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MULTIPLE EQUILIBRIA IN ECONOMIC GROWTH: A STUDY OF HEALTH-INDUCED POVERTY TRAPS

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Abstract

This study explores the self-reinforcing mechanisms possibly leading to multiple equilibria health-induced poverty traps in the context of health persistence. While theoretical models of multiple equilibria have been extensively studied, empirical validation remains relatively unexplored. We address this gap by extending the model proposed by Agénor (2015), which highlights the role of intergenerational transmission of poor health in the emergence of a health-induced poverty trap. We work with an endogenous probability of surviving from adulthood to old age which is dependent on health outcomes. We depart from Agénor's (2015) key assumption that reduces the system to one dimension, and instead study the two-dimensional system. The model is calibrated using data from India and its dynamics are studied using numerical methods. We find that, while multiple equilibria are a theoretical possibility, there is a unique steady-state equilibrium for plausible parameter ranges. The steady-state level of health status is sensitive to the degree of health persistence, and higher degrees of health persistence are associated with lower steady-state health results. Thus, while a multiple equilibria health-induced poverty trap cannot occur, the economy might converge to a steady-state equilibrium characterized by adverse health conditions that are transmitted to future generations, thus constituting a single equilibrium health-induced poverty trap. We perform a sensitivity analysis and find that an increase in the supply of public health services may not always be effective in improving the steady-state growth rate due to a crowding-out effect, despite the fact that it enhances health outcomes. The results are robust to alternative functional forms.

JEL codes: C62, C63, I15, 041

Keywords: Multiple equilibria; Poverty traps; Economic growth; Longevity; Health

Resumo

A dissertação explora os mecanismos que dão origem a múltiplos equilíbrios e armadilhas da pobreza num contexto de persistência das condições de saúde. Apesar de ser extensa a literatura dedicada ao estudo de modelos teóricos com múltiplos equilíbrios, a sua validação empírica permanece escassa. É analisado o papel da transmissão de condições adversas de saúde a gerações futuras na ocorrência de uma armadilha da pobreza através da extensão do modelo proposto por Agénor (2015). É considerada uma probabilidade de sobrevivência endógena dependente do estado de saúde da população. Esta abordagem diverge da simplificação adotada por Agénor (2015), que reduz o sistema a uma dimensão, sendo estudado o sistema dinâmico em duas dimensões. O modelo é calibrado com dados da Índia e a sua dinâmica é estudada através de métodos numéricos. Apesar da existência de múltiplos equilíbrios ser uma possibilidade teórica, existe apenas um equilíbrio para intervalos razoáveis dos parâmetros. O nível do estado de saúde em equilíbrio é sensível ao grau de persistência de saúde, sendo menor quanto maior o grau de persistência de saúde. Assim, apesar de não ser possível surgir uma armadilha da pobreza caracterizada por múltiplos equilíbrios, é possível que a economia convirja para um equilíbrio caracterizado por condições de saúde desfavoráveis que são transmitidas a gerações futuras, correspondendo a uma armadilha de pobreza caracterizada por um único equilíbrio estável. Um aumento da oferta de serviços públicos de saúde nem sempre é capaz de fomentar um aumento da taxa de crescimento de equilíbrio devido a um efeito de *crowding-out*, apesar de melhorar os resultados de saúde. Os resultados são robustos a formas funcionais alternativas da probabilidade de sobrevivência.

Códigos JEL: C62, C63, I15, 041

Palavras-chave: Múltiplos equilíbrios; Armadilhas da pobreza; Crescimento económico; Longevidade; Saúde

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1 Introduction

The concept of multiple equilibria, in contrast to the standard unique equilibrium, has long been a topic of discussion in economic theory. In the context of economic growth, Solow's (1956) influential paper alludes to the possibility of multiple equilibria under alternative functional forms of the production function.

In the presence of multiple long-run equilibria, a process of equilibrium selection must occur. We focus on systems exhibiting history dependence, meaning that an economy's initial conditions are sufficient to determine its long-run equilibrium, and there may exist initial conditions from which the economy will be directed towards undesirable equilibria (Barrett & Swallow, 2006).

This dissertation is situated at the intersection of the following strands of economic research: multiple equilibria, health and development. It aims to contribute to the literature by exploring an economy characterized by health persistence and determining under which conditions it is subject to a health-induced poverty trap, with a particular focus on empirical validation, a notable gap in current research. While theoretical models of multiple equilibria have been extensively studied, empirical validation remains insufficient, as many studies only suggest ways of avoiding undesirable equilibria without thoroughly examining the empirical plausibility of these multiple equilibria.

This is the case of Agénor (2015), whose model we extend and critically examine. The model describes a three-period overlapping generations (OLG) economy characterized by health persistence and explores potential self-reinforcing mechanisms between health and economic growth. This framework considers that health outcomes are transmitted across generations, and health persistence may be the root of persistent poverty.

We extend the model by simultaneously considering endogenous longevity and a two-dimensional dynamical system, unlike Agénor (2015), who makes a key assumption and reduces the system to one dimension, since studying the two-dimensional system becomes

analytically challenging when the endogeneity of the survival probability is introduced.

We consider an endogenous survival probability from adulthood to old age defined by a piecewise function across three intervals, each representing a stage of development. Based on relevant literature, we introduce a strictly increasing concave functional form which ensures continuity between intervals.

Since the system is defined across three intervals, we analytically study the model and derive conditions of existence and stability of interior equilibria in each of the intervals of the survival probability's domain. The introduction of endogenous longevity defined in a piecewise fashion allows for potential shifts in dynamics between intervals.

An important contribution of this dissertation is the calibration of the model to match data from India, which allows us to use numerical methods to numerically study the dynamics and complement the analytical analysis.

We determine whether the economy is subject to a multiple equilibria health-induced poverty trap, meaning there are multiple steady-state equilibria and the economy might converge to an equilibrium characterized by adverse health conditions, depending on its initial conditions. On the other hand, it might be subject to a single equilibrium health-induced poverty trap, meaning it converges to a unique steady-state equilibrium characterized by adverse health conditions that are transmitted to future generations, regardless of its initial conditions. This distinction has an important role in the design of economic policy.

Finally, we contribute with a sensitivity analysis, where we test whether the same results apply when the survival probability assumes different functional forms, namely a smooth one over the whole domain. Moreover, we test the way the economy's steady-state equilibrium reacts to different degrees of health persistence.

The remainder of this dissertation is organized as follows. Chapter 2 contextualizes the research within the existing literature on multiple equilibria and health-induced poverty traps. Chapter 3 presents the model. Chapter 4 studies the existence and local stability of equilibria, while Chapter 5 focuses on calibrating the model. Chapter 6 provides a sensitivity analysis. Finally, Chapter 7 offers concluding remarks.

2 Literature review

2.1 Historical context of multiple equilibria

Although the notion of a unique and typically stable equilibrium dominates economic discourse, considerations of multiple equilibria are not novel. To account for non-convergent growth, many researchers have adapted standard economic models to feature multiple attractors (Azariadis, 1996). Cerny (2010, p.90) broadly defines multiple equilibria as "the existence of multiple alternative potential future developmental pathways generated by a [system]".

One of the most remarkable mentions of this idea can be found in Solow's (1956) seminal paper, where it is referred to as a conceivable outcome under an alternative functional form of the production function.

While the formalization of multiple equilibria as a distinct concept in economic theory is relatively recent, its underlying ideas have been considered for centuries. Authors such as Graham and Temple (2006) suggest that Malthus' (1836) observation on how countries with nearly equal natural capabilities exhibited very different growth patterns can be regarded as one of the earliest reflections on multiple equilibria.

After World War II, several influential studies based on multiple equilibria and poverty traps as explanations for underdevelopment emerged, such as Nurkse's (1958) theory of the vicious circle of poverty and Nelson's (1956) suggestion that undeveloped economies might be trapped in a low-level stable equilibrium. These studies were central to development economics and significantly shaped its conceptual framework.

In the late 1960s, the rise of neoclassical economics led to a decline in the development of poverty trap theory, as attention shifted towards models focused on capital accumulation and technological progress, typically featuring a a single stable equilibrium (Fu et al., 2024).

The growing interest and advancements in endogenous growth theory in the early 1990s gave rise to influential papers on multiple equilibria (e.g., Azariadis, 1996; Galor, 1996; Krugman,

1995; Matsuyama, 1991). This trend persists in the 21st century, as advanced empirical methods allow researchers to revisit poverty trap theory with greater rigour (Fu et al., 2024).

Nowadays, literature on multiple equilibria is extensive and can be found in various fields of economic theory, such as economic growth and development (e.g., Barrett & Carter, 2013; Futagami & Konishi, 2019), game theory (e.g., Carrera, 2019; De la Croix & Docquier, 2012), monetary economics (e.g., Altermatt et al., 2025; Benhabib et al., 2016) and environmental economics (e.g., Dao & Edenhofer, 2018; Goenka et al., 2020; Varvarigos, 2014).

In the context of underdevelopment, the concept of multiple equilibria is useful when studying persistent differences in development patterns across nations. It is frequently used in studies concerning poverty traps, suggesting that underdeveloped economies are subject to self-reinforcing mechanisms that trap them in low-growth or low-level equilibria they cannot escape (Matsuyama, 1997). Multiple equilibria poverty trap models typically incorporate non-convexities and/or market failures, including externalities, increasing returns, threshold effects and imperfect competition (Santos, 2011). Although poverty traps and multiple equilibria can occur simultaneously, they are distinct concepts. Poverty traps are not limited to models with multiple equilibria, as they can also occur under a single low-level equilibrium; conversely, multiple equilibria may emerge outside of poverty contexts (Barrett & Carter, 2013).

In a single equilibrium poverty trap model, there is a unique steady-state equilibrium, as is common in economic theory, but it is located below a poverty line. However, most literature on poverty traps considers the existence of multiple steady-state equilibria, where it is possible for low-growth and high-growth equilibria to exist simultaneously. In this case, there exist tipping points at the boundaries of the basin of attraction of each stable equilibrium (Barrett et al., 2016).

2.2 Theoretical framework of multiple equilibria

The notion of multiple equilibria encompasses various phenomena with distinctive characteristics, making it crucial to distinguish between them in order to delimit the scope of this dissertation. Thus, the concepts of local and global indeterminacy, as well as history dependence, must be clarified.

Local indeterminacy arises when there is a continuum of equilibrium paths that converge to a unique long-run equilibrium starting from different initial conditions (Brito & Venditti, 2010). For given initial conditions, the trajectory converging to the unique long-run equilibrium

depends on the choice of the jump variables (Beyer & Farmer, 2003). Local indeterminacy is frequently found in studies about economic fluctuations and self-fulfilling expectations (e.g., Antoci et al., 2021).

On the other hand, global indeterminacy arises when, for given initial conditions, there are multiple equilibrium paths that converge to different stationary solutions (Brito & Venditti, 2010). Therefore, the economy's initial conditions are insufficient to determine its long-run dynamic behaviour, since it depends on the choice of the jump variables. This feature is commonly found in studies explaining underdevelopment as a coordination failure, in the sense that economies might converge to undesirable equilibria because agents fail to coordinate their actions (Slobodyan, 2005). Both types of indeterminacy can (and frequently do) occur simultaneously (e.g., Benhabib et al., 2018; Gori & Sodini, 2020).

As highlighted by Krugman (1991) and Matsuyama (1991), systems featuring multiple long-run equilibria undergo a process of equilibrium selection. It is essential to understand whether this process is history dependent or whether expectations play a role, since it determines how policy intervention should be designed.

Global indeterminacy represents the expectations-driven side of the literature, as opposed to the more straightforward history dependence literature. Under history dependence, the equilibrium selection solely depends on the economy's history, meaning that the economy's long-run equilibrium is unique and determinate for given initial conditions (Benhabib & Perli, 1994). In this case, the economy exhibits multiple steady-state equilibria, yet has a unique dynamic equilibrium trajectory starting from any initial conditions (Altermatt et al., 2025), meaning that multiple equilibria can exist in deterministic settings.

This dissertation focuses on such literature and, consequently, on the existence of a threshold (or possibly multiple thresholds), such that the long-run outcome depends on the position of the initial conditions relative to the critical thresholds associated with each equilibrium (Barrett & Swallow, 2006).

Since the economy's initial conditions determine its long-run equilibrium, there might exist initial conditions that direct it towards an undesirable equilibrium, characterized by low growth or a low level of output, for instance.

In such case, it might make sense for the government to intervene to prevent the economy from being stuck in or moving towards undesirable equilibria.

Government intervention must take into consideration the mechanisms giving rise to multiple

equilibria, and be substantial enough to ensure that the economy effectively experiences a change in its long-run equilibrium, rather than merely changing its position within the original equilibrium path (Agénor, 2017).

The government might try to direct the economy towards a high-growth equilibrium in (at least) one of the following ways: by altering its initial conditions, in order to position it on a converging path to the high-growth equilibrium, or by shifting the law of motion governing the system, such that the economy's initial conditions now position it in a path leading to the desired equilibrium (e.g., Agénor, 2015). Under certain circumstances, the latter approach might even reduce the set of equilibria.

Matsuyama (1991) defends that models with multiple equilibria should not be considered unrealistic or incapable of yielding useful predictions. Understanding the conditions and parameter values which yield multiple equilibria is insightful and relevant for policy design. However, it is worth noting that the likelihood of occurrence of multiple equilibria may be overstated in the literature. On the one hand, as noted by Hosoya (2023), the possibility of multiple equilibria is often limited to rather restrictive scenarios, such as very narrow and potentially empirically implausible parameter ranges. Kraay and Raddatz (2007) and Kraay and McKenzie (2014) find little evidence of multiple equilibria for many of the usual mechanisms suggested in the theoretical literature, thus reinforcing this idea.

On the other hand, some of the potential equilibria might be ruled out because they are associated with negative levels of investment, consumption, or other economic variables, rendering them economically meaningless. This point is illustrated by Afonso et al. (2014), who find that Hosoya (2012) proposes two candidates for equilibria, but one of them corresponds to negative levels of consumption, making it economically irrelevant.

2.3 Health and economic growth

In the context of health literature, some research studies the link between health and economic growth, as well as the potential emergence of self-reinforcing dynamics between them.

Health has been regarded as one of the main determinants of productivity and growth since the early 1990s, alongside education (in what can be considered a broad definition of human capital) (Bedir, 2016). While education has traditionally been seen as the primary source of human capital accumulation, its productive impact often depends on whether it is complemented by good health conditions, which enhance mental capacity and cognitive

development (Chakraborty & Das, 2005).

It is widely accepted that health, typically measured by life expectancy at birth and/or adult survival rates, and economic growth are positively linked.

On the one hand, improved health status enhances labour productivity by strengthening the physical and mental capacities of individuals, thus contributing to economic growth (Bucci et al., 2019; Siddiqui et al., 2020).

Additionally, a higher life expectancy broadens individuals' time horizons, producing a life-cycle effect that encourages greater accumulation of physical and human capital, by lowering the risk of not enjoying the future returns on current investments. This leads to an increase in savings and labour market participation (Agénor, 2015; Chakraborty, 2004; Gori & Sodini, 2020; Varvarigos, 2010; Zhang & Zhang, 2005). As stated by Lorentzen et al. (2008, pp. 81-82), "Development occurs only if people make provision for the future. If they see no future, there is no growth."

On the other hand, countries exhibiting sustained growth have more resources to invest in healthcare and improve the health status of the population. In contrast, limited growth restricts the capacity to improve the health status of the population, since individuals are more prone to face malnutrition and food insecurity, as well as difficulties accessing healthcare, and the government has limited funds to intervene (Agénor, 2015; Siddiqui et al., 2020).

Lorentzen et al. (2008) argue that a longer life expectancy extends individuals' time horizons and is associated with a higher growth rate. Similarly, He and Li (2020) investigate the link between life expectancy and GDP for 65 countries from 1980 to 2014, and find a positive correlation over the long run in most countries, though this relationship appears to be influenced by population aging.

Strittmatter and Sunde (2013) investigate the causal effect of health improvements on economic growth using a panel of 12 European countries from 1820 to 2010, and find that introducing public health systems has a significant positive effect on per capita and aggregate income growth.

Nevertheless, the positive link between health and economic growth has been contested in the literature, with some studies suggesting that the causal relationship might be negative or nonexistent.

Lee and Shin (2019) investigate the impact of population aging on economic growth using panel data for 142 countries from 1950 to 2014, and find that an increase in life expectancy, in combination with lower fertility, reduces the share of working-age population and increases

old-age dependency, which has a negative impact on economic growth. Maestas et al. (2023) expect that the rapidly aging population of the United States of America will slow down the country's economic growth by altering the economy's industrial structure.

Wang (2015) suggests that an increase in public health expenditure may lead to a crowding-out effect, whereby spending on other productive sectors, such as education or infrastructure, is reduced, potentially hindering economic growth.

Acemoglu and Johnson (2007) find a negative correlation between health and economic growth from 1940 to 2000, arguing that while better health may enhance labour productivity, lower mortality increases population size, thus reducing per capita inputs. This result was challenged by Bloom et al. (2014), who point out that excluding initial life expectancy from the growth regression might make the results inconsistent.

The relationship between productivity and health is thus multidimensional and influenced by several interacting mechanisms, as was mentioned by Ridhwan et al. (2022), who stress the importance of accounting for endogeneity bias and reverse causality, and find that studies that do not account for endogeneity seem to create an upward bias. Nonlinearities and threshold effects are also commonly found when studying this bidirectional link.

Yang (2020) finds that the impact of health expenditure on growth depends on the economy's level of human capital, becoming negative for low levels of human capital. Wang (2011) asserts that the effectiveness of promoting economic growth through increased health care expenditure varies by income level. In the long run, it has little impact on low and high income countries. In the short run, it positively affects economies with medium and high growth rates.

Cervellati and Sunde (2011) and Hirono (2021) point to a nonlinear effect of life expectancy on income growth per capita, which is contingent on the country's environment and stage of development. Cervellati and Sunde (2011) also argue that the conflicting results in the literature might result from differences in sample composition, particularly regarding the countries and observation periods considered.

A growing body of literature defends that economic activity negatively affects longevity and overall health due to the environmental damage caused by production (primarily through pollution), which may weaken the positive impact of rising income on life expectancy and influence the economy's dynamic behaviour (e.g., Fodha & Seegmuller, 2014; Mariani et al., 2010; Palivos & Varvarigos, 2017; Varvarigos, 2010).

Throughout the following chapters, we study the complementarities between improvements in

health status and economic growth, and find that improved health might not enhance growth, since it leads to a crowding-out effect associated with the public-private capital ratio.

2.4 Health-induced poverty traps

Our research then focuses on the potential self-reinforcing mechanisms leading to health-induced poverty traps. An economy whose population currently exhibits a poor health status may struggle to generate the wealth needed to improve health outcomes. Moreover, there might be intergenerational health persistence, meaning that such poor health outcomes are transmitted across generations, and health persistence may be the root of persistent poverty (Chakraborty & Das, 2005).

Epidemiological research finds that unfavorable intrauterine conditions lead to adverse health conditions at birth (Qian et al., 2017). In addition, these children continue to experience inferior health and human capital conditions throughout their lifetime (Almond & Currie, 2010; Black et al., 2007).

Bhalotra and Rawlings (2011) investigate intergenerational health persistence across time in developing countries, and find that the mother's childhood and current health status are likely to persist to the next generation, thus perpetuating health and wealth inequality.

In this context, government intervention, namely investment and provision of health services, might be recommended to help the economy escape this health-induced poverty trap (e.g., Artekin & Konya, 2020; Atilgan et al., 2017). However, such approach might not be effective in improving health outcomes since public funds are often used inefficiently, particularly in developing economies. Makuta and O'Hare (2015) find that the effect of public health spending on health outcomes depends on the quality of governance in a panel data regression comprising 43 countries in Sub-Saharan Africa between 1996 and 2011.

Some of the factors contributing to such inefficiency are poor governance and corruption, weak healthcare systems, and inadequate infrastructure and human resources (Grigoli & Kapsoli, 2018; Todaro & Smith, 2015). Agénor (2015) takes this into account and associates government spending with an efficiency parameter that measures the degree of resource waste in public intervention.

This study is related to the rich field of literature studying the interaction between health and economic growth in the context of overlapping generations models (e.g., Cipriani & Fiorini, 2021; Kuhn & Prettner, 2016; Prettner, 2013; Zhang & Zhang, 2005; and Zhang et al., 2023).

The increasing longevity observed in recent decades has motivated a rise in research examining the link between life expectancy and economic growth, such as Aísa and Pueyo (2004), Chen et al. (2024), Jouvet et al. (2010), Kunze (2014), Leung and Wang (2010) and Nishimura et al. (2018).

As noted by Jouvet et al. (2010), the mere introduction of endogenous longevity can create self-reinforcing dynamics that may lead to multiple equilibria. Chakraborty (2004) and Blackburn and Cipriani (2002) present seminal work in the class of OLG models where endogenous life expectancy, as determined by the survival probability, can be the source of development traps.

In Chakraborty (2004), the probability of surviving from the first period to the next is endogenous and a non-decreasing concave function of an individual's health status, which can be improved by public health investment. Agénor et al. (2014), Fanti and Gori (2014) and Okada (2020) also work with an endogenous survival probability which is improved through public health expenditure.

Blackburn and Cipriani (2002) consider a survival probability dependent on the economy's level of development and find multiple development regimes depending on its initial conditions. The survival probability is a piecewise function with two constant intervals, which was later adopted by Chen (2010) and Clootens (2017). The same results apply when the function assumes a smooth form, as was confirmed by Blackburn and Cipriani's (2002) numerical simulations. We perform a similar sensitivity analysis in Chapter 6.

Chakraborty and Das (2005) also consider a survival probability subject to thresholds, taking the form of a piecewise function with one constant interval and one interval dependent on the representative agent's private health investment during youth.

The presence of such thresholds means that the process of determining equilibria and studying their local stability is slightly different. Since the system is defined across multiple intervals, it is necessary to take admissibility into account and be mindful of the location of possible solutions in each subdomain (Prieur et al., 2013).

We thoroughly present our extension of Agénor's (2015) model in Chapter 3, which is closely related to the literature presented throughout this chapter. The model describes a three-period OLG economy characterized by intergenerational transmission of health outcomes, subjecting it to a potential health-induced poverty trap. We introduce endogenous longevity defined as piecewise function across three intervals, which is dependent on the average health status of the population.

3 The model

In this chapter, we present our extension of Agénor's (2015) model, making it clear when we depart from the baseline model's assumptions in Sections 3.4 and 3.5.

The model considers an OLG economy where a single marketed good is produced, which can be either consumed immediately, or stored to yield capital in the beginning of the subsequent period. Individuals have a three-period lifespan: childhood, adulthood and old age. They are certain to survive from childhood to adulthood, but face a survival probability from adulthood to old age ranging from 0 to 1.

In the baseline model introduced in Agénor (2015, Sec. 2), the two-dimensional system is solved under the assumption of an exogenous and constant survival probability from adulthood to old age. This assumption is relaxed in Agénor (2015, Sec. 4), where it is assumed that the survival probability is endogenously related to the average health status in the economy and defined using a piecewise function across three intervals. However, when the endogeneity of the survival probability is introduced, the key assumption $\chi = 1$ (which we explain in depth in Section 3.4, where we depart from it) is made to reduce the system to one dimension, since studying the two-dimensional system becomes analytically challenging.

We extend the model by simultaneously considering endogenous longevity and a two-dimensional system, where the average health status of the population and the public-private capital ratio are the state variables. The endogenous survival probability is related to the average health status of the population in the following way: In a first stage of development, the economy exhibits such poor health that it is incapable of translating health status improvements into higher survival probabilities. As the health status improves above the lowest threshold, the survival probability positively and concavely depends on the average health status, and becomes constant again as the economy crosses the highest threshold.

Due to the large number of variables and parameters, we present a non-exhaustive list of their respective meanings and restrictions in Table $B1$.

3.1 Households

An individual born in period t becomes an adult in period $t+1$ and reaches old age in period $t+2$. It is only during adulthood that individuals receive a time endowment normalized to 1, which they allocate to work, child rearing and leisure. This implies that wages earned during adulthood are the only source of income to finance both consumption in adulthood (which encompasses children's consumption during the initial period of life) and old age.

Adults devote a fraction ε_{t+1}^{W} of their one-unit time endowment to market work. Caring for a child requires a fixed time of $\varepsilon^{R} \in (0,1)$, as in Zhang and Zhang (2005). Considering an exogenous fertility rate $n \geq 1$, which represents the number of children each adult bears, leisure is thus defined as $1-\varepsilon_{t+1}^{W}-n\varepsilon^{R}$. All children are born with the same innate abilities and initial health status. At the end of period $t+1$, adults face a probability of survival to old age of $p_{t+1} \in (0,1)$, which is discussed in greater depth in Section 3.5 below.

Moreover, individuals are endowed with an initial stock of physical capital, K_0^P, at time $t=0$, which is held by an initial generation of retirees. Assuming that health does not directly provide utility, the expected lifetime utility at the beginning of period $t+1$ for an individual born in period t is expressed as the logarithmic utility function

$$U = \ln c_{t+1}^{t} + \eta_L \ln(1-\varepsilon_{t+1}^{W}-n\varepsilon^{R}) + p_{t+1}\frac{\ln c_{t+2}^{t}}{1+\rho}. \tag{3.1}$$

Equation (3.1) indicates that an individual born in period t derives utility from consumption and leisure during adulthood, respectively c_{t+1}^{t} and $1-\varepsilon_{t+1}^{W}-n\varepsilon^{R}$, and from consumption during old age, c_{t+2}^{t}, which occurs with a probability of p_{t+1}. [1] The parameter $\rho > 0$ represents the discount rate, and $\eta_L > 0$ represents the individual's relative preference for leisure.

In contrast to some studies, such as those by Zhang and Zhang (2005) and Cipriani and Fioroni (2021), parents do not derive utility from the survival of offspring nor from the duration of each surviving child's lifespan.

The budget constraints during adulthood and old age are given by, respectively,

$$c_{t+1}^{t} + s_{t+1} = (1-\tau)\varepsilon_{t+1}^{W} a_{t+1} w_{t+1}, \tag{3.2}$$

[1] The survival probability is constant and equal to p_m in Agénor's (2015) Equation (1). Hence, our Equation (3.1) represents an adjustment tailored for a context with endogenous longevity. The same applies to Equations (3.3) and (3.4).

$$p_{t+1}c_{t+2}^{t} = (1 + r_{t+2})s_{t+1}, \tag{3.3}$$

where $\tau \in (0, 1)$ denotes the effective income tax rate, a_{t+1} the individual labour efficiency, w_{t+1} the wage rate, r_{t+2} the rental rate of private capital, and s_{t+1} the savings, which can only be held in the form of physical capital.

Solving (3.2) for s_{t+1} and replacing the solution in (3.3) leads to the consolidated budget constraint

$$c_{t+1}^{t} + \frac{p_{t+1}c_{t+2}^{t}}{1 + r_{t+2}} = (1 - \tau)\varepsilon_{t+1}^{W} a_{t+1} w_{t+1}. \tag{3.4}$$

3.2 Firms

A continuum of identical firms, indexed by $i \in (0, 1)$, produce a unique good that cannot be stored and must either be consumed or used for investment. To produce this good, firms use the private inputs, effective labour and private capital (which they rent from the current generation of retirees), along with public capital. Public capital is non-excludable, but partially rival due to congestion effects, which depend on the aggregate private capital stock and the size of the adult population.

The output of firm i is produced according to a Cobb-Douglas technology:

$$Y_t^i = \left[\frac{K_t^I}{(K_t^P)^{\phi_K} N_t^{\phi_N}}\right]^{\alpha} (A_t \varepsilon_t^{W,i} N_t^i)^{\beta} (K_t^{P,i})^{1-\beta}, \tag{3.5}$$

where $K_t^{P,i}$ represents the firm-specific stock of capital, $K_t^P = \int_0^1 K_t^{P,i}\, di$ the aggregate private capital stock, K_t^I the stock of public capital in infrastructure, A_t the average, economy-wide labour efficiency, N_t^i the number of adult workers employed by firm i, N_t the total number of adults, ϕ_K , $\phi_N > 0$, $\alpha > 0$, and $\beta \in (0, 1)$.

Equation (3.5) indicates that production exhibits constant returns to scale in the firm-specific inputs, $A_t \varepsilon_t^{W,i} N_t^i$ and $K_t^{P,i}$. Furthermore, it highlights that public capital in infrastructure, K_t^I, is exogenous and impacts the production processes of all individual firms equally. However, the aggregate private capital stock and the size of the population, $(K_t^P)^{\phi_K} (N_t)^{\phi_N}$, imply that the productive effects of public capital are diminished by excessive use.

Each firm maximizes profits, Π_t^i , with respect to labour and private capital, taking prices, K_t^I and A_t as given:

$$\max_{N_t^i, K_t^{P,i}} \Pi_t^i = Y_t^i - r_t K_t^{P,i} - w_t \varepsilon_t^W A_t N_t^i.$$

Profit maximization results in

$$w_t = \beta Y_t^i / \varepsilon_t^W A_t N_t^i, \quad r_t = (1-\beta) Y_t^i / K_t^{P,i}, \tag{3.6}$$

meaning that private inputs are remunerated at their marginal product.

Since all firms and workers are identical, this translates into a symmetric equilibrium where

$$w_t = \beta Y_t / \varepsilon_t^W A_t N_t, \quad r_t = (1-\beta) Y_t / K_t^P. \tag{3.7}$$

Normalizing the number of firms to 1, aggregate output is expressed as

$$Y_t = \int_0^1 Y_t^i \, di = N_t^{\beta - \alpha\phi_N} (k_t^I)^\alpha (\varepsilon_t^W A_t)^\beta (K_t^P)^{1-\beta+\alpha(1-\phi_K)}, \tag{3.8}$$

where $k_t^I = K_t^I / K_t^P$ corresponds to the public-private capital ratio. As shown below, k_t^I, ε_t^W and A_t are constant in the steady state. To eliminate the scale effect associated with the population size and guarantee steady-state growth, the following assumption is made:

Assumption 1 $\beta - \alpha(1-\phi_K) = 0, \phi_N = \beta/\alpha$.

Under Assumption 1, aggregate output is given by

$$Y_t = (k_t^I)^\alpha (\varepsilon_t^W A_t)^\beta K_t^P. \tag{3.9}$$

3.3 Health status and labour efficiency

Health status in childhood, h_t^C, positively depends on parental health status, h_t^A, child-rearing time, ε^R, and access to public health services:

$$h_t^C = \bar{h}^C + (h_t^A)^\kappa (1+\varepsilon^R)^{\nu_C} (H_t^G)^{1-\nu_C}, \tag{3.10}$$

where $\bar{h}^C \geq 0$ represents the health status at birth, H_t^G denotes the supply of public health services, and $\kappa, \nu_C \in (0,1)$.

The parent's health can impact the child's health status either through in utero effects or by influencing the parent's ability to tend to the child. Health status in adulthood is assumed to solely depend on health status in childhood:

$$h_{t+1}^{A} = h_{t}^{C}. \tag{3.11}$$

Substituting (3.10) into (3.11) and considering that health status at birth, $\bar{h}^{C}$, is equal to 0 for simplicity leads to

$$h_{t+1}^{A} = (h_{t}^{A})^{\kappa}(1+\varepsilon^{R})^{\nu_C}(H_{t}^{G})^{1-\nu_C}. \tag{3.12}$$

For simplicity, it is further assumed that $\varepsilon^{R} = 0$, meaning that parents devote no time to rearing their children. Therefore, health status in adulthood is given by

$$h_{t+1}^{A} = (h_{t}^{A})^{\kappa}(H_{t}^{G})^{1-\nu_C}. \tag{3.13}$$

The fact that parental health status affects children's health, and health status in adulthood is entirely determined by health status in childhood, implies there is serial dependence in h_{t}^{A}. This serial dependence reveals the existence of health persistence, meaning that health outcomes are transmitted across generations.

Consequently, an economy currently facing adverse health conditions and, thus, high mortality and low productivity, not only faces difficulties in improving its current state, it also transmits such unfavourable conditions to future generations. Thus, health persistence might cause persistent poverty, and the government might wish to improve access to public health services in childhood to enhance overall health outcomes and avoid potential self-reinforcing dynamics. As we noted in Chapter 2, improving the population's health status through supplying public health services may be challenging due to inefficiencies in government spending.

Health persistence is a key feature of the model which plays a pivotal role in determining the economy's dynamic behaviour, as we illustrate in Chapter 4.

Health is notoriously difficult to measure, due to its multidimensional nature (Bhalotra & Rawlings, 2011). The variable h^{A} can, therefore, be interpreted as a broad representation of the health status in the economy, and it can be measured using various health indicators, such as the body mass index (BMI) or the daily intake of a vital nutrient.

In this model, the health status is not influenced by private health expenditure, despite the fact that it constitutes a significant portion of the total health expenditure in many economies. The World Health Organization [2] reports that domestic private health expenditure accounted for 15% to 30% of current health expenditure in most European Union countries in 2019, and exceeded 50% in many developing countries. In India, the economy for which we calibrate the model, domestic private health expenditure amounted to 64.38% of current health expenditure in 2019. [3]

For simplicity, labour efficiency is assumed to be linearly related to the health status:

$$a_t = h_t^A. \tag{3.14}$$

Although health does not directly provide utility, it contributes to the production of goods through labour augmentation.

3.4 Government

The government invests a total of G_t^I in infrastructure, and spends G_t^H and G_t^U on health and on unproductive services, respectively. All government services are provided free of charge. In order to finance its expenditure, the government taxes adults at the constant rate τ.

Health and infrastructure are productive categories of spending, while unproductive services are not. Thus, it is possible to conduct economic policy and change the allocation of public funds without trade-offs, as long as the increase in health or infrastructure expenditure is financed through a reduction in unproductive services spending. The government cannot borrow and must run a balanced budget in each period:

$$G_t = G_t^I + G_t^H + G_t^U = N_t \tau \varepsilon_t^W A_t w_t. \tag{3.15}$$

The public expenditure shares on the three categories of spending are constant fractions of the total government revenue:

$$G_t^h = \upsilon_h N_t \tau \varepsilon_t^W A_t w_t, \quad h = I, H, U, \tag{3.16}$$

[2] Domestic private health expenditure (PVT-D) as percentage of current health expenditure (CHE) (%) in World Health Organization.

[3] Varvarigos and Zakaria (2013) build an OLG model with endogenous fertility and both public and private health expenditure, where multiple equilibria can arise because of bidirectional effects between fertility, savings and the capital stock.

where $\upsilon_h \in (0,1)$. Combining (3.15) and (3.16) results in

$$\sum \upsilon_h = 1. \tag{3.17}$$

The flow spending on infrastructure and the existing stock of public capital are combined to produce new public capital. However, public capital is assumed to fully depreciate within each period, meaning that it is entirely used during period t, or immediately before its end, to produce the capital stock available in period $t+1$. The law of motion of the public capital stock in infrastructure is given by

$$K_{t+1}^I = (G_t^I)^\chi (K_t^I)^{1-\chi}, \tag{3.18}$$

where $\chi \in (0,1)$ is a technology parameter which measures the relative contribution that the public expenditure on infrastructure and the public capital stock in infrastructure in period t have on the production of the public capital stock in infrastructure available in period $t+1$.

Agénor (2015, Sec. 4) assumes χ = 1, which has a significant impact on the system. This key assumption implies that the current public capital stock in infrastructure does not contribute whatsoever to the next period's public capital stock, as the public capital stock in infrastructure in period $t+1$ is solely determined by public expenditure on infrastructure in period t. It is made in order to simplify the analytical study of the dynamical system when the probability of surviving from adulthood to old age is endogeneized, since it allows an otherwise two-dimensional system to be reduced to one dimension (see the dynamical system in (4.4)).

Hence, the setting where the survival probability from adulthood to old age is endogenous and the public capital stock in infrastructure in period $t+1$ is determined by both the public expenditure on infrastructure and the public capital stock in infrastructure in period t (meaning that $\chi \neq 1$) remains unexplored. We do not make such an assumption and study the two-dimensional system in Chapter 4.

The production of health services by the government exhibits constant returns to scale with respect to the stock of public capital in infrastructure, K_t^I, and the flow of government spending on health services, G_t^H:

$$H_t^G = \left[\frac{K_t^I}{(K_t^P)^{\phi_H}}\right]^\mu (\varphi G_t^H)^{1-\mu}, \tag{3.19}$$

where $\mu, \varphi \in (0,1)$ and $\phi_H > 0$.

As was mentioned in Chapter 2, government spending, for instance on health, is considered to be carried out with a certain degree of inefficiency. The coefficient φ is thus introduced to measure how efficient government spending on health is in the production of health services. Therefore, the quantity $1-\varphi$ measures the proportion of resources that are lost due to poor governance, for instance. Consequently, the ability to improve the health status through an increase in the supply of public health services depends on the magnitude of φ.

Although Agénor (2015) notes that public health services might be subject to congestion associated with the potential demand of medical assistance by children, nN_t, the only congestion effect considered is measured by the private capital stock.

Finally, to ensure that the health status is stationary, the following assumption is also considered:

Assumption 2 $\phi_H = \mu^{-1}$.

3.5 Adult survival probability

Individuals have a three-period lifespan: childhood, adulthood and old age, and are certain to survive from childhood to adulthood, but face a survival probability of $p_t \in (0,1)$ from adulthood to old age. As was mentioned in the introduction to Chapter 3, the survival probability from adulthood to old age is assumed to be exogenous for the entirety of Agénor's (2015) main analysis. Instead, we extend the endogenous survival probability the author proposes in the paper's briefly explored fourth section, which considers how public health expenditure affects the population's health status and, thus, the survival probability.

Preferably, the survival probability would be directly related to each individual's own health status, in which case individuals would internalize the impact of their time allocation decisions. However, given its complexity, the survival probability is assumed to depend on the average health status in the economy to allow for analytical treatment. Consider a survival probability defined by the following piecewise function:

$$p_t = \begin{cases} p_m & \text{for} \quad h_t^A < h_L^A, \\ f(h_t^A) & \text{for} \quad h_L^A \leq h_t^A < h_H^A, \\ p_M & \text{for} \quad h_t^A \geq h_H^A, \end{cases} \tag{3.20}$$

where $0 < p_m \leq f(h_t^A) \leq p_M < 1$.

To ensure $f(h_t^A)$ assumes a strictly increasing concave form and there is continuity between the intervals, we demand that f satisfies the following properties: $f(h_L^A) = p_m$, $f(h_H^A) = p_M$, $f' > 0$ and $f'' < 0$.

The function is defined across three intervals, each representing a stage of development. If the health status falls below h_L^A, the likelihood of surviving from adulthood to old age is constant and equal to p_m. Health status improvements are unable to translate to a higher survival probability until the lowest threshold is reached, possibly because of a high disease burden and limited access to healthcare. As the health status improves above this threshold, the survival probability positively and concavely depends on the average health status over the interval (h_L^A, h_H^A), and becomes constant and equal to p_M as the economy crosses the highest threshold, where the survival probability stabilizes at a maximum level regardless of further improvements in health status. At this stage of development, improvements in health status through public health expenditure become less effective in reducing adult mortality, as longevity approaches its saturation point (Fanti & Gori, 2014).

Equation (3.20) is illustrated in the first quadrant of Figure 3.1 and can be seen to resemble a S-shape relationship between the health status and the survival probability.

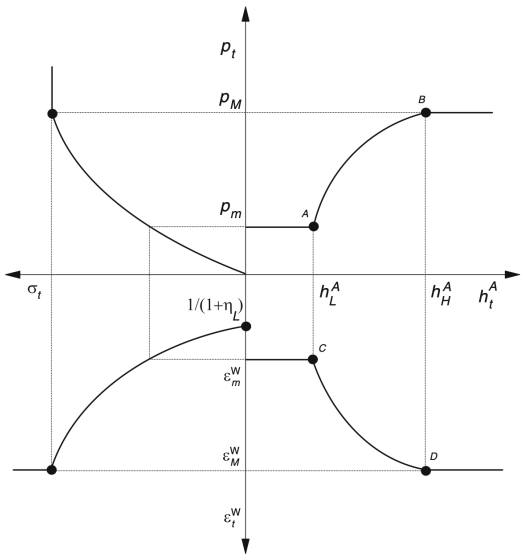

Figure 3.1: The relationship between h_t^A, p_t, σ_t and ε_t^W

The first quadrant illustrates the relationship between h_t^A and p_t. If h_t^A falls below h_L^A, p_t remains constant at p_m. In the range between h_L^A and h_H^A, there exists a positive and concave relationship between h_t^A and p_t. If h_t^A exceeds h_H^A, p_t remains constant at p_M. The remaining quadrants illustrate Equations (4.2) and (4.3). **Source:** Agénor (2015).

4 Existence and local stability of equilibria

Throughout this chapter, we analytically solve our extension of the model presented in Chapter 3, in order to derive conditions of existence and stability of interior equilibria in the three regions of the survival probability's domain.

As expressed in Equation (4.4) and thoroughly demonstrated in Appendix A.2, the dynamical system is influenced by the savings rate and the proportion of time devoted to market work, respectively σ_t and ε_t^W. As shown in Appendix A.1 and given by Equations (4.2) and (4.3), σ_t and ε_t^W are dependent on p_t. Hence, the fact that we define p_t as a piecewise function across three intervals implies that σ_t and ε_t^W and, consequently, the system's state variables, are also subject to these thresholds. Such relationships are illustrated in Figure 3.1.

We study the dynamics in each interval separately to find fixed points, determine under which conditions they are admissible solutions, i.e., whether they are located within their subdomain bounds, and study their local stability properties. We base our stability analysis on Galor (2007) and Gandolfo (2009).

Moreover, in the second interval, p_t and, consequently, σ_t and ε_t^W, are defined as functions of the state variable h_t^A, which has implications for the interval's Jacobian matrix, as we demonstrate in Subsection 4.2.2.

Based on similar literature, we propose a functional form for p_t that follows the required properties, namely a strictly increasing concave form and continuity between the intervals.

Given that this chapter is devoted to analytically solving the model and analysing the existence and stability of interior equilibria, it is crucial to introduce the definitions of a competitive equilibrium and a balanced growth path.

4.1 Market-clearing and equilibrium

In order for the asset market to clear, there can be no debt or inheritances between generations in net terms, meaning that the current private capital stock must be equal to the previous period's aggregate savings by adults:

$$K_{t+1}^P = N_t s_t. \tag{4.1}$$

Definition 4.1.1. *A competitive equilibrium for this economy is a sequence of consumption, savings and time allocated to market work* $\{c_{t+1}^t, c_{t+2}^t, s_t, \varepsilon_{t+1}^W\}_{t=0}^{\infty}$*, public and private capital stocks* $\{K_{t+1}^I, K_{t+1}^P\}_{t=0}^{\infty}$*, prices* $\{w_t, r_{t+1}\}_{t=0}^{\infty}$*, health status in childhood and adulthood* $\{h_t^C, h_{t+1}^A\}_{t=0}^{\infty}$*, a constant tax rate* τ *and constant spending shares* υ_I *and* υ_H*, such that, given the initial capital stocks* K_0^P *and* $K_0^I > 0$*, health status* h_0^C *and* $h_0^A > 0$*, individuals maximize utility, firms maximize profits, the product market clears, and the government budget is balanced.*[1]

Under symmetric equilibrium, individual labour efficiency must be equal to the economy-wide average efficiency level, meaning that $a_t = A_t$.

Definition 4.1.2. *A balanced growth equilibrium is a competitive equilibrium where* c_t^t*,* c_{t+1}^t*,* Y_t*,* K_t^P*, and* K_t^I*, all grow at the constant rate* γ*, health status in childhood and adulthood,* h_t^C *and* h_t^A*, are constant, and the rate of return on private capital,* r_t*, is also constant.*

4.2 Balanced growth path

Adults maximize (3.1) subject to (3.4) and (3.13), with respect to c_{t+1}^t, c_{t+2}^t, and ε_{t+1}^W, taking τ, H_t^G, and p_t as given. We solve the household problem in Appendix A.1. In equilibrium, considering a constant population ($n = 1$), the following values hold for the marginal propensity to save and the proportion of time devoted to market work, respectively σ and ε^W:

$$\sigma_t = \frac{p_t}{(1+\rho)+p_t} < 1, \tag{4.2}$$

$$\varepsilon_t^W = \frac{1}{1+\eta_L(1-\sigma_t)} = \frac{1+\rho+p_t}{(1+\rho)(1+\eta_L)+p_t} < 1. \tag{4.3}$$

[1]Definitions 4.1.1 and 4.1.2 are extracted from Agénor (2015), since the same definitions of competitive equilibrium and balanced growth equilibirum apply.

Consequently, the initial health status not only determines the initial probability of survival, p_0, but also the initial propensity to save, σ_0, and the initial proportion of time devoted to market work, ε_0^W.

As shown in Appendix A.2, the dynamic behaviour of the economy is described by the following first-order difference equation system:

$$\begin{cases} h_{t+1}^A = (\varphi \nu_H \tau \beta)^{\nu} \, (\varepsilon_t^W)^{\beta\nu} (h_t^A)^{\zeta_1} (k_t^I)^{\zeta_2} \\ k_{t+1}^I = \dfrac{(\beta \upsilon_I \tau)^{\chi} (\varepsilon_t^W)^{-\beta(1-\chi)}}{\beta \sigma_t (1-\tau)} (k_t^I)^{(1-\alpha)(1-\chi)} (h_t^A)^{-\beta(1-\chi)} \end{cases} \tag{4.4}$$

where $\nu = (1-\nu_C)(1-\mu)$, $\quad \zeta_1 = \kappa + \nu\beta \quad$ and $\quad \zeta_2 = \mu(1-\nu_C) + \nu\alpha$.

We denote the curve representing $k_{t+1}^I = k_t^I$ as KK, and that representing $h_{t+1}^A = h_t^A$ as HH, respectively (4.5) and (4.6). The steady-state values of k^I and h^A, respectively k^{I*} and h^{A*}, are obtained by solving

$$k^{I*} = \left\{ \frac{(\beta \upsilon_I \tau)^{\chi} (\varepsilon_t^W)^{-\beta(1-\chi)}}{\beta \sigma_t (1-\tau)} \right\}^{1/\Pi} (h^{A*})^{-\beta(1-\chi)/\Pi}, \tag{4.5}$$

$$k^{I*} = (\varphi \upsilon_H \tau \beta)^{-\nu/\zeta_2} \left(\varepsilon_t^W\right)^{-\beta\nu/\zeta_2} (h^{A*})^{(1-\zeta_1)/\zeta_2}, \tag{4.6}$$

where $\Pi \equiv 1 - (1-\alpha)(1-\chi) \in (0,1)$.

The steady-state values of h^A and k^I thus correspond to the intersections of the curves KK and HH.

As was previously mentioned, Agénor (2015) makes the key assumption $\chi = 1$ in order to reduce the system to one dimension, where the adult health status is the only state variable. Equation (4.4) shows that assuming $\chi = 1$ results in a constant k^I over time.

We do not make such an assumption and instead determine the conditions under which a health-induced poverty trap (either characterized by multiple interior equilibria or a single low-level equilibrium) might emerge in the two-dimensional system.

Since p_t and, consequently, σ_t and ε_t^W, are defined as piecewise functions of h_t^A, the curves KK

and HH will have three regions separated by the thresholds h_L^A and h_H^A (see (3.20), (4.2) and (4.3)).

In the first and third intervals, σ_t and ε_t^W are constant (while different). In the middle interval, however, σ_t and ε_t^W are functions of h_t^A, which changes the slopes of KK and HH within this region.

We can conclude from (4.5) that KK is always negatively sloped [2]. On the other hand, from (4.6), the slope of HH depends on the magnitude of ζ_1, which is critical in determining the number of intersections between KK and HH and, thus, the number of interior equilibria.

The central role of ζ_1 (= $\kappa + \nu\beta$) is justified by its high sensitivity regarding the size of κ, which measures the economy's degree of health persistence. Higher values of κ reflect a higher dependence of future health results on the current health status of the population. This manifests through the intergenerational transmission of good or poor health from parents to children, as can be seen in (3.10), and through the persistence of health conditions from childhood to adulthood, as can be seen in (3.11).

Higher values of κ and, consequently, ζ_1, reflect a higher degree of health persistence and, thus, it is more likely that the economy's initial health conditions dictate whether it enters a vicious cycle of self-reinforcing bad health conditions, or a prosperous cycle of good health and productivity. Therefore, economies with adverse initial health conditions could experience a health-induced poverty trap.

Scenario (i): when $\zeta_1 < 1$, i.e., when HH is positively sloped in the first and third intervals.

In this case, there is, at most, one intersection between KK and HH. In the second interval, the slope is negatively influenced by $\left(\varepsilon^W(h_t^A)\right)^{-\beta\nu/\zeta_2}$, and can, in principle, switch to negative. However, it is highly implausible that there is more than one intersection between KK and HH, as it would require an excessively large shift in the slope between the intervals.

For illustrative purposes only, we present an example of KK and HH where the slope of HH is positive[3]:

---

[2] In the first and third intervals, the power $-\beta(1-\chi)/\Pi$ only assumes negative values. In the second interval, the slope is influenced by ε_t^W and σ_t, which both positively depend on h_t^A, as can be seen in (4.2) and (4.3), and are associated with a negative power in (4.5), making the slope steeper in this interval.

[3] We believe this graphical example makes the presentation clearer, although it is purely illustrative and not based on the calibration we perform in Chapter 5. The same applies to Fig.4.2.

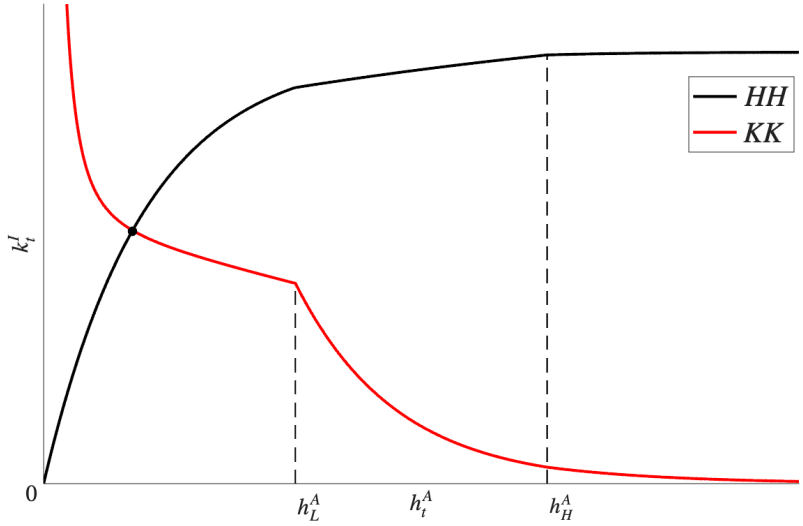

Figure 4.1: Illustrative KK and HH when $\zeta_1 < 1$

KK is a downward-sloping curve, while HH is upward-sloping. There is, at most, one intersection between the curves, which could be located in either interval depending on the values assumed by the model parameters.

Scenario (ii): when $\zeta_1 > 1$, i.e., when HH is negatively sloped.

The negative slope is steeper in the second interval due to the negative effect of $\varepsilon^W(h_t^A)$ and $\sigma(h_t^A)$, making it possible for multiple intersections between the curves to exist. The thresholds separating the three intervals are, thus, crucial to understanding why multiple equilibria are a theoretical possibility in this economy, as they allow for potential shifts in dynamics. Once again, we present a purely illustrative example of KK and HH where HH has a negative slope:

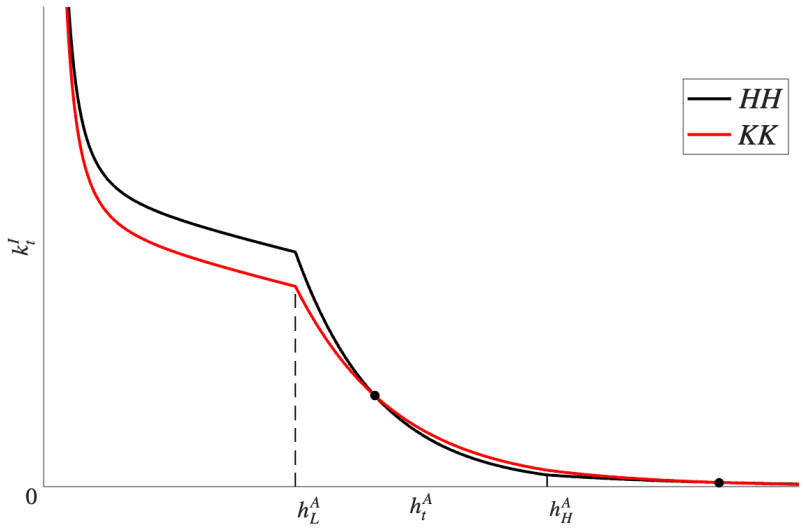

Figure 4.2: Illustrative KK and HH when $\zeta_1 > 1$

Both curves are downward-sloping. The shifts in slope between the intervals make it possible for multiple intersections to exist, depending on the values assumed by the model parameters.

4.2.1 First interval: $h_t^A < h_L^A$

For $h_t^A < h_L^A$, p_t is constant and equal to p_m, meaning that σ_t and ε_t^W are constant at the values given by (4.2) and (4.3), which we denote as σ_m and ε_m^W, respectively.

From (4.5) and (4.6), fixed points in the first interval solve

$$k^{I*} = \left\{ \frac{(\beta \upsilon_I \tau)^{\chi} (\varepsilon_m^W)^{-\beta(1-\chi)}}{\beta \sigma_m (1-\tau)} \right\}^{1/\Pi} (h^{A*})^{-\beta(1-\chi)/\Pi}, \tag{4.7}$$

$$k^{I*} = [\varphi \upsilon_H \tau \beta (\varepsilon_m^W)^{\beta}]^{-\nu/\zeta_2} (h^{A*})^{(1-\zeta_1)/\zeta_2}. \tag{4.8}$$

There is one interior solution, which we denote as (h_1^{A*}, k_1^{I*}):

$$h_1^{A*} = \left[\frac{\left\{ \frac{(\beta \upsilon_I \tau)^{\chi} (\varepsilon_m^W)^{-\beta(1-\chi)}}{\beta \sigma_m (1-\tau)} \right\}^{1/\pi}}{[(\varphi \upsilon_H \tau \beta)(\varepsilon_m^W)^{\beta}]^{-\nu/\zeta_2}} \right]^{\frac{1}{(1-\zeta_1)/\zeta_2 + \beta(1-\chi)/\Pi}} \tag{4.9}$$

$$k_1^{I*} = (h_1^{A*})^{(1-\zeta_1)/\zeta_2} \left[\varphi \upsilon_H \tau \beta (\varepsilon_m^W)^{\beta} \right]^{-\nu/\zeta_2} \tag{4.10}$$

Proposition 1. *The state* (h_1^{A*}, k_1^{I*}) *yields an admissible steady-state equilibrium if and only if* h_1^{A*} *satisfies* $0 < h_1^{A*} < h_L^A$.

This condition ensures that only solutions within the interval bounds are considered valid steady-state equilibria.

Regarding local stability, we show in Appendix A.3 that the dynamical system in (4.4) can be condensed into an autonomous, first-order linear difference equation system in $\hat{h}_t^A = \ln h_t^A$ and $\hat{k}_t^I = \ln k_t^I$, which can be written as

$$\begin{bmatrix} \hat{h}_{t+1}^A \\ \hat{k}_{t+1}^I \end{bmatrix} = \begin{bmatrix} a_{10} \\ a_{20} \end{bmatrix} + \begin{bmatrix} a_{11} & a_{12} \\ a_{21} & a_{22} \end{bmatrix} \begin{bmatrix} \hat{h}_t^A \\ \hat{k}_t^I \end{bmatrix}, \tag{4.11}$$

where

$$a_{10} = \ln\left[(\varphi \upsilon_H \tau \beta)^{\nu} (\varepsilon_m^W)^{\nu\beta}\right],$$

$$a_{20} = \ln\left\{\frac{(\beta \upsilon_I \tau)^{\chi} (\varepsilon_m^W)^{-\beta(1-\chi)}}{\beta \sigma_m (1-\tau)}\right\},$$

$a_{11} = \kappa + \beta(1-\nu_C)(1-\mu) \equiv \zeta_1 > 0,$

$a_{12} = (1-\nu_C)\left[\mu + \alpha(1-\mu)\right] \equiv \zeta_2 > 0,$

$a_{21} = -\beta(1-\chi) < 0,$

$a_{22} = (1-\alpha)(1-\chi) > 0.$

Let $\mathbf{A}$ denote the coefficient matrix in (4.11), and tr$\mathbf{A}$ and det$\mathbf{A}$ denote its trace and determinant, respectively. Thus, tr$\mathbf{A}$ and det$\mathbf{A}$ are given by

$$\text{tr}\mathbf{A} = \zeta_1 + (1-\alpha)(1-\chi) > 0, \tag{4.12}$$

$$\det\mathbf{A} = \zeta_1(1-\alpha)(1-\chi) + \zeta_2\beta(1-\chi) > 0. \tag{4.13}$$

Let λ_j, $j = 1, 2$, denote the eigenvalues of the Jacobian matrix $\mathbf{A}$ in equilibrium. The characteristic polynomial is therefore

$$p(\lambda) \equiv \lambda^2 - \lambda\,\text{tr}\mathbf{A} + \det\mathbf{A} = 0, \tag{4.14}$$

and the eigenvalues, λ_1 and λ_2, are given by

$$\lambda_{1,2} = \frac{\text{tr}\mathbf{A} \pm \sqrt{\text{tr}\mathbf{A}^2 - 4\det\mathbf{A}}}{2}. \tag{4.15}$$

The sign of the discriminant $\Delta = \text{tr}\mathbf{A}^2 - 4\det\mathbf{A}$ determines whether the eigenvalues are real or complex.

The system has a pair of complex eigenvalues $\lambda_1 = a + ib$ and $\lambda_2 = a - ib$, $a, b \in \mathbb{R}$, if $\Delta < 0$. In this case, the equilibrium is locally stable if the modulus of the eigenvalues, r, is smaller than one. Since $r = \sqrt{a^2 + b^2} = \sqrt{\lambda_1\lambda_2} = \sqrt{\det\mathbf{A}}$, the equilibrium is locally stable if $\det\mathbf{A} < 1$, and unstable otherwise.[4]

[4]Throughout our entire analysis, we assume to be dealing with hyperbolic equilibria, meaning that the Jacobian matrix has no eigenvalue of modulus one.

If $\Delta > 0$, the eigenvalues are real. Since tr$\mathbf{A} > 0$ and det$\mathbf{A} > 0$, then

$$p(-1) = 1 + \text{tr}\mathbf{A} + \text{det}\mathbf{A} > 0. \tag{4.16}$$

On the other hand, the sign of $p(1) = 1 - \text{tr}\mathbf{A} + \text{det}\mathbf{A}$ depends on the following parameter values

$$\begin{aligned} &1 - \zeta_1 - (1-\alpha)(1-\chi) + \zeta_1(1-\alpha)(1-\chi) + \zeta_2\beta(1-\chi) = \\ &= (1-\zeta_1)\Pi + \zeta_2\beta(1-\chi), \end{aligned} \tag{4.17}$$

where $\Pi \equiv 1 - (1-\alpha)(1-\chi) \in (0,1)$.

This means there are multiple possible stability scenarios:

(i) if $p(1) < 0$ and $p(-1) > 0$, the equilibrium is a saddle. This instability stems from the fact that one of the eigenvalues is not within the interior of the unit disk (i.e. $\{\lambda_1 > 1$ and $|\lambda_2| < 1\}$ or $\{|\lambda_1| < 1$ and $\lambda_2 < -1\}$).

(ii) if $p(1) > 0$ and $p(-1) > 0$, the equilibrium is locally stable if $|\text{tr}\mathbf{A}| < 2$, and unstable otherwise. If $|\text{tr}\mathbf{A}| < 2$, both eigenvalues are situated within the unit disk (i.e. $|\lambda_i| < 1, i = 1, 2$).

When $\zeta_1 < 1$, the conditions $p(1) > 0$ and $|\text{tr}\mathbf{A}| < 2$ are always satisfied, meaning that $\zeta_1 < 1$ is sufficient to ensure the equilibrium is locally stable.

4.2.2 Second interval: $h_L^A \leq h_t^A < h_H^A$

For $h_L^A \leq h_t^A < h_H^A$, p_t is positively and concavely related to the average health status in the economy. Recalling that $p_t = f(h_t^A)$, we get $f' > 0$ and $f'' < 0$. To ensure continuity across the intervals, we set $f(h_L^A) = p_m$ and $f(h_H^A) = p_M$.

Because the savings rate, σ_t, and the proportion of time devoted to market work, ε_t^W, are positively related to the survival probability, p_t (see (4.2) and (4.3)), they both depend on the health status in the economy. Since the survival probability depends on the health status in a piecewise fashion, σ_t and ε_t^W also exhibit a piecewise dependency on the health status. Such relationships are illustrated in Figure 3.1.

The functional forms relating σ_t and ε_t^W to h_t^A can be written as $\sigma_t = \sigma(h_t^A)$ and $\varepsilon_t^W = \varepsilon^W(h_t^A)$, respectively.

Expressing (4.2) as a function of h_t^A, we get

$$\sigma(h_t^A) = \frac{f(h_t^A)}{1+\rho+f(h_t^A)}, \tag{4.18}$$

and we can calculate the derivative

$$\frac{d\sigma(h_t^A)}{dh_t^A} = \frac{f'(h_t^A)(1+\rho)}{[1+\rho+f(h_t^A)]^2}, \tag{4.19}$$

which is positive since $f'(h_t^A) > 0$.

Equilibrium savings increase with the survival probability, as a longer expected lifespan extends the horizon over which savings yield returns, thus raising the incentive to save during adulthood. Similar results can be found in Chakraborty (2004), Fodha and Seegmuller (2014), Osang and Sarkar (2008) and Varvarigos (2010).

Formally, an increase in the survival probability raises the marginal utility of consumption during old age. To reestablish equilibrium, the marginal utility of consumption during adulthood must rise through higher savings and reduced consumption during adulthood (Palivos & Varvarigos, 2017).

Writing (4.3) as

$$\varepsilon^W(h_t^A) = \frac{1}{1+\eta_L(1-\sigma(h_t^A))}, \tag{4.20}$$

we obtain the following derivative

$$\frac{d\varepsilon^W(h_t^A)}{dh_t^A} = \frac{\eta_L \dfrac{d\sigma(h_t^A)}{dh_t^A}}{[1+\eta_L(1-\sigma(h_t^A))]^2} > 0. \tag{4.21}$$

Although the shape of $f(h_t^A)$ is known, multiple closed forms can be used to describe the relationship between p_t and h_t^A and, therefore, the relationships between σ_t, ε_t^W and h_t^A. The selection of closed forms impacts the analytical study of the system, since it determines whether a closed-form solution is attainable or only an implicit one.

Since our results might be sensitive to the chosen closed form and we wish to attain a certain level of generality, we start by considering a generic function for $f(h_t^A)$ and, therefore, $\sigma(h_t^A)$ and $\varepsilon^W(h_t^A)$, and present some brief considerations regarding the solution and its local stability. This standard practice is used by authors such as Dioikitopoulos (2014) and Jouvet et al. (2010), who study their systems analytically using a generic longevity function and only assume a closed form when calibrating the model. Thus, we are only able to reach an implicit solution.[5]

It is worth recalling that the variable h^A can be interpreted as a broad representation of the health status in the economy, and can be measured using various health indicators. The ambiguity around its exact meaning implies that, although we know h^A assumes positive values, there is no benchmark for the magnitude of the values it assumes.

However, the survival probability must only assume values from 0 to 1, meaning that we ought to be careful when selecting a closed-form expression to define $f(h_t^A)$ and ensure it remains within interval bounds. [6]

Considering generic functions rather than defining specific closed forms, the law of motion in the second interval is given by

$$\begin{cases} h_{t+1}^A = (\varphi \nu_H \tau \beta)^\nu \left(\varepsilon^W(h_t^A)\right)^{\beta\nu} (h_t^A)^{\zeta_1} (k_t^I)^{\zeta_2} \\ k_{t+1}^I = \dfrac{(\beta \upsilon_I \tau)^\chi (\varepsilon^W(h_t^A))^{-\beta(1-\chi)}}{\beta\sigma(h_t^A)(1-\tau)} (k_t^I)^{(1-\alpha)(1-\chi)} (h_t^A)^{-\beta(1-\chi)}, \end{cases} \tag{4.22}$$

and fixed points in the second interval, $(h_2^{A^*}, k_2^{I^*})$, solve

$$k^{I^*} = \left\{ \frac{(\beta \upsilon_I \tau)^\chi (\varepsilon^W(h^{A^*}))^{-\beta(1-\chi)}}{\beta\sigma(h^{A^*})(1-\tau)} \right\}^{1/\Pi} (h^{A^*})^{-\beta(1-\chi)/\Pi}, \tag{4.23}$$

$$k^{I^*} = \left[\varphi \upsilon_H \tau \beta (\varepsilon^W(h^{A^*}))^\beta\right]^{-\nu/\zeta_2} (h^{A^*})^{(1-\zeta_1)/\zeta_2}. \tag{4.24}$$

---

[5] If we wish to fully study the model analytically, we can we employ a linear approximation to both σ_t and ε_t^W, albeit with the understanding that this only represents a particular context. There is an interior solution $(h_2^{A^*}, k_2^{I^*})$ and the Jacobian matrix is given by

$$\begin{bmatrix} \beta\nu + \zeta_1 & \zeta_2 \\ -2\beta(1-\chi) - 1 & (1-\alpha)(1-\chi) \end{bmatrix}$$

[6] Given the one-unit time constraint, the proportion of time allocated to market work, ε_t^W, must also not assume values greater than 1. However, this condition is guaranteed if p_t does not exceed 1.

Proposition 2. *The state* $(h_2^{A^*}, k_2^{I^*})$ *yields an admissible steady-state equilibrium if and only if* $h_2^{A^*}$ *satisfies* $h_L^A \leq h_2^{A^*} < h_H^A$.

By performing a log-linearization of (4.22), which we demonstrate in Appendix A.3, we get the following Jacobian matrix evaluated at the steady-state equilibrium $(h_2^{A^*}, k_2^{I^*})$:

$$\begin{bmatrix} \zeta_1 + \beta\nu \left.\frac{d\ln\varepsilon^W(h^A)}{d\ln(h^A)}\right|_{h^A=h_2^{A^*}} & \zeta_2 \\ -\beta(1-\chi) - \beta(1-\chi) \left.\frac{d\ln\varepsilon^W(h^A)}{d\ln(h^A)}\right|_{h^A=h_2^{A^*}} - \left.\frac{d\ln\sigma(h^A)}{d\ln(h^A)}\right|_{h^A=h_2^{A^*}} & (1-\alpha)(1-\chi) \end{bmatrix} \tag{4.25}$$

It is clear that the Jacobian matrix in the second interval differs from that of the first and third intervals, which means the stability properties of equilibria belonging to these intervals might be different. The values assumed by the trace and determinant are contingent upon the chosen closed form for $f(h_t^A)$ and, consequently, $\sigma(h_t^A)$ and $\varepsilon^W(h_t^A)$.

In Chapter 5, we numerically compute this Jacobian matrix to study local stability in the second interval. The analogous considerations presented in Subsection 4.2.1 regarding the role of the trace and determinant of the Jacobian matrix (and, consequently, the discriminant and characteristic polynomial) in determining local stability apply.

We are interested in assuming a functional form which follows the previously imposed properties, such as $f' > 0$, $f'' < 0$ and continuity across intervals.

Authors such as Chakraborty (2004) and Dioikitopoulos (2014) work with the functional form $\phi(h_t^A) = h_t^A/(1 + h_t^A)$ over the whole domain of the survival probability function. We adapt it to ensure it satisfies $f(h_L^A) = p_m$ and $f(h_H^A) = p_M$ and get the following strictly increasing concave functional form:

$$f(h_t^A) = \left(\frac{p_M - p_m}{\frac{h_H^A}{1+h_H^A} - \frac{h_L^A}{1+h_L^A}}\right) \cdot \frac{h_t^A}{1+h_t^A} + \left[p_m - \left(\frac{p_M - p_m}{\frac{h_H^A}{1+h_H^A} - \frac{h_L^A}{1+h_L^A}}\right) \cdot \frac{h_L^A}{1+h_L^A}\right] \tag{4.26}$$

When we consider a nonlinear functional form for $f(h_t^A)$, the dynamics in (4.4) become too complex to study analytically, since when we substitute (4.26) in (4.18) and (4.20), σ_t and

ε_t^W also assume concave functional forms and we cannot obtain a closed-form solution to the system. Although this functional form does not allow us to analytically study the dynamics in this interval any further, it allows us to get more accurate results in Chapter 5, where we study the interval's dynamics numerically.

4.2.3 Third interval: $h_t^A \geq h_H^A$

For $h_t^A \geq h_H^A$, p_t is constant and equal to p_M, meaning that σ_t and ε_t^W are constant at the values given in (4.2) and (4.3), which we denote as σ_M and ε_M^W, respectively.

From (4.5) and (4.6), fixed points in the third interval solve

$$k^{I*} = \left\{ \frac{(\beta \upsilon_I \tau)^{\chi} (\varepsilon_M^W)^{-\beta(1-\chi)}}{\beta \sigma_M (1-\tau)} \right\}^{1/\Pi} (h^{A*})^{-\beta(1-\chi)/\Pi}, \tag{4.27}$$

$$k^{I*} = \left[\varphi \upsilon_H \tau \beta (\varepsilon_M^W)^{\beta} \right]^{-\nu/\zeta_2} (h^{A*})^{(1-\zeta_1)/\zeta_2}. \tag{4.28}$$

There is one interior solution, which we denote as (h_3^{A*}, k_3^{I*}):

$$h_3^{A*} = \left[\frac{\left\{ \dfrac{(\beta \upsilon_I \tau)^{\chi} (\varepsilon_M^W)^{-\beta(1-\chi)}}{\beta \sigma_M (1-\tau)} \right\}^{1/\Pi}}{\left[(\varphi \upsilon_H \tau \beta)(\varepsilon_M^W)^{\beta} \right]^{-\nu/\zeta_2}} \right]^{\frac{1}{(1-\zeta_1)/\zeta_2 + \beta(1-\chi)/\Pi}} \tag{4.29}$$

$$k_3^{I*} = (h_3^{A*})^{(1-\zeta_1)/\zeta_2} \left[\varphi \upsilon_H \tau \beta (\varepsilon_M^W)^{\beta} \right]^{-\nu/\zeta_2} \tag{4.30}$$

where $\nu = (1-\nu_C)(1-\mu)$.

Proposition 3. *The state (h_3^{A*}, k_3^{I*}) yields an admissible steady-state equilibrium if and only if h_3^{A*} satisfies $h_3^{A*} \geq h_H^A$.*

Given that the sole distinction between the first and third intervals lies in the constant values assumed by p_t, σ_t and ε_t, the Jacobian matrix in (4.11) remains identical across these intervals and only the elements a_{10} and a_{20} differ. Consequently, the fixed points in these intervals differ in value, yet share the same local stability properties.

5 Calibration

5.1 Baseline calibration

Throughout this chapter, we calibrate the model to match data from India and solve the system (4.4) numerically. With this numerical analysis, we are able to fully study the model's dynamics and explore the analytical results presented in Chapter 4.

We chose to calibrate the model using data from India for several compelling reasons. First, India represents a large country that can be reasonably approximated as a closed economy, which simplifies certain modelling assumptions. Second, the abundance of available data for India facilitates the empirical analysis. Finally, as a developing country, India provides a relevant context for the issues addressed by the model, such as intergenerational transmission of poor health and the possible occurrence of health-induced poverty traps.

To calibrate the model, we use data from a variety of sources, such as The World Bank's World Development Indicators (WDI) and Global Jobs Indicators Database (JOIN), the World Health Organization, research papers and official reports.

Since we are working with health-related variables and parameters, we use data referring to the most recent year available up to 2019. This approach helps mitigate potential biases associated with the COVID-19 pandemic, thus ensuring the integrity of our analysis by focusing on pre-pandemic trends.

Moreover, we found that health related parameters are often calibrated arbitrarily due to a lack of reliable estimates, as the ways of measuring health differ significantly across studies. We summarize the baseline calibration process in Table 5.1.

In the production sector, the output elasticities with respect to the public-private capital ratio and effective labour, α and β, are set to 0.16 and 0.70, respectively. We proxy α using an estimate of the output elasticity of public capital in India. While not identical, this estimate serves as a practical proxy given the lack of estimates for the output elasticity of the public-private capital

ratio. Chatterjee et al. (2021) report an output elasticity of public capital for formal sector firms of 0.16, which is in line with the long-run estimate by Bom and Ligthart (2014), based on a meta-regression analysis of core national public capital. The value of β is chosen so that the output elasticity with respect to private capital equals 0.3, a standard value commonly found in the literature and close to the estimate reported by Chatterjee et al. (2021) for India.

On the household side, as was previously mentioned, the proportion of time devoted to child rearing, ε^R, is set to 0, and the fertility rate, n, is set to 1.

The initial proportion of time individuals devote to market work, ε_0^W, is set to 0.544. This value is obtained by dividing the average weekly working hours in India in 2019 (for ages ranging from 15 to 64, retrieved from The World Bank's JOIN [1]), 49.5, by the total number of discretionary hours available in a week, which we estimate at 91. We derive this number by subtracting time spent on non-discretionary activities, such as sleep (8 hours per day, 56 hours per week) and personal care, including eating or showering (3 hours per day, 21 hours per week), from the 168 hours in a week (7 x 24 hours per day). This value is close to the estimate of 726 minutes spent in a day per person in self-care and maintenance in the Goverment of India's Time Use Survey for 2019.[2] With this adjustment, we avoid overestimating leisure time and more accurately capture the portion of time that individuals can allocate between market work and leisure.

The effective income tax rate, τ, is calculated by dividing the tax revenue as a percentage of GDP provided by the WDI database for 2018 [3] , 12.02 percent, by the share of labour income in the model, 0.7. This adjustment is made because τ represents the effective tax rate as a percentage of household income, rather than total output, as established through Equations (3.7) and (3.15). The value of τ is thus approximately 17.17 percent.

Similarly, the initial savings rate, σ_0, is calibrated to 0.35 by dividing the household gross savings rate, reported as a percentage of GDP at current market prices for 2018 - 2019 by the Government of India [4], 0.203, by the share of labour income in the model, 0.7, and the net effect of the effective income tax rate, $1-0.1717$. This adjustment is made because σ represents the savings rate as a percentage of disposable labour income, rather than GDP, as established through Equations (A.5) and (3.6).

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[1] Average weekly working hours, aged 15-64, total (JI.TLF.1564.WK.TM) in Global Jobs Indicators Database (JOIN).

[2] Statement 7: Average time (in minutes) spent in different activities in a day per person in Time Use Survey (TUS), January 2019-December 2019.

[3] Tax revenue (% of GDP) (GC.TAX.TOTL.GD.ZS) on World Development Indicators.

[4] Table 1.9: Household Sector Gross Domestic Saving (as a percentage of GDP at current market prices) in Economic Survey 2022-2023: Statistical Appendix (Government of India).

We calibrate the initial survival probability, p_0, using an estimate for the probability of dying. According to the World Health Organization [5], the probability of dying for individuals between 15 and 60 years of age in 2019 was 16.3 percent. The initial survival probability can therefore be measured as $p_0 = 1 - 0.163 = 0.837$.

Equation (4.2) can be solved backward for the value of ρ, which yields an intergenerational discount factor of approximately 0.5544. This results in an annual discount rate of approximately 2.2% when we interpret each period as 20 years. Similarly, from (4.3), the value of η_L is approximately equal to 1.29.

Regarding health conditions, the degree of intergenerational health persistence, κ, is set to 0.45, based on the implicit value used by Osang and Sarkar (2008). This value, which we explore in Section 6.3, was chosen arbitrarily by Osang and Sarkar (2008).

The health status elasticity with respect to the supply of public health services, $1 - \nu_C$, is set to 0.2, which means ν_C is equal to 0.8. There are no good benchmarks for the value of ν_C, so we follow Agénor et al. (2015) and assume a relatively low value for $1 - \nu_C$ to avoid overestimating the benefits of public health expenditure.

As for government spending, the value of υ_H is set to 3.85%, according to the Domestic general government health expenditure (% of general government expenditure) indicator in the WDI database for 2019. [6] We use the Government investment as a share of total government expenditures indicator presented in the OECD's Government at a Glance report for the year 2019 as a proxy for the parameter υ_I, which is therefore equal to 14.8%.[7] From (3.17), the share of government spending on unproductive items, υ_U, is equal to 81.35%.

The parameter φ, which measures the efficiency of public expenditure on health, is set to 0.38. There are no direct estimates for the value of φ, so we follow the approach employed by Agénor et al. (2015) and use the average value estimated by Dabla-Norris et al. (2012) for the efficiency of public investment for a group of Asian countries (Cambodia, Pakistan and Indonesia).

The elasticity of the supply of health services with respect to public health expenditure, $1 - \mu$, is set to 0.85, meaning that μ is equal to 0.15. Once again, we follow Agénor et al. (2015) since there are no good benchmarks in the literature and the parameters used by these authors have very similar meanings to ours.

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[5] Adult mortality rate (probability of dying between 15 and 60 years per 1000 population in World Health Organization.

[6] Domestic general government health expenditure (% of general government expenditure) in World Development Indicators.

[7] Government at a Glance 2021 (OECD National Accounts Statistics). Figure 2.33 in Government at a Glance 2021.

Table 5.1: Calibrated parameters

α	β	ε^R	n	τ	ρ	η_L	κ	ν_C
0.16	0.7	0	1	0.1717	0.5544	1.29	0.45	0.8
υ_H	υ_I	υ_U	φ	μ	p_m	p_M	h_L^A	h_H^A
0.0385	0.148	0.8135	0.38	0.15	0.7	0.93	0.1	0.5

The lowest level of the survival probability is equal to p_m, which corresponds to a first stage of development characterized by high mortality, and should therefore be calibrated according to data from particularly underdeveloped nations. We set p_m to 0.7, in line with the probability of dying for individuals between 15 and 60 years of age in 2019[8] for Afghanistan, Botswana, Congo, Malawi, Namibia and South Africa, which are all approximately 30%.

The highest level is equal to p_M and corresponds to the highest stage of development. We set it to 0.93, in line with the probability of dying for individuals between 15 and 60 years of age in 2019[9] for some of the world's most advanced economies, such as Canada, Denmark, Finland, France and Germany.

The thresholds h_L^A and h_H^A separate the three intervals in (3.20), meaning that the position of the economy's initial health status, h_0^A, with regard to h_L^A and h_H^A determines which interval it initially belongs to. Since h^A is quite an ambiguous variable, there are no reference values for these thresholds. We set $h_L^A = 0.1$ and $h_H^A = 0.5$. The values of h_L^A and h_H^A are critical, as they dictate whether a solution is an admissible steady-state equilibrium, as can be seen in Propositions 1, 2 and 3.

The functional form for $f(h_t^A)$ provided in Equation (4.26) is then given by

[8] Adult mortality rate (probability of dying between 15 and 60 years per 1000 population in World Health Organization.

[9] Adult mortality rate (probability of dying between 15 and 60 years per 1000 population in World Health Organization.

$$f(h_t^A) = \left(\frac{0.93 - 0.7}{\frac{0.5}{1+0.5} - \frac{0.1}{1+0.1}} \right) \cdot \frac{h_t^A}{1+h_t^A} + \left[0.7 - \left(\frac{0.93 - 0.7}{\frac{0.5}{1+0.5} - \frac{0.1}{1+0.1}} \right) \cdot \frac{0.1}{1+0.1} \right] =$$
$$= 0.94875 \frac{h_t^A}{1+h_t^A} + 0.61375. \tag{5.1}$$

We do not calibrate χ for now. Since one of the novelties of our study is the fact that we do not assume χ = 1 and instead study the two-dimensional system, we find it appropriate to analyse how different values of χ impact the system and its equilibria properties, particularly the steady-state level of h^A.

Subsequently, we calibrate χ in order to obtain an explicit solution and study its stability properties, as well as to perform a sensitivity analysis in Chapter 6. We examine the system's sensitivity regarding changes in a key policy making parameter: the share of government spending on health, υ_H, and test whether the same results apply when we employ alternative functional forms for $f(h_t^A)$ in (3.20).

5.2 Numerical simulations

We start by performing a simulation which allows us to express the steady-state level of h^A as a function of χ. Thus, we establish under which values of $\chi \in (0,1)$ the interior solution found for each interval in Chapter 4 is admissible, i.e., under which interval of χ values each of the Propositions 1, 2 and 3 is verified ($0 < h_1^{A^*} < h_L^A$, $h_L^A \leq h_2^{A^*} < h_H^A$ and $h_3^{A^*} \geq h_H^A$, respectively). This results in three regions, which we plot in Figure 5.1. In case of an overlap between any regions, multiple equilibria exist simultaneously.

We plot $h_1^{A^*}(\chi)$ and $h_3^{A^*}(\chi)$ by calibrating all parameters except χ in (4.9) and (4.29), respectively, thus expressing $h_1^{A^*}$ and $h_3^{A^*}$ as functions of χ [10]. To plot $h_2^{A^*}(\chi)$, we substitute (5.1) in (4.18) and (4.20) to achieve a closed form for $\sigma(h_t^A)$ and $\varepsilon^W(h_t^A)$ in (4.23) and (4.24), and numerically find potential $h_2^{A^*}$ by determining the intersections between these curves[11] for different values of χ. We provide the code executed using MATLAB in the Annex.

Figure 5.1 reveals how the steady-state level of h^A depends on the value of χ and, consequently, which interval the steady-state solution is located in depending on χ. It reveals that no values

[10] While in the first and third intervals we provided closed-form solutions, $(h_1^{A^*}, k_1^{I^*})$ and $(h_3^{A^*}, k_3^{I^*})$, the nature of the second interval only allowed us to provide am implicit solution for $(h_2^{A^*}, k_2^{I^*})$.

[11] This method is analogous to our analytical resolution of $h_1^{A^*}$ and $h_3^{A^*}$.

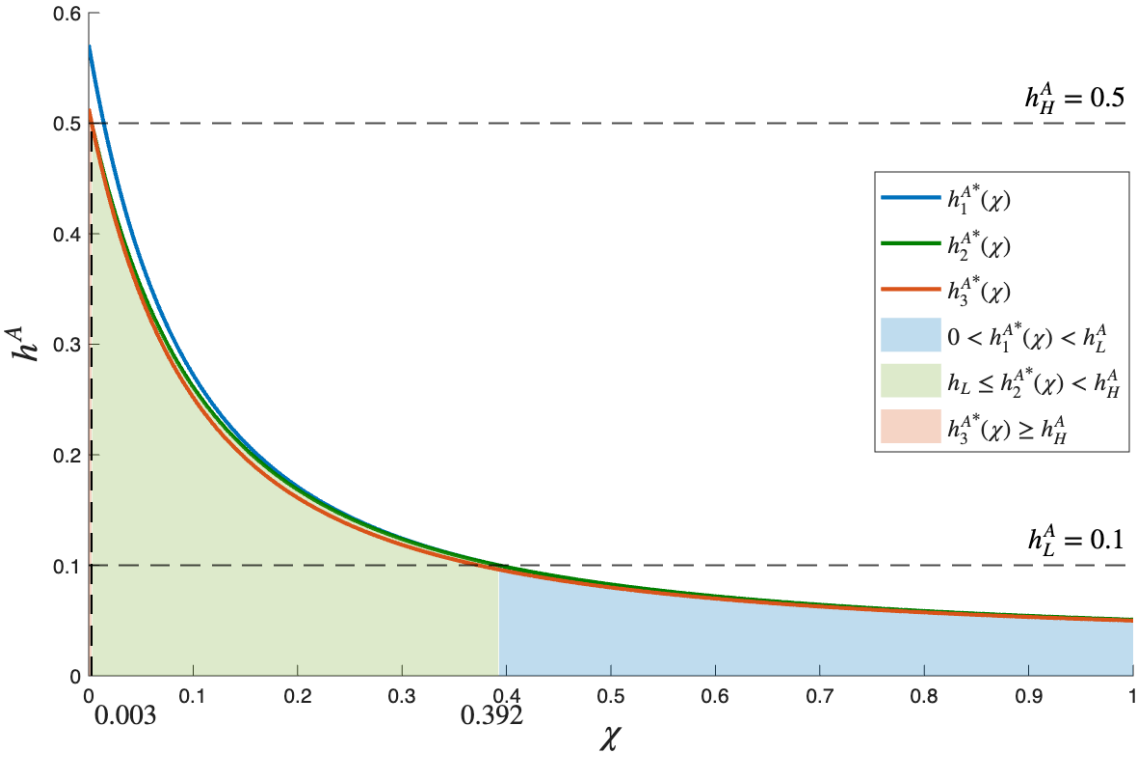

Figure 5.1: Steady-state level of h^A as a function of χ

There is one steady-state equilibrium for each value of χ. For $\chi < 0.003$, the equilibrium is situated in the third interval and h^{A^*} assumes its maximum levels. For $0.003 \leq \chi < 0.392$, the equilibrium is situated within the second interval's bounds. For $\chi \geq 0.392$, the equilibrium is characterized by low health resultts and belongs to the first interval.

of χ generate multiple equilibria, and there is one steady-state equilibrium for each level of χ. We can observe that the steady-state level of h^A decreases with χ, as can be inferred from (4.10) and (4.30), and only quite low values of χ allow the economy to achieve high health status results. The economy's steady-state equilibrium only corresponds to the advanced stage of development when χ approximates 0, making the low and intermediate stages of development (first and second intervals, respectively) more plausible scenarios.

Higher values of χ translate into lower steady-state health outcomes, making the economy settle into a state of high mortality and low productivity. Such steady-state equilibria characterized by adverse health conditions that are transmitted to future generations may be interpreted as single equilibrium health-induced poverty traps.

This hinders market participation and savings, since individuals have narrower time horizons and a smaller incentive to accumulate capital. For a majority of the possible values of χ, the steady-state level of h^A is characterized by low health results, which has an impact on the population's quality of life and on the steady-state growth rate.

Starting from (3.9) and substituting A_t and K_t^P by (3.14) and (A.10), respectively, we get

$$Y_{t+1} = (k_{t+1}^I)^\alpha \left(\varepsilon_{t+1}^W h_{t+1}^A\right)^\beta \beta\sigma_t(1-\tau)Y_t. \tag{5.2}$$

The steady-state growth rate is then given by

$$1 + \gamma = \left(k^I\right)^\alpha \left(\varepsilon^W h^A\right)^\beta \beta\sigma(1-\tau). \tag{5.3}$$

We examine how the steady-state levels of h^A and k^I and, thus, the steady-state growth rate, react to government intervention in Section 6.1.

We can conclude that, while we explored multiple equilibria as a theoretical possibility in Chapter 4, it is not empirically sound for this set of calibrated parameters.

This result stems from the central role of κ and, consequently, ζ_1, in determining the slope of HH and, therefore, the set of equilibria resulting from intersections between HH and KK, as we illustrated in Chapter 4.

From the calibrated parameters, we get $\zeta_1 = \kappa + (1-\nu_C)(1-\mu)\beta = 0.45 + (1-0.8)\cdot(1-0.15)\cdot 0.7 = 0.569$.

As we mentioned in Chapter 4, if $\zeta_1 < 1$, then the slope of HH is positive in the first and third intervals, and it is highly implausible that there is more than one intersection between KK and HH.

The value of ζ_1 is very sensitive regarding κ, which measures the economy's health persistence. Higher values of κ reflect a higher dependence of future health results on the current health status of the population, as manifested through intergenerational transmission of poor health from parents to children and the persistence of bad health from childhood to adulthood. Only high enough values of κ that ensure $\zeta_1 > 1$ and, consequently, a negatively sloped HH curve, render multiple equilibria a possibility, in principle (see Figure 4.2).

With this in mind, in Section 6.3 we perform an analogous simulation to the present one, where we explore how the steady-state level of h^A depends on κ. There is a threshold value of κ after which the slope of HH becomes negative because ζ_1 exceeds 1, yet there are no interior equilibria.

In order to obtain an explicit solution and study its stability properties, we need to calibrate the

final parameter. We set χ at 0.30[12] and determine the steady-state coordinates of h^A and k^I.

While we concluded in Chapter 4 that $\zeta_1 < 1$ is sufficient to ensure local stability if the steady-state equilibrium is situated within the first or third intervals, we have not yet studied stability in the second interval. The implicit nature of the system in the second interval did not allow us to provide explicit stability conditions, and we study the interval's stability numerically.

The Jacobian matrix evaluated at the steady-state equilibrium provides a good approximation of the local dynamics near this steady-state.

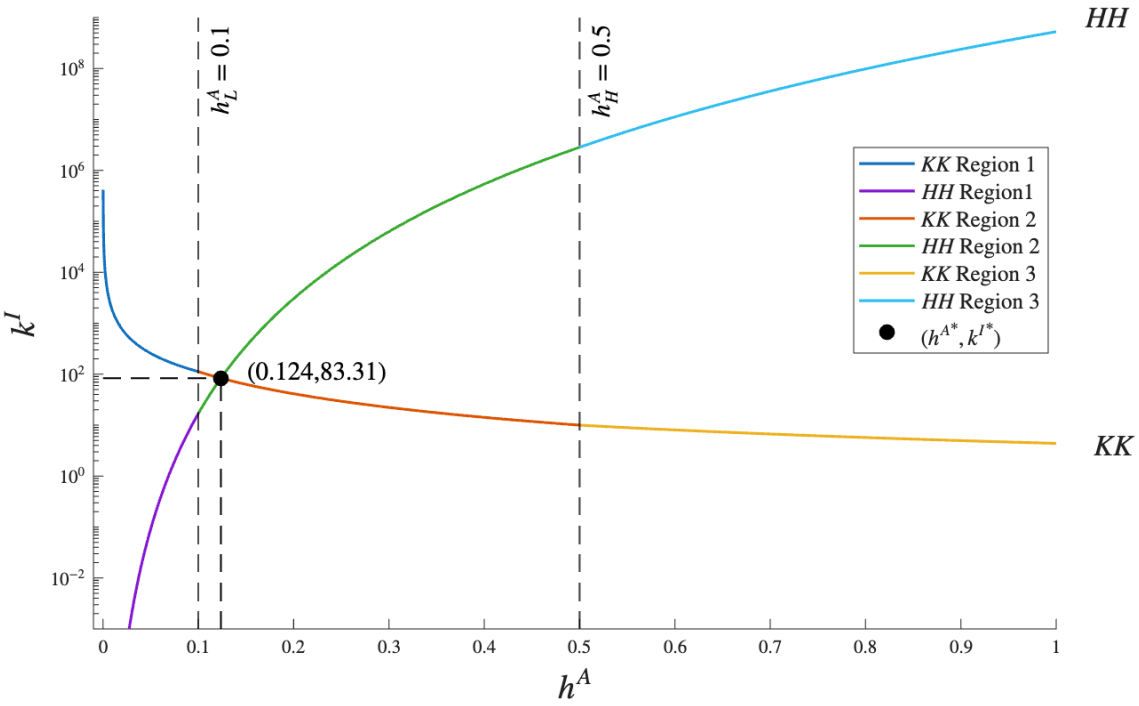

Figure 5.2: KK and HH for $\chi = 0.30$

KK and HH for $\chi = 0.30$. There are three regions, each corresponding to one of the three intervals where the system is defined. There is one steady-state equilibrium at $(h^{A*}, k^{I*}) = (0.124, 83.31)$.

Figure 5.2 displays that there is a unique steady-state equilibrium at $(h^{A*}, k^{I*}) = (0.124, 83.31)$ for $\chi = 0.30$.[13] This equilibrium belongs to the second interval and corresponds to an intermediate level of development, characterized by a steady-state growth rate of $(83.31)^{0.16} \cdot (0.531 \cdot 0.124)^{0.7} \cdot 0.7 \cdot 0.316 \cdot (1 - 0.1717) = 5.5\%$, taking into account (5.3).

From (5.1), (4.18) and (4.20), we get $p^* = 0.72$, $\sigma^* = 0.316$ and $\varepsilon^{W*} = 0.531$.

[12] We chose this value arbitrarily due to a lack of benchmarks in the literature. As is made clear by Figure 5.1, it has an impact on the steady-state values of h^A and k^I and, thus, on their position with regard to the interval thresholds.

[13] We provide the code executed using MATLAB in the Annex.

We compute the log-linearized Jacobian matrix (4.25) at the equilibrium $(0.124, 83.31)$ by numerically evaluating the log-linearized system's partial derivatives with respect to its state variables at the steady-state equilibrium, and come to

$$\begin{bmatrix} 0.57128159 & 0.0572 \\ -0.58792047 & 0.588 \end{bmatrix} \tag{5.4}$$

There is a pair of complex eigenvalues $\lambda_{1,2} = 0.579640794079751 \pm 0.183191633554258i$ with modulus $\sqrt{(0.579640794079751)^2 + (0.183191633554258)^2} \approx 0.61 < 1$. The steady-state equilibrium is a spiral sink and, thus, locally stable.[14]

Figure 5.3 reveals that the system is characterized by spiral convergence towards the steady-state equilibrium (h^{A^*}, k^{I^*}) in a clockwise motion. [15]An economy whose initial conditions do not coincide with the steady-state equilibrium will undergo a process of convergence towards it.

Our economy's initial conditions are given by $p_0 = 0.837$, $\sigma_0 = 0.35$ and $\varepsilon_0^W = 0.544$. Since $p_m \leq p_0 < p_M$, the economy is initially situated in the second interval, and we can determine the initial health status, h_0^A, using (5.1). We get $h_0^A = 0.308$.

Since we do not know k_0^I, we display three possible k_0^I for the given $h_0^A = 0.308$ in Figure 5.3. We also display the trajectory associated with alternative initial conditions to allow for a better understanding of the phase diagram.

Throughout this economy's adjustment process, the average health status of the population, h^A, will monotonically decrease until it reaches its steady-state level, 0.124, while the public-private capital ratio, k^I, might significantly decrease at first, and subsequently increase until it stabilizes at 83.31.

Therefore, the population's health status tends to considerably deteriorate across time and, consequently, so do the probability of survival from adulthood to old age, the savings rate and the labour market participation. This has a negative impact on the economy's growth rate due to the reduced h^A, σ and ε^W, as can be seen in (5.3). Unless k^{I^*} is sufficiently bigger than k_0^I to contradict this effect, the steady-state growth rate is lower than the economy's current one.

In this case, the government might wish to intervene to keep the economy from experiencing this undesirable trajectory. Since there is a unique and locally stable steady-state equilibrium, it

[14]The existence of complex eigenvalues opposes Agenór's (2015) assumption of real eigenvalues throughout his entire analysis.

[15]We provide the code written in Python and executed using Jupyter Notebook in the Annex.

is not possible to position the economy on a converging path to a more desirable equilibrium by altering its initial conditions, and the government must change the position of the equilibrium itself.

This can be achieved through direct public intervention concerning the tax rate, τ, and the government expenditure shares on health and infrastructure, υ_H and υ_I, respectively, or indirectly by improving the efficiency associated with health spending, φ. We explore this topic in Section 6.1.

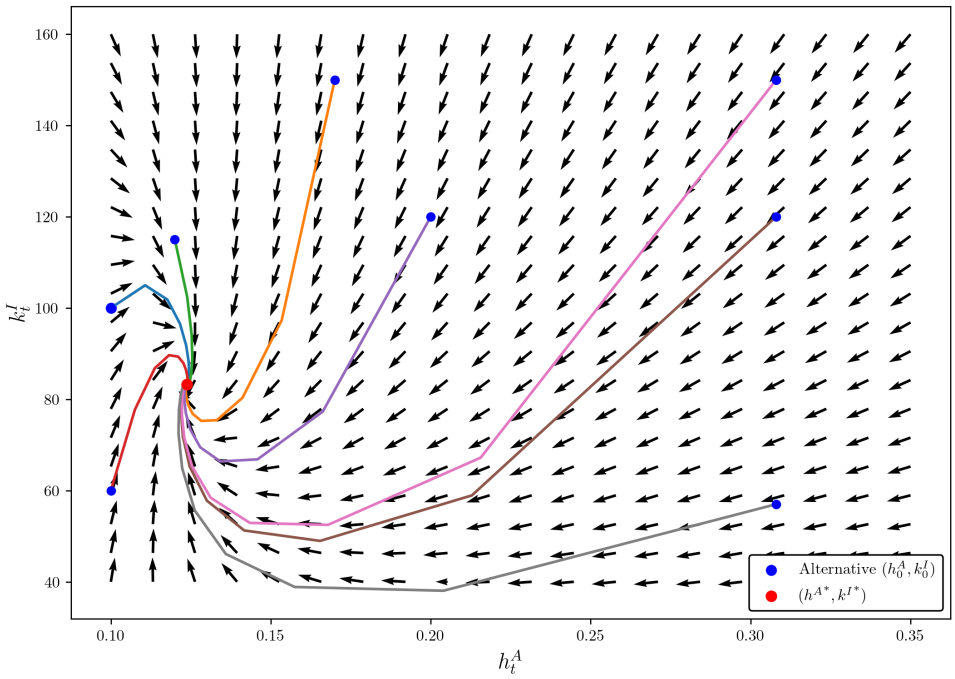

Figure 5.3: Phase diagram

The system exhibits spiral convergence towards the steady-state equilibrium (h^{A*}, k^{I*}) in a clockwise motion. The blue dots correspond to alternative initial conditions, in order to illustrate the law of motion of h^A and k^I.

6 Sensitivity analysis

In this chapter, we provide a sensitivity analysis regarding the results presented in Chapter 5. We find that the same results apply when we consider different functional forms relating the health status to the probability of survival from adulthood to old age. Moreover, we evaluate the impact of an increase in public health expenditure on the economy's steady-state growth rate, and test the way the steady-state equilibrium reacts to different degrees of health persistence. All of the numerical simulations we present in this chapter were executed using MATLAB.

6.1 Increase in public health expenditure

Let's consider an increase in the proportion of public expenditure devoted to health, υ_H, from 3.85% to 13.85%. This substantial increase in health expenditure is budget neutral, since it is financed through a decrease in public spending on unproductive items, υ_U.

The rise in the supply of public health services positively affects the health status of the population, h^A (see (3.12) and (3.19)), and, consequently, raises the probability of surviving from adulthood to old age. This, in turn, broadens individuals' time horizons and the incentive to participate in the labour market and save for old-age consumption, thus increasing ε^W and σ, respectively. This reaction has a positive impact on the growth rate (see (5.3)).

On the other hand, there is a negative indirect effect associated with k^I, which decreases as a result of the rise in υ_H and has a negative impact on the growth rate. The variable k^I translates the public-private capital ratio, since it is defined as the fraction of the public capital stock in infrastructure, K^I, and the aggregate private capital stock, K^P. An increase in υ_H has a negative impact on k^I because it increases K^P, the denominator.

The aggregate private capital stock, K^P, is determined by the population's investment, which is influenced by their labour income and propensity to save, as can be seen in (A.9).

Since a higher υ_H motivates a rise in labour market participation and propensity to save, it enhances investment and raises the aggregate private capital stock, K^P. However, the

public capital stock in infrastructure, K^I, which is the numerator, is mostly determined by government investment in infrastructure (see (3.18)), meaning that the shift in υ_H does not affect it directly. The reduction in k^I leads to a decrease in the supply of public health services, as can be seen in (3.19), which partially offsets the positive impact of the policy on h^A.

It is relevant to refer that k^I is not directly affected since there is no trade-off associated with the increase in υ_H due to the existence of an unproductive category of government expenditure. Otherwise, the government would have to reduce the proportion of public expenditure on infrastructure, υ_I, to finance the increase in υ_H, and the crowding-out effect we described would be more accentuated.

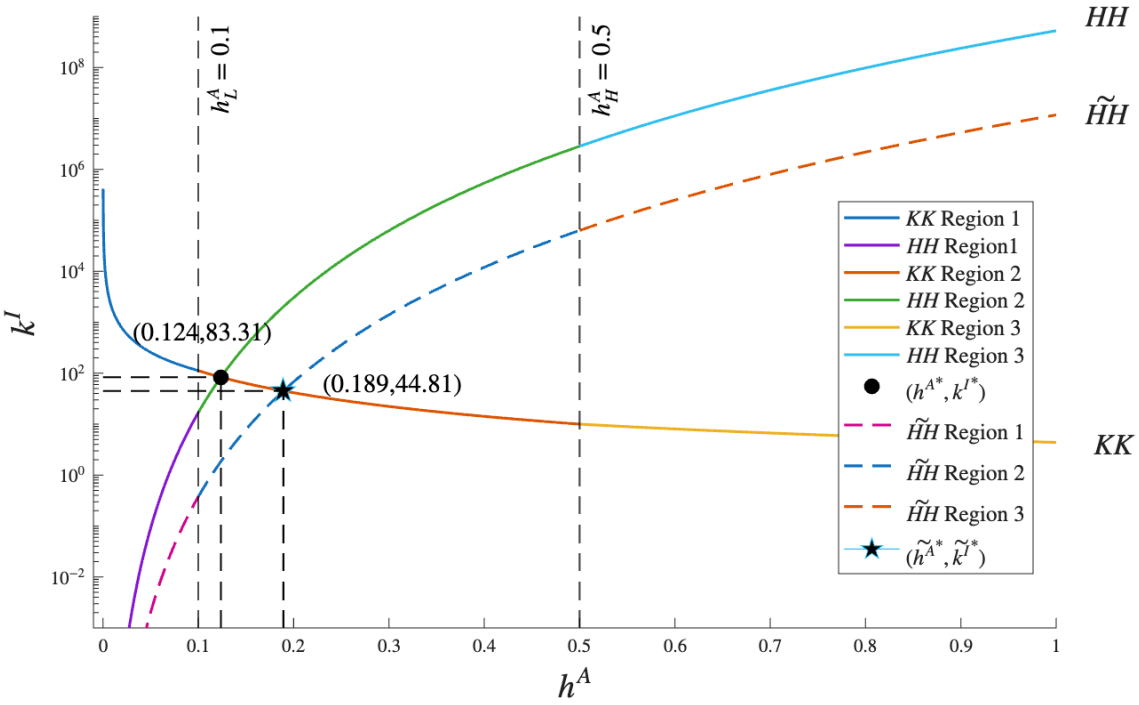

Figure 6.1: KK and HH for $\upsilon_H = 0.1385$

There is a downward shift of HH, which now corresponds to $\tilde{HH}$, while KK does not shift. There is a new steady-state equilibrium $(\tilde{h^{A*}}, \tilde{k^{I*}}) = (0.189, 44.81)$. Every other parameter is calibrated using the same values as Figure 5.2.

Figure 6.1 shows that the increase in υ_H causes a downward shift of HH, while KK does not move (see (4.5) and (4.6)). The new steady-state equilibrium is characterized by a higher h^A and a lower k^I, passing from $(h^{A*}, k^{I*}) = (0.124, 83.31)$ to $(\tilde{h^{A*}}, \tilde{k^{I*}}) = (0.189, 44.81)$. The steady-state values assumed by σ and ε^W are now 0.33 and 0.536, respectively.

The new steady-state growth rate is thus $(44.81)^{0.16}(0.536 \cdot 0.189)^{0.7} 0.7 \cdot 0.33(1-0.1717) = 7.08\%$, according to (5.3), which constitutes a rise from the original steady-state growth rate of 5.5%.

We can conclude that an increase in the proportion of public expenditure on health from 3.85% to 13.85% allows the government to simultaneously promote sustained growth and better health results for the population, since the crowding-out effect manifested through a decrease in the steady-state level of k^I is not sufficient to cancel out the positive impact of the economy's enhanced labour productivity, market participation and savings on growth. However, depending on the magnitude of the shift in HH, it is possible that an increase in υ_H could lead to a different outcome, as Figure 6.1 and Equation (5.3) illustrate.

The existence of such crowding-out effect makes it interesting to test different government policies, such as changes in the tax rate, or to consider exogenous changes to structural parameters, such as φ, which measures the efficiency of public expenditure on health. For instance, interventions involving τ cause a shift in both curves, as can be inferred from (4.5) and (4.6).

6.2 Alternative functional forms

In this section, we analyse whether the results presented in Chapter 5, namely the existence of a unique and locally stable equilibrium, apply when we consider alternative functional forms for the probability of surviving from adulthood to old age.

First, we employ alternative functional forms for $p_t = f(h_t^A)$ in (3.20), meaning that we are still considering piecewise forms. We then test smooth functional forms over the entire domain and examine whether this impacts the results.

We perform similar simulations to those presented in Chapter 5, where we look into the set of equilibria for an interval of values of $\chi \in (0, 1)$, as well as find intersections between KK and HH for each of the alternative functional forms for $f(h_t^A)$.

In the context of piecewise forms, we consider a square root functional form[1] and a linear functional form[2], and find that the same results apply in both cases. We ensure continuity between the intervals. The alternative functional forms in the second interval only have a very insignificant impact on the coordinates of the equilibrium, and every result is analogous to those presented in Chapter 5, including the occurrence of spiral convergence.

When we employ a smooth function over the whole domain, the same situation occurs. We experiment with a concave functional form similar to Chakraborty's (2004), $\bar{p} \cdot (h_t^A)/(1+h_t^A)$,

[1] $f(h_t^A) = 0.5139260913 + 0.5884173646 \cdot (h_t^A)^{0.5}$
[2] $f(h_t^A) = 0.575 \cdot h_t^A + 0.6425$

with $\bar{p} = 2$, and a logistic function $1/(1 + e^{-h_t^A})$, and find that it has no impact on the results aside from a deviation in the equilibrium coordinates.

Hence, our results are not sensitive to the choice of functional form. Regardless of the functional form we employ, the economy is not subject to a multiple equilibria poverty trap, and has a unique and locally stable steady-state equilibrium. Whether it is subject to a single equilibrium health-induced poverty trap depends on the parameter values, since they determine the location of the equilibrium and, thus, its position relative to a poverty line.

We are particularly interested in the influence of the degree of health persistence, κ, on the economy's steady-state equilibrium and on the potential emergence of a health-induced poverty trap. Thus, we explore the sensitivity of the steady-state equilibrium regarding κ in the following section.

6.3 Steady-state equilibrium as a function of κ

In Section 4.2, we illustrated how the degree of health persistence, κ, impacts the slope of HH and, consequently, the set of equilibria. Moreover, when calibrating κ in Chapter 5, we followed the implicit value used by Osang and Sarkar (2008), which the authors chose arbitrarily, given the lack of empirical estimates for this parameter.

Therefore, it is pertinent to explore how sensitive the economy's steady-state equilibrium is with respect to κ, and whether high enough values of κ that ensure $\zeta_1 > 1$ (and, consequently, a negatively sloped HH curve) result in a multiple equilibria poverty trap.

We perform a similar simulation to that underlying Figure 5.1, where we calibrate every parameter except κ, and present the results in Table 6.1. It reveals how the steady-state growth rate $(1 + \gamma)(\%)$ and health status (h^{A^*}) react to different degrees of health persistence.

Table 6.1 translates the intuition behind the dynamic role of κ we presented in Section 4.2. Lower values of κ are associated with higher steady-state levels of health status. As the elasticity of the current health status with regard to past health conditions increases, the more significant becomes the intergenerational transmission of health results and the parent-to-child externality.

As κ increases and the steady-state level of h^A decreases, so does the steady-state growth rate. The lower health status and, consequently, lower savings rate and labour market participation, have a negative impact on the steady-state growth rate, as can be inferred from (5.3). While there occurs a simultaneous increase in the steady-state public-private capital ratio (k^{I^*}), which has a positive impact on the growth rate, the net effect is negative.

Table 6.1: Steady-state growth rate and health status as a function of κ

$\boldsymbol{\kappa}$	0.1	0.2	0.3	0.4	0.5	0.6	0.7	0.8
$\boldsymbol{h^{A*}}$	0.289	0.245	0.198	0.149	0.098	0.051	0.015	0.001
$\boldsymbol{1+\gamma}$	9.12	8.27	7.29	6.16	4.86	3.31	1.73	0.4

After κ reaches approximately 0.83, KK and HH no longer intersect for positive levels of health status. Moreover, the steady-state value of h^A explodes when its exponent's denominator, $(1-\zeta_1)/\zeta_2 + \beta(1-\chi)/\Pi$, approaches 0 (see (4.9) and (4.29)).

Furthermore, the slope of HH becomes negative as κ crosses 0.89. While, in principle, a downward-sloping HH could correspond to a scenario of multiple steady-state equilibria, as we proposed on Figure 4.2, there is no intersection between the curves since there is a large gap between their vertical positions.

We conclude that the economy is not subject to a multiple equilibria health-induced poverty trap, regardless of the degree of health persistence. However, when the degree of health persistence is high, the steady-state equilibrium is characterized by poor health results that are transmitted to future generations, which results in low savings and low productivity, thus hindering growth.

Such a steady-state result may be interpreted as a single equilibrium health-induced poverty trap, since the economy converges to a state of self-reinforcing poor health and hindered growth regardless of its initial conditions.

7 Concluding remarks

While most economic literature assumes uniqueness of equilibria, a portion of research has devoted itself to studying the self-reinforcing dynamics leading to multiple equilibria. In systems exhibiting history dependence, there exist several attractors from which the equilibrium is determined based on the economy's initial conditions (Setterfield, 1997).

In the context of health literature, intergenerational health persistence is often pointed as a root of persistent poverty in developing countries (e.g., Bhalotra & Rawlings, 2011; Chakraborty & Das, 2005), in the sense that adverse health conditions are transmitted across generations, which might trap the economy in a state of poor health, productivity and growth.

The purpose of this dissertation has been to study an economy characterized by health persistence, which we present in Chapter 3, and determine under which conditions it is subject to a health-induced poverty trap.

We extend the model proposed by Agénor (2015), which considers a three-period OLG economy characterized by intergenerational health persistence, since health conditions are transmitted from parents to children and health status in adulthood is entirely determined by health status in childhood.

We work with an endogenous probability of surviving from adulthood to old age, which is related to the health status of the population in a piecewise fashion. Our approach diverges from Agénor's (2015), namely since we depart from a key assumption made by the author to reduce the system to one dimension, since studying the two-dimensional system becomes analytically challenging when the endogeneity of the survival probability is introduced.

The analytical study of the model presented in Chapter 4 allows us to derive conditions of existence and stability of interior equilibria in the three regions of the survival probability's domain, as well as the equilibrium behaviour of the propensity to save and the labour market participation. We find that multiple equilibria are a theoretical possibility, and the thresholds separating the three regions have a crucial role in this result. The introduction of endogenous

longevity defined in a piecewise fashion allows for potential shifts in dynamics across the three regions.

Furthermore, we find that the degree of health persistence, κ, has a critical role in this theoretical result, since a higher dependence of future health results on the current health status of the population increases the likelihood that the economy's initial health conditions dictate whether it enters a vicious cycle of self-reinforcing bad health conditions, or a prosperous cycle of good health and productivity.

We calibrate the model using data from India in Chapter 5 and numerically explore these theoretical results.

We introduce a closed-form expression for the survival probability and numerically study the model. We find that the economy is not subject to a multiple equilibria health-induced trap, since there is a unique steady-state equilibrium regardless of the value assumed by the technology parameter χ. However, higher values of χ translate to lower steady-state health outcomes, making the economy settle into a state of high mortality and low levels of productivity, savings and labour market participation. Steady-state equilibria characterized by adverse health conditions that are transmitted to future generations may be interpreted as single equilibrium health-induced poverty traps.

For a calibrated χ, there is a unique and locally stable steady-state equilibrium characterized by spiral convergence. The steady-state level of the population's health status is much lower than the economy's initial health status, which means there is a deterioration of health conditions in the convergence process, and possibly a reduction in the steady-state growth rate.

Finally, in Chapter 6 we perform a sensitivity analysis, where we examine the effectiveness of an increase in public health expenditure on rising the economy's steady-state health outcomes and growth rate.

Since there is a unique and locally stable equilibrium, the government might try to shift its position with this sort of direct intervention, as to avoid convergence to a low health status.

The increase in the supply of public health services positively affects the population's health status, which has a favorable impact on the steady-state growth rate since a higher survival probability enhances labour market participation and encourages savings. However, there is an indirect crowding-out effect associated with the negative impact that the reduced public-private capital ratio has on the growth rate, since higher savings translate into higher investment and an increased stock of private capital. In our simulation, the net effect is positive and the

intervention is able to simultaneously enhance the growth rate and the population's health status.

Additionally, we find that the same results apply when we consider alternative functional forms for the survival probability. While the results are not sensitive to the choice of closed form, they are sensitive to the degree of health persistence. We test how the steady-state level of health status reacts to different degrees of health persistence and find that higher degrees of health persistence are associated with lower steady-state health results, as was suggested earlier. Thus, higher degrees of health persistence are associated with persistent poor health and may lead to single equilibrium health-induced poverty traps.

The main limitation of this study regards the public-private capital ratio, k^I, which can reach exceedingly large values (as can be observed in Figure 5.2). This points to a lack of internal stabilizing mechanisms in the model, in order to avoid such explosive dynamics. We calibrate χ at 0.3 in order to anchor the steady-state value of k^I, while keeping the technology parameter at a reasonable level, to try to attenuate this effect, but we believe it poses as an opportunity for future research.

In addition, the fact that the survival probability is defined in a piecewise fashion creates a forced switch of dynamics between the intervals, which overstates the plausibility of a multiple equilibria health-induced poverty trap until one calibrates the model.

Moreover, the analysis can be extended in various directions. First, across our analysis, it has been assumed that survival from adulthood to old age is uncertain, while survival from childhood to adulthood is guaranteed. It might make sense to consider a non-zero probability of dying between the first and second periods of life, particularly in the context of countries characterized by high infant mortality rates.

We could also account for human capital accumulation and its impact on productivity through (3.14), which changes the output elasticity with respect to effective labour through (3.9) and $a_t = A_t$. This enables the inspection of the impact of health persistence on human capital outcomes mentioned in Section 2.4.

Finally, we could account for other determinants of health status, such as environmental quality, which continues to gain attention in the literature, and is often found to improve public health outcomes in the long run (e.g., Omri et al., 2024).

Appendix A
Mathematical demonstrations

A.1 Household optimization problem

From (3.1), and considering $\varepsilon^R = 0$, each individual maximizes

$$U = \ln c_{t+1}^t + \eta_L \ln(1 - \varepsilon_{t+1}^W) + p_{t+1} \frac{\ln c_{t+2}^t}{1+\rho}, \tag{A.1}$$

with respect to c_{t+1}^t, c_{t+2}^t, and ε_{t+1}^W, subject to (3.4).

The intertemporal household maximization problem is thus defined as

$$\max_{c_{t+1}^t, c_{t+2}^t, \varepsilon_{t+1}^W} U(c_{t+1}^t, c_{t+2}^t, \varepsilon_{t+1}^W) \qquad s.t. \qquad c_{t+1}^t + \frac{p_{t+1} c_{t+2}^t}{1+r_{t+2}} = (1-\tau)\varepsilon_{t+1}^W a_{t+1} w_{t+1}.$$

Using the Lagrangian Method to solve the optimization problem, the Lagrangian function is defined as

$$L(c_{t+1}^t, c_{t+2}^t, \varepsilon_{t+1}^W, \lambda) =$$

$$= U(c_{t+1}^t, c_{t+2}^t, \varepsilon_{t+1}^W) - \lambda[c_{t+1}^t + \frac{p_{t+1} c_{t+2}^t}{1+r_{t+2}} - (1-\tau)\varepsilon_{t+1}^W a_{t+1} w_{t+1}]$$

By calculating the partial derivatives of the Lagrangian function with respect to its four arguments, we obtain the first-order conditions of the optimization problem:

$$\begin{cases} \dfrac{\partial L}{\partial c^t_{t+1}} & = \dfrac{1}{c^t_{t+1}} - \lambda = 0, \\ \dfrac{\partial L}{\partial c^t_{t+2}} & = \dfrac{p_{t+1}}{1+\rho}\dfrac{1}{c^t_{t+2}} - \lambda\left(\dfrac{p_{t+1}}{1+r_{t+2}}\right) = 0, \\ \dfrac{\partial L}{\partial \varepsilon^W_{t+1}} & = -\dfrac{\eta_L}{1-\varepsilon^W_{t+1}} - \lambda[-(1-\tau)a_{t+1}w_{t+1}] = 0, \\ \dfrac{\partial L}{\partial \lambda} & = -c^t_{t+1} - \dfrac{p_{t+1}c^t_{t+2}}{1+r_{t+2}} + (1-\tau)\varepsilon^W_{t+1}a_{t+1}w_{t+1} = 0, \end{cases}$$

which is equal to

$$\begin{cases} \lambda & = \dfrac{1}{c^t_{t+1}}, \\ \dfrac{1}{(1+\rho)c^t_{t+2}} & = \dfrac{\lambda}{1+r_{t+2}} \\ \dfrac{\eta_L}{1-\varepsilon^W_{t+1}} & = \lambda(1-\tau)a_{t+1}w_{t+1}, \\ c^t_{t+1} + \dfrac{p_{t+1}c^t_{t+2}}{1+r_{t+2}} & = (1-\tau)\varepsilon^W_{t+1}a_{t+1}w_{t+1}. \end{cases}$$

The first-order conditions yield

$$\frac{c^t_{t+2}}{c^t_{t+1}} = \frac{1+r_{t+2}}{1+\rho}, \tag{A.2}$$

and

$$\frac{\eta_L}{1-\varepsilon^W_{t+1}} = \frac{(1-\tau)a_{t+1}w_{t+1}}{c^t_{t+1}}. \tag{A.3}$$

Subtituting (A.2) in (3.4) results in

$$c^t_{t+1} = \left[\frac{1+\rho}{1+\rho+p_{t+1}}\right](1-\tau)\varepsilon^W_{t+1}a_{t+1}w_{t+1}, \tag{A.4}$$

meaning that savings are equal to

$$s_{t+1} = 1 - c^t_{t+1} = \sigma_{t+1}(1-\tau)\varepsilon^W_{t+1} a_{t+1} w_{t+1}, \qquad \sigma_{t+1} = \frac{p_{t+1}}{1+\rho+p_{t+1}} < 1. \tag{A.5}$$

Substituting (A.4) in (A.3) yields

$$\frac{\eta_L}{1-\varepsilon^W_{t+1}} = \frac{1}{(1-\sigma_{t+1})\varepsilon^W_{t+1}}, \tag{A.6}$$

which, solving for ε^W_{t+1}, corresponds to

$$\varepsilon^W_{t+1} = \frac{1}{1+\eta_L(1-\sigma_{t+1})} < 1. \tag{A.7}$$

Substituting out for σ_{t+1}, using (A.5), yields

$$\varepsilon^W_{t+1} = \frac{1+\rho+p_t}{(1+\rho)(1+\eta_L)+p_t} < 1. \tag{A.8}$$

A.2 Derivation of the dynamical system

To analyse the dynamic behaviour of this economy, substitute (A.5) in (4.1) to give

$$K^P_{t+1} = N_t s_t = \sigma_t(1-\tau) N_t \varepsilon^W_t a_t w_t, \tag{A.9}$$

which, substituting for w_t using (3.7) and $a_t = A_t$, is equal to

$$K^P_{t+1} = \beta\sigma_t(1-\tau) Y_t. \tag{A.10}$$

From (3.9),

$$K^P_{t+1} = \beta\sigma_t(1-\tau)(k^I_t)^\alpha (\varepsilon^W_t)^\beta A^\beta_t K^P_t. \tag{A.11}$$

Using (3.16) and (3.18), we get

$$K_{t+1}^I = (\upsilon_I N_t \tau \varepsilon_t^W A_t w_t)^\chi (K_t^I)^{1-\chi}.$$

From (3.7), we get

$$K_{t+1}^I = (\upsilon_I \tau \beta Y_t)^\chi (K_t^I)^{1-\chi}.$$

Substituting for Y_t using (3.9) leads to

$$K_{t+1}^I = (\upsilon_I \tau \beta)^\chi \left[k_t^{I\alpha} (\varepsilon_t^W A_t)^\beta K_t^P\right]^\chi (K_t^I)^{1-\chi},$$

which is equal to

$$\begin{aligned} K_{t+1}^I &= (\beta \upsilon_I \tau)^\chi (\varepsilon_t^W)^{\beta\chi} \left[\frac{(k_t^I)^\alpha A_t^\beta K_t^P}{K_t^I}\right]^\chi K_t^I = \\ &= (\beta \upsilon_I \tau)^\chi (\varepsilon_t^W)^{\beta\chi} (k_t^I)^{-\chi(1-\alpha)} A_t^{\chi\beta} K_t^I. \end{aligned} \tag{A.12}$$

Combining (A.11) and (A.12) results in

$$k_{t+1}^I = \frac{K_{t+1}^I}{K_{t+1}^P} = \frac{(\beta \upsilon_I \tau)^\chi (\varepsilon_t^W)^{\beta\chi} (k_t^I)^{-\chi(1-\alpha)} A_t^{\chi\beta} K_t^I}{\beta \sigma_t (1-\tau)(k_t^I)^\alpha (\varepsilon_t^W)^\beta A_t^\beta K_t^P},$$

which, from (3.14) and $a_t = A_t$, is equal to

$$k_{t+1}^I = \frac{(\beta \upsilon_I \tau)^\chi (\varepsilon_t^W)^{-\beta(1-\chi)}}{\beta \sigma_t (1-\tau)} (k_t^I)^{(1-\alpha)(1-\chi)} (h_t^A)^{-\beta(1-\chi)}. \tag{A.13}$$

Regarding the dynamics of h_t^A, the expression (3.19) is equal to

$$H_t^G = \frac{(K_t^I)^\mu}{(K_t^P)^{\mu\phi_H}} (\varphi G_t^H)^{1-\mu}. \tag{A.14}$$

Taking into account Assumption 2 ($1 - \mu\phi_H = 0$) and (3.16),

$$H_t^G = \frac{(K_t^I)^\mu}{K_t^P} \left(\varphi \upsilon_H N_t \tau \varepsilon_t^W A_t w_t\right)^{1-\mu}.$$

From (3.7), we get

$$\begin{aligned} H_t^G &= \frac{(K_t^I)^\mu}{K_t^P} (\varphi \upsilon_H \tau \beta Y_t)^{1-\mu} = \\ &= (k_t^I)^\mu (\varphi \upsilon_H \tau \beta)^{1-\mu} \left(\frac{Y_t}{K_t^P}\right)^{1-\mu}. \end{aligned}$$

Substituting for H_t^G in (3.13) yields

$$h_{t+1}^A = (\varphi \upsilon_H \tau \beta)^\nu (h_t^A)^\kappa (k_t^I)^{\mu(1-\nu_C)} \left(\frac{Y_t}{K_t^P}\right)^\nu, \tag{A.15}$$

where $\nu = (1 - \nu_C)(1 - \mu)$.

From (3.9) and (3.14), we get

$$h_{t+1}^A = (\varphi \nu_H \tau \beta)^\nu (\varepsilon_t^W)^{\beta\nu} (h_t^A)^{\zeta_1} (k_t^I)^{\zeta_2}, \tag{A.16}$$

where $\zeta_1 = \kappa + \nu\beta \quad$ and $\quad \zeta_2 = \mu(1 - \nu_C) + \nu\alpha$.

Equations (A.13) and (A.16) form a first-order nonlinear difference equation system:

$$\begin{cases} h_{t+1}^A = (\varphi \nu_H \tau \beta)^\nu (\varepsilon_t^W)^{\beta\nu} (h_t^A)^{\zeta_1} (k_t^I)^{\zeta_2} \\ k_{t+1}^I = \dfrac{(\beta \upsilon_I \tau)^\chi (\varepsilon_t^W)^{-\beta(1-\chi)}}{\beta \sigma_t (1 - \tau)} (k_t^I)^{(1-\alpha)(1-\chi)} (h_t^A)^{-\beta(1-\chi)} \end{cases} \tag{A.17}$$

The steady-state values of k^I and h^A, respectively k^{I*} and h^{A*}, are obtained by solving the system (A.17) with respect to k^I and h^A:

$$k^{I^*} = \left\{ \frac{(\beta \upsilon_I \tau)^{\chi} (\varepsilon_t^W)^{-\beta(1-\chi)}}{\beta \sigma_t (1-\tau)} \right\}^{1/\Pi} (h^{A^*})^{-\beta(1-\chi)/\Pi},$$

$$k^{I^*} = [\varphi \upsilon_H \tau \beta (\varepsilon_t^W)^{\beta}]^{-\nu/\zeta_2} (h^{A^*})^{(1-\zeta_1)/\zeta_2},$$

where $\Pi \equiv 1 - (1-\alpha)(1-\chi) \in (0,1)$.

A.3 Stability analysis

Considering $\hat{h}_t^A = \ln h_t^A$ and $\hat{k}_t^I = \ln k_t^I$, the system (A.17) is given by

$$\begin{cases} \hat{h}_{t+1}^A = \ln(\varphi \upsilon_H \tau \beta)^{\nu} + \ln(\varepsilon_t^W)^{\beta\nu} + \zeta_1 \hat{h}_t^A + \zeta_2 \hat{k}_t^I \\ \hat{k}_{t+1}^I = \ln\left(\dfrac{(\beta \upsilon_I \tau)^{\chi}}{\beta(1-\tau)} \right) + \ln\left(\dfrac{(\varepsilon_t^W)^{-\beta(1-\chi)}}{\sigma_t} \right) + (1-\alpha)(1-\chi)\hat{k}_t^I - \beta(1-\chi)\hat{h}_t^A \end{cases} \tag{A.18}$$

In the first interval, $h_t^A < h_L^A$, and p_t, σ_t and ε_t^W are constant and equal to p_m, σ_m and ε_m^W, respectively. The system can be written in matrix form

$$\begin{bmatrix} \hat{h}_{t+1}^A \\ \hat{k}_{t+1}^I \end{bmatrix} = \begin{bmatrix} a_{10} \\ a_{20} \end{bmatrix} + \begin{bmatrix} a_{11} & a_{12} \\ a_{21} & a_{22} \end{bmatrix} \begin{bmatrix} \hat{h}_t^A \\ \hat{k}_t^I \end{bmatrix}, \tag{A.19}$$

where

$a_{10} = \ln[(\varphi \upsilon_H \tau \beta)^{\nu} (\varepsilon_m^W)^{\nu\beta}]$,

$$a_{20} = \ln\left\{ \frac{(\beta \upsilon_I \tau)^{\chi} (\varepsilon_m^W)^{-\beta(1-\chi)}}{\beta \sigma_m (1-\tau)} \right\},$$

$a_{11} = \kappa + \beta(1-\nu_C)(1-\mu) \equiv \zeta_1 > 0$,

$a_{12} = (1-\nu_C)[\mu + \alpha(1-\mu)] \equiv \zeta_2 > 0$,

$a_{21} = -\beta(1-\chi) < 0$,

$a_{22} = (1-\alpha)(1-\chi) > 0$.

Given that the sole distinction between the first and third intervals lies in the constant values assumed by p_t, σ_t and ε_t^W (p_m, σ_m and ε_m^W in the first interval, and p_M, σ_M and ε_M^W in the third interval, respectively), the Jacobian matrix remains identical across these intervals and only the elements a_{10} and a_{20} differ. Consequently, the fixed points in these intervals differ in level, yet they share the same stability properties.

However, in the second interval ($h_L \leq h_t^A < h_H^A$), p_t, σ_t and ε_t^W are endogenously related to h_t^A, such that $p_t = f(h_t^A)$, $\sigma_t = \sigma(h_t^A)$ and $\varepsilon_t^W = \varepsilon^W(h_t^A)$. This implies that the system (A.17) is equal to

$$\begin{cases} h_{t+1}^A = (\varphi \nu_H \tau \beta)^\nu [\varepsilon^W(h_t^A)]^{\beta\nu} (h_t^A)^{\zeta_1} (k_t^I)^{\zeta_2} \\ k_{t+1}^I = \dfrac{(\beta \upsilon_I \tau)^\chi [\varepsilon^W(h_t^A)]^{-\beta(1-\chi)}}{\beta(1-\tau)\sigma(h_t^A)} (k_t^I)^{(1-\alpha)(1-\chi)} (h_t^A)^{-\beta(1-\chi)} \end{cases} \tag{A.20}$$

From (A.20), it can be inferred that the Jacobian matrix in the second interval differs from that of the first and third intervals, which means the equilibria might not all have the same stability properties. The Jacobian matrix in the second interval is contingent upon the chosen closed form for $f(h_t^A)$ and, consequently, $\sigma(h_t^A)$ and $\varepsilon(h_t^A)$.

However, we can derive the Jacobian matrix in the second interval from the log-linearized system (A.18) and evaluate it at the steady-state equilibrium (h_2^{A*}, k_2^{I*}) without assuming a closed form, taking into account $\sigma_t = \sigma(h_t^A)$ and $\varepsilon_t^W = \varepsilon^W(h_t^A)$:

$$\begin{bmatrix} \zeta_1 + \beta\nu \left.\frac{d\ln\varepsilon^W(h^A)}{d\ln(h^A)}\right|_{h^A=h_2^{A*}} & \zeta_2 \\ -\beta(1-\chi) - \beta(1-\chi) \left.\frac{d\ln\varepsilon^W(h^A)}{d\ln(h^A)}\right|_{h^A=h_2^{A*}} - \left.\frac{d\ln\sigma(h^A)}{d\ln(h^A)}\right|_{h^A=h_2^{A*}} & (1-\alpha)(1-\chi) \end{bmatrix} \tag{A.21}$$

We can expand on the elements of the Jacobian matrix in the following way:

$$\left.\frac{d\ln\sigma(h^A)}{d\ln(h^A)}\right|_{h^A=h_2^{A*}} = \left.\frac{d\sigma(h^A)}{dh^A}\right|_{h^A=h_2^{A*}} \cdot \frac{h_2^{A*}}{\sigma^*}, \tag{A.22}$$

$$\left.\frac{d\ln\varepsilon^W(h^A)}{d\ln(h^A)}\right|_{h^A=h_2^{A*}} = \left.\frac{d\varepsilon^W(h^A)}{dh^A}\right|_{h^A=h_2^{A*}} \cdot \frac{h_2^{A*}}{\varepsilon^{W*}}, \tag{A.23}$$

which, from (4.19) and (4.21), respectively, yields

$$\left.\frac{d\ln\sigma(h^A)}{d\ln(h^A)}\right|_{h^A=h_2^{A*}} = \frac{f'(h_t^A)(1+\rho)}{[1+\rho+f(h_t^A)]^2}\cdot\frac{h_2^{A*}}{\sigma^*}, \tag{A.24}$$

$$\left.\frac{d\ln\varepsilon^W(h^A)}{d\ln(h^A)}\right|_{h^A=h_2^{A*}} = \frac{\eta_L\dfrac{d\sigma(h_t^A)}{dh_t^A}}{[1+\eta_L(1-\sigma(h_t^A))]^2}\cdot\frac{h_2^{A*}}{\varepsilon^{W*}}. \tag{A.25}$$

In Chapter 5, we calibrate the model and numerically explore this Jacobian matrix.

Appendix B
Complementary notes

Non-exhaustive list of variables and parameters:

η_L	relative preference for leisure	>0
ε^W	proportion of time devoted to market work	$(0,1)$
ε^R	proportion of time devoted to child rearing	$(0,1)$
n	fertility rate	>0
N	total number of adults	>0
p	probability of surviving from adulthood to old age	$(0,1)$
ρ	intergenerational discount factor	>0
τ	effective income tax rate	$(0,1)$
a	individual labour efficiency	>0
A	economy-wide labour efficiency	>0
r	rental rate of private capital	>0
Y	aggregate output	>0
K^I	stock of public capital in infrastructure	>0
K^P	stock of private capital	>0
k^I	public-private capital ratio	>0
α	output elasticity with respect to the public-private capital ratio	$(0,1)$

β	output elasticity with respect to effective labour	$(0,1)$
h^C	health status in childhood	>0
$\bar{h}^C$	health status at birth	>0
h^A	health status in adulthood	>0
H^G	supply of public health services	>0
κ	elasticity of health status in childhood with respect to parental health status (health persistence)	$(0,1)$
ν_C	elasticity of health status in childhood with respect to child-rearing time	$(0,1)$
G	total government expenditure	>0
G^I	government expenditure on infrastructure	>0
G^H	government expenditure on health services	>0
G^U	government expenditure on unproductive items	>0
υ_h	government expenditure shares on the three categories of spending, $h=I,H,U$	$(0,1)$
χ	technology parameter	$(0,1)$
μ	elasticity of the production of health services with respect to the public-private capital ratio	$(0,1)$
φ	measure of government expenditure efficiency	$(0,1)$
σ	marginal propensity to save	$(0,1)$

Table B1: Non-exhaustive list of variables and parameters, along with their respective meanings and restrictions.

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Annex

Annex A: Figure 5.1

The following code was executed using MATLAB. It corresponds to Figure 5.1 and displays how the value of χ affects the steady-state level of h^A and, consequently, the stage of development the economy's steady-state equilibrium is located in.

We calibrate every parameter except χ, which is defined as an array of values between 0 and 1. From (4.9) and (4.29), we calculate h_1^{A*} and h_3^{A*} for each value of χ. In the second interval, we find potential h_2^{A*} by finding the intersections between KK and HH, which we define as functions of h^A and χ. We plot the steady-state level of h^A for each possible value of χ, which results in three colored regions, each corresponding to an interval of the survival probability's domain.

```
% Figure 5.1: Steady state level of h^A as a function of chi

clearvars
clear all

% Parameters

alpha=0.16;          beta=0.7;          tau=0.1717;        rho=0.5544;
eta_L=1.29;          kappa=0.45;        nu_C=0.8;          upsilon_H=0.0385;
upsilon_I=0.148;     phi=0.38;          mu=0.15;           p_m=0.7;
p_M=0.93;            h_L=0.1;           h_H=0.5;

nu=(1-nu_C)*(1-mu);      zeta_1=kappa+nu*beta;      zeta_2=mu*(1-nu_C)+nu*alpha;

sigma_m=p_m/(1+rho+p_m);                sigma_M=p_M/(1+rho+p_M);
epsilon_m=1/(1+eta_L*(1-sigma_m));      epsilon_M=1/(1+eta_L*(1-sigma_M));

% Define chi as an array of values between 0 and 1

chi_array=linspace(0,1,1500);
Pi=1-(1-alpha)*(1-chi_array);

% Determine h_star1(chi) and h_star3(chi) numerically
% First interval

numerator_1=((((beta*upsilon_I*tau).^(chi_array)).*(epsilon_m.^(-beta.*(1-chi_array
        )))))./(beta*sigma_m*(1-tau))).^(1./Pi);
```

```
denominator_1=((phi*upsilon_H*tau*beta)*(epsilon_m^beta))^(-nu/zeta_2);

hA1_star=(numerator_1./denominator_1).^(1./((1-zeta_1)/zeta_2+beta.*(1-chi_array)./
        Pi));

% Third interval

numerator_3=((((beta*upsilon_I*tau).^(chi_array)).*(epsilon_M.^(-beta.*(1-chi_array
        )))).*(beta*sigma_M*(1-tau))).^(1./Pi);

denominator_3=((phi*upsilon_H*tau*beta)*(epsilon_M^beta))^(-nu/zeta_2);

hA3_star=(numerator_3./denominator_3).^(1./((1-zeta_1)/zeta_2+beta.*(1-chi_array)./
        Pi));

% Second interval: numerically find the root of KK-HH
% Define KK-HH

function func=f(h_eq,chi_func,Pi_func,beta,upsilon_I,tau,nu_C,mu,phi,upsilon_H,rho,
        eta_L,zeta_1,zeta_2)

    p=0.94875*h_eq./(1+h_eq)+0.61375;
    sigma=p./(1+rho+p);
    epsilon=1./(1+eta_L.*(1-sigma));

    KK=(((beta*upsilon_I*tau)^chi_func)*(epsilon^(-beta*(1-chi_func)))/(beta*sigma
        *(1-tau)))^(1/Pi_func)*h_eq^(-beta*(1-chi_func)/Pi_func);

    HH=((phi*upsilon_H*tau*beta*(epsilon^beta)))^(-(1-nu_C)*(1-mu)/zeta_2)*h_eq
        ^((1-zeta_1)/zeta_2);

    func=KK-HH;
end

% Solve KK-HH for every value of chi

hA2_star=nan(size(chi_array));

for i=1:numel(chi_array)
    chi_func=chi_array(i);
    Pi_func=Pi(i);

    for n=0.5;
        try
            eq=fzero(@(h_eq) f(h_eq,chi_func,Pi_func,beta,upsilon_I,tau,nu_C,mu,phi
                ,upsilon_H,rho,eta_L,zeta_1,zeta_2),n);
            if isreal(eq) && eq>0
                hA2_star(i)=eq;
                break;
            end
        catch
        end
    end


end

% Plots
```

```
figure('Color','w'); hold on;

% First interval

region1=hA1_star<h_L;
h1plot=plot(chi_array,hA1_star,'LineWidth',2,'Color',[0 0.4470 0.7410]);
hA1_star(~region1)=NaN;
hArea1= area(chi_array,hA1_star,'FaceAlpha',0.25,'FaceColor',[0 0.4470 0.7410],
        'EdgeColor','none');

% Second interval

region2=(hA2_star>h_L) & (hA2_star<h_H);
hsplot=plot(chi_array,hA2_star,'k-','LineWidth',2,'Color','[0 0.5 0]');
hA2_star(~region2)=NaN;
hArea2=area(chi_array,hA2_star,'FaceAlpha',0.25,'FaceColor',[0.4660 0.6740 0.1880],
        'EdgeColor','none');

% Third interval

region3=hA3_star>0.5;
h3plot=plot(chi_array,hA3_star,'LineWidth',2,'Color',[0.8500 0.3250 0.0980]);
hA3_star(~region3)=NaN;
hArea3=area(chi_array, hA3_star,'FaceAlpha',0.25,'FaceColor',[0.8500 0.3250
        0.0980],'EdgeColor','none');

% Threshold h_L=0.1

threshold_L=find(diff(region1)~=0,1,'first');
plot([chi_array(threshold_L) chi_array(threshold_L)],[0 hA1_star(threshold_L)],'k--
        ','LineWidth',1);
text(chi_array(threshold_L)*0.90,-0.035,sprintf('%.3f',chi_array(threshold_L)),
        'Interpreter','latex','FontSize',14);

% Threshold h_L=0.5

threshold_H=find(diff(region3)~=0,1,'first');
plot([chi_array(threshold_H) chi_array(threshold_H)],[0 hA3_star(threshold_H)],'k--
        ','LineWidth',1);
text(chi_array(threshold_H),-0.035,sprintf('%.3f',chi_array(threshold_H)),
        'Interpreter','latex','FontSize',14);

% Legend

yline(h_L,'k--','$h_L^A=0.1$','LineWidth',1,'Interpreter','latex','FontSize',14);
yline(h_H,'k--','$h_H^A=0.5$','LineWidth',1,'Interpreter','latex','FontSize',14);
xlabel('$\chi$','Interpreter','latex','FontSize',18);
ylabel('$h^A$','Interpreter','latex','FontSize',18);
legend([h1plot,hsplot,h3plot,hArea1,hArea2,hArea3],{'${h^A_1}^*(\chi)$','${h^A_2
        }^*(\chi)$','${h^A_3}^*(\chi)$','$0<{h^A_1}^*(\chi)<h_L^A$','$h_L\leq {h^A_2
        }^*(\chi)<h_H^A$','${h^A_3}^*(\chi)\geq h_H^A$'},'Interpreter','latex',
        'Location','best','FontSize',12);
grid off;
```

Annex B: Figure 5.2

The following code was executed using MATLAB. It corresponds to Figure 5.2 and displays HH and KK for $\chi = 0.3$.

We define and plot HH and KK in the three intervals of the survival probability's domain and find the intersection of the curves, which corresponds to the steady-state equilibrium $(h^{A*}, k^{I*}) = (0.124, 83.31)$.

```
% Figure 5.2: KK and HH for chi=0.3

clearvars
clear all

% Parameters

alpha= 0.16;          beta=0.7;          tau=0.1717;          rho=0.5544;
eta_L=1.29;           kappa=0.45;        nu_C=0.8;            upsilon_H=0.0385;
upsilon_I=0.148;      phi=0.38;          mu=0.15;             p_m=0.7;
p_M=0.93;             chi=0.3;           h_L=0.1;             h_H=0.5;

nu=(1-nu_C)*(1-mu);                      zeta_1=kappa+nu*beta;
zeta_2=mu*(1-nu_C)+nu*alpha;             Pi=1-(1-alpha)*(1-chi);

sigma_m=p_m/(1+rho+p_m);                     sigma_M=p_M/(1+rho+p_M);
epsilon_m=1/(1+eta_L*(1-sigma_m));           epsilon_M=1/(1+eta_L*(1-sigma_M));

% Define KK and HH in the first and third intervals

KK=@(epsilon,sigma,h) (((beta*upsilon_I*tau)^chi.*(epsilon).^(-beta*(1-chi))./
    (beta*sigma*(1-tau))).^(1/Pi).*h.^(-beta*(1-chi)/Pi);

HH=@(epsilon,h) ((phi*upsilon_H*tau*beta*(epsilon.^beta)).^(-(1-nu_C)*(1-mu)/
        zeta_2)).*h.^((1-zeta_1)/zeta_2);

% Define KK and HH in the second interval

KK2=@(h) (((beta*upsilon_I*tau)^chi.*(((1+rho+(0.94875*h./(1+h)+0.61375))./
    ((0.94875*h./(1+h)+0.61375)+(1+rho)*(1+eta_L)))).^(-beta*(1-chi))./(beta*((
    0.94875*h./(1+h)+0.61375)./ ...
    (1+rho+(0.94875*h./(1+h)+0.61375)))*(1-tau))).^(1/Pi).*h.^(-beta*(1-chi)/Pi);

HH2=@(h) ((phi*upsilon_H*tau*beta*(((1+rho+(0.94875*h./(1+h)+0.61375))./
    ((0.94875*h./(1+h)+0.61375)+(1+rho)*(1+eta_L)))).^beta).^(-(1-nu_C)*(1-mu)/
    zeta_2)).*h.^((1-zeta_1)/zeta_2);

% First interval: 0 <= h^A < h_L

h1=linspace(0.0001,h_L,300);
kk1=KK(epsilon_m,sigma_m,h1);
hh1=HH(epsilon_m,h1);

% Second interval: h_L <= h^A < h_H

h2=linspace(h_L,h_H,400);
kk2=KK2(h2);
```

```
hh2=HH2(h2);

% Third interval: h^A >= h_H

h3=linspace(h_H,1.0,300);
kk3=KK(epsilon_M,sigma_M,h3);
hh3=HH(epsilon_M,h3);

% Plots

figure('Color','w'); hold on;

kk1_plot=plot(h1,kk1,'-','LineWidth',1.5);
kk2_plot=plot(h2,kk2,'-','LineWidth',1.5);
kk3_plot=plot(h3,kk3,'-','LineWidth',1.5);

hh1_plot=plot(h1,hh1,'-','LineWidth',1.5);
hh2_plot=plot(h2,hh2,'-','LineWidth',1.5);
hh3_plot=plot(h3,hh3,'-','LineWidth',1.5);

xline(h_L,'k--','$h_L^A=0.1$','LineWidth',1,'Interpreter','latex','FontSize',14);
xline(h_H,'k--','$h_H^A=0.5$','LineWidth',1,'Interpreter','latex','FontSize',14);
ylabel('$k^I$','Interpreter','latex','FontSize',18);
xlabel('$h^A$','Interpreter','latex','FontSize',18);

axis([-0.01 1 0.001 1000000000])
text(1+0.026,1000000000,'$HH$','FontSize',16,'Interpreter','latex');
text(1+0.036,4,'$KK$','FontSize',16,'Interpreter','latex');

% Intersection

func=@(h_eq) KK2(h_eq)-HH2(h_eq);
h_star=fzero(func, 0.5);
k_star=KK2(h_star);

eq=plot(h_star,k_star,'ko','MarkerFaceColor','k','MarkerSize',8);
plot([h_star h_star],[min(ylim) k_star],'k--','LineWidth',1,'HandleVisibility',
        'off');
plot([0 h_star],[k_star k_star],'k--','LineWidth',1,'HandleVisibility','off');
text(h_star*1.2,k_star*1.4, sprintf('(%0.2f,%0.2f)',h_star,k_star),'FontSize',14,
        'Interpreter','latex', 'Color','k');

% Transform Y axis into log scale

ax=gca;
ax.YScale='log';
ax.TickLabelInterpreter='latex';

% Legend

legend([kk1_plot,hh1_plot,kk2_plot,hh2_plot,kk3_plot,hh3_plot,eq],{'$KK$ Region 1',
        '$HH$ Region1','$KK$ Region 2','$HH$ Region 2','$KK$ Region 3','$HH$ Region
        3','$({h^A}^*, {k^I}^*)$'},'Interpreter','latex','Location','best','FontSize',
        12);
grid off;

display(sprintf('(%0.9f,%0.9f)',h_star,k_star));
```

Annex C: Figure 5.3

The following code was written in Python and executed using Jupyter Notebook. It corresponds to Figure 5.3 and illustrates the system's phase diagram.

We define the difference equation system in order to determine the dynamic behavior of the state variables, h_t^A and k_t^I, by adding the time component and achieving h_{t+1}^A and k_{t+1}^I as functions of h_t^A and k_t^I. We plot the trajectory starting from alternative initial conditions using a quiver plot and reveal spiral convergence towards the steady-state equilibrium.

```
# Figure 5.3: Phase diagram

import numpy as np
import matplotlib.pyplot as plt

# Parameters

alpha=0.16
beta=0.7
tau=0.1717
chi=0.30
rho=0.5544
eta_L=1.29
kappa=0.45
nu_C=0.8
mu=0.15
phi=0.38
upsilon_I=0.148
upsilon_H=0.0385

nu=(1-nu_C)*(1-mu)
zeta_1=kappa+nu*beta
zeta_2=mu*(1-nu_C)+nu*alpha

# p, epsilon and sigma are a function of h in the second interval

def f(h):
    return 0.94875*h/(1+h)+0.61375

def epsilon(h):
    p=f(h)
    return (1+rho+p)/((1+rho)*(1+eta_L)+p)

def sigma(h):
    p=f(h)
    return p/(1+rho+p)

# Define HH and KK

def HH(h,k):
    return ((phi*upsilon_H*tau*beta)**nu)*(epsilon(h)**(beta*nu))*(h**zeta_1)*
        (k**zeta_2)

def KK(h,k):
    return ((beta*upsilon_I*tau)**chi)*(epsilon(h)**(-beta*(1-chi)))/(beta*sigma(h)
```

```
        *(1-tau))*(k**((1-alpha)*(1-chi)))*(h**(-beta*(1-chi)))


# Define the dynamic behavior of h and k: h_t+1=HH(h_t) and k_t+1=KK(k_t)

h2=np.linspace(0.1,0.35,20)
k2=np.linspace(40,160,20)

H,K=np.meshgrid(h2,k2)

dH=HH(H,K)-H
dK=KK(H,K)-K

dH=dH/np.sqrt(dH**2+dK**2)
dK=dK/np.sqrt(dH**2+dK**2)

plt.figure(figsize=(10,7),dpi=250)
plt.quiver(H,K,dH,dK,angles="xy")

# Add the time component and define the difference equation system

def DE(h0,k0,T=50):
    h_0,k_0=[h0],[k0]
    for t in range(1,T):
        h_1=HH(h_0[t-1],k_0[t-1])
        k_1=KK(h_0[t-1],k_0[t-1])
        h_0.append(h_1)
        k_0.append(k_1)
    return np.array(h_0), np.array(k_0)

# Plot the trajectory of alternative initial conditions

initial_conditions=[(0.10,100),(0.17,150),(0.12,115),(0.1,60),(0.2,120),(0.308,
        120),(0.308,150),(0.308,57)]

for h0, k0 in initial_conditions:
    h_0,k_0=DE(h0,k0)
    plt.plot(h_0,k_0,lw=1.7)
    plt.scatter(h0,k0,color="blue" s=25,zorder=2)

# Steady-state equilibrium

h_star=0.123722013
k_star=83.310831598

# Plot

plt.scatter(0.10,100,color="blue",label="Alternative $(h^A_0,k^I_0)$",zorder=1)
plt.scatter(h_star,k_star,color="red",s=40,label="$({h^A}^*,{k^I}^*)$",zorder=2)
plt.xlabel("$h_t^A$",fontsize=14)
plt.ylabel("$k_t^I$",fontsize=14)
plt.rcParams.update({"text.usetex":True,"font.family":"serif","font.serif":
        ["Garamond"],})
plt.legend(frameon=True,fancybox=True,edgecolor="black",facecolor="white",
        framealpha=1,loc="lower right")
plt.grid(False)

plt.savefig("phase.png")
```

```
plt.show()
```

Annex D: Figure 6.1

The following code was executed using MATLAB. It corresponds to Figure 6.1 and illustrates the impact of an increase in public health expenditure on the economy's steady-state equilibrium.

We plot HH and its shift due to the intervention, while KK is not affected. We define and plot HH, $\tilde{HH}$ and KK in the three intervals of the survival probability's domain and find the intersections of the curves, thus finding the equilibrium points before and after the intervention.

```
% Figure 6.1: KK and HH for upsilon_H=0.1385

clear all
clearvars

% Parameters

alpha= 0.16;          beta=0.7;          tau=0.1717;          rho=0.5544;
eta_L=1.29;           kappa=0.45;        nu_C=0.8;            upsilon_H=0.0385;
upsilon_I=0.148;      phi=0.38;          mu=0.15;             p_m=0.7;
p_M=0.93;             chi=0.3;           h_L=0.1;             h_H=0.5;
upsilon_H_alt=0.1385;

nu=(1-nu_C)*(1-mu);                      zeta_1=kappa+nu*beta;
zeta_2=mu*(1-nu_C)+nu*alpha;             Pi=1-(1-alpha)*(1-chi);

sigma_m=p_m/(1+rho+p_m);                     sigma_M=p_M/(1+rho+p_M);
epsilon_m=1/(1+eta_L*(1-sigma_m));           epsilon_M=1/(1+eta_L*(1-sigma_M));

% Define KK and HH in the first and third intervals

KK=@(epsilon,sigma,h) (((beta*upsilon_I*tau)^chi.*(epsilon).^(-beta*(1-chi))./
    (beta*sigma*(1-tau))).^(1/Pi).*h.^(-beta*(1-chi)/Pi);

HH=@(epsilon,h) ((phi*upsilon_H*tau*beta*(epsilon.^beta)).^(-(1-nu_C)*(1-mu)/
        zeta_2)).*h.^((1-zeta_1)/zeta_2);

% Define KK and HH in the second interval

KK2=@(h) (((beta*upsilon_I*tau)^chi.*(((1+rho+(0.94875*h./(1+h)+0.61375))./
    ((0.94875*h./(1+h)+0.61375)+(1+rho)*(1+eta_L)))).^(-beta*(1-chi))./(beta*((
    0.94875*h./(1+h)+0.61375)./ ...
    (1+rho+(0.94875*h./(1+h)+0.61375)))*(1-tau))).^(1/Pi).*h.^(-beta*(1-chi)/Pi);

HH2=@(h) ((phi*upsilon_H*tau*beta*(((1+rho+(0.94875*h./(1+h)+0.61375))./
    ((0.94875*h./(1+h)+0.61375)+(1+rho)*(1+eta_L)))).^beta).^(-(1-nu_C)*(1-mu)/
    zeta_2)).*h.^((1-zeta_1)/zeta_2);

% Define KK and HH for alternative upsilon_H

KK_alt=@(epsilon,sigma,h) (((beta*upsilon_I*tau)^chi.*(epsilon).^(-beta*(1-chi))./
    (beta*sigma*(1-tau))).^(1/Pi).*h.^(-beta*(1-chi)/Pi);
```

```
HH_alt=@(epsilon,h) ((phi*upsilon_H_alt*tau*beta*(epsilon.^beta)).^(-(1-nu_C)*(1-
        mu)/zeta_2)).*h.^((1-zeta_1)/zeta_2);


KK2_alt=@(h) (((beta*upsilon_I*tau)^chi.*(((1+rho+(0.94875*h./(1+h)+0.61375))./
    ((0.94875*h./(1+h)+0.61375)+(1+rho)*(1+eta_L)))).^(-beta*(1-chi)))./(beta*((
    0.94875*h./(1+h)+0.61375)./(1+rho+(0.94875*h./(1+h)+0.61375)))*(1-tau)))
    .^(1/Pi).*h.^(-beta*(1-chi)/Pi);

HH2_alt=@(h) ((phi*upsilon_H_alt*tau*beta*(((1+rho+(0.94875*h./(1+h)+0.61375))./
    ((0.94875*h./(1+h)+0.61375)+(1+rho)*(1+eta_L)))).^beta).^(-(1-nu_C)*(1-mu)/
    zeta_2)).*h.^((1-zeta_1)/zeta_2);

% First interval: 0 <= h^A < h_L

h1=linspace(0.0001,h_L,300);
kk1=KK(epsilon_m,sigma_m,h1);
hh1=HH(epsilon_m,h1);

% Second interval: h_L <= h^A < h_H

h2=linspace(h_L,h_H,400);
kk2=KK2(h2);
hh2=HH2(h2);

% Third interval: h^A >= h_H

h3=linspace(h_H,1.0,300);
kk3=KK(epsilon_M,sigma_M,h3);
hh3=HH(epsilon_M,h3);

% Plots

figure('Color','w'); hold on;

kk1_plot=plot(h1,kk1,'-','LineWidth',1.5);
kk2_plot=plot(h2,kk2,'-','LineWidth',1.5);
kk3_plot=plot(h3,kk3,'-','LineWidth',1.5);

hh1_plot=plot(h1,hh1,'-','LineWidth',1.5);
hh2_plot=plot(h2,hh2,'-','LineWidth',1.5);
hh3_plot=plot(h3,hh3,'-','LineWidth',1.5);

xline(0.1,'k--','$h_L^A=0.1$','LineWidth',1,'Interpreter','latex','FontSize',14);
xline(0.5,'k--','$h_H^A=0.5$','LineWidth',1,'Interpreter','latex','FontSize',14);
ylabel('$k^I$','Interpreter','latex','FontSize',18);
xlabel('$h^A$','Interpreter','latex','FontSize',18);

axis([-0.01 1 0.001 1000000000])
text(1+0.026,1000000000,'$HH$','FontSize',16,'Interpreter','latex');
text(1+0.036,4,'$KK$','FontSize',16,'Interpreter','latex');
text(1+0.026,10000000*1.8,'$\tilde{HH}$','FontSize',16,'Interpreter','latex');

% Plot alternative HH

hh1_alt=HH_alt(epsilon_m,h1);
hh1_alt_plot=plot(h1,hh1_alt,'--','LineWidth',1.5);
```

```
hh2_alt=HH2_alt(h2);
hh2_alt_plot=plot(h2,hh2_alt,'--','LineWidth',1.5);

hh3_alt=HH_alt(epsilon_M,h3);
hh3_alt_plot=plot(h3,hh3_alt,'--','LineWidth',1.5);

% Intersection

func=@(h_eq) KK2(h_eq)-HH2(h_eq);
h_star=fzero(func,0.5);
k_star=KK2(h_star);

eq=plot(h_star,k_star, 'ko','MarkerFaceColor','k','MarkerSize',8);
plot([h_star h_star],[min(ylim) k_star],'k--','LineWidth',1,'HandleVisibility','off');
plot([0 h_star],[k_star k_star],'k--','LineWidth',1,'HandleVisibility','off');
text(h_star*0.23,k_star*8, sprintf('(%0.3f,%0.2f)',h_star,k_star),'FontSize',14,
        'Interpreter','latex', 'Color','k');

% Intersection for upsilon_H_alt

func_alt=@(h_eq_alt) KK2_alt(h_eq_alt)-HH2_alt(h_eq_alt);
h_star_alt=fzero(func_alt,0.5);
k_star_alt=KK2_alt(h_star_alt);

eq_alt=plot(h_star_alt,k_star_alt,'-p','MarkerFaceColor','k','MarkerSize',14);
plot([h_star_alt h_star_alt],[min(ylim) k_star_alt],'k--','LineWidth',1,
        'HandleVisibility','off');
plot([0 h_star_alt],[k_star_alt k_star_alt],'k--','LineWidth',1,'HandleVisibility',
        'off');
text(h_star_alt*1.2,k_star_alt*1.4, sprintf('(%0.3f,%0.2f)',h_star_alt,k_star_alt),
        'FontSize',14,'Interpreter','latex', 'Color','k');

% Transform Y axis into log scale

ax=gca;
ax.YScale='log';
ax.TickLabelInterpreter='latex';

% Legend

legend([kk1_plot,hh1_plot,kk2_plot,hh2_plot,kk3_plot,hh3_plot,eq,hh1_alt_plot,
        hh2_alt_plot,hh3_alt_plot,eq_alt],{'$KK$ Region 1','$HH$ Region 1',
        '$KK$ Region 2','$HH$ Region 2','$KK$ Region 3','$HH$ Region 3',
        '$({h^A}^*,{k^I}^*)$', '$\tilde{HH}$ Region 1','$\tilde{HH}$,Region 2',
        '$\tilde{HH}$ Region 3','$(\tilde{h^A}^*,\tilde{k^I}^*)$'},'Interpreter',
        'latex','Location','best','FontSize',12);
grid off;
```