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The role of consumption and the financing of health investment under epidemic shocks*

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Abstract

We study the behavior of consumption and health investment resulting from shocks undermining health capital accumulation. We examine the effects on subsequent life cycle of long-lived shocks undermining health with either an acceleration of health capital deterioration, or a decrease in health investment efficiency. We also address the issue of the financing of health investment. We provide new evidence based on nonparametric estimations which show complex non-linear interplay between life expectancy and health expenditure. We then develop a benchmark model where consumption and health capital enter additively in the utility function, featuring independence between the returns from ordinary consumption and health. Then, we depart from this setup by assuming non-additive preferences meaning that ordinary consumption also is crucial for health. We show that a shock undermining health which increases health expenditures and weakens the income base, not only affects savings but also compromises the consumption capacity, the human and physical capital of the economy, and undercuts the process of economic development. We also show that the magnitude of the effects strongly depends on the assumed preferences.

JEL classification: E21; I12; O10
Keywords: Ordinary consumption; health investment; saving; non-parametric estimation

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1 Introduction

'... These efforts will also uncover some so far unmeasured positive return
– in the way of greater health and longevity, greater mobility,
more leisure, less income inequality and the like.'
(Kuznets, 1973)

One of the most challenging tasks policy makers are facing in both developed and developing countries is the design of an 'efficient' health care system to improve population welfare. In the US for instance, this unachieved goal is commonly acknowledged to be as high a priority as that of Administration. In Europe where health care systems have already been put to the test, inefficiency usually leads governments to regularly introduce a variety of new schemes (generalization of generic drugs, dropped from the market of inefficient drugs, obligation for people to have a family doctor, etc.) in order to reduce health spending and to improve the health care system. In the developing world, the economic consequences of diseases like AIDS, Malaria, etc. (sharp decline in life expectancy, fall in productivity, etc.) reinforced the idea of formulating a well adapted health care system without large long-run costs in terms of income per capita as recently pointed out by Acemoglu and Johnson (2007). Understanding the economic mechanisms that drive the design and implementation of such policies is a key issue. The aim of this study is to highlight some of these economic mechanisms, mainly those related to ordinary consumption, savings and the financing of health expenditure within the face of epidemic shocks.

Improvement of health is an important social and economic objective, which has obvious direct returns in the sense it favors longer and better life, but also a large indirect effect through the acceleration of economic growth.[1] Among the well known indicators of health status in a country, mortality and life expectancy are the usual candidates, each of them with their advantages and drawbacks. In fact, the interplay between economic growth and health has been and still is a source of important debate in the literature since evidently, health is a human capital. In an illuminating assessment of this question, Sen (1998) pointed out the crucial connection between mortality and growth, and highlighted the ability of countries to reduce mortality as a test of their economic performance. Sen (1998) also outlined that the forces that contribute to an increase or a reduction of mortality often have economic causes.[2] Moreover, the increase of health expenditures in both developed and developing countries calls for a debate on health policies focusing on limiting the growth of health spending as a rational response to changing economic conditions notably the growth of income per capita as life expectancy increases. The role of health expenditure in improving longevity was recently studied by Hall and Jones (2007) who showed that spending on health to improve longevity led individuals to procure additional periods of utility, and that the marginal utility of life extension does not decline.[3]

This paper contributes to the current debate by highlighting the behavior of consumption and health investment under epidemic shock with a special emphasis on the financing of health expenditure. In this set up, we consider two specifications for the preferences: i) a specification where consumption and health capital enter additively in the utility function, and ii) the case of non-additive preferences meaning that ordinary consumption is also crucial for health.

In Grossman's (1972) standard model, health is considered as a capital stock that increases with investment, by buying into health services, medical goods, or spending time on health related activities. However, it also decreases naturally through the ageing process. As pointed out by Gjerde et al. (2001), there are three key reasons why people want to improve health:

[1] See e.g., Chalkley and Malcomson (1998) for a nice contribution to the debate.

[2] See also Cervelatti and Sunde (2005, 2008) and de la Croix (2008).

[3] See the recent study of Dolan and Kahnemann (2008) on interpretations of utility in the context of non-market goods such as health. See also Bleichrodt and Pinto (2005) for some applications of non-expected utility in the health area.

i) Since health directly enters the individual's utility, it can be considered as a consumption commodity.

ii) Health determines the total amount of time available for monetary and non-monetary activities. Therefore it is an investment commodity.

iii) Being healthy lengthens the life span and lessens the likelihood of premature mortality. Thus health is a determinant factor of longevity.

Ehrlich and Chuma (1990) specified a demand function for quantity and quality of life. Their model is based on Grossman (1972)'s type specification using the consumption-investment commodity aspect of health. The authors calculated that optimal health and longevity are correlated to the endowment of wealth, rather than necessarily current income. Gjerde et al. (2001) analyzed the impact of adaptating to a falling health state on the demand for health and medical care by integrating adaptation processes in the pure consumption model of Grossman (1972). They also introduced the uncertainty of longevity and their simulation experiments showed that adaptation affects health by lowering the incentives to invest in health, as well as smoothening the optimal health stock path over life cycle.

One key feature in the traditional modeling strategy *a la* Grossman is that preferences are separable in health and ordinary consumption. As a result, the returns from these two goods are independent. However, recent evidence stressed the fact that ordinary consumption is also crucial for health. According to the *World Health Organization* 2002 report, while overweight status and obesity coexist with stunting and micronutrient malnutrition, undernutrition has long been considered to represent both a consequence and cause of poor human health, underdevelopment, and underachievement throughout life.[4] Undernutrition with respect to energy, protein, etc. can adversely affect the quality of life, impair resistance to infection and diseases, and decrease span of life.

At this stage of our motivation, let us briefly put forward some specific aspects of health which may help understanding of our approach. Epidemics and malnutrition often occur in tandem. Indeed, poor nutrition increases the risk and progression of disease and in turn, disease exacerbates malnutrition. Curtis (2004) outlined a possible relationship between nutrition and scarlet fever. The author suggested that poor nutrition during pregnancy may have caused women to give birth to children who were particularly susceptible to scarlet fever.[5] On the other hand, according to the World Initiative for Soy in Human Health (WISHH), 'HIV/AIDS-infected people may need 50-100% more protein than uninfected people.' Bobat et al. (1997) studied prenatal HIV-infected children in Italy. The authors concluded that breastfeeding not only protects infants from common childhood illnesses, but it also could slow down the progression to AIDS for HIV-infected children. However, the advantages of breastfeeding were lost by the time children reached five years of age.[6] d'Albis and Augeraud-Véron (2008) considered that the dynamics of optimal prevention depends on the relationship between the epidemiological characteristics of disease, labor productivity and intergenerational equity. They demonstrated that a minimal level of labor productivity is necessary to reduce the prevalence rate of the epidemic in the long-run. Boucekkine and Laffargue (2008) developed a general theory to study the economic and demographic impact of epidemics. The authors characterized analytically the short and medium run consequences of epidemics for population size, age pyramid, economic performance and income distribution.[7]

Many factors may contribute to disease evolution making the identification of single causes very difficult. However, the observations above suggest that improved nutrition and the prompt

[4]See e.g. the recent study of Nonnemaker et al. (2008).

[5]See e.g. Blaum (2005) for an empirical assessment of the role of pregnancy employment on health at birth.

[6]See also Tanaka (2005) for a study of the interplay between parental leave and child health.

[7]See also Boucekkine et al. (2008), Geoffard and Philipson (1996,1997), Gersovitz and Hammer (2004,2005), Kremer (1996).

treatment of infections in HIV infected individuals may delay the onset of AIDS. There is evidence from several studies that macronutrients play a role in HIV disease progression. Friis and Michaelsen (1998) focused on HIV-infected men in the USA. They suggested that high intakes of riboflavin, vitamin E and iron, and possibly vitamin A, C and thiamin were associated with reduced disease progression. Moreover, Fawzi et al. (1998) produced data from a randomized, controlled vitamin A trial among pre-school children with acute pneumonia in Tanzania. They concluded that vitamin A prolonged the life expectancy of the HIV infected, suggesting that vitamin A may play a role in slowing the course of HIV infection in children.

VanMaanen (1988) led two exploratory studies with elderly US and UK people in various age-groups. He found that the perception of health, by the self proclaimed 'healthy' elderly American was more 'a state of mind'. Whereas the 'un-healthy' elderly British person interpreted it as 'the state of absence of disease'. In each case, health maintenance behavior patterns valued were: balanced nutrition and physical exercise, etc. Subsequently, good nutrition throughout adult life will help protect against diseases such as diabetes, coronary heart problems, strokes and some cancers. For instance, the March 2003 report of the East Midlands Regional Assembly stated that, healthy eating could lead to a 20% reduction in deaths from chronic diseases, and appropriate dietary advice can prevent physical and mental deterioration, and improve the quality of life of older people.

Studies on human capital allow us to analyze the effects of nutrition on labor productivity. Fogel (1997) stated that nutritional improvements contributed to between 20 and 30% of the income per capita growth in England during 1780-1979. The functionality of the individual and his capacity to work in a productive way depended partly on her/his nutrition. In the same way, Seshadri and Tara (1989) showed that in India, iron deficiency in children affects their cognitive capacity and their performance at school. Therefore, consumption of un-healthy goods plays an important role when addressing health.

In this study, we consider a model in which marginal utility of consumption depends on health capital and vice-versa. For simplification, we consider that the lifetime of individuals is infinite, thus removing any uncertainty on that side. However this facet of our approach is closely related to the framework of Grossman (1972) and Ehrlich and Chuma (1990) in that health capital is still a determinant of lifetime utility. In this complex, we study agents' behavior facing an epidemic shock which either accelerates the depreciation of health capital or decreases the productivity or efficiency of investment in health.

At a first glance, one may observe that these two actions are interconnected but we cannot make conclusions about the net effects on health investment. Hence, it will be very difficult, at least analytically, to slice on the net effect of a high health deterioration rate and low health productivity on health investment. It is important to note that not all diseases have the same effects on individuals. For instance, we can distinguish between virulent epidemics which can kill in a very short period of time, and strongly affect an individual's health capital and life expectancy on the one hand and epidemics which do not kill in the short term, but confine people to bed, or constitute a handicap for the rest of their life on the other.[8] Therefore, to simplify and to avoid potential ambiguities in our modelling which entails the analysis of all factors, including epidemics that can undermine health capital, we assume that we have two separate and different epidemic shocks: one that accelerates the health deterioration rate, and another that decreases the efficiency of health investment.

One of the main economic implications of an epidemic is a probable and significant distortion in saving behavior. For example Freire (2002) studied the impact of AIDS on household savings in South Africa. The author found that the pandemic, and the associated sharply declin-

[8]Among those diseases we can quote Plague, Spanish Influenza, Cholera, Meningitis, Virus of Marburg (discovered in Marburg in the north of Germany in 1967). The second kind of epidemic can be assimilated to endemic diseases. The effect of these diseases is that, many people die but many people survive but are reduced to poverty because of the loss of productivity, and their lives are blighted by frequent bouts of illness. Among them we find AIDS, paludism, Leishmaniasis (the second-largest parasitic killer in the world after malaria).

ing life expectancy is likely to shift savings downward for a while, therefore restricting economic growth and standard of living in the medium and long-run. Chakraborty (2004) considered the problem of public investment in health within the framework of overlapping generation models. The author showed that in poor countries where life expectancy is weak, individuals are more likely to discount the future and thus less inclined to save. Cuddington and Hancock (1994) also stated that, health expenditure induces a decrease in savings at the expense of capital accumulation. However, this is questionable since health expenditure is detrimental to ordinary consumption. Therefore, there is an overriding issue as to how to deal with savings in the context of epidemics.[9]

In this paper, we provide new non-parametric evidence on the relation between life expectancy and health expenditure. Then, we pay particular attention to the way health enters the utility function, and to the impact of epidemics on saving decisions, investment in health capital and ordinary consumption. We shall see that the picture is quite sophisticated, depending on the preferences postulated. The chief outcome of this paper demonstrates that a shock undermining health (increasing health expenditure and weakening the income base), not only affects savings, but also compromises the consumption capacity as well as the human and physical capital of the economy undercutting the process of economic development. We show that the magnitude of these effects depends on the assumed preferences.

The remainder of the paper proceeds as follows. To support our theoretical framework, Section 2 presents empirical outlines and estimation results. We provide a brief literature review of empirical findings and then we propose a non-parametric analysis of the interplay between health expenditure and life expectancy. Section 3 studies the benchmark model with separable health and consumption. Section 4 is devoted to the alternative model with multiplicative interaction between health and ordinary consumption. In both cases, we deal with a general equilibrium framework. Section 5 summarizes our findings and Section 6 concludes the study.

2 Empirical background

In the previous section, we emphasize the importance of the connection between health investment and life expectancy. In this section, we would like to say a little bit more and elaborate on some empirical facts. Over the last few centuries, it has been recognized that improvements in health care are the main driving force of life expectancy in both developed and developing countries. How does health care spending impact life expectancy? Firstly, we provide a short literature review on some empirical facts and then, we provide an empirical non-parametric examination of the relationship between health expenditure and life expectancy, accounting for possible endogeneity issues (or feedback effects). Up till now, empirical contributions have been based on parametric specifications (see e.g, Shaw et al., 2005) which may have the drawback of skipping out non-linearities inducing severe mis-specification problems.

2.1 A brief review of some facts

There are several channels through which diseases can affect the economy. Some recent contribution have advocated the human capital channel. For example, Corrigan et al. (2005) have used a calibrated OLG model to analyze the effect of the drop in life expectancy on investment. They found a significant increase in the number of orphans as a result of AIDS. McDonald and

[9]Looking at this from a different perspective, Acharya and Balvers (2004) revisited the concept of intertemporal preferences replacing the unobservable concept of utility function by an observable health function. They showed that the rate of time preference varies in an intuitive way with changes in conditional lifetime, initial wealth age, and the marginal productivity of consumption in affecting health. Dusansky and Koç (2006) emphasized the role of uncertainty examining the interconnection between individual consumer's demand for medical care and choice of health insurance coverage (see also Cameron et al., 1988 and Dardanoni and Wagstaff, 1990). It is worthwhile to mention that those aspects of the issue are out of the scope of this study.

Roberts (2006) used an econometric model combining growth and health capital equations.[10] Applied on African countries, the model predicted the substantial effects of the epidemic: the marginal impact on income per capita of a one percent increase in HIV prevalence rate is minus 0:59%. The authors concluded that while the human and social costs of the HIV/AIDS epidemic are major causes for concern, their results indicate that the macroeconomic effects of the epidemic are by no way negligible. Thornton (2008) evaluated an experiment in which individuals in rural Malawi were randomly assigned monetary incentives to learn their HIV results after being tested.

In a highly controversial paper, Young (2005) claimed that AIDS severely lowers fertility for two main reasons: On the one hand, the epidemic has undoubtedly reduced willingness to engage in unprotected sexual activity. On the other, the high mortality of adult males and the resulting scarcity of labor are likely to increase the value a woman's availability. Both channels are arguably strong enough to induce a long-lasting decrease in fertility, which may cause future consumption per capita to rise. Using a Barro-Becker based empirical model, the author found that in the case of South-Africa, this decreasing fertility engine is so strong that it dominates the human capital channel put forward by Corrigan et al. (2005). Hence, AIDS might well be interpreted as a 'gift of the dying' for future South African generations. Nonetheless, such a finding has been challenged by some authors, including the very interesting paper of Kalemli-Ozcan (2006) who studied the fertility issues on a panel of 44 African countries between 1985-2000. The author showed that the HIV/AIDS epidemic affects the total fertility rate positively and the school enrollment rates negatively.

Several studies have elaborated on the relevance of the interdependence between health investment, life expectancy and economic growth as well as outlining the crucial role of public policies. In this respect, country historical figures are very interesting to put forward. As outlined by Jones (2001), in 1998, the U.S. spent 13.% of its GDP on goods and services related to health care which represents a big increase compared to the 5.1% spending in 1960. The 1998 spending shares were 10.6% in Germany, 9.6% in France, 9.5% in Canada, 7.4% in Japan, and 6.7% in the U.K. At the same time, the pattern of life expectancy increase is the opposite, considering that U.S. life expectancy at birth was 57.1 years in 1929, 68.2 years in 1950 and up to 75.5 years by 1990.

Shaw et al. (2005) studied life expectancy production function for a sample of OECD countries using a parametric specification. The authors found that pharmaceutical consumption has positive impact on life expectancy for middle age and older. Shaw et al. (2005) also noted that this relation is sensitive to age distribution. Peltzman (1987) used a GLS regression of life expectancy at birth on wealth and government health spending and found only health to be a significant determinant. Miller and Frech (2000) used OECD age strata data and found that the determinants of life expectancy in each stratum regression were wealth, pharmaceutical and non-pharmaceutical medical expenditures.

The literature also provides strong evidence support as to how healthiness is closely connected to economic development. Gallup and Sachs (2001) argued that wiping out malaria in sub-Saharan Africa could increase the continent's per capita growth rate by as much as 2.6% by year. In the U.S. between 1980 and 2000, the annual number of deaths fell by 16%, life expectancy increased across all age groups by an average of 5%, and the number of hospital days fell by 56%.[11] Additionally, using the value of a statistical life method[12], the Medical Technology Assessment and Policy (MEDTAP) revealed in their 2002 report that, annual health care

[10] See also Bloom and Mahal (1997) for a very careful econometric study on the effect of epidemics on economic growth.

[11] However, as noticed by Bhargava et al. (2001), life expectancy in a country is a broad measure of population health, though it needs not accurately reflect the productivity of the labor force.

[12] The value of life is the cost of reducing the (average) number of deaths by one. When deciding on the appropriate level of health care spending, a typical method is to equate the marginal cost of the health care to the marginal benefits received. In order to obtain a marginal benefit amount, some estimation of the dollar value of life is required.

expenditure per person increased by $2,254 in the U.S. between 1980 and 2000. This report also stated that for every additional $1 spent on health care, the value of health gains ranged from $2.40 to $3.00, and at least a 40% increase in life expectancy is directly attributable to additional health care. However, as mentioned by Hall and Jones (2007) how much of this increase is exactly due to increased health spending is unclear, but the large gains in life expectancy clearly represent one of the major accomplishments of the 20th century. One way to explain for this increase in share of health expenditure should be to consider health as a superior good. As people get richer, consumption rises but they devote an increasing share of resources to health care.[13]

It worthwhile to note that all the above mentioned empirical contributions are based on parametric specifications, which may have the potential drawback of mis-specification. In what follows, we provide a non-parametric appraisal of the relation between life expectancy and health expenditure.

2.2 Non-parametric analysis

The complex interdependence between health care spending and life expectancy advocates the empirical modelling of the relationship using a flexible non-parametric framework. By focusing on the effect of health expenditure, it is not our intention to deny the role of other determinants of life expectancy. We are aware that not allowing for all possible determinants such as wealth, lifestyle variables (alcohol, tobacco, etc.), GDP, age distribution, etc., may be viewed as a limitation. However, several arguments can be put forward in support of our choice.

The first and obvious one, concerns the methodology. In this respect, it is important to note that using panel methods sweeping country effects away allows us to control implicitly for any time invariant determinant. The second obvious and more important point is that, we are not concerned here with obtaining the paramount predictions for life expectancy but with the *shape* of the relationship between the latter and health expenditure. In this respect, determinants of life expectancy which are not correlated with health expenditure become irrelevant. Moreover the impact of determinants which *are* correlated with health expenditure (for example GDP, age distribution, lifestyle variables such as consumption of alcohol and tobacco) will be captured via health expenditure.[14] Depending on the question asked, this can be seen as a drawback or as an advantage. It is a drawback if we purport to determine the ceteris paribus impact of health expenditure on life expectancy – *but what list of regressors would guarantee this?* It is an advantage if we are interested in the global effect of health expenditure, including indirect effects linked with omitted variables. Finally, we truly believe that over last few centuries, improvements in health care are the main driving force of life expectancy in both developed and developing countries.[15]

[13] Newhouse (1992) has pointed out the crucial role played by the development of new technologies in the rise of health spending. In fact, the invention of new and expensive medical technologies causes health spending to rise over time.

[14] Remark: in our case, as will be described in the data section, we use health expenditure in percentage of GDP. In that case, there is a clear strong correlation between health variable and GDP which prevents the use of GDP as additional regressor. As regard to age distribution, it is obvious that the same is likely to be highly correlated with health expenditure. For instance it is well known that ageing populations usually need more health care than younger population. The same can be applied to lifestyle variables. Indeed, consumption of alcohol and tobacco are likely to increase health expenditure whereas regular physical exercises could reduce health expenditure and lengthens the life span.

[15] As outlined in Kirkwood (2008), during the nineteenth and early twentieth centuries, the increase in life expectancy was driven mainly by improvements in sanitation, housing and education, causing a steady decline in early and mid-life mortality, which was chiefly due to infections. This trend continued with the development of vaccines and then antibiotics. By the latter half of the twentieth century, there was little room for further reduction in early and mid-life mortality. The continuing increase is due almost entirely to a new phenomenon: the decline in late-life mortality as a consequence of health improvement.

2.2.1 Data

We use unbalanced panel data including both developed and developing countries spanning the period 1960-2000. Life expectancy data is extracted from the *World Development Indicators* database. Health expenditure data is collected from the *Health, Nutrition and Population* database. We use total health expenditure in percentage of GDP. Total expenditure on health is defined as the sum of expenditure on activities that – through application of medical, paramedical, and nursing knowledge and technology – have the objective of: i) promoting health and preventing disease; ii) curing illness and reducing premature mortality; iii) caring for persons affected by chronic illness who require nursing care; iv) caring for persons with health-related impairments, disability, and handicaps who require nursing care; v) assisting patients to die with dignity; vi) providing and administering public health; vii) providing and administering health programmes, health insurance and other funding arrangements. By means of this definition, general public safety measures such as technical standards monitoring and road safety are not considered as part of expenditure on health. Activities such as food and hygiene control and health research and development are considered health-related, but are not included in total health expenditure.

Insert Table 1 here

Table 1 summarizes descriptive statistics which take into account the panel structure of the sample by reporting the decomposition of the standard deviation into, between and within country magnitudes. The within patterns refer to deviation from each country's average over time, whereas the between refers to a country's average. We observe that the between standard deviation is approximately more that the double of the within counterpart.

Insert Figure 1 here

To get a precise picture of the distribution of variables, we compute the non-parametric Kernel[16] density estimates. Figure 1 shows a bi-modal distribution for both life expectancy and health expenditure. There are two quite remarkable features in these distributions. We observe a high proportion of countries who dedicate a weak part of their GDP to health care spending, and a weak proportion of countries who dedicate an important part of their GDP to health care spending. At the same time, a small proportion of countries have a weak density of life expectation, and a high proportion of countries have a high density of life expectation. The last picture clearly reflects a stylized fact of the economic and demographic transition. In what follows, we investigate the interplay between life expectancy and health expenditure.

2.2.2 Econometric specification

We use a Generalized Additive Model (hereafter GAM) for panel data.[17] The appeal of the GAM is that it has the advantage of avoiding the 'curse of dimensionality' which appears in non-parametric regressions when many explanatory variables are accounted for. Moreover, the statistical properties (optimal rate of convergence and asymptotic distribution) of the estimator is well known (see e.g., Stone, 1980). The structure of the model is given by

$$y_{it} = \sum_{j=1}^{p} m_j(\mathbf{x}_{it}^j) + \mu_i + u_{it}, \qquad i = 1, \cdots, N, \quad t = 1, \cdots, T \tag{1}$$

where y_{it} denotes the response variable (here life expectancy at birth), $\mathbf{x}_{it}^j$s are j explanatory variables for $j = 1, \cdots, p$ (here $\mathbf{x}$ denotes health expenditure), the m_j are unknown univariate

[16] We use the Epanechnikov kernel which is known to have optimal properties (see e.g., Wand and Jones, 1995).
[17] See e.g., Hastie and Tibshirani (1990) and Stone (1985) for further details on GAM.

functions to be estimated, μ_i is unobserved individual specific effects. We assume that errors u_{it} are independent and identically distributed, but no restriction is placed on the temporal variance structure. To account for possible endogeneity, that is, potential feedbacks from life expectancy we use the *predeterminedness* assumption: $\mathbb{E}(u_{it}|\mathbf{x}_{it}, \mathbf{x}_{i,t-1}, \ldots, \mathbf{x}_{i1}) = 0$, which is fairly much weaker than the *strict exogeneity* assumption: $\mathbb{E}(u_{it}|\mathbf{x}_{i1}, \ldots, \mathbf{x}_{iT}) = 0$, $i = 1, \cdots, N$, $t = 1, \ldots, T$, often made when working with panel data. Now, observe that the possibility to sweep away the unobserved effect μ_i allows us to keep unspecified the joint distribution of $\mathbf{x}_{it}$ and μ_i, and that of μ_i and u_{it}. Thus, we make no assumption on $\mathbb{E}(\mu_i|\mathbf{x}_{i\ell_1}, \cdots, \mathbf{x}_{i\ell_K})$ for any set of dates $\ell_1, \cdots, \ell_K$ in $\{1, \cdots, T\}$[18] The unobserved effect μ_i can be eliminated by first differentiation:

$$y_{it} - y_{i,t-1} = \sum_{j=1}^{p} m_j(\mathbf{x}_{it}^j) - \sum_{j=1}^{p} m_j(\mathbf{x}_{i,t-1}^j) + e_{it} \tag{2}$$

where $e_{it} = u_{it} - u_{i,t-1}$. The first difference assumption (FDA): $\mathbb{E}(\eta_{it}|\mathbf{x}_{it}^j, \mathbf{x}_{i,t-1}^j) = 0$, $i = 1, \cdots, N$, $t = 2, \cdots, T$ identifies the functions:

$$\mathbb{E}\left[y_{it} - y_{i,t-1}|\mathbf{x}_{it}^j, \mathbf{x}_{i,t-1}^j\right] = \sum_{j=1}^{p} m_j(\mathbf{x}_{it}^j) - \sum_{j=1}^{p} m_j(\mathbf{x}_{i,t-1}^j) \tag{3}$$

It should be noted that a special case under which FDA is satisfied is strict exogeneity which drives the within estimator for parametric panel models (see e.g., Wooldridge, 2002). Moreover, strict exogeneity would preclude any feedback from the current value of life expectancy on future values of health expenditure, which is not a realistic hypothesis. It is also worth noting that predeterminedness is neither necessary nor sufficient for FDA to hold. It is not sufficient since under predeterminedness alone, we have:

$$\mathbb{E}(e_{it}|\mathbf{x}_{it}, \mathbf{x}_{i,t-1}, \cdots, \mathbf{x}_{i1}) = -\mathbb{E}(u_{i,t-1}|\mathbf{x}_{it}, \mathbf{x}_{i,t-1}, \cdots, \mathbf{x}_{i1})$$

which will not be null in general.[19] This calls for an extension of the predeterminedness yielding (3): $\mathbb{E}(u_{it}|\mathbf{x}_{i,t+1}, \mathbf{x}_{it}, \mathbf{x}_{i,t-1}, \cdots, \mathbf{x}_{i1}) = 0$, $i = 1, \cdots, N$, $t = 1, \ldots, T-1$ with predeterminedness still holding for $t = T$. In our case, this only precludes feedback from the current value of life expectancy on next year's value of health expenditure, but not on later values. In other words, we allow possible feedback from current values of life expectancy on values of health expenditure starting from $t + 2$, which thus appears as a fairly weak condition compared to the strict exogeneity. In practice, we base our estimation on the 'smooth backfitting algorithm' (see e.g., Mammen et al., 1999 and Nielsen and Sperlich, 2005). We denote $\hat{m}(\mathbf{x}_{it})$ and $\hat{m}(\mathbf{x}_{i,t-1})$ the estimates of $m(\mathbf{x}_{it})$ and $m(\mathbf{x}_{i,t-1})$ respectively.

2.2.3 Estimation results

Estimation results of relation (2) based on the extended predeterminedness assumption are reported in Figure 2. A first glance of estimates gives the impression that the effect of health expenditure on life expectancy is highly non-linear. To go beyond this first impression, we test for the significance of non-linearity in the econometric specification. We use the 'gain' statistic (see, Hastie and Tibshirani, 1990 for details).[20] The 'gain' is computed as $90.74 > \chi^2(18.998) = 10.11$

[18] See Arellano and Honoré (2001, Chap. 53).

[19] Notice that this will be zero under the strict exogeneity assumption.

[20] The 'gain' is the difference in normalized deviance between the GAM and the parametric linear model. A large 'gain' indicates a lot of non-linearity, at least as regard the statistical significance. The distribution of this statistic is approximated by a chi-square χ^2 ($df = df_g - df_l$), where df_g denotes the degree of freedom of the GAM. It is computed as the trace of $2\mathbf{S} - \mathbf{S}\mathbf{S}'$ where $\mathbf{S}$ is the smoothing matrix, and df_l is the degree of freedom of the parametric linear model. Here we use the first difference linear model $y_{it} - y_{i,t-1} = \beta(\mathbf{x}_{it} - \mathbf{x}_{i,t-1}) + \varepsilon_{it} - \varepsilon_{i,t-1}$, which is then estimated by ordinary least squares. In that case, $\mathbf{S}$ turns out to be the matrix of orthogonal projection: $\mathbf{S} = \mathbf{Z}\left(\mathbf{Z}'\mathbf{Z}\right)^{-1}\mathbf{Z}'$, where $\mathbf{Z}$ denotes the matrix of regressors which does stack up elements of $\mathbf{x}_{it} - \mathbf{x}_{i,t-1}$.

at the 5% level. As a result, there is a strong evidence of non-linearity which means that our approximation is preferred to the linear specification.

Insert Figure 2 here

Our empirical specification is flexible enough to account for the complex way health expenditure impacts life expectancy. The key message which emerges from Figure 2 is that the relationship between life expectancy and health expenditure, while roughly increasing, has quite varying concavity depending on the value of health expenditure. Let us consider the two graphs $\hat{m}(\mathbf{x}_{it})$ and $\hat{m}(\mathbf{x}_{i,t-1})$ within the range of significancy (i.e., range where the confidence bands do not contained the zero line). In the case of $\hat{m}(\mathbf{x}_{it})$, we observe that for low values of health expenditure, an additional increase in the same will firstly reduce life expectancy. This reduction is followed by a strong increase of life expectancy for intermediate values of health expenditure. The curve becomes flat within the range of significancy for high values of health expenditure.

The experience of life expectancy with respect to low values of health expenditure is quite surprising. In fact we can imagine, as outlined by Sen (1998) – when comparing growth of GDP per head and life expectancy – the explanation of our result lies in a lagged relation, so that increases in health expenditure can be seen as determining the corresponding life expectancy increase in the future. Indeed, this is now clearly observed in the graph of $\hat{m}(\mathbf{x}_{i,t-1})$ where the decreasing part previously observed in the graph of $\hat{m}(\mathbf{x}_{it})$ no longer appears.

Hereafter, we study the effects on subsequent life cycle of long-lived shocks undermining health with either an acceleration of health capital deterioration, or a decrease in health investment efficiency.

3 The benchmark model

Most studies investigating the interaction between health and the ways it affects utility, use an additive structure, that is health status and ordinary consumption are additively separable in the utility function. Our first approach consists in exploring this framework. However, in contrast to the literature, we add a final good sector to better understand the life cycle aspect of the issue. In doing so, we assume that the marginal utility of consumption is independent from health status. However, this assumption is questionable since, as we stated previously, health and consumption are interrelated. Therefore we first introduce this primary approach as a benchmark. In the next section, we will relax this supposition by considering a specification that accounts for the fact that health and consumption are correlated. Health status is a broad concept, it goes beyond the mere presence or absence of disease. In our case, the term health status refers to the general health situation of the individual.[21]

3.1 The consumer's problem

We assume (like Hall and Jones, 2007) that health status and consumption at time z are additively separable in utility. Then the agent has to maximize the following lifetime utility

$$\int_z^\infty V(C(z), M(z))e^{-\rho s}ds = \int_z^\infty \left[U(C(z)) + \varphi(M(z))\right] e^{-\rho s}ds \tag{4}$$

where $V(C(z), M(z))$ is the instantaneous utility derived from consumption goods $C(z)$, and stock of health capital $M(z)$, and ρ is the time preference rate. We assume that $\varphi(M(z))$ is an strictly increasing and concave function in M, and can be considered as the amount of healthy time. The law of motion of non-human assets and health are respectively given by

$$\dot{A}(z) = r(z)A(z) + w(z) - C(z) - m(z) \tag{5}$$

$$\dot{M}(z) = \psi(m(z)) - \delta_M M(z) \tag{6}$$

[21] Indeed, the way people report their health status is directly related to their use of medical services, which include visits to doctors or dentists, hospital stays, medicinal prescription, etc.

where $r(z)$ is the interest rate. We assume that each individual supplies one unit of work per unit of time, then $w(z)$ is the wage rate, $m(z)$ is the flow of gross investment in stock of health capital $M(z)$. The health investment $m(z)$ is produced with a decreasing-returns-to-scale technology via the function $\psi(m(z))$.

Consistent with Ehrlich and Chuma (1990), we consider that the stock of health capital can be maintained or increased through purposive investments $m(z)$. However, health is submitted to a natural biological deterioration at the rate δ_M. Thus, in contrary to Ehrlich and Chuma (1990), we assume a constant rate of health depreciation. However, the greater the health that one intends to maintain in later years, the earlier one must initiate significant investments in counteracting the depreciation of health.

Individuals maximize lifetime utility (4), subject to the state variables $\dot{A}(z)$ and $\dot{M}(z)$ in equations (5) and (6) respectively. The Hamiltonian and optimality conditions of this optimal control problem are

$$\begin{aligned} J &= [U(C(z)) + \varphi(M(z))]\, e^{-\rho z} \\ &+ \lambda_A e^{-\rho z}\, [r(z)A(z) + w(z) - C(z) - m(z)] \\ &+ \lambda_M e^{-\rho z}\, [\psi(m(z)) - \delta_M M(z)] \end{aligned}$$

The first order conditions (hereafter FOC) associated to this problem are given as:

$$\frac{\partial J}{\partial C(z)} = U_C e^{-\rho z} - \lambda_A e^{-\rho z} = 0 \tag{7}$$

$$\frac{\partial J}{\partial m(z)} = \lambda_M \psi'(m(z)) e^{-\rho z} - \lambda_A e^{-\rho z} = 0 \tag{8}$$

$$\frac{\partial J}{\partial M(z)} = [\dot{\lambda}_M - \rho\lambda_M] e^{-\rho z} = \lambda_M \delta_M e^{-\rho z} - \varphi'(M(z)) e^{-\rho z} \tag{9}$$

$$\frac{\partial J}{\partial A(z)} = e^{-\rho z}[\rho\lambda_A - \dot{\lambda}_A] = r(z)\lambda_A e^{-\rho z} \Longrightarrow \dot{\lambda}_A = \lambda_A(\rho - r(z)) \tag{10}$$

The additional transversality conditions are:

$$\lim_{z\to\infty} \lambda_A(z) e^{-\rho z} A(z) = 0 \tag{11}$$

$$\lim_{z\to\infty} \lambda_M(z) e^{-\rho z} M(z) = 0 \tag{12}$$

We specify constant-relative-risk-aversion (or CRRA) functions for $U(C(z))$ and $\varphi(M(z))$, and a decreasing return in health investment for function $\psi(m(z))$ as:

$$U(C(z)) = \frac{(C(z))^{1-\sigma_1}}{1-\sigma_1} \tag{13}$$

$$\varphi(M(z)) = b\frac{(M(z))^{1-\sigma_2}}{1-\sigma_2} \tag{14}$$

$$\psi(m(z)) = \pi(m(z))^{\alpha} \tag{15}$$

with $\sigma_1 > 0$, $\sigma_2 > 0$, $b > 0$ and $0 < \alpha < 1$. $U(C(z))$ and $\varphi(M(z))$ are strictly increasing and concave respectively in $C(z)$ and $M(z)$. $\psi(m(z))$ represents the health investments function, which is concave in $m(z)$, reflecting the assumed diminishing returns in health investment. π is the productivity or efficiency of health investment. Increased health care productivity not only shifts the health production function upward, but causes each unit of health care to have a larger contribution to health as well. From Eq. (8) we can derive the marginal value of health capital relative to the marginal value of ordinary consumption, that is

$$\frac{\lambda_M}{\lambda_A} = \frac{1}{\psi'(m(z))} = \frac{1}{\pi\alpha(m(z))^{\alpha-1}} \tag{16}$$

Ehrlich and Chuma (1990) also assumed that the consumer is choosing death when his stock of capital $M(z)$ is under a certain minimal level $M_{\min}$. In our setup, we assume $M_{\min} = 0$. By doing so, we end up with a standard infinite time model, provided $M(z) \geq 0, \forall z$. This simplification will allow us to tackle much more comfortably the sophisticated optimization problem. Equations (7) and (10) yield the traditional Euler relation:

$$\begin{aligned} & \dot{C} = \frac{U_C(\rho - r)}{U'_C} \\ \Rightarrow \quad & \frac{\dot{C}}{C} = \frac{r - \rho}{\sigma_1} \end{aligned} \tag{17}$$

From (8) and (9) we obtain:

$$\begin{aligned} \frac{\dot{m}}{m} &= \frac{\delta_M + r}{1 - \alpha} - \frac{\varphi'(M)\psi'(m)}{(1 - \alpha)U_C} \\ &= \frac{\delta_M + r}{1 - \alpha} - \frac{bM^{-\sigma_2}\pi\alpha m^{\alpha - 1}}{(1 - \alpha)C^{-\sigma_1}} \end{aligned} \tag{18}$$

The negative sign on the second term of the right hand-side of (18) implies that when the value of health increases, then health investment goes upward. Indeed, $\pi\alpha m^{\alpha-1}$ is the inverse of the unit value of health which lowers the whole term when this value increases, ensuring an increase in m. Let us now introduce the firm problem.

3.2 The firm's problem

We now proceed to the producer side. We consider a representative firm with neoclassical Cobb-Douglas technology:

$$F(K, L) = Y(z) = B(z)K(z)^{\varepsilon} \left(L(z)\right)^{1-\varepsilon}, \qquad 0 < \varepsilon < 1$$

where $F(K, L)$ denotes the production function, $Y(z)$, $K(z)$ and $L(z)$ are respectively output, capital input and labor input per time unit respectively, while $B(z)$ is the technological level which growths at a constant rate. Let us denote $\hat{k} = \frac{K}{L}$ the ratio capital labor. Then the output per labor can be rewritten:

$$f(\hat{k}) = \frac{Y(z)}{L(z)} = B(z)\hat{k}(z)^{\varepsilon}$$

The maximization of the profit function under perfect competition allows to equalize the marginal cost of each factor with its marginal benefit. Therefore,

$$r(z) = \varepsilon B(z)\hat{k}(z)^{\varepsilon - 1} - \delta \tag{19}$$

$$w(z) = f(\hat{k}(z)) - \hat{k}(z)f'(\hat{k}(z)) = (1 - \varepsilon)B(z)\hat{k}(z)^{\varepsilon} \tag{20}$$

where $\delta \geq 0$ is the capital depreciation rate and w is the wage rate. Combining the demand and the supply sides, we can characterize the equilibrium of the economy as follows.

3.3 Equilibrium

In a closed economy with no public debt, aggregate financial wealth equals, by definition, the value of the capital stock. This implies that $A(z) = K(z)$ for all z. We can therefore write

$$\frac{\dot{\hat{k}}(z)}{\hat{k}(z)} = \frac{\dot{K}(z)}{K(z)} - n = \frac{F\left(K, L\right) - \delta K(z) - C(z) - m(z)}{K(z)} - n$$

and

$$\begin{aligned}\dot{\hat{k}}(z) &= f(\hat{k}(z)) - \hat{C}(z) - \hat{m}(z) - (\delta + n)\,\hat{k}(z) \\ &= B(z)\hat{k}(z)^{\varepsilon} - \hat{C}(z) - \hat{m}(z) - (\delta + n)\,\hat{k}(z) \end{aligned} \tag{21}$$

where $\hat{C}(z)$ and $\hat{m}(z)$ are respectively the consumption and health expenditure per labor, and n (which for simplicity will be assumed null) is the population growth rate. Therefore, we are able to summarize the dynamics of the economy by the following non-trivial four dimensional system:

$$\begin{cases} \frac{\dot{\hat{C}}(z)}{\hat{C}(z)} = \frac{r(z)-\rho}{\sigma_1} \\ \frac{\dot{\hat{m}}(z)}{\hat{m}(z)} = \frac{\delta_M + r(z)}{1-\alpha} - \frac{bM(z)^{-\sigma_2}\pi\alpha m(z)^{\alpha-1}}{(1-\alpha)C(z)^{-\sigma_1}} \\ \dot{\hat{M}}(z) = \pi(\hat{m}(z))^{\alpha} - \delta_M \hat{M}(z) \\ \dot{\hat{k}}(z) = B(z)\hat{k}(z)^{\varepsilon} - \hat{C}(z) - \hat{m}(z) - \delta\hat{k}(z) \end{cases} \tag{22}$$

with $\hat{k}(0)$ and $\hat{M}(0)$ given, plus the transversality conditions. The steady-state values of $\hat{C}$, $\hat{m}$, $\hat{M}$, and $\hat{k}$ are obtained by equalizing $\dot{\hat{C}}$, $\dot{\hat{m}}$, $\dot{\hat{M}}$, $\dot{\hat{k}}$ to zero. We obtain:

$$\hat{C} = B\hat{k}^{\varepsilon} - \hat{m} - \delta\hat{k} \tag{23}$$

$$\hat{m}^{\frac{\alpha}{\sigma_2}+1-\alpha} = \left(\frac{\delta_M}{\pi}\right)^{\frac{1}{\sigma_2}} \frac{b\pi\alpha(B\hat{k}^{\varepsilon} - \hat{m} - \delta\hat{k})^{\sigma_1}}{\delta_M + r} \tag{24}$$

$$\hat{M} = \frac{\pi\hat{m}^{\alpha}}{\delta_M} \tag{25}$$

$$\hat{k} = B^{\frac{1}{1-\varepsilon}} \left(\frac{\delta + \rho}{\varepsilon}\right)^{\frac{1}{\varepsilon-1}} \tag{26}$$

The following proposition characterizes the solution of the system and the positivity of $\hat{m}$ and $\hat{C}$.

Proposition 1 *There is a unique solution $\hat{m}$ to the Equation (24), with $\hat{m} < B\hat{k}^{\varepsilon} - \delta\hat{k}$.*

Proof. The proof is quite intuitive. Indeed, the left hand side of the equation (24) is strictly increasing in $\hat{m}$, while the right hand side is strictly decreasing, and the latter is equal to zero when $\hat{m} = B\hat{k}^{\varepsilon} - \delta\hat{k}$ and equal to $(\frac{\delta_M}{\pi b})^{\frac{1}{\sigma_2}} \frac{b\pi\alpha(B\hat{k}^{\varepsilon} - \delta\hat{k})^{\sigma_1}}{\delta_M + r}$ if $\hat{m} = 0$. Hence, there is a unique solution $\hat{m}$ to equation (24), with $\hat{m} < B\hat{k}^{\varepsilon} - \delta\hat{k}$ which therefore means that $\hat{m}$ and $\hat{C}$ are always positive. □

3.4 Static comparative

In this section we study the static comparative of the model. We consider the effect of modifications in the parameters of the endogenous variables along the balanced growth path. We will consider two cases: the case where B is independent of the determinants of health capital. Then we examine an ad-hoc case where B is dependent on such determinants.

3.4.1 B independent of health capital

The following proposition characterizes the behavior of health investment and consumption with respect to health depreciation rate.

Proposition 2 *Health investment (resp. consumption) is a strictly increasing (resp. decreasing) function of the health depreciation rate δ_M if and only if $\sigma_2 < 1 + \frac{\rho}{\delta_M}$. In contrast, savings are unaltered by changes in δ_M.*

Proof. See appendix. □

Two comments are in order with respect to the above result. Firstly, the lower σ_2, the less marginal utility with respect to health capital (and thus to health investment) will drop if $\hat{M}$ or $\hat{m}$ is raised in response to the deterioration of health depreciation. Henceforth, low values of σ_2 are compatible with increasing health investments. In contrast, large values of σ_2 are likely to make the marginal welfare cost of such investment prohibitive.

Secondly, the larger δ_M, the lower the incentives to invest in health, compared to investment in physical capital (which depreciation is kept constant). This explains why the condition for increasing health investment becomes more and more stringent when δ_M increases indefinitely. Indeed, as δ_M rises, the range of the values of σ_2 needed for increasing health investment becomes smaller. It tends to $]0;1]$ when δ_M tends to infinity. Now notice that the marginal rate of investment in physical capital, that's $B\varepsilon\hat{k}^{\varepsilon-1} - \delta$, is unaffected by changes in δ_M. This is due to the fact that when epidemics do not affect total factor productivity (B) in our model, then gross investment $\hat{i}$ is equal to $\delta\hat{k}$ which is independent of δ_M. Therefore, in the steady state, savings do not get modified by changes in δ_M. It follows that when $\sigma_2 < 1 + \frac{\rho}{\delta_M}$, the rise in health investment in response to the epidemic shock is entirely paid by a decrease in consumption, which goes in odds with Cuddington and Hancok (1994)'s working assumption.

Proposition 3 *: When health productivity π decreases, an increase in health investment occurs if and only if $\sigma_2 < 1$, while consumption decreases and savings remain unchanged. If $\sigma_2 = 1$, there is a neutral effect on the economic variables, and if $\sigma_2 > 1$ health expenditure decreases.*

Proof. See appendix. □

A few comments are in order here regarding the interpretation of the model. A decrease in π has two effects on health investment $\hat{m}$. On one hand, it decreases the efficiency of health investment as featured in equation (25), therefore causing $\hat{m}$ to drop. On the other hand, a lower π increases the marginal value of health capital relative to the marginal value of the consumption good as one can notice in Equation (16), inducing an incentive to invest more in health capital. However, since a marginal drop in $\hat{m}$ is valued by the utility term $M^{-\sigma_2}$, the negative effect will dominate the positive one for σ_2 high enough, here $\sigma_2 > 1$. If $\sigma_2 = 1$, there is just a compensation between the two effects which yields a neutral impact. For low values of σ_2 allowing investment in health, the latter is always financed by a reduction in ordinary consumption, while savings remain constant. An important finding from this model is the undeniable fact that face with an epidemic shock, whether people reduce or increase their health expenditure, their saving remains constant, and an increase in health investment is moderately financed by a reduction of their consumption.

3.4.2 The case of productivity-decreasing epidemics

We now suppose that the productivity B depends on the health productivity π and the health depreciation rate δ_M in an ad-hoc way. We will assume (like Cuddington and Hancok, 1994) that a decreasing health capital induces lower productivity. More precisely, we will assume that all factors pushing down this capital has a negative effect on B. Therefore, we make the hypothesis that the health productivity π positively affects B, while the latter is negatively affected by δ_M. The following result holds.

Proposition 4 *Health investment (resp. consumption) increases (resp. decreases) in response to the epidemic shock δ_M if and only if*

$$\sigma_2 < \frac{1}{1 - B_{\delta_M}\sigma_1\hat{k}^{\varepsilon}(\delta_M + r)(B\hat{k}^{\varepsilon} - \hat{m} - \delta\hat{k})^{-1}} + \frac{\rho}{\delta_M - \delta_M B_{\delta_M}\sigma_1\hat{k}^{\varepsilon}(\delta_M + r)(B\hat{k}^{\varepsilon} - \hat{m} - \delta\hat{k})^{-1}}$$

while savings always decrease.

Proof. See appendix. □

This result is in line with the Proposition (2), however, it is worse in the case of health investment. Indeed, if δ_M increases infinitely, instead of $\sigma_2 < 1 + \frac{\rho}{\delta_M}$, we now need σ_2 in $]0; x[$ with $x = \frac{1}{1-B_{\delta_M}\sigma_1\hat{k}^{\varepsilon}(\delta_M+r)(B\hat{k}^{\varepsilon}-\hat{m}-\delta\hat{k})^{-1}} < 1$ to allow a rise in $\hat{m}$, where $B_{\delta_M} = \frac{\partial B}{\partial \delta_M} < 0$. It happens that the interval of evolution of σ_2 which would favor $\hat{m}$ is reduced by the additional resource constraint through the productivity B. On the ordinary consumption side, when it occurs, the increase in health expenditure is totally compensated by a fall in consumption (see proof in the appendix). However, on the other side we observe a fall in the saving level, since the capital stock is negatively affected through the negative effect of δ_M on the productivity B. Compared to the case where the productivity B is not affected by the health parameters, at this point, to finance an increase in health expenditure, in addition to the fall in consumption, savings drop because of the loss of resources generated by the loss of productivity. Furthermore, if the epidemic has a large effect (i.e. $B_{\delta_M} \to -\infty$), health investment will never increase (the necessary and sufficient condition of proposition (4) tends to $\sigma_2 \leq 0$).

Proposition 5 *A downshift of the health productivity π is followed by an increase in health investment if and only if $\sigma_2 < \frac{1}{1+B_{\pi}\sigma_1\hat{k}^{\varepsilon}\pi(B\hat{k}^{\varepsilon}-\hat{m}-\delta\hat{k})^{-1}}$, meanwhile consumption and savings decrease.*

Proof. See appendix. □

Let us comment on the meaning of this result. The interval in which the values of σ_2 are favorable to an increase in health expenditure is now reduced. Indeed we now need $\sigma_2 < \frac{1}{1+B_{\pi}\sigma_1\hat{k}^{\varepsilon}\pi(B\hat{k}^{\varepsilon}-\hat{m}-\delta\hat{k})^{-1}}$ instead of $\sigma_2 < 1$ to increase $\hat{m}$, where $B_{\pi} = \frac{\partial B}{\partial \pi} > 0$. In other words, from now on, even for σ_2 in $\left]\frac{1}{1+B_{\pi}\sigma_1\hat{k}^{\varepsilon}\pi(B\hat{k}^{\varepsilon}-\hat{m}-\delta\hat{k})^{-1}}; 1\right]$ a fall in π is followed by a fall in $\hat{m}$. This phenomenon is certainly due to the fact that the agent has also a resource problem. Indeed a fall of π, has a negative effect on the wages through the productivity B, thus reducing his financial possibilities. In addition, given that there is also a negative impact on the gross investment in physical capital, we can expect a reduction of saving. As in Proposition (4), a drop in consumption and savings will make it possible to pay the increase in health expenditure.

To conclude this section, it is worth noting that while some studies like Cuddington and Hancok (1994) showed that an increase in health expenditure is inevitably accompanied by a decrease in savings, our study demonstrated that health expenditure can be fully financed by a decrease in saving and consumption, or by a reduction of consumption while saving remains unaltered. However, in this benchmark model, the determination of the relationship between health investment (and therefore consumption and savings) and the health parameters δ_M and π entirely depends on the health elasticity σ_2. This constraint may be due to the fact that health and ordinary consumption enter utility in an additive way. Therefore, marginal utility of consumption is independent of health capital and vice-versa. We develop hereafter an alternative model where consumption is also a determinant factor of good health, as largely previously explained.

4 An alternative model: The case of non-additive utility

We move from the above pattern, where consumption and health enter into the utility function in an additive way. Remember that in the later case the marginal utility of consumption is independent from health, and this does not fit in for instance with the notion that good nutrition is also important for health. Indeed, undernutrition can affect quality of life, weaken resistance to infection and diseases, and cause life expectancy to drop. Epidemics and malnutrition often operate together. Indeed, poor nutrition increases the risk and progression of disease and in

turn, disease exacerbates malnutrition. Evidence from Friis and Michaelsen (1998) suggested that good nutrition and prompt treatment of infections in HIV infected individuals may delay the onset of AIDS and reduce the progression of the disease. Also, healthy eating might lead to a reduction in mortality from chronic diseases, and appropriate dietary advice can prevent physical and mental deterioration, and improve the quality of life. Therefore, ordinary consumption is also crucial for health. The alternative model we propose below seeks to account for this important aspect.

4.1 Model set-up

The individual's lifetime utility is now specified as follows:

$$\int_z^\infty V(C(z), M(z))e^{-\rho s}ds = \int_z^\infty U(C(z))\varphi(M(z))e^{-\rho s}ds \tag{27}$$

In order for the function $V(\cdot)$ to fulfil the standard property of positive marginal utility, we have to assume $\sigma_1 < 1$, $\sigma_2 < 1$. We disregard the case $\sigma_1 = \sigma_2 = 1$ because it imposes further constraints on the values of economic variables. Indeed, when $\sigma_1 = 1$, then $V_M = \ln(C)\varphi'(M)$, which requires $C > 1$. Therefore, we focus on the case $\sigma_1 < 1$, $\sigma_2 < 1$, which allows us to compare results with the analogue benchmark model.

Individuals seek to maximize lifetime utility (27), which depends on a consumption stream $C(z)$ subject to the state variables $\dot{A}(z)$ and $\dot{M}(z)$ in Equations (5) and (6) respectively. The hamiltonian of this problem is given by:

$$\begin{aligned} J &= U(C(z))\varphi(M(z))e^{-\rho z} \\ &+ \lambda_A e^{-\rho z}\left[r(z)A(z) + w(z) - C(z) - m(z)\right] \\ &+ \lambda_M e^{-\rho z}\left[\psi(m(z)) - \delta_M M(z)\right] \end{aligned}$$

The optimality conditions (FOC) are:

$$\frac{\partial J}{\partial C(z)} = U_C\varphi(M(z))e^{-\rho z} - \lambda_A e^{-\rho z} = 0 \tag{28}$$

$$\frac{\partial J}{\partial m(z)} = \lambda_M\psi'(m(z))e^{-\rho z} - \lambda_A e^{-\rho z} = 0 \tag{29}$$

$$\frac{\partial J}{\partial M(z)} = [\dot{\lambda}_M - \rho\lambda_M]e^{-\rho z} = \lambda_M\delta_M e^{-\rho z} - U(C(z))\varphi'(M(z))e^{-\rho z} \tag{30}$$

$$\frac{\partial J}{\partial A(z)} = e^{-\rho z}[\rho\lambda_A - \dot{\lambda}_A] = r(z)\lambda_A e^{-\rho z} \Longrightarrow \dot{\lambda}_A = \lambda_A(\rho - r(z)) \tag{31}$$

with the transversality conditions:

$$\lim_{z\to\infty} \lambda_A(z)e^{-\rho z}A(z) = 0 \tag{32}$$

$$\lim_{z\to\infty} \lambda_M(z)e^{-\rho z}M(z) = 0 \tag{33}$$

We use the same functional forms as in the benchmark set-up for $U(C(z))$, $\varphi(M(z))$ and $\psi(m(z))$. However, for $\varphi(M(z))$, we get rid of the parameter b which is unimportant in what follows. Equation (29) allows to find the shadow price $\frac{\lambda_M}{\lambda_A} = g(t) = \frac{1}{\psi'(m(z))}$ which is the unit value of health capital. Moreover, a continuous stock equilibrium condition for health can be derived from Equation (30):

$$\frac{\dot{\lambda}_M}{\lambda_A} = \frac{\lambda_M}{\lambda_A}(\rho + \delta_m) - \frac{U(C(z))\varphi'(M(z))}{\lambda_A} \tag{34}$$

Combining Equations (31) and (34), we obtain:

$$g\left(\delta_m + r - \tilde{g}\right) = \frac{1}{\lambda_A(0)} U(C(z))\varphi'(M(z))e^{\int_0^z (r(s)-\rho)ds} \tag{35}$$

with $\tilde{g} = \frac{\dot{g}}{g}$ and $g = \frac{1}{\pi\alpha}(m(z))^{1-\alpha}$. Equation (35) states that the instantaneous user cost of health capital should equal the instantaneous marginal benefit. The explicit expression of the shadow price can be derived from Equation (35) as

$$g = \bar{g}e^{\int_z^\infty (\delta_m + r(s))ds} + \frac{e^{\int_z^\infty (\delta_m + r(s))ds}}{\lambda_A(0)} \int_z^\infty \left[U(C(v))\varphi'(M(v))e^{\int_0^v (r(s)-\rho)ds}\right] e^{-\int_v^\infty (\delta_m + r(s))ds} dv \tag{36}$$

where $\bar{g}$ is an integration constant, reflecting the limit value of g when z tends to infinity (assuming that such a limit value is finite). Equation (36) means that the value of health capital is determined by the asymptotic value of life extension (the first term of the right-hand side), and the value of healthy life or the discounted value of health benefits (second term).

We can now explore the connection between the path of health investment and the optimal consumption over life cycle. Using Equation (28), we obtain $\lambda_A = U_C\varphi(M(z))$ and

$$\frac{\dot{\lambda}_A}{\lambda_A} = \rho - r(z) = \frac{\dot{C}(z)U'_C}{U_C} + \frac{\dot{M}(z)\varphi'(M(z))}{\varphi(M(z))}$$

Then the path of the optimal consumption is:

$$\dot{C}(z) = -\frac{U_C}{U'_C}(r(z) - \rho) - \frac{U_C}{U'_C}\frac{\varphi'(M(z))}{\varphi(M(z))}\dot{M}(z) \tag{37}$$

The first term on the right-hand side is the slope of the consumption path times the difference between the rate of interest and time preference. The second term reflects the interaction between the individual's health and his capacity to consume. Next we replace U_C, U'_C and $\varphi'(M(z))$ by their respective expressions in (37) for the elasticity parameters to show up:

$$\begin{aligned} \frac{\dot{C}(z)}{C(z)} &= \frac{1}{\sigma_1}(r - \rho) + \frac{1 - \sigma_2}{\sigma_1}\frac{\dot{M}(z)}{M(z)} \\ &= \frac{1}{\sigma_1}(r - \rho) + \frac{1 - \sigma_2}{\sigma_1}\left(\frac{\psi(m)}{M(z)} - \delta_M\right) \end{aligned} \tag{38}$$

In the typical Ramsey setup, we have the following optimal rule:[22]

$$r = \rho - \frac{U'_C C}{U_C}\frac{\dot{C}}{C}$$

This means that the interest rate should cover the impatience rate (or rate of time preference) and the marginal utility loss as captured by the term $-\frac{U'_C C}{U_C}\frac{\dot{C}}{C}$, where $\frac{U'_C C}{U_C}$ is the elasticity of marginal utility with respect to consumption. Equation (37) can be rewritten as:

$$r = \rho - \frac{U'_C C}{U_C}\frac{\dot{C}}{C} - \frac{\varphi'(M(z))}{\varphi(M(z))}M(z)\frac{\dot{M}(z)}{M(z)}$$

In this model, the interest rate should also account for the possible loss or gain in health capital due to any marginal change in consumption and savings. This reflects the dependence between

[22]The Keynes-Ramsey standard term known as the difference between the interest rate and the pure rate of time preference.

the (marginal) welfare impacts of consumption and health investment. We shall now extract the optimal law of motion for health investment m. Using Equation (29), we obtain:

$$\begin{aligned}\frac{\dot{\lambda}_M}{\lambda_M} &= \frac{\dot{\lambda}_A}{\lambda_A} - \frac{\dot{m}\psi''(m)}{\psi'(m)} \\ &= \rho - r(z) + (1-\alpha)\frac{\dot{m}(z)}{m(z)} \end{aligned} \tag{39}$$

From equation (30) we get

$$\begin{aligned}\frac{\dot{\lambda}_M}{\lambda_M} &= -\frac{U(C(z))\varphi'(M)\psi'(m)}{\lambda_A} + \delta_M + \rho \\ &= -\frac{U(C(z))\varphi'(M)\psi'(m)}{U_C\varphi(M)} + \delta_M + \rho \end{aligned} \tag{40}$$

Combining these two equations yields the path of the optimal health investment:

$$\frac{\dot{m}(z)}{m(z)} = \frac{r+\delta_M}{1-\alpha} - \frac{U(C(z))}{\varphi(M)}\frac{\varphi'(M)\psi'(m)}{(1-\alpha)U_C} \tag{41}$$

$$= -\frac{1-\sigma_2}{1-\sigma_1}\frac{\pi\alpha m^{\alpha-1}}{1-\alpha}\frac{C(z)}{M(z)} + \frac{r+\delta_M}{1-\alpha} \tag{42}$$

The next proposition states required concavity conditions for $U(C)$ and $\varphi(M)$.

Proposition 6 *The Mangassarian sufficient conditions for our optimal control problem are fulfilled if and only if* $\frac{\sigma_1\sigma_2}{(1-\sigma_1)(1-\sigma_2)} - 1 > 0$.

Proof. See appendix. □

4.2 Equilibrium

The dynamics of the economy is driven at equilibrium by the following system

$$\begin{cases} \frac{\dot{\hat{C}}(z)}{\hat{C}(z)} = \frac{1}{\sigma_1}(\varepsilon B\hat{k}_z^{\varepsilon-1} - \delta - \rho) + \frac{1-\sigma_2}{\sigma_1}\frac{\dot{\hat{M}}(z)}{\hat{M}(z)} \\ \frac{\dot{\hat{m}}(z)}{\hat{m}(z)} = -\frac{1-\sigma_2}{1-\sigma_1}\frac{\pi\alpha\hat{m}^{\alpha-1}}{1-\alpha}\frac{\hat{C}(z)}{\hat{M}(z)} + \frac{1}{1-\alpha}(r+\delta_M) \\ \dot{\hat{M}}(z) = \pi(\hat{m}(z))^\alpha - \delta_M\hat{M}(z) \\ \dot{\hat{k}}_z = B\hat{k}_z^{\varepsilon} - \hat{C} - \hat{m} - \delta\hat{k}_z \end{cases} \tag{43}$$

with $\hat{k}(0)$ and $\hat{M}(0)$ given, plus the transversality conditions. The steady-state values of $\hat{C}$, $\hat{m}$, $\hat{M}$, and $\hat{k}$ are obtained by equalizing $\dot{\hat{C}}$, $\dot{\hat{m}}$, $\dot{\hat{M}}$, $\dot{\hat{k}}$ to zero. We therefore get:

$$\hat{C} = \frac{(\delta_M+r)(1-\sigma_1)B^{\frac{1}{1-\varepsilon}}\left[\left(\frac{\delta+\rho}{\varepsilon}\right)^{\frac{\varepsilon}{\varepsilon-1}} - \delta\left(\frac{\delta+\rho}{\varepsilon}\right)^{\frac{1}{\varepsilon-1}}\right]}{\alpha\delta_M(1-\sigma_2)+(1-\sigma_1)(\delta_M+r)} \tag{44}$$

$$\hat{m} = \frac{\alpha\delta_M(1-\sigma_2)B^{\frac{1}{1-\varepsilon}}\left[\left(\frac{\delta+\rho}{\varepsilon}\right)^{\frac{\varepsilon}{\varepsilon-1}} - \delta\left(\frac{\delta+\rho}{\varepsilon}\right)^{\frac{1}{\varepsilon-1}}\right]}{\alpha\delta_M(1-\sigma_2)+(1-\sigma_1)(\delta_M+r)} \tag{45}$$

$$\hat{M} = \frac{\pi\hat{m}^\alpha}{\delta_M} \tag{46}$$

$$\hat{k} = B^{\frac{1}{1-\varepsilon}}\left(\frac{\delta+\rho}{\varepsilon}\right)^{\frac{1}{\varepsilon-1}} \tag{47}$$

Within the structure of the system, studying the stability properties analytically is simply unbearable. Instead, we resort to numerical simulations with a set of reasonable parameterizations and the necessary corroborating sensitivity tests. We always end up with four eigenvalues with two real negative parts and two real positive parts, suggesting that saddle path properties are fulfilled.[23]

4.3 Comparative statics

We now elaborate on how the health parameters (δ_M, π) affect the steady-state values, and notably the health investment variable ($\hat{m}$), and the consequences on consumption, capital stock and savings.

Proposition 7 *Given condition of Proposition (6), a high health deterioration rate of health causes health expenditure to increase, and the latter is fully financed by a fall in consumption, and saving remains unaffected.*

Proof. See appendix. □

Let us reiterate that with the benchmark model we get the same reaction only for σ_2 low enough, and more precisely when δ_M is high enough. On the contrary, in this case individuals react in face of an epidemic shock by spending more on health, independently of the size of σ_2, provided that the concavity requirements stated in proposition (6) are fulfilled. Without doubt, under the conditions of proposition (6) we have a positive effect of δ_M on $\hat{m}$. This positive effect can be explained by the fact that in the alternative framework, the marginal utility of consumption depends on health capital. Indeed, in order to enjoy consumption, one should be healthy, and a deterioration of health now negatively affects the marginal utility drawn from ordinary consumption. Therefore, one should invest more in health to maintain welfare optimal. This increase is paid by a fall in consumption, and since the individual's resources remain unchanged, if not improved, then there is no impact on gross investment in physical capital and savings remain constant.

Proposition 8 *Given Proposition (6), the effect of a decrease in the health productivity π on health investment, consumption and savings is neutral.*

Proof. The proof is intuitive. Indeed, a decrease in π has two effects as in the benchmark model: first, a fall in π decreases the marginal efficiency of health investment, therefore pushing this variable downward. But on the other hand, decreasing π also increases the marginal value of health capital relative to the marginal value of consumption, making it more desirable to acquire. However, contrarily to the benchmark model where for high values of σ_2 the negative effect dominates the positive one, in this alternative model, the second effect just offsets the first one. These two effects can be clearly identified in Equation (46) as well as the expression of the unit value of health capital $g = \frac{1}{\psi'(\hat{m})} = \frac{1}{\alpha\pi\hat{m}^{\alpha-1}}$. The rational of this neutral effect is that, here a decrease in health expenditure in response to a lower π (as it is the case in the benchmark model) is more harmful, since in turn we will have a negative impact on the marginal utility of consumption.

The conclusions emerging from the alternative model are different from those resulting from the benchmark model. The foremost and common result from the two models is that rising health expenditure does not always lead to a decrease in savings. However, while the benchmark model results completely rely on the health elasticity σ_2 as for the evolution of the health investment $\hat{m}$ in response to an epidemic shock, in the alternative model, an increase in δ_M induces purely a growth in health expenditure. Moreover, we have a neutral effect when π decreases, without any elasticity constraint.

[23] To simplify presentation, we do not report simulation results. Results are available from the authors upon request.

4.4 The case of productivity-decreasing epidemics

As in the benchmark model, we take into account the epidemic shock in the household's choices, via the channel of the productivity. We then assume that the level of technology $B(z)$ or the productivity of individuals depends on the health parameters. We suppose a function $B(\delta_M, \pi)$ which depends on δ_M and π. Then B is an increasing function in the health productivity π, and decreases with the health deterioration rate δ_M. Then, the following result can be highlighted

Proposition 9 *The effect of health deterioration rate on health investment is positive if and only if* $\sigma_2 < 1 + \frac{(1-\sigma_1)\left[\delta_M B_{\delta_M}(\delta_M+\rho)+\rho(1-\varepsilon)\right]}{\alpha\delta_M^2 B_{\delta_M}}$*, and consumption and savings always decrease.*

Proof. See appendix. □

In contrast to the case where the productivity B is independent of δ_M, here the optimality of increasing health expenditure in the face of epidemics does depend on the values of σ_1 and σ_2. Clearly, the negative effect on productivity has an adverse effect on income, which in turn induces a decrease in ordinary consumption and health investment. Such effects can dominate those pointed out in the previous subsection where B is independent of health parameters. This can be clearly deduced from the necessary and sufficient conditions in Proposition (9). In case of epidemics with a tenus productivity effect, that is when B_{δ_M} is close to zero, the limit condition becomes $\sigma_2 < \infty$, that is $\hat{m}$ will increase whatever elasticities' values, provided that the Mangassarian condition is checked. In contrast, if the epidemic has strong adverse effects on productivity, say $B_{\delta_M} \to -\infty$, then the condition of Proposition (9) violates the Mangassarian restriction in Proposition (6). Indeed, the limit condition becomes $\sigma_2 < \frac{(1-\sigma_1)(\delta_M+\rho)}{\alpha\delta_M}$. Therefore $\sigma_2 < 1$ if and only if $\frac{(1-\sigma_1)(\delta_M+\rho)}{\alpha\delta_M} < 0$, which in turn implies that $\sigma_1 > 1$. Although the Mangassarian condition is a sufficient condition, the latter results mean that the range of elasticities' values yielding an increase in health expenditure in the face of epidemic will shrink as the adverse productivity effect becomes tougher. On the physical capital side, due to the negative effect on B, we have a fall in gross investment which causes savings to decrease. In this case, the role of public health expenditure becomes crucial.

Proposition 10 *In the presence of a decrease in* π*, health expenditure increases if and only if* $\sigma_2 > 1$*. Thus health expenditure should drop when* π *decreases in the alternative model, since* $\sigma_2 < 1$ *is imposed for marginal utility of* M *and* C *to be positive.*

Proof. See appendix. □

According to Proposition (6), Proposition (10) completely violates the concavity condition. Therefore, we cannot expect an increase in health expenditure when the health productivity π decreases. Indeed, the negative effect on the productivity undermines individuals resources, forcing them to undercut their health investment, consumption and savings. Here, the negative effect from the marginal efficiency of health dominates the positive one induced by the increase in the unit value of health. Indeed, as featured in Equation (41), marginal utilities with respect to M and C depend on the utility levels of C and M in this order (due to the non-separable preferences). Therefore the negative effect of decreasing efficiency in the production of health capital is magnified.

5 Summary of findings and comparison with prior studies

In this section, we summarize the key findings that emerge from our study. From an empirical point of view, the estimation of the relation between life expectancy and health expenditure shows a highly non-linear curvature. These findings cannot be obtained within the parametric

specifications considered by Peltzman (1987), Miller and Frich (2000) and Shaw et al. (2005). Our econometric specification allows us to estimate both the effects of current and lagged values of health expenditure on life expectancy. While roughly increasing, the relation has quite varying concavity depending on the value of health expenditure. It is clear that the effect of health expenditure on life expectancy is less pronounced when the former is already high. Observe that this finding is consistent with Shaw et al. (2005) who showed that the actual predicted effect of pharmaceutical expenditure on life expectancy is decreasing in age. On the contrary, we find that this effect is strong for intermediate values of health expenditure.

From the theoretical side, our findings can be gathered in two main strands.

i) *When productivity is independent of health parameters*

In the case of an acceleration of the health deterioration rate (δ_M) (resulting for instance from epidemic shock), in the benchmark model, the reaction of health investment entirely relies on the elasticity of the marginal utility of health σ_2. Low values of σ_2 (or when δ_M high enough) are compatible with increasing health expenditure, while high values of σ_2 are likely to make the marginal welfare cost of such investment prohibitive. Indeed, the lower σ_2, the less marginal utility with respect to health capital will also drop. In the alternative model, individuals react to such shock by increasing their health expenditure, independently of the size of σ_2. This behaviour can be interpreted by the fact that marginal utility of consumption now depends on health capital.

When we consider a loss of health productivity, we have two effects. Firstly, a decrease in health productivity (π) induces a loss in health investment efficiency, which causes health expenditure to drop. That's what we call the negative effect. Secondly, a weak value of π increases the marginal value of health capital relative to the marginal value of consumption goods, which in turn induces an incentive to invest in health. This is the positive effect. In the benchmark model, the positive effect dominates the negative one for σ_2 low enough, while in the alternative model, one of the two effects compensates the other, and the resulting impact is neutral.

However, in both models, regardless of the two kind of shocks, a relevant result is contrary to that of Cuddington and Hancok (1994). They predicted a decrease in savings while health expenditure increases. In our framework, irrespective of the increase in health investment, savings remain unchanged. And in case it increases, the latter is fully financed by a cut in consumption.

ii) *When productivity depends on health parameters*

In the benchmark model, a high rate of health deterioration induces an increase in health expenditure only for very low values of σ_2. In that case, if the epidemics have large negative effects on productivity, health investment does not increase. However, in the alternative model, findings depend on whether epidemics have tenuous or large effects on productivity. In the former case, health expenditure will increase whatever the elasticities' values. In the case of epidemics strongly affecting productivity, the range of elasticities' values yielding an increase in health expenditure in face of an epidemic will shrink as the adverse productivity effect becomes tougher.

With a loss of health productivity (a decrease in π), while in the benchmark model we obtain an increase in health expenditure for σ_2 low enough, this expenditure should drop in the alternative model (with multiplicative interaction between health and consumption). Indeed, in the latter model, marginal utilities with respect to health capital and consumption depend on the utility levels of consumption and health. Therefore, the negative effect of decreasing efficiency in the production of health capital is magnified.

Finally, when productivity depends on health parameters, an increase in health expenditure is found to be accompanied with a loss in both consumption and savings, due to the loss of productivity.

As evident from above, the assumption of Cuddington and Hancok (1994) is certainly not fulfilled. An epidemic shock which stimulates increasing health expenditure and weakens the income base, not only affects savings, but it also compromises the consumption capacity, as well as the human and physical capital of the economy, and also undercuts the process of economic development. In such cases, the role of public health expenditure becomes crucial in increasing the average health level of individuals, which depends on the quality of the health sector, which in turn depends on the amount of public resources devoted to this sector. This finding can be linked in some sense to study of Hazan and Zoabi (2006) who incorporated health into a model extending the basic Ben-Porath mechanism. The authors considered health and education as integrated inputs in the production of human capital. The showed that increased health can not only increase the return on quality but also the return on quantity so that the transition from stagnation to growth becomes possible.

6 Conclusion

This paper addresses the issue of health, in interaction with problems in ordinary consumption and physical capital investment caused by shocks undermining health capital accumulation. We examine, in particular, the effects on the subsequent life cycle of long-lived epidemics with either an acceleration of health capital deterioration, or a decrease in health investment efficiency. We also address the question of the financing of health investment, that is, is an increase in health expenditure always paid by a decrease in savings?

We thus develop a benchmark model in which we suppose, as usual, that consumption and health capital enter utility in an additive way, namely the marginal utility of consumption is independent of health and vice-versa. We also study an alternative model, where we consider that ordinary consumption is also crucial for health. Indeed, undernutrition can adversely affect quality of life, impair resistance to infection and diseases, and decrease the span of life. We show that a shock undermining health which increases health expenditure and weakens the income base, not only affects savings but also compromises the consumption capacity, the human and physical capital of the economy, and undercuts the process of economic development. We also show that the magnitude of these effects strongly depends on the assumed preferences.

Several challenges remain to be addressed, among which, i) the optimal choices of public health policies, holding account the age structure of population, ii) the effects of a decrease in mortality benefiting young people and/or old people, iii) the distortions entailed by private health expenditure on savings, education and the productivity of agents in poor countries.

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Appendix

Proof of proposition 2
With Equation (24) can be rewritten in terms of output as:

$$f = \hat{m}^{\frac{\alpha}{\sigma_2}+1-\alpha} - \left(\frac{\delta_M}{\pi}\right)^{\frac{1}{\sigma_2}} \frac{b\pi\alpha(B\hat{k}^{\varepsilon} - \hat{m} - \delta\hat{k})^{\sigma_1}}{\delta_M + r} = 0$$

Relying on the the implicit functions Theorem we obtain:

$$\begin{aligned}
\frac{\partial \hat{m}}{\partial \delta_M} &= -\frac{\frac{\partial f}{\partial \delta_M}}{\frac{\partial f}{\partial \hat{m}}} \\
&= \frac{\left(\frac{\delta_M}{\pi}\right)^{\frac{1}{\sigma_2}} b\pi\alpha(B\hat{k}^{\varepsilon} - \hat{m} - \delta\hat{k})^{\sigma_1} \left(\frac{\delta_M + r}{\sigma_2 \delta_M} - 1\right)}{(\delta_M + r)\left[(\frac{\alpha}{\sigma_2} + 1 - \alpha)m^{\frac{\alpha}{\sigma_2}-\alpha}(\delta_M + r) + \sigma_1 \left(\frac{\delta_M}{\pi}\right)^{\frac{1}{\sigma_2}} b\pi\alpha(B\hat{k}^{\varepsilon} - \hat{m} - \delta\hat{k})^{\sigma_1 - 1}\right]}
\end{aligned}$$

Now, observe that $\frac{\partial \hat{m}}{\partial \delta_M} > 0$ if $\frac{\delta_M + r}{\sigma_2 \delta_M} - 1 > 0$, which means that $\sigma_2 < 1 + \frac{\rho}{\delta_M}$. Proceeding in the same way with respect to $\hat{C}$, we have:

$$\begin{aligned}
\frac{\partial \hat{C}}{\partial \delta_M} &= -\frac{\frac{\partial f}{\partial \delta_M}}{\frac{\partial f}{\partial \hat{C}}} \\
&= -\frac{\left(\frac{\delta_M}{\pi}\right)^{\frac{1}{\sigma_2}} b\pi\alpha\hat{C}^{\sigma_1} \left(\frac{\delta_M + r}{\sigma_2 \delta_M} - 1\right)}{(\delta_M + r)\left[(\frac{\alpha}{\sigma_2} + 1 - \alpha)m^{\frac{\alpha}{\sigma_2}-\alpha}(\delta_M + r) + \sigma_1 \left(\frac{\delta_M}{\pi}\right)^{\frac{1}{\sigma_2}} b\pi\alpha(B\hat{k}^{\varepsilon} - \hat{m} - \delta\hat{k})^{\sigma_1 - 1}\right]}
\end{aligned}$$

In that case, if $\sigma_2 = \sigma_1 = 1$, and then:

$$\hat{m} = \frac{\delta_M b\alpha(B\hat{k}^{\varepsilon} - \delta\hat{k})}{\delta_M(1 + b\alpha) + r}$$

It follows that:

$$\frac{\partial \hat{m}}{\partial \delta_M} = \frac{b\alpha(B\hat{k}^{\varepsilon} - \delta\hat{k})\left[\delta_M(1 + b\alpha) + r\right] - \delta_M b\alpha(B\hat{k}^{\varepsilon} - \delta\hat{k})(1 + b\alpha)}{\left[\delta_M(1 + b\alpha) + r\right]^2}$$

As a result, $\frac{\partial \hat{m}}{\partial \delta_M} > 0$ if and only if $r > 0$, which is always justified, and $\frac{\partial \hat{C}}{\partial \delta_M} = -\frac{\partial \hat{m}}{\partial \delta_M}$ $\square$

Proof of proposition 3
The derivative of $\hat{m}$ with respect to π is given by

$$\begin{aligned}
\frac{\partial \hat{m}}{\partial \pi} &= -\frac{\frac{\partial f}{\partial \pi}}{\frac{\partial f}{\partial \hat{m}}} \\
&= \frac{\left(\frac{\delta_M}{\pi}\right)^{\frac{1}{\sigma_2}} b\alpha(B\hat{k}^{\varepsilon} - \hat{m} - \delta\hat{k})^{\sigma_1} \left(1 - \frac{1}{\sigma_2}\right)}{(\frac{\alpha}{\sigma_2} + 1 - \alpha)m^{\frac{\alpha}{\sigma_2}-\alpha}(\delta_M + r) + \sigma_1 \left(\frac{\delta_M}{\pi}\right)^{\frac{1}{\sigma_2}} b\pi\alpha(B\hat{k}^{\varepsilon} - \hat{m} - \delta\hat{k})^{\sigma_1 - 1}}
\end{aligned}$$

Then $\frac{\partial \hat{m}}{\partial \pi} < 0$ if and only if $\sigma_2 < 1$, which ends the first part of the proof. Similarly, we have:

$$\begin{aligned}
\frac{\partial \hat{C}}{\partial \pi} &= -\frac{\frac{\partial f}{\partial \pi}}{\frac{\partial f}{\partial \hat{C}}} \\
&= -\frac{\left(\frac{\delta_M}{\pi}\right)^{\frac{1}{\sigma_2}} b\alpha(B\hat{k}^{\varepsilon} - \hat{m} - \delta\hat{k})^{\sigma_1} \left(1 - \frac{1}{\sigma_2}\right)}{(\frac{\alpha}{\sigma_2} + 1 - \alpha)m^{\frac{\alpha}{\sigma_2}-\alpha}(\delta_M + r) + \sigma_1 \left(\frac{\delta_M}{\pi}\right)^{\frac{1}{\sigma_2}} b\pi\alpha(B\hat{k}^{\varepsilon} - \hat{m} - \delta\hat{k})^{\sigma_1 - 1}}
\end{aligned}$$

Therefore, $\frac{\partial \hat{C}}{\partial \pi} > 0$ if and only if $\sigma_2 < 1$. If $\sigma_2 = \sigma_2 = 1$, $\frac{\partial \hat{m}}{\partial \pi} = \frac{\partial \hat{C}}{\partial \pi} = 0$ $\square$

Proof of proposition 4
Recall that we are dealing with the case of productivity B depending on health productivity and π and the health depreciation rate δ_M. Thus, we have

$$\begin{aligned}\frac{\partial \hat{m}}{\partial \delta_M} &= -\frac{\frac{\partial f}{\partial \delta_M}}{\frac{\partial f}{\partial \hat{m}}} \\ &= \frac{\left(\frac{\delta_M}{\pi}\right)^{\frac{1}{\sigma_2}} b\pi\alpha(B\hat{k}^{\varepsilon} - \hat{m} - \delta\hat{k})^{\sigma_1}\left[\frac{\delta_M+r}{\sigma_2\delta_M} - 1 + B_{\delta_M}\sigma_1\hat{k}^{\varepsilon}(\delta_M+r)(B\hat{k}^{\varepsilon} - \hat{m} - \delta\hat{k})^{-1}\right]}{(\delta_M+r)\left[(\frac{\alpha}{\sigma_2}+1-\alpha)m^{\frac{\alpha}{\sigma_2}-\alpha}(\delta_M+r) + \sigma_1\left(\frac{\delta_M}{\pi}\right)^{\frac{1}{\sigma_2}} b\pi\alpha(B\hat{k}^{\varepsilon} - \hat{m} - \delta\hat{k})^{\sigma_1-1}\right]}\end{aligned}$$

It then follows that $\frac{\partial \hat{m}}{\partial \delta_M} > 0$ if and only if $\frac{\delta_M+r}{\sigma_2\delta_M} - 1 + B_{\delta_M}\sigma_1\hat{k}^{\varepsilon}(\delta_M+r)(B\hat{k}^{\varepsilon} - \hat{m} - \delta\hat{k})^{-1} > 0$, that is $\sigma_2 < \frac{1}{1-B_{\delta_M}\sigma_1\hat{k}^{\varepsilon}(\delta_M+r)(B\hat{k}^{\varepsilon}-\hat{m}-\delta\hat{k})^{-1}} + \frac{\rho}{\delta_M - \delta_M B_{\delta_M}\sigma_1\hat{k}^{\varepsilon}(\delta_M+r)(B\hat{k}^{\varepsilon}-\hat{m}-\delta\hat{k})^{-1}}$ $\square$ Also,

$$\begin{aligned}\frac{\partial \hat{C}}{\partial \delta_M} &= -\frac{\frac{\partial f}{\partial \delta_M}}{\frac{\partial f}{\partial \hat{C}}} \\ &= -\frac{\left(\frac{\delta_M}{\pi}\right)^{\frac{1}{\sigma_2}} b\pi\alpha(B\hat{k}^{\varepsilon} - \hat{m} - \delta\hat{k})^{\sigma_1}\left[\frac{\delta_M+r}{\sigma_2\delta_M} - 1 + B_{\delta_M}\sigma_1\hat{k}^{\varepsilon}(\delta_M+r)(B\hat{k}^{\varepsilon} - \hat{m} - \delta\hat{k})^{-1}\right]}{(\delta_M+r)\left[(\frac{\alpha}{\sigma_2}+1-\alpha)m^{\frac{\alpha}{\sigma_2}-\alpha}(\delta_M+r) + \sigma_1\left(\frac{\delta_M}{\pi}\right)^{\frac{1}{\sigma_2}} b\pi\alpha(B\hat{k}^{\varepsilon} - \hat{m} - \delta\hat{k})^{\sigma_1-1}\right]}\end{aligned}$$ $\square$

Proof of proposition 5
We have:

$$\begin{aligned}\frac{\partial \hat{m}}{\partial \pi} &= -\frac{\frac{\partial f}{\partial \pi}}{\frac{\partial f}{\partial \hat{m}}} \\ &= \frac{\left(\frac{\delta_M}{\pi}\right)^{\frac{1}{\sigma_2}} b\alpha(B\hat{k}^{\varepsilon} - \hat{m} - \delta\hat{k})^{\sigma_1}\left[1 - \frac{1}{\sigma_2} + B_{\pi}\sigma_1\hat{k}^{\varepsilon}\pi(B\hat{k}^{\varepsilon} - \hat{m} - \delta\hat{k})^{-1}\right]}{(\frac{\alpha}{\sigma_2}+1-\alpha)m^{\frac{\alpha}{\sigma_2}-\alpha}(\delta_M+r) + \sigma_1\left(\frac{\delta_M}{\pi}\right)^{\frac{1}{\sigma_2}} b\pi\alpha(B\hat{k}^{\varepsilon} - \hat{m} - \delta\hat{k})^{\sigma_1-1}}\end{aligned}$$

Then we can see easily that $\frac{\partial \hat{m}}{\partial \pi} < 0$ if and only if $1 - \frac{1}{\sigma_2} + B_{\pi}\sigma_1\hat{k}^{\varepsilon}\pi(B\hat{k}^{\varepsilon} - \hat{m} - \delta\hat{k})^{-1} < 0$, that is $\sigma_2 < \frac{1}{1+B_{\pi}\sigma_1\hat{k}^{\varepsilon}\pi(B\hat{k}^{\varepsilon}-\hat{m}-\delta\hat{k})^{-1}}$. Also,

$$\begin{aligned}\frac{\partial \hat{C}}{\partial \pi} &= -\frac{\frac{\partial f}{\partial \pi}}{\frac{\partial f}{\partial \hat{C}}} \\ &= -\frac{\left(\frac{\delta_M}{\pi}\right)^{\frac{1}{\sigma_2}} b\alpha(B\hat{k}^{\varepsilon} - \hat{m} - \delta\hat{k})^{\sigma_1}\left[1 - \frac{1}{\sigma_2} + B_{\pi}\sigma_1\hat{k}^{\varepsilon}\pi(B\hat{k}^{\varepsilon} - \hat{m} - \delta\hat{k})^{-1}\right]}{(\frac{\alpha}{\sigma_2}+1-\alpha)m^{\frac{\alpha}{\sigma_2}-\alpha}(\delta_M+r) + \sigma_1\left(\frac{\delta_M}{\pi}\right)^{\frac{1}{\sigma_2}} b\pi\alpha(B\hat{k}^{\varepsilon} - \hat{m} - \delta\hat{k})^{\sigma_1-1}}\end{aligned}$$ $\square$

Proof of proposition 6
The condition stated is obtained by establishing the concavity of the objective function in (C, M). The concavity of the state functions and the positivity of λ_A and λ_M are straightforward. Using the Hamiltonian

$$H = U(C(z))\varphi(M(z))e^{-\rho z} + \lambda_A e^{-\rho z}\left[r(z)A(z) + w(z) - C(z) - m(z)\right] + \lambda_M e^{-\rho z}\left[\psi(m(z)) - \delta_M M(z)\right]$$

it is straightforward to obtain with some calculus that the positivity of the determinant of the Hessian matrix of the above Hamiltonian requires

$$C^{-2\sigma_1}M^{-2\sigma_2}\left[\frac{\sigma_1\sigma_2}{(1-\sigma_1)(1-\sigma_2)} - 1\right] > 0$$

That is either $\sigma_1 < 1$ and $\sigma_2 < 1$ or $\sigma_1 > 1$ and $\sigma_2 > 1$ $\square$

Proof of proposition 7
The proof is based on the sign of the derivatives $\frac{\partial \hat{m}}{\partial \delta_M}$ and $\frac{\partial \hat{C}}{\partial \delta_M}$. We have:

$$\frac{\partial \hat{m}}{\partial \delta_M} = \frac{r\alpha B^{\frac{1}{1-\varepsilon}}\left[\left(\frac{\rho+\delta}{\varepsilon}\right)^{\frac{\varepsilon}{\varepsilon-1}} - \delta\left(\frac{\rho+\delta}{\varepsilon}\right)^{\frac{1}{\varepsilon-1}}\right](1-\sigma_2)(1-\sigma_1)}{\left[\alpha\delta_M(1-\sigma_2)+(1-\sigma_1)(\delta_M+r)\right]^2} > 0$$

and,

$$\frac{\partial \hat{C}}{\partial \delta_M} = -\frac{r\alpha B^{\frac{1}{1-\varepsilon}}\left[\left(\frac{\rho+\delta}{\varepsilon}\right)^{\frac{\varepsilon}{\varepsilon-1}} - \delta\left(\frac{\rho+\delta}{\varepsilon}\right)^{\frac{1}{\varepsilon-1}}\right](1-\sigma_2)(1-\sigma_1)}{\left[\alpha\delta_M(1-\sigma_2)+(1-\sigma_1)(\delta_M+r)\right]^2} < 0 \quad \square$$

Proof of proposition 9
We have:

$$\begin{aligned}\frac{\partial \hat{m}}{\partial \delta_M} &= \frac{\alpha\delta_M(1-\sigma_2)B^{\frac{\varepsilon}{1-\varepsilon}}\left[\left(\frac{\rho+\delta}{\varepsilon}\right)^{\frac{\varepsilon}{\varepsilon-1}} - \delta\left(\frac{\rho+\delta}{\varepsilon}\right)^{\frac{1}{\varepsilon-1}}\right]B_{\delta_M}\left[\alpha\delta_M(1-\sigma_2)+(1-\sigma_1)(\delta_M+r)\right]}{(1-\varepsilon)\left[\alpha\delta_M(1-\sigma_2)+(1-\sigma_1)(\delta_M+r)\right]^2} \\ &+ \frac{r\alpha B^{\frac{1}{1-\varepsilon}}\left[\left(\frac{\rho+\delta}{\varepsilon}\right)^{\frac{\varepsilon}{\varepsilon-1}} - \delta\left(\frac{\rho+\delta}{\varepsilon}\right)^{\frac{1}{\varepsilon-1}}\right](1-\sigma_2)(1-\sigma_1)}{\left[\alpha\delta_M(1-\sigma_2)+(1-\sigma_1)(\delta_M+r)\right]^2}\end{aligned}$$

and,

$$\begin{aligned}\frac{\partial \hat{C}}{\partial \delta_M} &= \frac{(\delta_M+r)_M(1-\sigma_1)B^{\frac{\varepsilon}{1-\varepsilon}}\left[\left(\frac{\rho+\delta}{\varepsilon}\right)^{\frac{\varepsilon}{\varepsilon-1}} - \delta\left(\frac{\rho+\delta}{\varepsilon}\right)^{\frac{1}{\varepsilon-1}}\right]B_{\delta_M}\left[\alpha\delta_M(1-\sigma_2)+(1-\sigma_1)(\delta_M+r)\right]}{(1-\varepsilon)\left[\alpha\delta_M(1-\sigma_2)+(1-\sigma_1)(\delta_M+r)\right]^2} \\ &- \frac{r\alpha B^{\frac{1}{1-\varepsilon}}\left[\left(\frac{\rho+\delta}{\varepsilon}\right)^{\frac{\varepsilon}{\varepsilon-1}} - \delta\left(\frac{\rho+\delta}{\varepsilon}\right)^{\frac{1}{\varepsilon-1}}\right](1-\sigma_2)(1-\sigma_1)}{\left[\alpha\delta_M(1-\sigma_2)+(1-\sigma_1)(\delta_M+r)\right]^2} < 0 \quad \square\end{aligned}$$

Proof of proposition 10
The conditions ate checked for $\frac{\partial \hat{m}}{\partial \pi}$, and $\frac{\partial \hat{C}}{\partial \pi}$ as:

$$\frac{\partial \hat{m}}{\partial \pi} = \frac{\alpha\delta_M(1-\sigma_2)B^{\frac{\varepsilon}{1-\varepsilon}}\left[\left(\frac{\rho+\delta}{\varepsilon}\right)^{\frac{\varepsilon}{\varepsilon-1}} - \delta\left(\frac{\rho+\delta}{\varepsilon}\right)^{\frac{1}{\varepsilon-1}}\right]B_\pi}{(1-\varepsilon)\left[\alpha\delta_M(1-\sigma_2)+(1-\sigma_1)(\delta_M+r)\right]^2}$$

So $\frac{\partial \hat{m}}{\partial \pi} < 0$ if and only if $1-\sigma_2 < 0$, then $\sigma_2 > 1$. Similarly,

$$\frac{\partial \hat{C}}{\partial \pi} = -\frac{(\delta_M+r)(1-\sigma_1)B^{\frac{\varepsilon}{1-\varepsilon}}\left[\left(\frac{\rho+\delta}{\varepsilon}\right)^{\frac{\varepsilon}{\varepsilon-1}} - \delta\left(\frac{\rho+\delta}{\varepsilon}\right)^{\frac{1}{\varepsilon-1}}\right]B_\pi}{(1-\varepsilon)\left[\alpha\delta_M(1-\sigma_2)+(1-\sigma_1)(\delta_M+r)\right]^2} \quad \square$$

Table 1: Descriptive statistics: Life expectancy, health expenditure in % of GDP

	Mean	Std.dev.	Min.	Max.	Obs.
Life expectancy					
overall	63.14	11.50	31.22	79.99	$nT = 2396$
between		10.88	34.92	76.77	$n = 201$
within		3.81	39.39	77.82	
Health expenditure					
overall	5.47	2.27	.86	13.30	$nT = 1762$
between		2.12	1.35	12.93	$n = 189$
within		.83	1.61	10.92	

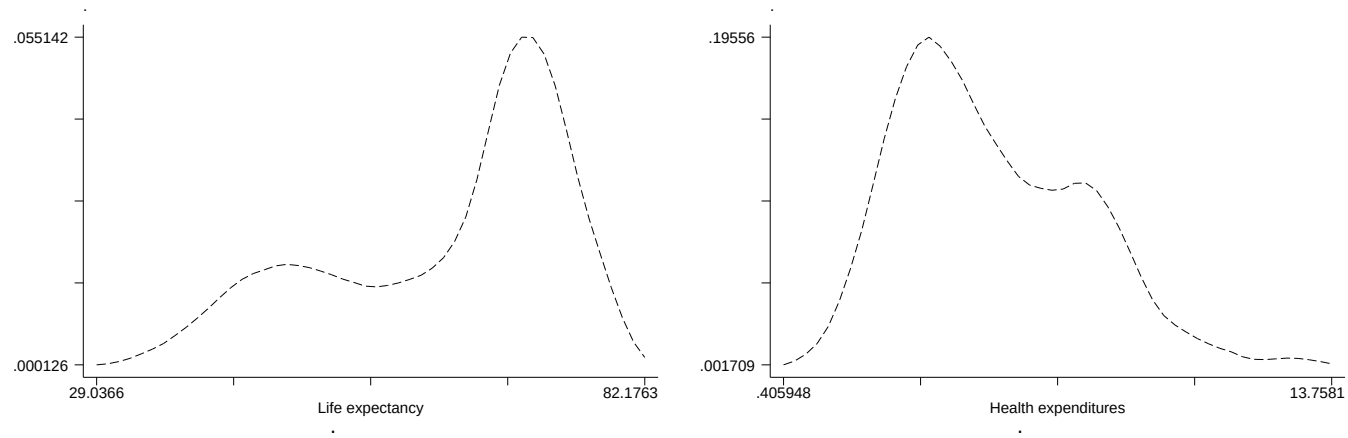

Figure 1: Kernel density estimates. [left]: life expectancy. [right]: health expenditure in percentage of GDP.

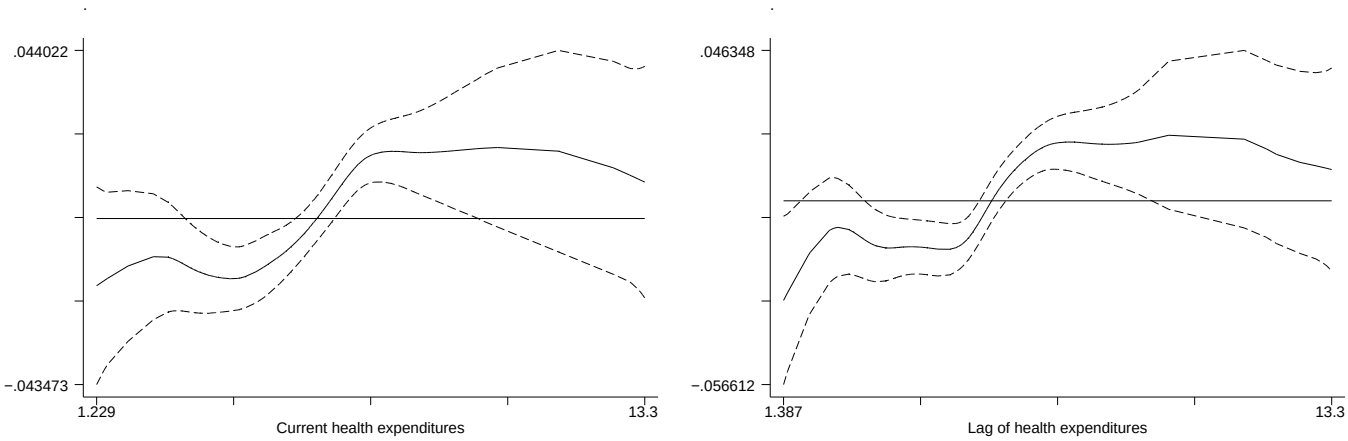

Figure 2: Non-parametric estimation of health expenditure effects on life expectancy. The solid line represents the non-parametric fit. Dashed lines are 95% bootstrap pointwise confidence intervals. The straight solid line is the zero line. [left]: $\hat{m}(\mathbf{x}_{it})$; [right]: $\hat{m}(\mathbf{x}_{i,t-1})$.

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