot,\cdot)=\frac{\gamma\beta\delta aZ'\left(p_{t+1}\right)p_{t+1}}{\{1+\beta[1+\delta Z\left(p_{t+1}\right)]+\gamma\}^2 k_t}>0. \quad \text{(A13)}$$

From (A10), we can also derive

$$J_{qn_t}(\cdot,\cdot)=1-\frac{\gamma\beta\delta Z'\left(\tau(1-a)Ak_t^a(1-qn_t)^{1-a}\right)\tau(1-a)^2Ak_t^a(1-qn_t)^{-a}}{\{1+\beta[1+\delta Z\left(\tau(1-a)Ak_t^a(1-qn_t)^{1-a}\right)]+\gamma\}^2}, \quad \text{(A14)}$$

to which we can substitute (A11) to get

$$J_{qn_t}(\cdot,\cdot)=1-\frac{\gamma\beta\delta(1-a)Z'\left(p_{t+1}\right)p_{t+1}}{\{1+\beta[1+\delta Z\left(p_{t+1}\right)]+\gamma\}^2(1-qn_t)}. \quad \text{(A15)}$$

Substituting (1.18) and (A11) in (A15), we can write the latter as

$$J_{qn_t}(\cdot,\cdot)=1-\frac{\gamma}{1+\beta[1+\delta Z\left(p_{t+1}\right)]+\gamma}\frac{(1-a)\beta\delta Z'\left(p_{t+1}\right)p_{t+1}}{1+\beta[1+\delta Z\left(p_{t+1}\right)]}>0, \quad \text{(A16)}$$

which is positive because $Z'\left(p_{t+1}\right)p_{t+1}<Z\left(p_{t+1}\right)$ holds. Now, we can combine the results in (A16) and (A12), and apply the implicit function theorem to (A10). This yields

$$\frac{dqn_t}{dk_t}=-\frac{J_{k_t}(\cdot,\cdot)}{J_{qn_t}(\cdot,\cdot)}<0, \quad \text{(A17)}$$

Therefore, given that q is a fixed parameter, we can conclude that $n_t = n(k_t)$ such that $n'(k_t) < 0$. Finally, we can use the previous analysis to write (1.17) as

$$s_t = \frac{\beta[1+\delta Z\left(\tau(1-a)Ak_t^a(1-qn(k_t))^{1-a}\right)]}{1+\beta[1+\delta Z\left(\tau(1-a)Ak_t^a(1-qn(k_t))^{1-a}\right)]}(1-\tau)(1-a)Ak_t^a(1-qn(k_t))^{1-a},$$

from which it is straightforward to establish that $ds_t / dk_t > 0$. $\square$.

4. Proof to Proposition 2

From equations (1.18) and (1.20), we can establish that $n(0) = \dfrac{\gamma / q}{1+\beta(1+\delta)+\gamma}$ and $n(\infty) = \dfrac{\gamma / q}{1+\beta(1+\delta\overline{\varepsilon})+\gamma}$. Combining these with equation (1.22), we see that $\psi(0) = 0$ and $\psi(\infty) = \infty$. Furthermore, it is

$$\begin{aligned}\psi'(k_t) = \eta\Bigg\langle & \frac{\beta[1+\delta Z\left(\tau(1-a)Ak_t^a(1-qn(k_t))^{1-a}\right)]}{1+\beta[1+\delta Z\left(\tau(1-a)Ak_t^a(1-qn(k_t))^{1-a}\right)]}\frac{\theta(k_t)}{n(k_t)} + \\ & \frac{k_t^a(1-qn(k_t))^{1-a}}{n(k_t)}\frac{\beta\delta Z'\left(\tau(1-a)Ak_t^a(1-qn(k_t))^{1-a}\right)\tau(1-a)A\theta(k_t)}{\{1+\beta[1+\delta Z\left(\tau(1-a)Ak_t^a(1-qn(k_t))^{1-a}\right)]\}^2} - \\ & \frac{\beta[1+\delta Z\left(\tau(1-a)Ak_t^a(1-qn(k_t))^{1-a}\right)]}{1+\beta[1+\delta Z\left(\tau(1-a)Ak_t^a(1-qn(k_t))^{1-a}\right)]}\frac{k_t^a(1-qn(k_t))^{1-a}}{[n(k_t)]^2}n'(k_t)\Bigg\rangle,\end{aligned} \tag{A18}$$

where

$$\theta(k_t) = ak_t^{a-1}(1-qn(k_t))^{1-a} - k_t^a(1-a)qn'(k_t)(1-qn(k_t))^{-a}. \tag{A19}$$

Clearly, as $qn'(k_t) > 0 \Rightarrow \theta(k_t) > 0$. Hence, $\psi'(k_t) > 0$. Now, combine (A13) and (A16) to write (A17) as

$$\frac{dqn_t}{dk_t} = -\frac{\gamma\beta\delta a Z'\left(p_{t+1}\right)p_{t+1}\{1+\beta[1+\delta Z\left(p_{t+1}\right)]\}}{\{1+\beta[1+\delta Z\left(p_{t+1}\right)]+\gamma\}\{1+\beta[1+\delta Z\left(p_{t+1}\right)]\}-\gamma\beta\delta(1-a)Z'\left(p_{t+1}\right)p_{t+1}} \times \frac{1}{k_t\{1+\beta[1+\delta Z\left(p_{t+1}\right)]+\gamma\}}.$$

Given $Z'(0)=\varphi$ and $Z'(\infty)=0$, we can combine the expression above together with (A11), $n(0)=\dfrac{\gamma/q}{1+\beta(1+\delta)+\gamma}$ and $n(\infty)=\dfrac{\gamma/q}{1+\beta(1+\delta\overline{\varepsilon})+\gamma}$ to establish that

$$n'(k_t)=\begin{cases}-\infty & \text{for} \quad k_t=0 \\ 0 & \text{for} \quad k_t\to\infty\end{cases}. \tag{A20}$$

Combining (A19) and (A20) with (A18), we infer that

$$\psi'(k_t)=\begin{cases}\infty & \text{for} \quad k_t=0 \\ 0 & \text{for} \quad k_t\to\infty\end{cases}. \tag{A21}$$

Thus, we conclude that there must be *at least one* $\hat{k}\in(0,\infty)$ such that $\hat{k}=\psi(\hat{k})$ and $\psi'(\hat{k})<1$, i.e., $\hat{k}$ is a stable steady-state equilibrium. □ .

5. FOCs and work of solutions for utility maximisation in Section 1.5.1.

Substitute (1.23)-(1.25) in (1.1) so as to rewrite utility function as an unconstrained function as follows

$$\max_{s_t,n_t,x_t} u^t = \ln\left[(1-\tau)(1-qn_t)\omega_t - s_t - x_t\right] + \gamma\ln(n_t) + \beta\ln\left[Hx_t^{\delta\varepsilon_{t+1}}\right] + \beta\ln\left[r_{t+1}s_t\right] \tag{A22}$$

The optimisation problem of an individual is to maximise (A22). Taking ω_t, r_{t+1} and ε_{t+1} as given, we take the first derivatives of (A22) with respect to s_t, x_t and n_t separately and set each of them equal to zero. Therefore, we have the FOCs as follows

$$s_t : \frac{\beta}{s_t} - \frac{1}{(1-\tau)(1-qn_t)\omega_t - s_t - x_t} = 0. \tag{A23}$$

$$n_t : \frac{\gamma}{n_t} - \frac{q(1-\tau)\omega_t}{(1-\tau)(1-qn_t)\omega_t - s_t - x_t} = 0. \tag{A24}$$

$$x_t : \frac{\beta\delta\varepsilon_{t+1}}{x_t} - \frac{1}{(1-\tau)(1-qn_t)\omega_t - s_t - x_t} = 0. \tag{A25}$$

Rearrange (A25) so that

$$x_t = \frac{1}{1+\beta\delta\varepsilon_{t+1}}\left[\beta\delta\varepsilon_{t+1}(1-\tau)(1-qn_t)\omega_t - \beta\delta\varepsilon_{t+1}s_t\right]. \tag{A26}$$

Now, substitute out (A26) in (A24). This give us

$$n_t = \frac{\gamma(1-\tau)\omega_t - \gamma s_t}{q(1-\tau)\left[1+\beta\delta\varepsilon_{t+1}+\gamma\right]\omega_t}. \tag{A27}$$

Next, combine (A26) and (A27) in (A23) to yields

$$s_t = \frac{\beta}{1+\beta[1+\delta\varepsilon_{t+1}]+\gamma}(1-\tau)\omega_t. \tag{A28}$$

Substitute (A28) in (A27) to obtain

$$n_t = \frac{\gamma / q}{1+\beta[1+\delta\varepsilon_{t+1}]+\gamma}. \tag{A29}$$

Substitute (A28) and (A29) in (A26) to get

$$x_t = \frac{\beta\delta\varepsilon_{t+1}}{1+\beta[1+\delta\varepsilon_{t+1}]+\gamma}(1-\tau)\omega_t. \tag{A30}$$

Finally, using (1.16), we can rewrite (A28), (A29) and (A30) as equations (1.26), (1.27) and (1.28) respectively.

6. FOCs and work of solutions for profit maximisation of firms in Section 1.5.2

Using (1.30) and firm's cost function i.e. $\omega_t L_t + r_t K_t$, we can write profit function as

$$\max_{L_t, K_t} \pi_t = K_t^a (A_t L_t)^{1-a} - (\omega_t L_t + r_t K_t). \tag{A31}$$

The optimisation problem of a firm is to maximise (A31). Take the first derivative of (A31) with respect to L_t and K_t separately and set each of them equal to zero. Therefore, the FOCs of (A31) with respect to L_t and K_t are

$$L_t : (1-a)K_t^a L_t^{-a} A_t^{1-a} - \omega_t = 0, \tag{A32}$$

$$K_t : aK_t^{a-1}(A_t L_t)^{1-a} - r_t = 0. \tag{A33}$$

Given (1.9), (1.31) and $k_t = \dfrac{K_t}{N_t}$, we can establish equilibrium wage (i.e. equation 1.32) and interest rate from (A32) and (A33) as $\omega_t = \dfrac{(1-a)\Psi^{1-a}}{1-qn_t} k_t$ and $r_t = a\Psi^{1-a} = \hat{r}$ respectively.

7. FOCs and work of solutions for utility maximisation in Section 1.5.2

Given the budget constraint that consists of equation (1.29), $c_t = (1-\tau)(1-qn_t)\omega_t - s_t$ and $c_{t+1} = r_{t+1}s_t - x_{t+1}$, we can rewrite equation (1.1) as an unconstraint utility function as follows

$$\max_{s_t, n_t, x_{t+1}} u^t = \ln\left[(1-\tau)(1-qn_t)\omega_t - s_t\right] + \beta \ln\left[r_{t+1}s_t - x_{t+1}\right] + \beta \ln\left[(1-\delta)p_{t+1} + f(\delta p_{t+1})x_{t+1}\right] + \gamma \ln n_t \tag{A34}$$

The optimisation problem of an individual is to maximise (A34). Taking ω_t, r_{t+1} and p_{t+1} as given, the FOCs for s_t, n_t and x_{t+1} are

$$s_t : \frac{\beta r_{t+1}}{r_{t+1}s_t - x_{t+1}} - \frac{1}{(1-\tau)(1-qn_t)\omega_t - s_t} = 0, \tag{A35}$$

$$n_t : \frac{\gamma}{n_t} - \frac{q(1-\tau)\omega_t}{(1-\tau)(1-qn_t)\omega_t - s_t} = 0, \tag{A36}$$

$$x_{t+1} : \frac{f(\delta p_{t+1})}{(1-\delta)p_{t+1} + f(\delta p_{t+1})x_{t+1}} - \frac{1}{r_{t+1}s_t - x_{t+1}} = 0. \tag{A37}$$

Rearrange (A37) so as to obtain

$$x_{t+1} = \frac{r_{t+1}s_t}{2} - \frac{(1-\delta)p_{t+1}}{2f(\delta p_{t+1})}. \tag{A38}$$

Next, we use $r_{t+1} = \hat{r} = a\Psi^{1-a}$, (1.6), (1.9), (1.15) and (1.32) in (A35) and substitute out (A38) in (A35) to obtain expression (1.33). That is

$$s_t = \frac{\left[2\beta(1-\tau) - \frac{\hat{r}^{-1}(1-\delta)\tau}{f\left(\delta\tau(1-a)\Psi^{1-a}k_t\right)}\right]}{1+2\beta}(1-a)\Psi^{1-a}k_t.$$

Now, substitute (1.33) back in (A38), and combine together with $r_{t+1} = \hat{r} = a\Psi^{1-a}$, (1.6), (1.9), (1.15) and (1.32) so as to obtain expression (1.35). That is

$$x_{t+1} = \frac{\left[\beta\hat{r}(1-\tau) - \frac{(1+\beta)(1-\delta)\tau}{f\left(\delta\tau(1-a)\Psi^{1-a}k_t\right)}\right]}{1+2\beta}(1-a)\Psi^{1-a}k_t.$$

Rearrange (A36) yields

$$\frac{\gamma}{qn_t(1-\tau)\omega_t} = \frac{1}{(1-\tau)(1-qn_t)\omega_t - s_t}. \tag{A39}$$

Substitute (A38) in (A35) and then equate (A35) with (A39) results in

$$s_t = \frac{q}{\gamma}\left(2\beta(1-\tau)\omega_t\right)n_t - \frac{(1-\delta)p_{t+1}}{r_{t+1}f(\delta p_{t+1})}. \tag{A40}$$

Next, given (1.32) for ω_t, substituting (1.6), (1.9) and (1.15) in (A40), we attain

$$qn_t\left((1-\tau)r_{t+1}f(\delta p_{t+1})(1+\gamma+2\beta) + \gamma(1-\delta)\tau\right) = \gamma\left((1-\tau)r_{t+1}f(\delta p_{t+1}) + (1-\delta)\tau\right). \tag{A41}$$

Divide through (A41) by $(1+\gamma+2\beta)\hat{r}f(\delta\tau(1-a)\Psi^{1-a}k_t)(1-\tau)$ and rearrange the terms. Finally, using $r_{t+1} = \hat{r} = a\Psi^{1-a}$ in (A41), yields expression (1.34). That is

$$n_t = \frac{1}{q} \times \frac{\dfrac{\gamma}{1+2\beta+\gamma} + \dfrac{\gamma[\hat{r}(1+2\beta+\gamma)(1-\tau)]^{-1}(1-\delta)\tau}{f\left(\delta\tau(1-a)\Psi^{1-a}k_t\right)}}{1 + \dfrac{\gamma[\hat{r}(1+2\beta+\gamma)(1-\tau)]^{-1}(1-\delta)\tau}{f\left(\delta\tau(1-a)\Psi^{1-a}k_t\right)}} = n(k_t).$$

Appendix B

Appendix for Chapter 2

1. Profit Maximisation by Intermediate and Final Good Producers

Final goods producers will choose quantities for y_{it} so as to maximise profits $\left(\int_0^1 y_{it}^{\frac{\sigma-1}{\sigma}} di\right)^{\frac{\sigma}{\sigma-1}} - \int_0^1 P_{it} y_{it} di$. The first order condition can be eventually written as

$$\left(\int_0^1 y_{it}^{\frac{\sigma-1}{\sigma}} di\right)^{\frac{\sigma}{\sigma-1}-1} y_{it}^{\frac{\sigma-1}{\sigma}-1} = P_{it}. \tag{B1}$$

Multiplying (B1) by y_{it} and integrating both sides of the resulting expression leads to

$$\left(\int_0^1 y_{it}^{\frac{\sigma-1}{\sigma}} di\right)^{\frac{\sigma}{\sigma-1}-1} \left(\int_0^1 y_{it}^{\frac{\sigma-1}{\sigma}} di\right) = \int_0^1 P_{it} y_{it} di. \tag{B2}$$

Now, we can divide (B1) and (B2) by parts and get

$$\frac{y_{it}^{\frac{\sigma-1}{\sigma}-1}}{\int_0^1 y_{it}^{\frac{\sigma-1}{\sigma}} di} = \frac{P_{it}}{\int_0^1 P_{it} y_{it} di}, \tag{B3}$$

in which we can substitute (2.3) to get

$$\frac{y_{it}^{\frac{\sigma-1}{\sigma}-1}}{Y_t^{\frac{\sigma-1}{\sigma}}} = \frac{P_{it}}{\int_0^1 P_{it} y_{it} di}. \tag{B4}$$

Given Equation (2.3), the price level $P_t = \left(\int_0^1 P_{it}^{1-\sigma} di\right)^{1/(1-\sigma)}$ implies that

$$\int_0^1 P_{it} y_{it} di = P_t Y_t. \tag{B5}$$

Substitute (B5) in (B4) and rearrange terms in order to solve for y_{it}. Eventually, we get

$$y_{it} = \left(\frac{P_{it}}{P_t} \right)^{-\sigma} Y_t \,, \tag{B6}$$

which is the result of Equation (2.11), after we use the notation $p_{it} = \frac{P_{it}}{P_t}$ to indicate the relative price.

Next, substitute (2.11) in (2.4), we have

$$\max_{p_{it}} \pi_{it}^{variable} = \left(p_{it}(1-\tau p_{it}) - m_t \right) p_{it}^{-\sigma} Y_t. \tag{B7}$$

The optimisation problem of an entrepreneur is to maximise (B7). Therefore, the FOC for (B7) with respect to p_{it} is

$$p_{it} : Y_t \left(-\sigma p_{it}^{-\sigma-1} (p_{it}(1-\tau\mu_{it}) - m_t) + p_{it}^{-\sigma}(1-\tau\mu_{it}) \right) = 0. \tag{B8}$$

Rearrange (B8) so that we can obtain (2.12). That is

$$p_{it} = \frac{\sigma}{(\sigma-1)(1-\tau\mu_{it})} m_t. \tag{2.12}$$

2. Cost Minimisation Problem of Entrepreneur

The cost minimising problem is $\min_{L_{it},\,K_{it}} w_t L_{it} + R_t K_{it}$ subject to (2.1). It can be solved using Langrangean

$$\Lambda_t = w_t L_{it} + R_t K_{it} + m_t \left[y_{it} - B K_{it}^{\beta} (A_t L_{it})^{1-\beta} \right]. \tag{B10}$$

Therefore, the FOCs of (B10) with respect to L_t and K_t are

$$L_t : w_t - m_t(1-\beta)BK_{it}^{\beta}L_{it}^{-\beta}A_t^{1-\beta} = 0, \tag{B11}$$

and

$$K_t : \mathrm{R}_t - m_t\beta BK_{it}^{\beta-1}(A_tL_{it})^{1-\beta} = 0, \tag{B12}$$

respectively.

Now, it is straightforward that (B11) and (B12) yield wage rate and gross interest rate as in (2.13) and (2.14) respectively.

3. Household Utility Maximisation Problem

Substituting $c_t^{t-1} = (1+h_t^{young})w_t(1-qn_t) - s_t$ and $c_{t+1}^{t-1} = (1+h_{t+1}^{old})r_{t+1}s_t$ in (2.6). Therefore, we have unconstrained utility function as follows

$$\max_{s_t, n_t} U^t = \ln\left[(1+h_t^{young})w_t(1-qn_t) - s_t\right] + \gamma \ln n_t + \psi_t \ln\left[(1+h_{t+1}^{old})r_{t+1}s_t\right]. \tag{B13}$$

The optimisation problem of an individual is to maximise (B13). Taking ψ_t, w_t and r_{t+1} as given, the FOCs of (B13) are

$$s_t : \frac{\psi_t}{s_t} - \frac{1}{(1+h_t^{young})w_t(1-qn_t) - s_t} = 0, \tag{B14}$$

$$n_t : \frac{\gamma}{n_t} - \frac{q(1+h_t^{young})w_t}{(1+h_t^{young})w_t(1-qn_t) - s_t} = 0. \tag{B15}$$

Rearrange terms in (B14) to obtain equation (2.20) as follows

$$s_t = \frac{\psi_t}{1+\psi_t}(1+h_t^{young})w_t(1-qn_t).$$

Next, substitute (2.20) in (B15) and rearrange terms yield equation (2.21). That is

$$n_t = \frac{\gamma}{q(1+\gamma+\psi_t)}.$$

4. The Goods Market Equilibrium

The goods market equilibrium requires

$$Y_t = C_t + I_t + g_t, \tag{B16}$$

where C_t is the total consumption expenditure and I_t is investment. With regards to the latter, full depreciation implies that $I_t = K_{t+1}$. As for consumption, it is composed of the consumption expenditures of the current young and the current (surviving) old, as well as the consumption spending of entrepreneurs. Formally, $C_t = N_t c_t^{t-1} + \psi_{t-1} N_{t-1} c_t^{t-2} + \pi_t$. Substituting these in (B16) we have

$$Y_t = N_t c_t^{t-1} + \psi_{t-1} N_{t-1} c_t^{t-2} + \pi_t + K_{t+1} + g_t. \tag{B17}$$

Now, let us substitute (2.19), (2.32) and the budget constraint $c_t^{t-1} = (1+h_t^{young}) w_t (1-qn_t) - s_t$ in Equation (B17) to get

$$Y_t = N_t (1+h_t^{young}) w_t (1-qn_t) - N_t s_t + \psi_{t-1} N_{t-1} c_t^{t-2} + K_{t+1} + \frac{1}{\sigma} y_t \ . \tag{B18}$$

Further substitution of $y_t = Y_t$, $k_t = \dfrac{K_t}{N_t}$, (2.18) and (2.29) yields

$$y_t = \frac{\sigma-1}{\sigma}(1-\beta) B\Theta^{1-\beta} K_t (1-qn_t) - N_t s_t + \psi_{t-1} N_{t-1} c_t^{t-2} + K_{t+1} + \frac{1}{\sigma} y_t \Rightarrow$$

$$y_t = \frac{\sigma-1}{\sigma}(1-\beta) y_t - N_t s_t + \psi_{t-1} N_{t-1} c_t^{t-2} + K_{t+1} + \frac{1}{\sigma} y_t. \tag{B19}$$

Now, check that by virtue of (2.18), (2.24), (2.28), (2.31) and the constraint $c_t^{t-2} = (1+h_t^{old})r_t s_{t-1}$, we have

$$\psi_{t-1}N_{t-1}c_t^{t-2} =$$

$$\psi_{t-1}N_{t-1}(1+h_t^{old})r_t s_{t-1} =$$

$$\psi_{t-1}N_{t-1}(1+h_t^{old})\frac{R_t}{\psi_{t-1}}\frac{K_t}{N_{t-1}} =$$

$$(1+h_t^{old})R_t K_t =$$

$$\left(1+\frac{\tau\mu_t}{1-\tau\mu_t}\right)(1-\tau\mu_t)\frac{\sigma-1}{\sigma}\beta B\Theta^{1-\beta}K_t(1-qn_t) =$$

$$\frac{\sigma-1}{\sigma}\beta y_t. \tag{B20}$$

Since (B20) establishes that $\psi_{t-1}N_{t-1}c_t^{t-2} = \frac{\sigma-1}{\sigma}\beta y_t$, we can substitute back in (B19) to get

$$y_t = \frac{\sigma-1}{\sigma}(1-\beta)y_t - N_t s_t + \psi_{t-1}N_{t-1}c_t^{t-2} + K_{t+1} + \frac{1}{\sigma}y_t \Rightarrow$$

$$y_t = \frac{\sigma-1}{\sigma}(1-\beta)y_t - N_t s_t + \psi_{t-1}N_{t-1}c_t^{t-2} + K_{t+1} + \frac{1}{\sigma}y_t \Rightarrow$$

$$y_t = \frac{\sigma-1}{\sigma}(1-\beta)y_t - N_t s_t + \frac{\sigma-1}{\sigma}\beta y_t + K_{t+1} + \frac{1}{\sigma}y_t \Rightarrow$$

$$K_{t+1} = N_t s_t,$$

that holds by virtue of the financial market equilibrium condition in (2.24).

5. Derivation of Government Consumption, g_t in 2.32

The equilibrium that is symmetric across entrepreneurs implies that $P_{it} = P_t$, $K_{it} = K_t$, $L_{it} = L_t$, $\mu_{it} = \mu_t$ and $y_{it} = y_t$ for every i. Therefore, we can rewrite 2.7 as

$$\tau\mu_t y_t = h_t^{young} w_t (1 - qn_t) N_t + h_t^{old} r_t s_{t-1} \psi_{t-1} N_{t-1} + g_t \tag{B20}$$

Now, substituting 2.16, 2.17 and 2.31 in 2.7 yields

$$\tau\mu_t y_t = \frac{\tau\mu_t}{1-\tau\mu_t}(1-\tau\mu_t)\frac{\sigma-1}{\sigma}(1-\beta)BK_t^{\beta} A_t^{1-\beta} L_t^{-\beta}(1-qn_t)N_t$$

$$+\frac{\tau\mu_t}{1-\tau\mu_t}(1-\tau\mu_t)\frac{\psi_{t-1}}{\psi_{t-1}}\frac{\sigma-1}{\sigma}\beta BK_t^{\beta-1}(A_t L_t)^{1-\beta} s_{t-1} N_{t-1} \tag{B21}$$

$$+g_t.$$

Furthermore, using $s_{t-1}N_{t-1} = K_t$ in (B21), we obtain

$$\tau\mu_t y_t = \frac{\tau\mu_t}{1-\tau\mu_t}(1-\tau\mu_t)\frac{\sigma-1}{\sigma}(1-\beta)BK_t^{\beta} A_t^{1-\beta} L_t^{-\beta}(1-qn_t)N_t$$

$$+\frac{\tau\mu_t}{1-\tau\mu_t}(1-\tau\mu_t)\frac{\psi_{t-1}}{\psi_{t-1}}\frac{\sigma-1}{\sigma}\beta BK_t^{\beta-1}(A_t L_t)^{1-\beta} K_t \Rightarrow$$

$$+g_t.$$

$$\tau\mu_t y_t = \tau\mu_t \frac{\sigma-1}{\sigma}(1-\beta) y_t + \tau\mu_t \frac{\sigma-1}{\sigma}\beta y_t + g_t. \tag{B22}$$

Finally, rearrange the terms so as to obtain g_t. That is

$$g_t = \tau\mu_t y_y - \tau\mu_t \frac{\sigma-1}{\sigma} y_y \Rightarrow$$

$$g_t = \tau\mu_t \frac{1}{\sigma} y_y.$$

6. Derivation of economic growth equation, $\Omega(\mu_t)$ in 2.33

Substituting (2.20), (2.22), (2.27) and (2.31) in 2.26) yields

$$k_{t+1} = \frac{\frac{\Psi(1/\mu_t)}{1+\Psi(1/\mu_t)}(1+\frac{\tau\mu_t}{1-\tau\mu_t})(1-\tau\mu_t)\frac{\sigma-1}{\sigma}(1-\beta)B\Theta^{1-\beta}k_t(1-q\frac{\gamma}{q[1+\gamma+\Psi(1/\mu_t)]})^{-\beta}}{\frac{\gamma}{q[1+\gamma+\Psi(1/\mu_t)]}} \times$$

$$(1-q\frac{\gamma}{q[1+\gamma+\Psi(1/\mu_t)]})$$

(B23)

Divide through both sides of (B23) by k_t and simplify the terms, we have

$$\frac{k_{t+1}}{k_t} = \frac{(\sigma-1)(1-\beta)B\Theta^{1-\beta}q}{\sigma\gamma}\Psi(1/\mu_t)\left[\frac{1+\gamma+\Psi(1/\mu_t)}{1+\Psi(1/\mu_t)}\right]^{\beta}. \quad \text{(B24)}$$

Finally, subtract both sides of (B24) by 1. That is

$$\frac{k_{t+1}}{k_t} - 1 = \Omega(\mu_t) = \frac{(\sigma-1)(1-\beta)B\Theta^{1-\beta}q}{\sigma\gamma}\Psi(1/\mu_t)\left[\frac{1+\gamma+\Psi(1/\mu_t)}{1+\Psi(1/\mu_t)}\right]^{\beta} - 1. \quad (2.33)$$

APPENDIX C

Appendix for Chapter 3

1. First order conditions for individuals' utility maximisation.

Substitute (3.3), (3.4) and (3.5) in (3.2) yield unconstrained utility function as follows

$$\max_{s_t, x_{t+1}} U_t = \ln[w_t + \varrho(1-\gamma)F_t - s_t] + \beta\ln[H(x_{t+1} + \gamma F_{t+1})^{\varepsilon}] +$$
$$\beta\ln[r_{t+1}s_t + (1-\varrho)(1-\gamma)F_{t+1} - x_{t+1}] \tag{C1}$$

The individual's optimisation problem is to maximise (C1). Now, taking w_t, r_{t+1} and F_{t+1} as given, we take partial derivatives of (C1) with respect to s_t and x_{t+1} separately and set each of them equal to zero. Hence, we have the first order conditions (FOCs) for s_t and x_{t+1} respectively

$$s_t : \frac{1}{w_t + \varrho(1-\gamma)F_t - s_t} = \frac{\beta r_{t+1}}{r_{t+1}s_t + (1-\varrho)(1-\gamma)F_{t+1} - x_{t+1}} \tag{C2}$$

$$x_{t+1} : \frac{1}{r_{t+1}s_t + (1-\varrho)(1-\gamma)F_{t+1} - x_{t+1}} = \frac{\varepsilon}{x_{t+1} + \gamma F_{t+1}} \tag{C3}$$

Rearrange (C2), we have

$$s_t = \frac{1}{1+\beta}\left[\beta\left(w_t + \varrho(1-\gamma)F_t\right) + \frac{x_{t+1}}{r_{t+1}} - \frac{(1-\varrho)(1-\gamma)F_{t+1}}{r_{t+1}}\right] \tag{C4}$$

Similarly, rearrange (C3) we get

$$x_{t+1} = \frac{1}{1+\varepsilon}\left(\varepsilon r_{t+1}s_t + \varepsilon(1-\varrho)(1-\gamma)F_{t+1} - \gamma F_{t+1}\right) \tag{C5}$$

We substitute (C4) in (C5). Eventually, we get equation (3.9)

$$x_{t+1} = \frac{\beta\varepsilon r_{t+1}\left[w_t + \varrho(1-\gamma)F_t\right]}{1+\beta(1+\varepsilon)} - \frac{\left[\gamma(1+\beta) - \beta\varepsilon(1-\varrho)(1-\gamma)\right]}{1+\beta(1+\varepsilon)}F_{t+1}.$$

Now, we can utilise equation (3.9) in (C4) to obtain equation (3.8) as follows

$$s_t = \frac{\beta(1+\varepsilon)\left[w_t + \varrho(1-\gamma)F_t\right]}{1+\beta(1+\varepsilon)} - \frac{\left[1-\varrho(1-\gamma)\right]F_{t+1}}{\left[1+\beta(1+\varepsilon)\right]r_{t+1}}.$$

2. First order conditions for profit maximisation of firms.

Profit function for any firm can be written as

$$\max_{L_t, K_t} \pi_t = AK_t^a(Z_tL_t)^{1-a} - (w_tL_t + r_tK_t). \tag{C6}$$

The optimisation problem of any firm is to maximise (C6). Now, taking Z_t as given, we can obtain the FOCs of L_t and K_t by taking the first derivative of (C6) with respect to L_t and K_t separately and equate each of them to zero. Therefore we have the FOCs as follows

$$L_t : w_t - (1-a)AK_t^{a}Z_t^{1-a}L_t^{-a} = 0, \tag{C7}$$

and

$$K_t : r_t - aAK_t^{-a}(Z_tL_t)^{1-a} = 0. \tag{C8}$$

Profit maximisation of firms requires marginal products of inputs equal their marginal costs. Therefore, we can rewrite (C7) and (C8) as

$$w_t = (1-a)AK_t^a Z_t^{1-a} L_t^{-a}, \tag{C7a}$$

and

$$r_t = aAK_t^{a-1}(Z_t L_t)^{1-a}. \tag{C8b}$$

respectively. Substituting Z_t using equation (3.7) in both (C7a) and (C8b), and writing them in per worker term (i.e. $k_t = \frac{K_t}{L_t}$) leads to equation (3.10) and (3.11) respectively. That is

$$w_t = (1-a)A\theta^{1-a}k_t,$$

and

$$r_t = aA\theta^{1-a}.$$

3. Sufficient but not necessary condition of positive x_{t+1}.

We need to find the threshold of f to ensure that $x_{t+1} > 0$. We begin with substituting (3.1) in (3.9). Then, we combine (3.10) and (3.11) in (3.9) to get

$$x_{t+1} = \frac{A\theta^{1-a}k_t}{1+\beta(1+\varepsilon)} \times \{.\}, \tag{C9}$$

Where $\{.\} =$

$$a\beta\varepsilon A\theta^{1-a}\left[(1-a)+\varrho(1-\gamma)f\right] - \frac{k_{t+1}}{k_t}\left[(1+\beta)\gamma - \beta\varepsilon(1-\varrho)(1-\gamma)\right]f. \tag{C10}$$

For $x_{t+1} > 0$, it has to be $\{.\} > 0$. Given (3.14), it is

$$\frac{k_{t+1}}{k_t} = \frac{a\beta(1+\varepsilon)A\theta^{1-a}\left[(1-a)+\varrho(1-\gamma)f\right]}{a\left[1+\beta(1+\varepsilon)\right]+\left[1-\varrho(1-\gamma)\right]f}. \tag{C11}$$

Now, substitute (C11) in (C10), we get

$$a\beta\varepsilon A\theta^{1-a}\left[(1-a)+\varrho(1-\gamma)f\right] - \frac{a\beta(1+\varepsilon)A\theta^{1-a}\left[(1-a)+\varrho(1-\gamma)f\right]}{a\left[1+\beta(1+\varepsilon)\right]+\left[1-\varrho(1-\gamma)\right]f} \times$$

$$\left[(1+\beta)\gamma - \beta\varepsilon(1-\varrho)(1-\gamma)\right]f. \tag{C12}$$

For $\{.\} > 0$, expression (C12) has to be positive. Hence,

$$a\beta\varepsilon A\theta^{1-a}\left[(1-a)+\varrho(1-\gamma)f\right]$$

$$>$$

$$\frac{a\beta(1+\varepsilon)A\theta^{1-a}\left[(1-a)+\varrho(1-\gamma)f\right]}{a\left[1+\beta(1+\varepsilon)\right]+\left[1-\varrho(1-\gamma)\right]f}\left[(1+\beta)\gamma - \beta\varepsilon(1-\varrho)(1-\gamma)\right]f.$$

$$\Rightarrow$$

$$a\varepsilon\left[1+\beta(1+\varepsilon)\right]+\varepsilon\left[1-\varrho(1-\gamma)\right]f+\beta\varepsilon(1+\varepsilon)(1-\varrho)(1-\gamma)f > (1+\varepsilon)(1+\beta)\gamma f. \tag{C13}$$

For (C13) to hold, it is therefore sufficient (though not necessary) that it holds when $\gamma = 1$. That is

$$a\varepsilon\left[1+\beta(1+\varepsilon)\right] > (1+\varepsilon)(1+\beta)f. \tag{C14}$$

Hence, from (C14), eventually we get

$$f < a\varepsilon = \widehat{f}. \tag{C15}$$

So, it is sufficient to assume that f is sufficiently small to ensure that the above holds. Then, $x_{t+1} > 0 \;\; \forall t.$ $\square$.

4. Proof of positive s_t.

We can attempt to find a threshold of f for which $s_t > 0$. First, we substitute (3.1), (3.10) and (3.11) in (3.8). Thus, we have the following.

$$s_t = \frac{\beta(1+\varepsilon)A\theta^{1-a}k_t\left[(1-a)+\varrho(1-\gamma)f\right]}{1+\beta(1+\varepsilon)} - \frac{\left[1-\varrho(1-\gamma)\right]fk_{t+1}}{\left[1+\beta(1+\varepsilon)\right]a}. \tag{C16}$$

As we can see from (C16), it is very clear that $s_t > 0$ iff

$$\beta(1+\varepsilon)A\theta^{1-a}k_t\left[(1-a)+\varrho(1-\gamma)f\right] > \frac{\left[1-\varrho(1-\gamma)\right]fk_{t+1}}{a} \tag{C17}$$

Rearrange (C17) as follows

$$\beta(1+\varepsilon)A\theta^{1-a}\left[(1-a)+\varrho(1-\gamma)f\right] > \frac{\left[1-\varrho(1-\gamma)\right]f}{a}\frac{k_{t+1}}{k_t} \tag{C18}$$

Given (3.14), we substitute (C11) into (C18). This yields

$$\begin{gathered}\beta(1+\varepsilon)A\theta^{1-a}\left[(1-a)+\varrho(1-\gamma)f\right] \\ > \\ \frac{\left[1-\varrho(1-\gamma)\right]f}{a}\times\frac{a\beta(1+\varepsilon)A\theta^{1-a}\left[(1-a)+\varrho(1-\gamma)f\right]}{a\left[1+\beta(1+\varepsilon)\right]+\left[1-\varrho(1-\gamma)\right]f}\end{gathered} \tag{C19}$$

Rearrange (C19), we have

$$a\left[1+\beta(1+\varepsilon)\right]+\left[1-\varrho(1-\gamma)\right]f>\left[1-\varrho(1-\gamma)\right]f$$

$$\Rightarrow$$

$$a\left[1+\beta(1+\varepsilon)\right]>0$$

This shows that for any value of $f>0$, s_t is always positive. $\square$.

5. FOCs and work of solutions for utility maximisation in Section 3.5

Substitute (3.3)-(3.4) and (3.15) into (3.2). Therefore, we have unconstrained utility function as follows

$$\max_{s_t, x_{t+1}} U_t = \ln[w_t+\varrho(1-\gamma)F_t-s_t\,]+\beta\ln[H(x_{t+1}+\varphi\gamma F_{t+1})^{\varepsilon}]+ \\ \beta\ln[r_{t+1}s_t+(1-\varrho)(1-\gamma)F_{t+1}-x_{t+1}] \tag{C20}$$

The optimisation of individual is to maximise (C20). Taking w_t, r_{t+1} and F_{t+1} as given, we take partial derivatives of (C20) with respect to s_t and x_{t+1} separately and set each of them equal to zero. Hence, we have the first order conditions (FOCs) for s_t and x_{t+1} as follows

$$s_t:\frac{1}{w_t+(1-\gamma)\varrho F_t-s_t}=\frac{\beta r_{t+1}}{r_{t+1}s_t+(1-\gamma)(1-\varrho)F_{t+1}-x_{t+1}}, \tag{C21}$$

$$x_{t+1}:\frac{\varepsilon}{x_{t+1}+\varphi\gamma F_{t+1}}=\frac{1}{r_{t+1}s_t+(1-\gamma)(1-\varrho)F_{t+1}-x_{t+1}}. \tag{C22}$$

Next, we rearrange (C22) so as to obtain

$$x_{t+1} = \frac{1}{(1+\varepsilon)}\left([\varepsilon(1-\gamma)(1-\varrho)-\varphi\gamma]F_{t+1}+\varepsilon r_{t+1}s_t\right). \tag{C23}$$

Using (C23) in (C21) yields (3.17). Substituting out (3.17) back into (C23) we have (3.18.)

6. Profit Maximisation Problem of Firm

Using (3.16) and firm's cost function i.e. $w_t L_t + r_t K_t$, we can write profit function as

$$\max_{L_t, K_t} \pi_t = AK_t^a (Z_t L_t)^{1-a} - (w_t L_t + r_t K_t). \tag{C24}$$

The optimisation problem of a firm is to maximise (C24). Take the first derivative of (C24) with respect to L_t and K_t separately and set each of them equal to zero. Consequently, the FOCs of (C24) with respect to L_t and K_t are

$$L_t : w_t - (1-a)A_t K_t^{a} Z_t^{1-a} L_t^{-a} = 0, \tag{C25}$$

and

$$K_t : r_t - aA_t K_t^{-a}(Z_t L_t)^{1-a} = 0. \tag{C26}$$

Given (3.17) and $k_t = \dfrac{K_t}{L_t}$, we can establish equilibrium wage rate from (C24) and interest rate from (C26) as (3.18) and (3.19) respectively.

7. Proof of Proposition 3.

As $\frac{\partial \Phi(f)}{\partial f}$ sign depends on the sign of $V(f)$, it is useful to analyse the properties of $V(f)$ in equation (3.25). It is obvious that $V(0) = \lambda a(1-a)(1+\beta(1+\varepsilon)) > 0$ and $V(\infty) = \infty$. Now, taking the first derivative of $V(f)$, we obtain the following

$$V'(f) = 2\lambda(1-\gamma)\varrho[(1-\gamma)(1-\varrho)+\varphi\gamma]f + (1+\lambda)a(1+\beta(1+\varepsilon))(1-\gamma)\varrho$$
$$-(1-\lambda)(1-a)[(1-\gamma)(1-\varrho)+\varphi\gamma]. \tag{C27}$$

From (C27), we can have

$V'(0) = (1+\lambda)a(1+\beta(1+\varepsilon))(1-\gamma)\varrho - (1-\lambda)(1-a)[(1-\gamma)(1-\varrho)+\varphi\gamma]$ and

$V'(\infty) = \infty$.

Depending on conditions, we can have $V'(0)$ to be positive or negative.

Precisely,

$V'(0) > 0$ iff $(1+\lambda)a(1+\beta(1+\varepsilon))(1-\gamma)\varrho > (1-\lambda)(1-a)[(1-\gamma)(1-\varrho)+\varphi\gamma]$.

Hence, $V(f) > 0 \ \forall f$. Therefore, as $V(f) > 0$, this implies that $\frac{\partial \Phi(f)}{\partial f} > 0$.

(See Figure C1).

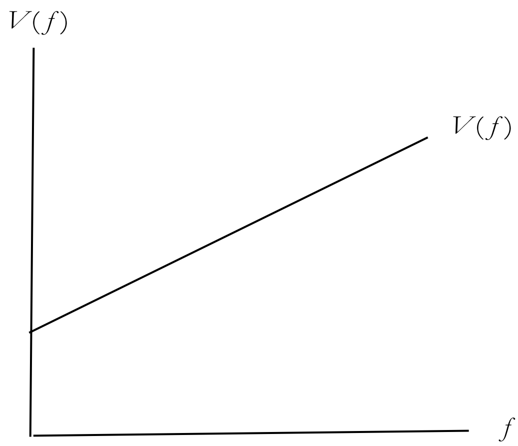

Figure C 1

□.

8. Proof of Proposition 4.

Similar to the proof of Proposition 2, the sign of $\dfrac{\partial\Phi(f)}{\partial f}$ sign depends on the sign of $V(f)$.

$V'(0)<0$ iff $(1+\lambda)a(1+\beta(1+\varepsilon))(1-\gamma)\varrho<(1-\lambda)(1-a)[(1-\gamma)(1-\varrho)+\varphi\gamma]$

holds. Hence, there is $\tilde{f}$ such that $V'(\tilde{f})=0$.

This entails

$$2\lambda(1-\gamma)\varrho[(1-\gamma)(1-\varrho)+\varphi\gamma]\tilde{f}$$
$$+\{(1+\lambda)a(1+\beta(1+\varepsilon))(1-\gamma)\varrho-(1-\lambda)(1-a)[(1-\gamma)(1-\varrho)+\varphi\gamma]\}=0. \tag{C28}$$

After some straightforward calculation of (C28), we have;

$$\tilde{f} = \frac{(1-\lambda)(1-a)[(1-\gamma)(1-\varrho)+\varphi\gamma]-(1+\lambda)a(1+\beta(1+\varepsilon))(1-\gamma)\varrho}{2\lambda(1-\gamma)\varrho[(1-\gamma)(1-\varrho)+\varphi\gamma]} > 0. \quad \text{(C29)}$$

Next, we need to determine the sign of $V(\tilde{f})$ to assist us in determination of growth impact of f . To do this, we substitute back (C29) into (3.25) and find the value of λ for which $V(\tilde{f})$ can be positive or negative. We have

$$V(\tilde{f}) = \frac{1}{4\lambda(1-\gamma)\varrho[(1-\gamma)(1-\varrho)+\varphi\varrho]} \times \{.\}, \quad \text{(C30)}$$

where $\{.\} =$

$$\begin{aligned} &4\lambda^2 a(1-a)(1+\beta(1+\varepsilon))(1-\gamma)\varrho[(1-\gamma)(1-\varrho)+\varphi\varrho]- \\ &\left\{(1-\lambda)(1-a)[(1-\gamma)(1-\varrho)+\varphi\varrho]-(1+\lambda)a(1+\beta(1+\varepsilon))\right\}^2. \end{aligned} \quad \text{(C31)}$$

From (C31), $V(\tilde{f}) < 0$ implies that

$$4\lambda^2 a(1-a)(1+\beta(1+\varepsilon))(1-\gamma)\varrho[(1-\gamma)(1-\varrho)+\varphi\varrho]$$

$$<$$

$$\left\{(1-\lambda)(1-a)[(1-\gamma)(1-\varrho)+\varphi\varrho]-(1+\lambda)a(1+\beta(1+\varepsilon))\right\}^2.$$

(C32)

Using (C32), we can find the threshold of λ for which $V(\tilde{f}) < 0$. That is

$$\lambda < \frac{1}{\left\{2a(1-a)((1+\beta(1+\varepsilon))(1-\gamma)\varrho[(1-\gamma)(1-\varrho)+\varphi\varrho]\right\}^{1/2}+(1-a)[(1-\gamma)(1-\varrho)+\varphi\varrho]+a((1+\beta(1+\varepsilon))}$$

$$\times$$

$$(1-a)[(1-\gamma)(1-\varrho)+\varphi\varrho]-a((1+\beta(1+\varepsilon))=\bar{\lambda}\in(0,1). \tag{C33}$$

Therefore, $\lambda < \bar{\lambda} \;\Rightarrow\; V(\tilde{f})<0$. Consequently, there are f_1 and f_2 for which $V(f_1)=V(f_2)=0$ and $f_1<\tilde{f}<f_2$. For any $f<f_1$ and $f>f_2$, $V(f)>0$. $V(f)>0$ implies $\frac{\partial\Psi(f)}{\partial f}>0$. However, for $f_1 \le f \le f_2 \;\Rightarrow\; V(f)<0$. Hence, $\frac{\partial\Psi(f)}{\partial f}<0$. (See Figure C2).

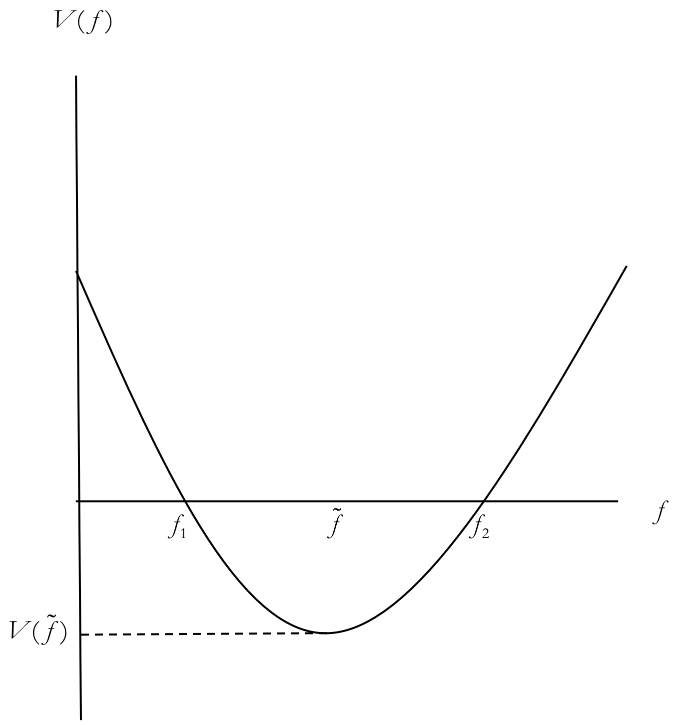

Figure C2

Nevertheless, if $\lambda \geq \bar{\lambda} \Rightarrow V(\hat{f}) \geq 0 \Rightarrow \frac{\partial \Psi(f)}{\partial f} > 0$. (See Figure C3).

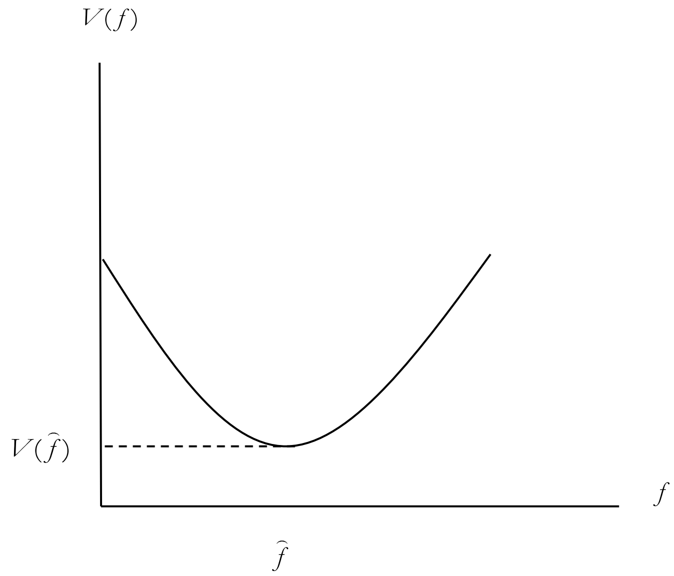

Figure C3

$\square$.

9. Proof of Proposition 5

We can determine the growth impact of γ by analysing $M(\gamma)$ in equation (3.27).

Suppose that $1-\varphi < \varrho$. Furthermore,

$$M(0) = \lambda[(1-a)+\varrho f][a(1+\beta(1+\varepsilon)+(1-\varrho)f] > 0 \text{ and,}$$

$$M'(\gamma) = -2\lambda\varrho[\varphi-(1-\varrho)]\gamma f^2 -$$

$$\left\{(1+\lambda)\varrho[a(1+\beta(1+\varepsilon)+(1-\varrho)f]+(1-\lambda)[(1-a)+\varrho f][\varphi-(1-\varrho)]\right\} f < 0.$$

(C34)

Equation (C34) implies that $M'(0) < 0$ and $M'(1) < 0$. Thus, there is γ^g such that $M(\gamma^g) = 0$. Therefore, for $\gamma < \gamma^g$, we have $M(\gamma) > 0$. On the other hand, for $\gamma^g \leq \gamma \leq 1$, we have $M(\gamma) < 0$. $M(\gamma) > 0 \Rightarrow \dfrac{\partial \Psi(f)}{\partial \gamma} > 0$ and $M(\gamma) < 0 \Rightarrow \dfrac{\partial \Psi(f)}{\partial \gamma} < 0$. (See Figure C4).

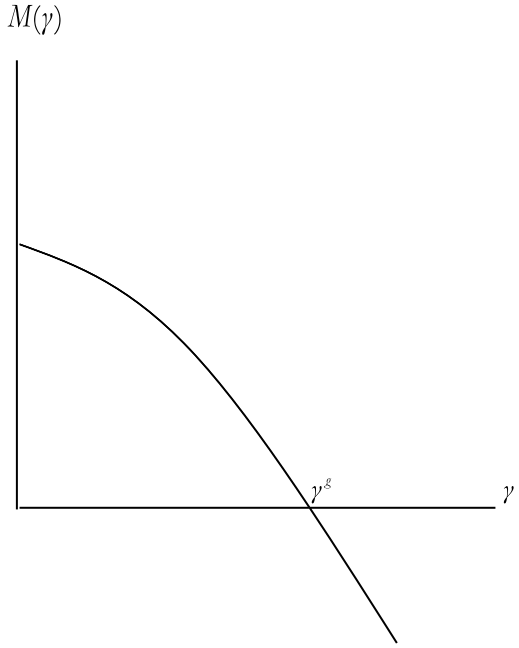

Figure C4

☐ .

10. Proof of Proposition 6

Let $1-\varphi \geq \varrho$, we have $M(0) = \lambda[(1-a)+\varrho f][a(1+\beta(1+\varepsilon)+(1-\varrho)f] > 0$.

Furthermore, we also have $M(1) > 1$ iff ;

$$(1-\lambda)[(1-a)+\varrho f][(1-\varrho)-\varphi] > (1+\lambda)\varrho[a(1+\beta(1+\varepsilon)+(1-\varrho)f] \qquad \text{(C35)}$$

Applying condition in (C35) also leads to $M'(\gamma) > 0$. Hence, we have upward sloping $M(\gamma)$ for $\gamma \in (0,1)$ (i.e. $M(\gamma) > 0$) . This implies $\frac{\partial \Psi(f)}{\partial \gamma} > 0$. (See Figure C5).

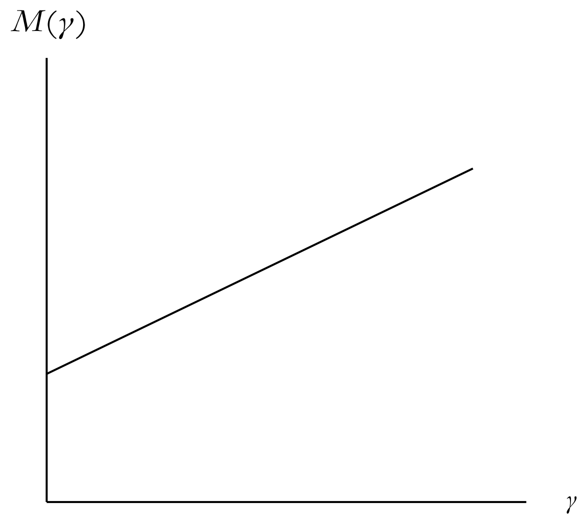

Figure C5

$\square$.

11. Proof of Proposition 7

We take the first derivative of $\Psi(f)$ with respect to φ as follows;

$$\frac{\partial \Psi(f)}{\partial \varphi} = \frac{a\beta(1+\varepsilon)\theta^{1-a}\gamma^{\lambda} f^{\lambda}}{(a(1+\beta(1+\varepsilon)) + [(1-\gamma)(1-\varrho) + \varphi\gamma] f)^2} \times [(1-a) + (1-\gamma)\varrho f] \times \{.\},$$

(C36)

where

$$\{.\} = \lambda[a(1+\beta(1+\varepsilon)+[(1-\gamma)(1-\varrho)+\varphi\gamma]f]+(1-\varphi)\gamma f. \qquad \text{(C37)}$$

The sign of (C36) depends on the sign of $\{.\}$ in (C37). From this, we can derive the threshold of φ, $\overline{\varphi}$. That is

$$\overline{\varphi} = \frac{\lambda a(1+\beta(1+\varepsilon))+[\lambda(1-\gamma)(1-\varrho)+\gamma]f}{(1+\lambda)\gamma f} \in (0,1). \qquad \text{(C38)}$$

Therefore, we have $\dfrac{\partial\Psi(f)}{\partial\varphi}<0 \left(\dfrac{\partial\Psi(f)}{\partial\varphi}>0\right)$ iff $\varphi<\overline{\varphi}$ $(\varphi\geq\overline{\varphi})$. □.

12. Proof of Proposition 8

We take the first derivative of $\Psi(f)$ with respect to ϱ as follows

$$\frac{\partial\Psi(f)}{\partial\varphi} = \frac{a\beta(1+\varepsilon)\theta^{1-a}\gamma^{\lambda}f^{\lambda}}{(a(1+\beta(1+\varepsilon))+f[(1-\gamma)(1-\varrho)+\varphi\gamma])^2}\times\{1+a(1+\beta(1+\varepsilon))+[(1-\gamma)+\varphi\gamma]f\}>0.$$

(C39)

□.