Non-communicable chronic diseases
and health capital

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Abstract

We develop a model where there is an intergenerational externality concerning obesity and NCDs with two channels of transmission. On the one hand parents affect the inherited health capital and on the other hand they affect the probability of their children being obese/catch up a chronic disease when old. Parents are not fully altruistic and when they make their choices they do not fully account for the impact of their choices on their children’s health. Consequently, the negative externality results on a lower health capital level and a higher level of unhealthy activities at the decentralized equilibrium, when contrasted to the optimal choices. Taxes on unhealthy activities or subsidies on health investments can be used to restore optimal health capital and unhealthy activities levels. Our model is consistent with different development regimes.

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1 Introduction

According to Abegunde and Stanciole (2006), chronic non-communicable diseases (NCDs) are increasing worldwide, accounting for over half of the total deaths in the world, 87% in high income countries and 85% in France. Moreover, according to the World Health Organization (WHO) it is projected that 388 million people will die of NCDs in the next ten years. Even if NCDs have been commonly associated to the elderly of wealthy countries, at the present time NCD are actually the major cause of death all over the world, except for Sub-Saharan Africa.

Obesity and overweight are considered major risk factors of NCDs in particular cardiovascular disease (mainly heart disease and stroke), type 2 diabetes, musculoskeletal disorders like osteoarthritis, and some types of cancers as endometrial, breast and colon. Besides death, obesity leads to substantial disability.

We provide an economic modelling of NCDs and obesity encompassing the intergenerational communicability of eating and physical activity habits. The novelty of our approach relies on the fact that we will present a theoretical framework that includes a set of dynamical equations describing the behavioural and intergenerational transmission mechanism of the epidemics of NCDs and obesity. The epidemics transmission mechanism has already been modelled for communicable diseases (Young (2005) and McDonald and Roberts (2006)) but to our knowledge not yet to NCDs. Our contribution is also in view of understanding which policy instruments can be used to implement the social optimum. Besides that, we pretend to contribute to the analysis of cross-country and regional differences associated to the incidence of the epidemics of NCDs and obesity. Indeed, following the set-up introduced by Blackburn and Cipriani (2002) and Mariani et al. (2009), we study the existence of different development regimes linked with health capital and the epidemics of NCDs/obesity.

In general, chronic diseases have been widely studied from a medical perspective, paying special attention to the mathematical modeling of the epidemics of communicable chronic diseases (CCD). A typical example in
epidemiology is the well-known Compartmental Model (see for instance Kermack and McKendrick (1927, 1932 and 1933), Bailey (1975), Anderson and May (1992), and Kuznetsov and Piccardi (1994)). However, from a theoretical point of view, the economic mechanisms behind NCDs epidemics and, in particular, obesity epidemics, are still far from being understood. Although there are several empirical studies that focus on the relationship between economics and the prevalence of NCDs (see for instance Cumming (1936), Lave and Seskin (1971), Cropper (1981), Mitchell (1990), and Suhrcke et al. (2006)), there is a general agreement about the lack of explicit economic modeling of the problem (Boucekkine et al. (2008) for a survey). According to the same author, there is a rising literature filling the gap between empirics and theory. This new literature distinguishes between short and long-lived epidemics. A short-lived epidemic takes place in a very short period. After that, the economy returns to its initial epidemiological environment. Then, short-lived epidemics are usually modeled as shocks on the initial conditions of the economy. The Black Death (Herlihy (1997), Hansen and Prescott (2002)) and the Spanish Flu (Boucekkine et al. (2008)) are examples of this kind of epidemics. However, long-lived epidemics are associated with long periods of disease. Therefore, the theory behind this kind of epidemics is much more complicated since the effects of long-lived epidemics cannot be reduced to a story of initial conditions shocks. HIV is an important example of long-lived epidemics, which devotes great attention due to its impact in sub-Saharan Africa (see for instance Young (2005) and McDonald and Roberts (2006)). NCDs are another example of long-lived epidemics, with growing incidence in both developed and developing countries. However, the lack of knowledge about the dynamical mechanisms behind NCDs epidemics and economic growth calls for further theoretical effort. This is, indeed, the aim of the part one of this project. The starting point will be the available theoretical models on long-lived CCDs epidemics, mainly HIV, which we must adapt to the NCDs and obesity realities.

We agree that by definition a NCDs is not biologically communicable. Although, “No epidemic has ever been resolved by paying attention to the affected individual”, a famous George W. Albee’s quote, that is of much
adequacy to the obesity epidemics. In reality there is evidence of obesity peer and family effects. Indeed, Christakis and Fowler (2007) and Trogdon et al. (2008) find clusters of obesity in social networks and identify peer effects. There is however a discussion concerning the causes of obesity clusters and if they are due more to environmental external factors than person-to-person relation (Cohen-Cole and Fletcher (2008), and Fowler and Christakis (2008)). Authors agree anyway that obesity clusters.

Family ties and in particular those between parents and children are also very important determinants for obesity. Indeed the probability of a younger than 10 years old child of becoming obese more than doubles with parental obesity (Branca et al. (2007)). It is unquestionably that there is a genetic component but this is estimated to explain just between 25% and 40% (Bouchard (1996)). Additionally, Chen and Li (2009) find that a mother’s education determines the health of adopted children and that the effect is similar to own birth sample. Therefore, the main effect of the mother’s education on child health is not genetics but post-natal nurturing. Accordingly, we will introduce a transmission mechanism of the NCDs and obesity that is not biological but social. Indeed, in part one of the project we thus aim to contribute to the understanding of the economics of the obesity epidemics by means of providing a theoretical framework that encompasses the intergenerational communicability of eating and physical activity habits.

We present an overlapping generations model in which agents live three periods: childhood, adulthood and old age. Consumption and saving decisions are made at adulthood and therefore parents make their own decisions and those of their children. The intergenerational communicability of obesity and NCDs is introduced by assuming that a child’s probability of being obese when old depends on her parents’ health capital or, in other words, on her health capital at the first period of life. Additionally, another intergenerational channel is present because we assume that individuals “inherit” their parents’ health capital. That is to say that the dynamics of the economy is based on health capital accumulation. Health capital will depend on past health capital but we aim to allow individuals’ consumption of goods that decrease health capital accumulation such as saturated fats and sugar and
to consume others that increase health capital accumulation such as sport. Health capital will depend on past health capital but individuals’ consumption of certain goods decrease health capital accumulation, such as saturated fats and sugar, and consume others that increase health capital accumulation, such as sport. Assuming that individuals are not perfectly altruist, an externality arises and therefore the decentralized equilibrium is inferior to the social planner solution. We then analyse how policy instruments such as a tax on sugar and on saturated fat or a subsidy on sport can be used to recover the social optimum. Our model also captures the existence of different development regimes in this model. Consequently policy instruments may as well be used to escape the low development regime.

Our paper is organized as follows. In Section ?? we present the model and in Section ?? we provide the welfare analysis. Section ?? concludes.

2 The model

2.1 Setup

Let us assume a discrete-time infinity-horizon economy populated by overlapping generations of agents living for three periods: childhood, adulthood, and old age. Time is indexed by $t = 0, 1, 2, ..., \infty$, and all decisions are taken in the adult period of life. We also consider identical agents within each generation and no population growth (the size of each generation is normalized to 1). In this paper we assume that individuals might suffer from a non-communicable chronic disease (NCD) at the old age. Indeed, this will depend on their health capital, broadly defined as the conditions that prevent an individual to be struck by a NCD.\footnote{Commonly accepted proxies for NCD-related health capital are height (see, among others, Deaton, 2008; Bozolli et al., 2008; and Steckel, 1995, 2008) and body mass index (BMI) (see, for instance, Revicki and Israel, 1986; and WHO, 2004).}

Individual preferences are described by a utility function $U_t(c_t, v_t, h_{t+1})$, such that $\partial U_t(\cdot)/\partial c_t > 0$, $\partial U_t(\cdot)/\partial v_t > 0$, $\partial U_t(\cdot)/\partial h_{t+1} > 0$, $\partial^2 U_t(\cdot)/\partial c_t^2 < 0$, $\partial^2 U_t(\cdot)/\partial v_t^2 < 0$, $\partial^2 U_t(\cdot)/\partial h_{t+1}^2 < 0$, $\lim_{c_t \to 0} \partial U_t(\cdot)/\partial c_t = +\infty$, $\lim_{v_t \to 0} \partial U_t(\cdot)/\partial v_t = ...$
Agents care about consumption \((c_t)\) and unhealthy activities \((v_t)\). Moreover, following Grossman (1972, 2000), they are also concerned about their health capital \((h_{t+1})\) when old. In order to get closed-form solutions, we assume the following function:

\[
U_t(c_t, v_t, h_{t+1}) = \mu \ln c_t + \lambda \ln v_t + (1 - \pi_t)\gamma \ln h_{t+1} + \pi_t\gamma(1 - \phi)\ln h_{t+1},
\]

(1)

where \(\mu > 0\) and \(\lambda > 0\) represent respectively the weight agents give to consumption and unhealthy activities, \(\gamma > 0\) stands for their concern about future health capital, and \(\pi_t\) denotes the probability of suffering from a NCD at old age. Consistently with the extensive medical literature on NCDs (see, for instance, Dietz, 1998; McElroy et al., 2004; Needham and Crosoe, 2005; and Kuh and Ben-Shlomo, 2005), we assume that \(\pi_t\) is endogenously determined by the health capital. Moreover, Case and Paxson (2008) also point out that it is widely accepted the crucial role of early life health (in particular, childhood) in determining health and economic outcomes at older ages (see also de la Croix and Licandro, 2007). Therefore, we consider in this paper that the probability of suffering from a NCD is a function of the inherited health capital \(\pi_t = \pi(h_t)\), such that \(\partial\pi(h_t)/\partial h_t < 0\), \(\lim_{h_t \to 0} \pi(h_t) = \pi\) and \(\lim_{h_t \to \infty} \pi(h_t) = \pi\), with \(0 < \pi < \bar{\pi} < 1\). Finally, \(\phi \in [0, 1]\) represents the disutility of suffering from a NCD, which may be interpreted as time loss due to treatment, hospital attendance, etc.\(^2\)

Adult agents allocate their exogenous income \((w_t)\) among consumption, unhealthy activities, and health investments \((m_t)\) as medical care, physical activity, etc. The corresponding budget constraint is

\[
w_t = c_t + v_t + m_t.
\]

(2)

As in Grossman (1972, 2000), our model assumes that health capital accumulates over time. In particular, we consider the following law of motion:

\[
h_{t+1} = (1 - \delta)h_t + \sigma m_t - \alpha v_t,
\]

(3)

\(^2\)Notice that our setup also allows for two extreme cases: mortal disease \((\phi = 1)\), and when the disability of NCD is negligible \((\phi = 0)\).
where $0 < \delta < 1$ and $\sigma, \alpha > 0$. In this expression, $\delta$ represents the depreciation rate of health capital, $\sigma$ is the effectiveness of health investment, and $\alpha$ is the reduction of health conditions due to the unhealthy activities of individuals.\(^3\)

At this moment it is worthwhile to consider the following observation. From a medical perspective, a NCD does not exhibit transmission among individuals. However, there is significant evidence of the existence of a behavioural transmission mechanism of NCDs which may be related to the agent’s economic choices. For instance, Branca et al. (2007) find that parental obesity more than doubles the probability of adult obesity among children younger than 10 years old. Moreover, Chen and Li (2009) show that the main effect of the mother’s education on child health is not genetics but post-natal nurturing. In this paper, we will study one dimension of the problem: the inter-generational transmission of NCDs. Indeed, equation (??) considers that the individual choices will affect health capital at the old age. However, agents are not able to internalize the external effects of their choices on future generations and, in particular, on the “bequested” probability of suffering from a NCD ($\pi_{t+1} = \pi(h_{t+1})$). Therefore, as we will see latter, agents may enjoy lower health conditions than it would be socially optimal.\(^4\)

### 2.2 Individual behaviour

Agents maximize $U_t(c_t, v_t, h_{t+1})$ subject to (??), (??), $c_t > 0$, $v_t > 0$, $m_t > 0$ and $h_t > 0$, where $w_t$ and $h_t$ are taken as given. For a general utility function,

\(^3\)Consistently with our definition of health capital, we assume that a NCD strike do not reduce $h_{t+1}$. However, it induces a utility loss thought the parameter $\phi$.

\(^4\)Since our paper focuses on inter-generational externalities, we can assume that $\pi_t$ is fully determined by the inherited health capital. However, we are aware that a more complete setup might also consider that agents can modify their current health capital through their actions. This would introduce an additional dimension to the problem which will be left for a future research.
the corresponding optimal condition is
\[
\frac{\partial U_t}{\partial c_t} = \frac{\partial U_t}{\partial v_t} - \alpha \frac{\partial U_t}{\partial h_{t+1}}. \tag{4}
\]
Considering the functional form (??), the optimal choices are
\[
m_t = \frac{(1 - \alpha)[\lambda \sigma + \mu(\sigma + \alpha)]h_t - \sigma[\gamma(\sigma + \alpha)(1 - \phi \pi_t) + \lambda \alpha]w_t}{-\sigma(\sigma + \alpha)[\lambda + \mu - \gamma(1 - \phi \pi_t)]}, \tag{5}
\]
\[
v_t = \frac{\lambda[(1 - \delta)h_t + \sigma w_t]}{(\sigma + \alpha)[\lambda + \mu + \gamma(1 - \phi \pi_t)]}, \tag{6}
\]
and
\[
c_t = \frac{\mu[(1 - \delta)h_t + \sigma w_t]}{\sigma[\lambda + \mu + \gamma(1 - \phi \pi_t)]}. \tag{7}
\]
From the equations (??), (??) and (??), we can observe that income has a positive effect on consumption, unhealthy activities and health investment. Moreover, greater inherited health conditions increase consumption and unhealthy activities, but decrease health investment: if inherited health conditions improve, investment in health capital is less needed. Regarding the probability of suffering from a NCD, the greater the probability the lower the value of old age for an individual. Therefore, \(\pi_t\) has positive effect on consumption and unhealthy activities, but a negative one on health investment. Similarly, a greater disutility of NCD reduces health investment, while consumption and unhealthy activities rise.

### 2.3 Dynamics

The dynamics of our economy are completely characterized by the evolution of health capital. By substituting equations (??), (??) and (??) into (??), we get the corresponding transition function \(\varphi(h_t)\):
\[
h_{t+1} = \frac{\gamma[(1 - \phi \pi_t)][(1 - \delta)h_t + \sigma w]}{\gamma + \mu + \gamma[1 - \phi \pi_t]} \equiv \varphi(h_t), \tag{8}
\]
where income is assumed to be constant, \(w_t = w\), for the sake of simplicity. In this paper we focus on a steady-state equilibrium \(h^*\) defined as a fixed

\footnote{Since agents inherit their current health conditions, our results would not change if \(h_t\) is also introduced in the utility function.}
point of the transition function, i.e., $h^* = \varphi(h^*)$.\(^6\) One can easily check that $h^*$ is stable (unstable) if $\varphi'(h^*) < 1 (> 1)$.

In our framework, the shape of the transition function is crucial to characterize our equilibrium. Indeed, if $\varphi(\cdot)$ is concave, there is a unique stable steady-state $h^*$ (see Figure <number>). In this case, we can conclude from equation (8) that $h^*$ is positively affected by the income ($w$), the effectiveness of health investment ($\sigma$) and the individual concern about future health capital ($\gamma$). However, a greater disutility of suffering from a NCD ($\phi$) will reduce the steady-state value of health capital.

As in Blackburn and Cipriani (2002), our model allows for multiple stable steady-states if the transition function displays one or more inflection points. Indeed, if $\phi(h_t)$ is a convex-concave function, our economy may end up in either high ($h^*_{H}$) or low ($h^*_{L}$) health capital, depending on the initial conditions $h_0$ (see Figure <number>). Several functional forms of $\pi(h_t)$ can provide a convex-concave $\phi(h_t)$. In particular, following Mariani et al. (2010), an analytically tractable illustration of this case is a step-function approximation of a convex-concave $1 - \pi(h_t)$ (the probability of not suffering from a NCD) (see Figures <number> and <number>).

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\(^6\)Mathematically, this corresponds to a stationary point of the equilibrium difference equation (??).
Multiple steady states may justify the existence of regional asymmetries in what concerns obesity and NCDs in general. Even restricting ourselves to Europe it is large the difference among countries. In 2005 in France, for example, 8% of men and 7% of women are obese, contrasting enormously with 21% of men and 24% of obese women in the United Kingdom (WHO (2009)). Blancfower and Oswald (2008) have also found evidence of country asymmetries with respect to hypertension. Additionally a step function seems appropriate to represent the problem at stake. Indeed physicians make often use of thresholds to identify diseases and critical health conditions. Examples are the thresholds for diabetes, blood pressure, obesity (BMI), etc....

We therefore assume the following step function for the probability of the disease:

\[ \varphi(h^*) < 1 \]
\[ \varphi'(h^*) < 1, \quad \varphi'(h^*) > 1 \]

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7The following quote gives also support to our assumption “cirrhosis in the liver may not result in a clinical effect until over 50% of the liver has been replaced by fibrous tissue.” Yuill and Miller (2008), Encyclopedia of Hearth, online on National Library of Medicine.
The transition function defined by Equation (9) is therefore composed by two branches and given by

\[
\pi(h_t) = \begin{cases} 
\pi_H & \text{if } h_t < h_c \\
\pi_L & \text{if } h_t \geq h_c 
\end{cases}
\] 

(9)

\[
\varphi(h_t) = \begin{cases} 
\frac{\gamma(1-\phi\pi_H)(1-\delta)h_t + \sigma w}{\gamma(1-\phi\pi_H)+\lambda+\mu} & \text{if } h_t < h_c \\
\frac{\gamma(1-\phi\pi_L)(1-\delta)h_t + \sigma w}{\gamma(1-\phi\pi_L)+\lambda+\mu} & \text{if } h_t \geq h_c 
\end{cases}
\] 

(10)

We assume an exogenous value for \( h_c \). The dynamics of the model admits therefore two stable steady states that are given by

\[
h_{\pi_H}^* = \frac{\gamma(1 - \phi \pi_H)\sigma w}{\delta \gamma(1 - \phi \pi_H) + \lambda + \mu} 
\]

(11)

\[
h_{\pi_L}^* = \frac{\gamma(1 - \phi \pi_L)\sigma w}{\delta \gamma(1 - \phi \pi_L) + \lambda + \mu}. 
\]

(12)

Figure <number> represents the two possible steady states. The low steady state is associated with a low level of health capital and a high probability of the disease while the high one is associated with a high level of health capital and low probability of the disease.

At this stage it may help understand what may cause the increase of the health capital steady state level. Permanent increases of income \( w \) or of the medical technology \( \sigma \) produce an upward parallel movement. On the other
hand a permanent decrease of the cost of NCD $\phi$ or of $\pi$ shift the curves upwards in a rotated movement. Therefore these are the variables the planner may use to restore a social optimum. If the effects are large enough it may be even possible to avoid the low steady state. Figure <number> illustrate these effects.

Suf. $\uparrow$ of $w$ or medical technology ($\sigma$) Suf. $\downarrow$ of $\pi$ or cost of NCD ($\phi$)

3 Welfare Analysis

In our model agents do not fully perceive the consequences of their choices and consequently an (intergenerational) externality arises. We now contrast the descentralized solution with the social planner solution. We will proceed in two stages. First we adopt the golden rule allocation, as defined by Chichilnisky et al. (1995) and used by Mariani et al. (2009) among others. The green golden rule allocation is the optimal solution for a “myopic” social planner treating all generations symmetrically and maximizing welfare in each period. Second we look at the dynamic optimal solution of a fully forward looking planner. The advantage of the first relies on its tractability as we can use closed form solutions.

The myopic social planner aims to maximize aggregate utility in each period subject to the budget constraint, the health capital law of motion,
and $c_t > 0$, $v_t > 0$, $m_t > 0$ and $h_t > 0$.

$$\max U(c, v, h)$$

subject to:

\[
w = c + m + v \\
\]

\[
h = (1 - \delta)h + \sigma m - \alpha v
\]

The solution to this problem is

\[
\frac{\partial U}{\partial v} = \frac{\partial U}{\partial c} + \frac{\alpha}{\delta} \left[ \frac{\partial U}{\partial h} + \frac{\partial U}{\partial \pi} \frac{\partial \pi}{\partial h} \right].
\] (13)

Comparing (13) with the decentralized solution given by (9) at the steady state, one can easily observe that the two coincide when $\delta \to 1$ and $\partial \pi / \partial h \to 0$ simultaneously. This means that when the two externality transmission channels are neutralized the decentralized solution coincides with the golden rule solution. At this point it should be clear that we are present an externality mechanism (parents’ choices affect their children’s utility) through two channels. The first channel consists on the fact that children’s inherit their parents’ health capital. The greater $\delta$ the greater the health capital depreciation from one period to the other, and therefore the weaker is the genetic influence. The second channel consists on the fact that the probability of catching up a disease depends on one’s health capital as a child (or to say in differently, on the health capital of one’s parents). Therefore, for the externality to disappear both channels must be inactive.

The planner can restore the Golden Rule by imposing a tax on unhealthy activities (or equivalently, a subsidy on health investments), where we suppose that the tax revenue is given back to individuals in the form of a lump-sum transfer. In the decentralized problem the budget constraint is replaced by

\[
w + T = c + m + (1 + t)v,
\] (14)

with $T = \tau v$. It can be checked that the tax restoring the golden rule solution is given by:
\[
\tau = \frac{\alpha}{\delta \sigma} \left[ (1 - \delta) + \frac{\partial U}{\partial \pi} \frac{\partial \pi}{\partial h} \right].
\] (15)

Again, if the intergenerational externality is absent \((\delta \to 1\) and \(\partial \pi / \partial h \to 0\)) then there is no need for distortion and \(\tau = 0\).

In our setting, assuming the utility function as given by (??) and the step function (??) allow us to have closed form solutions for the Golden Rule. As expected, the health capital of equilibrium under the Golden Rule is higher than the decentralized equilibrium. Indeed, the myopic planner solves the following problem for \(v\) and \(h\)

\[
\max U(c, v, h) = \mu \ln(c) + \lambda \ln(v) + (1 - \pi) \gamma \ln(h) + \pi \gamma (1 - \phi) \ln(h)
\]

subject to:

\[
w = c + m + v \\
h = (1 - \delta)h + \sigma m - \alpha v.
\]

The solution is given by \(h^g\) and \(v^g\) defined as

\[
h^g = \frac{\gamma \sigma (1 - \phi \pi) w}{\delta (\lambda + \mu) + \gamma \delta (1 - \phi \pi)},
\] (16)

\[
v^g = \frac{\lambda \sigma w}{(\sigma + \delta) \left[ \lambda + \mu + \gamma (1 - \phi \pi) \right]}.
\] (17)

Contrasting (??) with either (??) or (??) (depending on the value of \(\pi\)) it comes out clear that \(h^g > h^*\) and therefore the health capital accumulated at the steady state of the decentralized solution is suboptimal. In accordance, the level of unhealthy activities is above the optimal \(v^g < v^*\), as it becomes apparent from the contrast of (??) to (??). Finally, in our specific scenario the Golden Rule and the decentralized solution coincide as long as health capital is not inherited \((\delta \to 1)\). This is so because by imposing a step function we have eliminated one of the channels of transmission. The relevant tax is in this case given by

\[
\tau = \frac{1 - \delta \alpha}{\delta \sigma}.
\] (18)
3.1 Full Forward Looking Planner

The analysis can be extended by supposing the planner also cares about the transition process. The conclusions are consistent with the Golden Rule solution. In this case the social planner solves the following Langrangean

\[ \mathcal{L} = \beta^{-1} U_{-1} + \sum_{t=0}^{\infty} \beta^t \left\{ U_t(c_t, v_t, h_{t+1}) + \lambda_{t+1} [(1 - \delta) h_t + \sigma w - (\sigma + \alpha) v_t - \sigma c_t - h_{t+1}] \right\} \]  

The FOC is given by

\[
\frac{\partial U_t}{\partial c_t} = \frac{\partial U_t}{\partial v_t} - \alpha \left[ \frac{\partial U_t}{\partial h_{t+1}} + \frac{\partial U_{t+1}}{\partial \pi_{t+1}} \frac{\partial \pi_{t+1}}{\partial h_{t+1}} + \frac{\beta^t}{\beta^{t+1}} \lambda_{t+2} (1 - \delta) \right], 
\]

and again the optimal solution and the decentralized one coincide as long as the two externality channels are neutralized. The optimal tax is given by

\[
\tau_t = \frac{\beta^{t+1}}{\beta^t} \frac{\alpha}{\sigma} \left[ \frac{\partial U_{t+1}}{\partial \pi_{t+1}} \frac{\partial \pi_{t+1}}{\partial h_{t+1}} + (1 - \delta) \lambda_{t+2} \right], 
\]

that is driven to zero as long as \( \delta \to 1 \) and \( \partial \pi / \partial h \to 0 \).

4 Concluding remarks

We have developed a model where there is an intergenerational externality concerning obesity and NCDs with two channels of transmission. On the one hand parents affect the inherited health capital and on the other hand they affect the probability of their children being obese/catch up a chronic disease when old. Only when the two channels are neutralized the externality is absent. Parents are not fully altruistic and when they make their choices they do not fully account for the impact of their choices on their children’s health. Consequently, the negative externality results on a lower health capital level and a higher level of unhealthy activities at the decentralized equilibrium, when contrasted to the optimal choices. Taxes on unhealthy activities or subsidies on health investments can be used to restore optimal health capital and unhealthy activities levels.
Our model is able to predict different development regimes where some economies would be at the low steady state, associated with a low level of health capital and a high probability of the disease, while others would be at the high one, associated with a high level of health capital and low probability of the disease. Different development regimes can be an explanation for obesity/chronic disease prevalence rates asymmetries across countries and regions. We have introduced different development regimes by means of a step function for the probability of getting the NCD.

Several remarks can be made to our modelization. The first concerns the fact that individuals cannot affect directly their probability of disease that instead depends on their parents health capital. We agree that such assumption is more realistic, although our choice was consequent. We wanted, at a first stage, to isolate the transmission mechanism of NCDs from parents to children. Our guess is that letting $\pi$ to depend on individuals’ choices would reduce the importance of the intergenerational externality.

The second concerns income that we have supposed to be exogenous. It is in our interest to develop the analogous model supposing endogenous income. Finally we have supposed homogenous individuals. Although we believe that relying this assumption complicates considerably the analysis we think it is worth trying.

Another question concerns the empirical validation of our assumptions, mainly the relevance of the transmission channel and for the existence of different development regimes. Its is our aim to contribute to this literature using the SECODIP database for France.
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