Intergenerational Transmission of Non-Communicable Chronic Diseases

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Abstract

We introduce a theoretical framework that contributes to the understanding of the non-communicable chronic diseases (NCDs) epidemics: even if NCDs are not "biologically" communicable, they may spread due to the transmission of unhealthy activities such as unhealthy diet, physical inactivity, and smoking. In particular, we study the intergenerational dimension of this mechanism. We find that, due to the "social" transmission of NCDs, agents choose lower health conditions and higher unhealthy activities than what is socially optimal. Taxes on unhealthy activities, that may subsidize health investments, can be used to restore the social optimum. Finally, we also observe that our model is consistent with the existence of regional asymmetries regarding the prevalence of obesity and NCDs.

Keywords: Health capital, Chronic diseases and obesity, Social transmission.
Journal of Economic Literature: H21; H23; I18.

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1 Introduction

Non-communicable chronic diseases (NCDs) are "diseases or conditions (...) that affect individuals over an extensive period of time and for which there are no known causative agents that are transmitted from one affected individual to another." (Daar et al., 2007, p. 494). Despite being non-infectious NCDs are considered an epidemic because of their high prevalence rates. Indeed, according to Abegunde and Stanciole (2006), NCDs are increasing worldwide accounting for over half of the total deaths in the world, and 87% in high income countries. Moreover, the World Health Organization (WHO) points out that, even if NCDs have been commonly associated with the elderly of wealthy countries, at the present time NCDs are actually the major cause of death all over the world, except for Sub-Saharan Africa. In this regard, WHO (2008) estimates 35 million deaths each year due to NCDs. Besides death, NCDs also lead to substantial disabilitiy. Examples of NCDs are cardiovascular diseases, i.e., mainly heart disease and stroke, cancers, respiratory diseases, diabetes, and musculoskeletal disorders like osteoarthritis. In this paper, we provide an economic modelling of NCDs' epidemic mechanism enhancing its intergenerational determinants. Our contribution is also in view of understanding NCDs' economic implications and which policy instruments can be used to enhance welfare. Additionally, we attempt to contribute to the analysis of cross-country differences associated with the incidence of the epidemics of NCDs.

In general, epidemics have been widely studied from a medical perspective, paying special attention to the mathematical modeling of the epidemics of infectious diseases. 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, there is a general agreement regarding the lack of explicit epidemics' economic modeling (Boucekkine et al., 2008 for a survey). Indeed, even if the epidemics mechanism has already been modeled for infectious diseases (Young, 2005; McDonald and Roberts, 2006; and Philipson, 2000), to our knowledge it has not yet been modeled for NCDs. Still, 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). In contrast, from a

theoretical point of view, the economic mechanisms behind NCDs' epidemics are still far from being understood.

The literature on the epidemics' economic modeling distinguishes between short and long-lived epidemics. A short-lived epidemic takes place in a very short period, after which the economy returns to its initial epidemiological environment. Therefore, short-lived epidemics are usually modeled as shocks on the initial conditions of the economy. Examples are the Black Death (Herlihy, 1997; and Hansen and Prescott, 2002) and the Spanish Flu (Boucekkine et al., 2008). On the other hand, long-lived epidemics are associated with long periods of disease and hence their effects cannot be reduced to a story of initial conditions shocks. As a result, the theory rationalizing this kind of epidemics turns out to be more complex. An example is HIV (see for instance Young, 2005; McDonald and Roberts, 2006; and Boucekkine et al., 2009). NCDs are indeed another example of long-lived epidemics. However, because they are non-infectious, the lack of knowledge about the dynamical mechanisms behind NCDs epidemics and economics calls for further theoretical effort. Our aim is precisely to contribute to the economic modeling of NCDs epidemics' dynamical mechanism.

The main causes of NCDs are genetics and age, as well as modifiable risk factors, such as unhealthy diet, physical inactivity, and smoking (WHO, 2005). The novelty of our approach is to provide a theoretical framework that incorporates the social transmission of the modifiable risk factors. That is to say, that even if NCDs are not infectious, we identify a transmission mechanism of its modifiable risk factors that contributes to explaining the epidemic dimension of NCDs. More precisely, we introduce an intergenerational transmission mechanism of the NCDs that being social is not biological.

We present an overlapping generations model in which agents live for three periods (childhood, adulthood and old age), and where the dynamics of the economy is based on health capital accumulation (Grossman, 1972). All economic decisions are made at adulthood and therefore parents decide upon their consumption levels and those of their children. More specifically, parents decide upon consumption levels of unhealthy goods (as for example salt, tobacco, saturated fat) and prevention ones (physical activity, medical care, etc.) that affect the level of health capital of the following period. The intergenerational

NCDs' transmission is introduced through two different effects. The first assumes that children inherit their parents' health capital that is affected by their parents' choices of consumption of unhealthy and prevention goods. The second assumes that a child's probability of suffering from a NCD when old depends on her parents' health capital or, in other words, on her own health capital at the first period of life, also affected by her parents' choices. Assuming that individuals are not perfectly altruist, an externality arises since parents do not fully account for the effects of their choices on their children's health. Therefore the decentralized equilibrium is inferior to the social planner solution. We then analyse how policy instruments such as a tax on the unhealthy good, that may subsidize health investments, can be used to recover the social optimum. Our model also captures the existence of different development regimes linked to the presence of health thresholds. This provides therefore a reasoning for different regional NCDs prevalence rates. Moreover, we also study the role of health education or informational programs on the level of health capital, NCDs prevalence rates, and how it affects the optimal policy.

Our paper is organized as follows. In Section 2 we review the literature that provides evidence on NCDs social transmission in general and intergenerational transmission in particular. In Section 3 we present the model. Section 4 considers the decentralized solution and, in Section 5, we provide the welfare analysis. Finally, Section 6 concludes.

2 Evidence on NCDs' social transmission

Our economic modeling of NCDs' social transmission relies on three main assumptions. First, the modifiable risk factors such as unhealthy diet, physical inactivity, and smoking are socially transmissible. In particular, we assume an intergenerational transmission mechanism consisting of parents deciding upon their children's consumption of unhealthy and prevention goods. Second, parents' choices affect their children's health capital and, third, their children's probability of suffering from NCDs at old age. In this section we review the literature that provides evidence on these assumptions.

The idea that modifiable risk factors are socially transmitted is not new.

For instance, Eisenberg (2004) analyses the effect of a user-friend's move or graduation on a young person's marijuana use, smoking, and binge drinking and finds that all decrease. Family ties and in particular those between parents and children are also very important in determining children's propensity in engaging in modifiable risk factors. Powell and Chaloupka (2005) find that teenagers are more likely to smoke if their parents do, although genetics could be playing a role. Overcoming the genetics determinant, Sacerdote (2007) uses data on adoptees to investigate the intergenerational transmission of modifiable risk factors on young adults offsprings and concludes that drinking behavior is transmitted equally well towards adoptees and nonadoptees. Regarding unhealthy eating and in particular disinhibited overeating, Francis et al. (2007) find that girls with both parents overweight had the highest increases in disinhibited overeating and BMI. Other authors have investigated to what extent mealtime environment and in particular parents' feeding styles contribute to the child's unhealthy eating. In this respect, Zeller et al. (2007) have shown that mothers of obese children experience more mealtime challenges and both parents experience less mealtime interactions. Additionally, obese mothers are more likely to offer their children food in response to emotional distress, use food as a form of reward, encourage children to eat beyond what was wanted, and control less high-fat foods intakes (Wardle et al., 2002). Still, there is an ongoing discussion concerning income classes' and race-related differences (Powers et al., 2006), as well as sex-related ones (Jahnke and Warschburger, 2008). All in all and with few exceptions, the medical and economic literature seems to agree on the intergenerational transmission of modifiable risk factors.

Evidence on parents' choices affecting their children's health capital is plentiful. Even if health capital is a complex concept (Grossman, 1972 and 2000) commonly used proxies are height (see, among others, Case and Paxson, 2008; Deaton, 2008; Deaton and Arora 2009; Bozolli et al., 2008; and Steckel, 1995 and 2008; Silventoinen, 2003) and body mass index (BMI) (see, for instance, Revicki and Israel, 1986; and WHO, 2004). Therefore, evidence on parents' choices affecting their children's height or BMI, as well as evidence on correlation between parents' and children's heights or BMIs give support to our assumption. In this regard, Chen and Li (2009) conclude that mother's education is an important determinant on a child's health, as measured by height-

for-age-z-score. Additionally, the authors find the effect to be similar between adoptees and own-birth children putting therefore in evidence that behavior, just as genetics, is a channel of health transmission. Currie and Moretti (2003) also find that mothers attending college had a significant impact on children's health but Lindeboom et al. (2009) do not find any evidence of increasing the school leaving age of one year and offsprings' health. The latter authors remark that parents' education affecting children's health may be present only at sufficiently high education levels. Concerning obesity, a prominent example of parents' choices affecting children's health is tobacco consumption by pregnant women. Indeed, despite causing low birth-weights, it contributes to children's obesity at several ages (see for example Adams et al., 2005; Lake et al. 1997; Mamun et al., 2006; and Mendez et al., 2008). Moreover, there is a common agreement that children of obese parents are more likely to be obese at all ages, including adulthood (among others, Branca et al., 2007; and Abu-Rmeileh et al., 2008). Part of obesity's transmission is obviously due to genetics. Still, in the last 30 years obesity has doubled in the US (Cutler et al., 2003) and in many European countries it has increased threefold (WHO, 2009). Obviously such obesity prevalence increases cannot be supported by a similar genetic change (Hill and Peters, 1998). Accordingly, Bouchard (1996) estimates genetics to explain just between 25% and 40% of obesity rates increase, in accordance with Sacerdote's (2007) finding (46%).

Finally, there is evidence of parent's choices affecting their children's probability of suffering from NCDs at old age (see, for instance, Osmond and Barker, 2000). Indeed, low birth weight is associated with increased probability of coronary heart diseases and diabetes in later life: the mechanism at stake is that fetal growth restriction, due amongst others to maternal smoking and unhealthy diet, may imply a reprograming of the metabolism. Barker and Clark (1997) and Godfrey and Barker (2000) survey this literature, and Victoria et al. (2008), Montgomery and Ekbom (2002), and Eriksson et al. (2001) provide further results. Also. van den Berg et al. (2007) using historical data for the Netherlands find evidence that early life exposure to the 1846-47 famine results in lower survival rates at old ages for men.

3 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 NCD at the old age, and this will depend on their health capital.

Individual preferences are described by an expected lifetime utility function $U_t(c_t, v_t, h_{t+1}, \pi_t)$, where $\partial U_t(\cdot)/\partial c_t > 0$, $\partial U_t(\cdot)/\partial v_t > 0$, $\partial U_t(\cdot)/\partial h_{t+1} > 0$, $\partial^2 U_t(\cdot)/\partial c_t^2 < 0$, $\partial^2 U_t(\cdot)/\partial v_t^2 < 0$, $\partial^2 U_t(\cdot)/\partial h_{t+1}^2 < 0$, $\lim_{c_t \to 0} \partial U_t(\cdot)/\partial c_t = +\infty$, $\lim_{v_t \to 0} \partial U_t(\cdot)/\partial v_t = +\infty$, and $\lim_{h_{t+1} \to 0} \partial U_t(\cdot)/\partial h_{t+1} = +\infty$. Agents care about consumption c_t and unhealthy activities v_t, which encompass modifiable risk factors such as smoking, alcohol, consumption of fattening food and sugar-sweetened beverages. Moreover, following Grossman (1972, 2000), they are also concerned about their health capital when old h_{t+1}. We assume that individuals may suffer from a NCD at old age with a probability π_t. In particular, as in Blackburn and Cipriani (2002), we can consider the following function in order to get closed-form solutions:

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

where $\mu > 0$ and $\lambda > 0$ represent, respectively, the weight that agents give to consumption and unhealthy activities, $\gamma > 0$ stands for their concern about future health capital, and $\phi \in [0, 1]$ represents the disutility of suffering from a NCD, which as a result of disease's morbidity and time loss because of treatment reduces utility driven from health capital.[1]

Consistently with the extensive medical literature on NCDs reviewed in Section 2 we assume that the probability of suffering from a NCD is a function of the inherited health capital, or in other words, the health capital at childhood (for an alternative economic modeling of early life conditions as determinants of health and economic outcomes see de la Croix and Licandro,

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[1] Notice that our setup also allows for two extreme cases: mortal disease ($\phi = 1$), and negligible morbidity ($\phi = 0$).

2007). Therefore $\pi_t = \pi(h_t)$, such that $\partial \pi(h_t)/\partial h_t < 0$, $\lim_{h_t \to 0} \pi(h_t) = \pi_H$ and $\lim_{h_t \to \infty} \pi(h_t) = \pi_L$, with $0 < \pi_L < \pi_H < 1$.

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

$$w_t = c_t + v_t + m_t. \tag{2}$$

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

$$h_{t+1} = (1 - \delta)h_t + \sigma m_t - \alpha v_t, \tag{3}$$

where $0 < \delta < 1$ and $\sigma, \alpha > 0$. In this expression, δ represents the depreciation rate of health capital, σ is the effectiveness of health investment, and α is the reduction of health conditions due to the unhealthy activities of individuals. Consistently with Grossman (1972, 2000), we assume that δ is not affected by illness. However, a disease induces utility loss through the parameter ϕ in equation (1).[2]

Equation (3) considers that agents may affect their health capital at the old age h_{t+1} through health investment and unhealthy activities. However this also means that individual choices modify their children's inherited health capital as well. As a result the intergenerational transmission of NCDs occurs through two different channels: A *direct* channel, since parents' choices have a direct impact on their children's inherited health capital (h_{t+1}), and an *indirect* channel, since parents' choices impact their children's probability of developing a NCD ($\pi(h_t)$).[3] Therefore, if individuals do not internalize these effects NCDs will spread to future generations. Thus, we model NCDs' epidemics based on a intergenerational transmission mechanism related to modifiable risk factors,

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[2]Notice that, in our model, h_{t+1} is the stock of health capital at the beginning of period t+1, and the expected utility of health capital when old is thus given by $(1 - \pi_t)\gamma \ln h_{t+1} + \pi_t \gamma (1 - \phi) \ln h_{t+1}$. An alternative modeling of the effect of the disease could consider a reduction of h_{t+1} in equation (3). However, this would imply that children also inherit the disease, which is not appropriate for NCDs because they are non-communicable diseases by definition.

[3]Evidence supporting these two assumptions has been reviewed in Section 2.

still acknowledging that NCDs are non-infectious diseases and, therefore, do not exhibit "biological" transmission among individuals.[4]

Finally, we also consider that agents may have a limited perception of the effect of the unhealthy choices on their own health and face a "perceived" law of motion of health capital

$$h_{t+1}^p = (1 - \delta)h_t + \sigma m_t - \epsilon \alpha v_t, \tag{4}$$

where $0 < \epsilon < 1$ represents agent's health information level. This assumption is consistent with evidence of poor nutritional knowledge not only of the population in general (Vereecken and Maes, 2010; Grimes et al., 2009; and Schwartz et al. 2005) but also of physicians (Flynn et al., 2003; and Makowske and Feinman, 2005). Additionally, there is evidence that greater nutritional knowledge is related to better nutrition (Kolodinsky et al., 2007; Pollard et al. 2010; and Vereecken and Maes, 2010). Still, when as is the case for NCDs the health-risk has mainly long-term effects, limited perception of health effects frequently arises (for a theoretical contribution see for instance Cremer et al., 2010; and for empirical evidence see for example Davison et al., 1991; Frankel et al., 1991; and Brownell et al., 2009; which relates sugar-sweetened beverages and obesity).

4 Decentralized solution

Let us first study the decentralized solution. Individuals choose consumption, unhealthy activities and health investments that maximize their utility subject to their budget constraint and the "perceived" law of motion of health capital. Knowing that, we can completely characterized the dynamics of the economy by taking the "true" law of motion of health capital (3).

4.1 Individual behaviour

Agents maximize $U_t(c_t, v_t, h_{t+1}^p, \pi_t)$ subject to (2), (4), $c_t > 0$, $v_t > 0$, $m_t > 0$ and $h_t > 0$, where w_t and h_t are taken as given. For a general utility

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[4]We have already pointed out in Section 2 that genetics is not the main factor explaining the epidemics of NCDs (see for instance Hill and Peters, 1998; Bouchard, 1999; and Sacerdote, 2007).

function, the corresponding first order conditions (FOCs) are summarized in the following expression:[5]

$$\frac{\partial U_t}{\partial v_t} = \frac{\partial U_t}{\partial c_t} + \epsilon\alpha\frac{\partial U_t}{\partial h_{t+1}^p}. \tag{5}$$

Considering the functional form (1), the optimal choices are

$$m_t = \frac{\sigma[\gamma(\sigma + \epsilon\gamma)(1 - \phi\pi_t) + \epsilon\lambda\alpha]w_t - (1 - \delta)[(\lambda + \mu)\sigma + \epsilon\mu\delta]h_t}{\sigma(\sigma + \epsilon\alpha)[\lambda + \mu + \gamma(1 - \phi\pi_t)]}, \tag{6}$$

$$v_t = \frac{\lambda[(1 - \delta)h_t + \sigma w_t]}{(\sigma + \epsilon\alpha)[\lambda + \mu + \gamma(1 - \phi\pi_t)]} \tag{7}$$

and

$$c_t = \frac{\mu[(1 - \delta)h_t + \sigma w_t]}{\sigma[\lambda + \mu + \gamma(1 - \phi\pi_t)]}. \tag{8}$$

From (6)-(8) we can observe that, all other things being equal, income (w_t) has a positive effect on consumption, unhealthy activities and health investment. Moreover, greater inherited health conditions (h_t) will increase consumption and unhealthy activities, but will 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, π_t has positive effect on consumption and unhealthy activities, but a negative one on health investment. Similarly, a greater disutility of NCD (ϕ) will reduce health investment, while consumption and unhealthy activities will rise. Finally, the more informed is an agent regarding the negative effect of her unhealthy behavior (ϵ) the higher the investment in health and the lower the consumption of unhealthy goods.[6]

4.1.1 Dynamics

The dynamics of our economy are completely characterized by the evolution of health capital, as given by the "true" law of motion. By substituting (6)-(8)

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[5]Since agents inherit their current health conditions, our results would not change if h_t is also introduced in the utility function.

[6]Notice that, due to the assumption of additive separable preferences (1), ϵ has not a direct effect on c_t. However, as we will see in Section 4.1.1, ϵ affects c_t along both the short and long-run equilibrium.

into (3), we get the corresponding transition function:

$$h_{t+1} = \frac{[\gamma(\sigma + \epsilon\alpha)(1 - \phi\pi(h_t)) - (1 - \epsilon)\lambda\alpha][(1 - \delta)h_t + \sigma w]}{(\sigma + \epsilon\alpha)[\lambda + \mu + \gamma(1 - \phi\pi(h_t))]} \equiv \varphi(h_t), \quad (9)$$

where income is assumed to be constant ($w_t = w$) for the sake of simplicity. Since we are interested in positive interior solutions, Proposition 1 establishes sufficient condition for $\varphi(h_t) > 0$.

Proposition 1 *If NCDs' morbidity is high enough ($\phi > \tilde{\phi}$, where $\tilde{\phi} = 1 - \lambda\alpha/\gamma\sigma$) agents should be sufficiently informed about the effect of the unhealthy activities ($\epsilon > \tilde{\epsilon}$, where $\tilde{\epsilon} = \frac{\lambda\alpha - (1-\phi)\gamma\sigma}{\lambda\alpha + (1-\phi)\gamma\alpha}$) and be sufficiently concerned about their future health conditions ($\gamma > \lambda\alpha/\sigma$) to ensure positive health capital at old age ($\varphi(h_t) > 0$). Otherwise, ($\phi < \tilde{\phi}$), health capital at old age is always positive ($\varphi(h_t) > 0$).*

Proof. From Equation (9) we find that $\varphi(h_t) > 0 \Leftrightarrow \pi_t < \tilde{\pi}$, where $\tilde{\pi} = \frac{1}{\phi}[1 - \frac{(1-\epsilon)\lambda\alpha}{\gamma(\sigma+\epsilon\alpha)}]$. We consider all possible probabilities of disease ($0 < \pi_t < 1$) and therefore restrict ourselves to the case $\tilde{\pi} > 1$. Consequently, $\tilde{\pi} > 1 \Rightarrow \varphi(h_t) > 0$. This sufficient condition can be rewritten as $\epsilon > \tilde{\epsilon}$, where $\tilde{\epsilon}$ is defined as in the proposition. Since ϵ is assumed to be strictly positive, it is easy to see from the definition of $\tilde{\epsilon}$ that the condition $\lambda\alpha - (1-\phi)\gamma\sigma < 0$ implies $\tilde{\epsilon} < 0 \Rightarrow \epsilon > \tilde{\epsilon} \Leftrightarrow \tilde{\pi} > 1 \Rightarrow \varphi(h_t) > 0$. Note also that $\lambda\alpha - (1-\phi)\gamma\sigma < 0 \Leftrightarrow \phi < \tilde{\phi}$, where $\tilde{\phi}$ is defined as in the proposition. Hence, if $\lambda\alpha - (1-\phi)\gamma\sigma < 0$, $\phi < \tilde{\phi}$ is a sufficient condition for $\varphi(h_t) > 0$. Otherwise, if $\lambda\alpha - (1-\phi)\gamma\sigma > 0$ ($\Leftrightarrow \phi > \tilde{\phi}$) the sufficient condition becomes $\epsilon > \tilde{\epsilon}$ (notice that $\gamma > \lambda\alpha/\sigma$ is needed to well define $\tilde{\phi}$) ∎

Proposition 1 ensures positive health capital when NCDs' morbidity is high enough. In this case individuals should be sufficiently informed about unhealthy activities health effects and be concerned about their future health conditions.

In this paper we focus on the steady-state equilibrium h^*, which is defined as a fixed point of the transition function, i.e., $h^* = \varphi(h^*)$.[7] One can easily verify that h^* is stable (unstable) iff $\varphi'(h^*) < 1(> 1)$. As in Azariadis (1996),

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[7]Mathematically, this corresponds to a stationary point of the equilibrium difference equation (9).

and Azariadis and Stachurski (2005), we can assume the following step function for the probability of disease in order to get further analytical results:

$$\pi(h_t) = \begin{cases} \pi_H & \text{if} \quad h_t < h^c \\ \pi_L & \text{if} \quad h_t \geq h^c, \end{cases} \tag{10}$$

where h^c is an exogenous health threshold. According to this functional form, if the health conditions of an individual are low (high) enough (h^c) the probability of suffering from a NCD will be high (low). The existence of health thresholds is well established in the medical literature. Indeed physicians make often use of thresholds to identify diseases and critical health conditions (see, for instance, Yuill and Miller, 2008: "cirrhosis in the liver may not result in a clinical effect until over 50% of the liver has been replaced by fibrous tissue"). Other well known examples are the thresholds for diabetes, blood pressure, obesity (BMI), etc.

Taking (10), the corresponding transition function is therefore composed by two branches given by

$$\varphi(h_t) = \begin{cases} \frac{[\gamma(\sigma+\epsilon\alpha)(1-\phi\pi_H)-(1-\epsilon)\lambda\alpha][(1-\delta)h_t+\sigma w]}{(\sigma+\epsilon\alpha)[\lambda+\mu+\gamma(1-\phi\pi_H)]} \equiv \varphi_{\pi_H}(h_t) & \text{if} \quad h_t < h^c \\ \frac{[\gamma(\sigma+\epsilon\alpha)(1-\phi\pi_L)-(1-\epsilon)\lambda\alpha][(1-\delta)h_t+\sigma w]}{(\sigma+\epsilon\alpha)[\lambda+\mu+\gamma(1-\phi\pi_L)]} \equiv \varphi_{\pi_L}(h_t) & \text{if} \quad h_t \geq h^c. \end{cases} \tag{11}$$

In this case, we can prove that the dynamics of the model admits two stable steady-states:

Proposition 2 *Assuming the functional form (10) and the conditions established in Proposition 1, there exist two stable steady-states $0 < h^*_{\pi_H} < h^c < h^*_{\pi_L}$, given by*
$$h^*_{\pi_i} = \frac{[\sigma\psi_{\pi_i} - (1-\epsilon)\lambda\alpha]w}{\gamma\delta\psi_{\pi_i} + \sigma(\lambda+\mu) + \alpha[\lambda(1-\delta) + \epsilon(\mu+\lambda\delta)]},$$
where $\psi_{\pi_i} = \gamma(\sigma+\epsilon\alpha)(1-\phi\pi_i)$ and $i = \{H, L\}$. (If instead $0 < h^*_{\pi_H} < h^*_{\pi_L} < h^c$, the only steady-state is $h^*_{\pi_H}$. Finally, if $0 < h^c < h^*_{\pi_H} < h^*_{\pi_L}$ the only steady-state is $h^*_{\pi_L}$).

Proof. It is easy to see that $0 < \partial\varphi(h_t)/\partial h_t < 1$ for all $h_t > 0$, under the same conditions of Proposition 1. Therefore, assuming the step function (10), there exist two stable steady-states $h^*_{\pi_i}$ for $i = \{H, L\}$ if $h^*_{\pi_H} < h^c < h^*_{\pi_L}$ (notice that $h^*_{\pi_H} < h^*_{\pi_L}$ since $\partial\varphi(h_t)/\partial\pi_t < 0$). The closed-forms of the steady-state values are provided by computing the fixed points of the transition function

(11). Additionally one can easily check that if the health capital threshold is high enough so that $0 < h^*_{\pi_H} < h^*_{\pi_L} < h^c$, the economy admits only a steady-state given by $h^*_{\pi_H}$. Otherwise, if the health capital threshold is low enough $0 < h^c < h^*_{\pi_H} < h^*_{\pi_L}$ the only steady-state is $h^*_{\pi_L}$ ∎

Since $\varphi_{\pi_i}(h_t)$ is monotonically increasing in h_t, we can see that $h^*_{\pi_i}$ is positively affected by the income (w), the effectiveness of health investment (σ), the individual concern about future health capital (γ), and agent's health information level (ϵ). However, a greater disability of NCDs (ϕ) will reduce the steady-state value of health capital.[8]
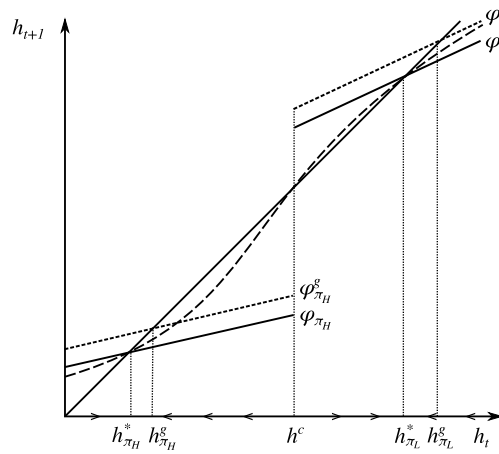

Figure 1: Dynamics

Figure 1 represents the two possible steady-states, assuming $0 < h^*_{\pi_H} < h^c < h^*_{\pi_L}$. The *high* (*low*) steady-state is associated with a high (low) level of health capital and a low (high) probability of suffering from a NCD. Indeed, if the initial health conditions are high (low) enough $(h_0 > (<)h^c)$, the probability of being struck by a NCD is low (high). Therefore, agents will give a higher (lower) value to their old age. Consequently, this will induce a higher (lower) investment in health and lower (higher) consumption and unhealthy activities, and the economy will end up in the high (low) steady-state.

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[8]Notice that if $\varphi(h_t)$ is concave and monotonically increasing in h_t, there is a unique stable steady-state h^*. In this case, we can also conclude from Equation (9) that h^* is positively affected by w, σ, γ, and ϵ. Moreover, the steady-state value is negatively affected by ϕ too.

Finally, we can point out that multiple steady states may support the existence of regional asymmetries in what concerns obesity and NCDs in general. Even restricting ourselves to Europe, we can identify meaningful differences 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). Blanchflower and Oswald (2008) have also found evidence of country asymmetries with respect to hypertension.

5   Welfare implications of NCDs

In this section we show that a major consequence of the social transmission mechanism of NCDs is that individual choices are socially non-optimal. We calculate the social optimum and compare it with the decentralized solution characterized in the previous section. In addition to this analysis, Section 5.2 considers the golden rule defined by Chichilnisky et al. (1995). This allocation may be considered as a constrained social optimum in which the planner maximizes the aggregate surplus at the steady state, ignoring thus the transition process. It has the advantage of allowing us to provide further analytical results. Finally, in Section 5.3, we consider the implications of relaxing the assumption that individuals cannot affect their own probability of disease, i.e., we assume $\pi(h_{t+1})$ instead of $\pi(h_t)$.

5.1   Full-fledge forward-looking planner

We characterize the social optimum by means of considering a full-fledge forward-looking planner, which maximizes a social welfare function $\beta^{-1}U_{-1} + \sum_{t=0}^{\infty} \beta^t U_t(c_t, v_t, h_{t+1}, \pi_t)$ subject to (2), (3), $c_t > 0$, $v_t > 0$, $m_t > 0$ and $h_t > 0$, where w and h_0 are taken as given, and $\beta \in (0, 1)$ represents the inter-temporal discount rate. The corresponding Lagrangian is

$$\mathcal{L} = \beta^{-1}U_{-1} + \sum_{t=0}^{\infty} \beta^t \left[U_t(c_t, v_t, h_{t+1}, \pi_t) + \iota_{t+1}\Omega_t \right], \tag{12}$$

where $\Omega_t = (1 - \delta)h_t + \sigma w - (\sigma + \alpha)v_t - \sigma c_t - h_{t+1}$ and $\iota_{t+1} > 0$ is the Lagrangian multiplier (shadow price of health capital). The corresponding

FOCs are summarized in

$$\frac{\partial U_t}{\partial v_t} = \frac{\partial U_t}{\partial c_t} + \alpha \left\{ \frac{\partial U_t}{\partial h_{t+1}} + \beta \left[\frac{\partial U_{t+1}}{\partial \pi_{t+1}} \frac{\partial \pi_{t+1}}{\partial h_{t+1}} + \iota_{t+2}(1-\delta) \right] \right\}. \qquad (13)$$

Comparing this expression with the FOC of the decentralized economy (5), we can conclude that the individual choices are socially non-optimal. Indeed, for c_t and $h_{t+1} = h_{t+1}^p$ given, agents choose too much unhealthy activities than what is socially optimal (notice that $\partial^2 U_t(\cdot)/\partial v_t^2 < 0$). Our model points out two sources of inefficiency. First, there is an intergenerational externality due to the social transmission of NCDs. Agents do not consider the *direct* effect of their individual choices on future health conditions, $\beta \iota_{t+2}(1-\delta)$. Moreover, they do not account for the *indirect* effect of their individual behaviour on the future generation through the probability of suffering from a NCD, $\beta \frac{\partial U_{t+1}}{\partial \pi_{t+1}} \frac{\partial \pi_{t+1}}{\partial h_{t+1}}$. Finally, a limited perception of the consequences of their unhealthy consumption (ϵ) will introduce an additional inefficiency. In fact, only if, simultaneously, there is no (social) transmission of NCDs ($\delta \to 1$ and $\frac{\partial \pi_{t+1}}{\partial h_{t+1}} = 0$) and agents have a high level of health information ($\epsilon \to 1$), (5) and (13) coincide and the decentralized solution is thus socially optimal. Still, notice that the social transmission of NCDs generates inefficiency even under the case of low misperception ($\epsilon \to 1$).

The previous result raises the question of how to implement the social optimum. In this paper we study the case of a tax on unhealthy activities. Real world examples encompass tobacco and alcohol taxation and the so called *fat tax*. A fat tax is a surcharge placed upon fattening foods and sugar-sweetened beverages with the aim of discouraging their consumption. Indeed the consumption of these products is considered a NCD modifiable risk factor since it contributes to an unhealthy diet and their increased consumption is associated with obesity epidemics (see for instance Vartanian et al., 2007; and Ludwig et al., 2001). Currently, fat taxes are under debate in several countries. In 2009, 33 states in the USA taxed soft drinks (Brownell et al., 2009). Moreover, France is considering plans to impose a fat tax on junk food (see IGF, 2008; Bonnet et al., 2009; and Allais et al., 2010). Non surprisedly, part of the food and beverage industry strongly criticizes this policy due to sales reduction (see, for instance, Brownell et al., 2009; and Vartanian et al., 2007). One of their main arguments considers that fat taxes are against individual freedom: contrary to tobacco and alcohol, the consumption of fattening food

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and soft drinks does not involve negative externalities as second hand smoke and drunk driving accidents (Rudd, 2009). Nevertheless, authors as Brownell et al. (2009) and Finkelstein et al. (2009) have identified external effects due to the rise of obesity-related medical expenditures. In this regard, our paper contributes to this literature by pointing out another external effect (an intergenerational externality) related to the social transmission of NCDs that may justify the usage of this kind of taxes. However, several drawbacks of fat taxes have been already identified in the litterature. A commonly-raised problem is the regressive nature of the policy (Allais et al., 2010). Nevertheless, several authors (see for instance, Brownell et al., 2009; Jacobson and Brownell, 2000; and Rudd, 2009) point out that such a problem is minimized if the revenues of the fat taxes are used in the benefit of the poor. In this direction, despite that redistribution concerns are beyond the scope of our paper (for an analytical dynamic setup of income distribution under epidemics, see Boucekkine and Laffargue, 2010), we model fat taxes where the corresponding revenues are used to finance healthy activities.

Let us consider the decentralized problem with a tax (τ_t) on unhealthy activities. We use the corresponding tax revenues to subsidize (s_t) the healthy activities (see for instance Cremer et al., 2010, for another theoretical contribution). Individuals maximize $U_t(c_t, v_t, h_{t+1}^p, \pi_t)$ subject to (4) and the modified budget constraint

$$w = c_t + (1 - s_t)m_t + (1 + \tau_t)v_t, \tag{14}$$

taking s_t and τ_t as given. Finally, at the equilibrium, $s_t m_t = \tau_t v_t$ for all $t \geq 0$. The corresponding FOC is

$$\frac{\partial U_t}{\partial v_t} = \frac{\partial U_t}{\partial c_t} + \left(\sigma \frac{\tau_t}{1 - s_t} + \epsilon \alpha \right) \frac{\partial U_t}{\partial h_{t+1}^p}, \tag{15}$$

Since at the social optimum $h_{t+1} = h_{t+1}^p$, we get the trajectory for the optimal policy by equating (13) and (15):

$$\frac{\tau_t}{1 - s_t} = \frac{\alpha}{\sigma} \left\{ (1 - \epsilon) + \beta \left[\frac{\frac{\partial U_{t+1}}{\partial \pi_{t+1}} \frac{\partial \pi_{t+1}}{\partial h_{t+1}} + (1 - \delta)\iota_{t+2}}{\frac{\partial U_t}{\partial h_{t+1}}} \right] \right\}. \tag{16}$$

As it is clear from the previous expression, this optimal policy considers the two sources of inefficiency described before, i.e., the intergenerational externality (direct and indirect effects) and the limited perception of the agents. Indeed,

as one can expect, in absence of transmission mechanism ($\delta \to 1$ and $\frac{\partial \pi_{t+1}}{\partial h_{t+1}} = 0$) and misperception ($\epsilon \to 1$) the optimal tax and subsidy vanish.[9]

5.2 Golden rule

Let us now consider the golden rule allocation defined in Chichilnisky et al. (1995). As in John and Peccenino (1994), this case may be interpreted as a particular social planner problem where we maximize the aggregate utility at the steady-state. The main advantage of this solution is that it allows us to provide further analytical results regarding social welfare.

The social planner maximizes $U(c, v, h, \pi)$ subject to (2) and (3) at the steady-state, $c > 0$, $v > 0$, $m > 0$ and $h > 0$. The corresponding FOC is provided by

$$\frac{\partial U}{\partial v} = \frac{\partial U}{\partial c} + \frac{\alpha}{\delta} \left(\frac{\partial U}{\partial h} + \frac{\partial U}{\partial \pi} \frac{\partial \pi}{\partial h} \right). \tag{17}$$

Similar to the case of full-fledge forward-looking planner, the FOC of the decentralized economy (5), at the steady-state, does not coincide with equation (17) because of the intergeneration transmission of NCDs and the misperception problem. As before, we can implement the golden rule by means of a tax (τ) on the unhealthy consumption and a subsidy (s) on the healthy activities. Taking $h_{t+1} = h_{t+1}^p$, we get the corresponding policy by equating (15) at the steady state with (17):

$$\frac{\tau}{1-s} = \frac{\alpha}{\sigma} \left[\left(\frac{1}{\delta} - \epsilon \right) + \frac{1}{\delta} \frac{\frac{\partial U}{\partial \pi} \frac{\partial \pi}{\partial h}}{\frac{\partial U}{\partial h}} \right]. \tag{18}$$

As it is clear from (17) and (18), under a high level of health information ($\epsilon \to 1$) and no intergenerational transmission ($\delta \to 1$ and $\frac{\partial \pi}{\partial h} = 0$) both FOCs coincide and, therefore, tax and subsidy become zero (notice that, at the equilibrium, $sm = \tau v$).

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[9]Notice that the optimal solution can be also decentralized by means of a tax on unhealthy activities and a lump-sum transfer T_t of the corresponding tax revenues to the agents (i.e., the modified budget constraint would be $w + T_t = c_t + m_t + (1 + \tau_t) v_t$ and, at the equilibrium, $T_t = \tau_t v_t$). In our model, this case is equivalent to consider $s_t = 0$.

5.2.1 Golden rule in our setting

Let us consider the functional forms (1) and (10). Taking (2) and (3) at the steady-state, and the equation (17), the golden rule values for health capital and unhealthy activities are respectively:

$$h_{\pi_i}^g = \frac{\gamma\sigma(1-\phi\pi_i)w}{\delta[(\lambda+\mu)+\gamma(1-\phi\pi_i)]} \tag{19}$$

and

$$v_{\pi_i}^g = \frac{\lambda\sigma w}{(\sigma+\alpha)[(\lambda+\mu)+\gamma(1-\phi\pi_i)]}, \tag{20}$$

for $i = \{H, L\}$.[10] As it is clear from Proposition 2, the individual choices are different than the golden rule allocation. Indeed, we can claim that due to the transmission mechanism of NCDs and the misperception problem agents will choose too many unhealthy activities and too little health capital:

Proposition 3 *At the steady-state, under Proposition 2, individuals choose higher quantity of unhealthy activities and lower health capital than the golden rule allocation.*

Proof. Taking (7) at the steady state and (20), it is easy to see that $v_{\pi_i}^* > v_{\pi_i}^g$ since $\epsilon < 1$ and, from Proposition 2, $h_{\pi_i}^* > 0$, for $i = \{H, L\}$. Let us now prove that $h_{\pi_i}^* < h_{\pi_i}^g$, for $i = \{H, L\}$. To do that it is sufficient to prove that $\varphi_{\pi_i}(h_t) < \varphi_{\pi_i}^g(h_t)$, for all $h_t > 0$, where $\varphi_{\pi_i}^g(h_t) = (1-\delta)h_t + \sigma m_{\pi_i}^g - \alpha v_{\pi_i}^g$ (the corresponding π_i-branch for the golden rule problem). From (2) and (3) in the golden rule problem, it is easy to get $m_{\pi_i}^g = \frac{[\lambda\alpha+\gamma(\sigma+\alpha)(1-\phi\pi_i)]w}{(\sigma+\alpha)[(\lambda+\mu)+\gamma(1-\phi\pi_i)]} > 0$. Therefore, taking (20), $\varphi_{\pi_i}^g(h_t) = \frac{[(\lambda+\mu)+\gamma(1-\phi\pi_i)](1-\delta)h_t+\gamma\sigma(1-\phi\pi_i)w}{(\lambda+\mu)+\gamma(1-\phi\pi_i)}$. Rearranging terms in (11) we get $\varphi_{\pi_i}(h_t) = \frac{[\gamma(1-\phi\pi_i)-(\frac{1-\epsilon}{\sigma+\epsilon\alpha})\lambda\alpha][(1-\delta)h_t+\sigma w]}{(\lambda+\mu)+\gamma(1-\phi\pi_i)}$. Finally, by comparing these two expressions, it is easy to conclude that $\varphi_{\pi_i}(h_t) < \varphi_{\pi_i}^g(h_t)$, for all $h_t > 0$ ∎

Furthermore, from equation (2), we can also conclude that agents choose too much consumption ($c_{\pi_i}^* > c_{\pi_i}^g$) and too little health investment ($m_{\pi_i}^* <$

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[10]Without lost of generality, this paper focuses on the case of a golden rule allocation associated to each steady-state established in Proposition 2. As it is clear from Figure 1, multiplicity arises if $h_{\pi_H}^g < h^c < h_{\pi_L}^g$ (notice that $h_{\pi_H}^g < h_{\pi_L}^g$ since $\partial h_{\pi_i}^g/\partial\pi_i < 0$). However, if $h^c < h_{\pi_H}^g$, the golden rule allocation is unique and given by $h^g = h_{\pi_L}^g$. In this case, it is easy to see that the results provided in this section remain the same.

$m_{\pi_i}^g$) at the steady-state. From (18) and assuming the step function (10), the corresponding policy to implement the golden rule is now given by

$$\frac{\tau_{\pi_i}}{1 - s_{\pi_i}} = \frac{\alpha}{\sigma}\left(\frac{1}{\delta} - \epsilon\right). \tag{21}$$

Moreover, we can establish the following proposition:

Proposition 4 *In our setting, the golden rule allocation can be decentralized by means of a tax τ_{π_i} on the unhealthy consumption, and a subsidy s_{π_i} on the healthy activities, which is financed by the τ_{π_i} revenues. The corresponding closed forms are given by*

$$\tau_{\pi_i} = \frac{\tilde{\tau}}{1 + \frac{v_{\pi_i}^g}{m_{\pi_i}^g}\tilde{\tau}} \tag{22}$$

and

$$s_{\pi_i} = \tau_{\pi_i}\frac{v_{\pi_i}^g}{m_{\pi_i}^g}, \tag{23}$$

where $\tilde{\tau} = \frac{\alpha}{\sigma}\left(\frac{1}{\delta} - \epsilon\right)$ and $\frac{v_{\pi_i}^g}{m_{\pi_i}^g} = \frac{\lambda\sigma}{\lambda\alpha + \gamma(1 - \phi\pi_i)(\sigma + \alpha)}$.

Proof. From (21) we know that $\frac{\tau_{\pi_i}}{1 - s_{\pi_i}}$ decentralizes the golden rule allocation. Since $s_{\pi_i}m_{\pi_i}^g = \tau_{\pi_i}v_{\pi_i}^g$, we get (22) and (23). Finally, from (20) and the formula of $m_{\pi_i}^g$ (see the proof of Proposition 3) we obtain the expression for $\frac{v_{\pi_i}^g}{m_{\pi_i}^g}$ ∎

Finally, from this proposition we can also conclude that the greater the misperception problem (i.e, the lower ϵ) the greater should be the tax and the subsidy. This result points out the importance of considering agent's health information level to study the economic impact of this kind of policies. In this regard, Allais et al. (2010) have predicted little effect of fat taxes on French consumers. However, they also recognise that their study does not include the effect of informational programs. Indeed, Pollard et al. (2009) concluded about the substantial effectiveness of the "Go for 2&5" (2 fruits and 5 vegetables a day) campaign in Australia. Moreover, Bonnet et. al (2009), using the same data base as Allais et al. (2010), showed that the estimated price elasticities of individual consumption are significant and may justify a tax on high density and cheap energy categories of food such as junk food as effective policy to reduce obesity and overweight.[11] For further empirical results in the same direction see, for instance, Mytton et al. (2007) and Epstein et al. (2007).

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[11]Among other things, they found that a 10% increase of junk food prices together with a 10% reduction of fruits and vegetables prices would induce a reduction of the proportion of

5.3 Further analytical results

As we have observed in Section 1, our set-up considers $\pi_t = \pi(h_t)$. Still, it may be reasonably argued that individuals also affect their own probability of disease. For this reason, let us now study the case $\pi_t = \pi(h_{t+1})$: agents may modify their own probability of suffering from a NCD, which is determined just before arriving to the old-age. The FOC corresponding to the decentralized solution is given by

$$\frac{\partial U_t}{\partial v_t} = \frac{\partial U_t}{\partial c_t} + \epsilon\alpha\left(\frac{\partial U_t}{\partial h_{t+1}^p} + \frac{\partial U_t}{\partial \pi_t}\frac{\partial \pi_t}{\partial h_{t+1}^p}\right). \tag{24}$$

Comparing this expression with (5), one can observe that agents consider the effect of the individual choices on the probability of suffering from a NCD because they can modify their own π_t. Let us now study the social optimum. The FOC corresponding to the full-fledge social planner is given by

$$\frac{\partial U_t}{\partial v_t} = \frac{\partial U_t}{\partial c_t} + \alpha\left[\frac{\partial U_t}{\partial h_{t+1}} + \frac{\partial U_t}{\partial \pi_t}\frac{\partial \pi_t}{\partial h_{t+1}} + \beta\xi_{t+2}(1-\delta)\right], \tag{25}$$

where $\xi_{t+2} > 0$ is the corresponding Lagrangian multiplier. From (24) and (25), taking $h_{t+1} = h_{t+1}^p$, it is easy to see that part of the effect of the social transmission of NCDs is internalized. Indeed, the *indirect* effect of the individual behaviour on the probability of suffering from a NCD partially vanishes if agents can modify π_t. However, agents completely neglect the *direct* effect on the future generations, $\beta\xi_{t+2}(1-\delta)$. Moreover, as before, the misperception problem (ϵ) adds an additional source of inefficiency.

Similar to the previous case, we can decentralized the social planner solution by means of a tax (τ_t) on the unhealthy activities, that we use to subsidize (s_t) the healthy activities. The individuals' FOC is now given by

$$\frac{\partial U_t}{\partial v_t} = \frac{\partial U_t}{\partial c_t} + \left(\sigma\frac{\tau_t}{1-s_t} + \epsilon\alpha\right)\left(\frac{\partial U_t}{\partial h_{t+1}^p} + \frac{\partial U_t}{\partial \pi_t}\frac{\partial \pi_t}{\partial h_{t+1}^p}\right), \tag{26}$$

and, taking $h_{t+1} = h_{t+1}^p$, the optimal policy is provided by equating (25) and (26):

$$\frac{\tau_t}{1-s_t} = \frac{\alpha}{\sigma}\left[(1-\epsilon) + \beta(1-\delta)\left(\frac{\xi_{t+2}}{\frac{\partial U_t}{\partial h_{t+1}} + \frac{\partial U_t}{\partial \pi_t}\frac{\partial \pi_t}{\partial h_{t+1}}}\right)\right]. \tag{27}$$

overweight (children: -33:64%; adult males: -8.78%; and adult females: -11.65%) and obese (children: -30.88%; adult males: -11.13%; and adult females: -20.61%).

Let us compare this expression with Equation (16). In the case of $\pi_t = \pi(h_t)$, the optimal policy includes, in addition to the misperception problem, both the *direct* and *indirect* effect of the individual choices on future generations' health. However, if agents can modify their own probability of suffering from a NCD, we only need to correct the *direct* effect and ϵ. Moreover, from (27) it is clear that $\frac{\partial U_t}{\partial \pi_t} \frac{\partial \pi_t}{\partial h_{t+1}}$ allows us to reduce the optimal policy (for instance, a lower fat tax in the case of a lump-sum transfer). Finally, under a high level of health information ($\epsilon \to 1$), (24) and (25) coincide under absence of transmission mechanism of NCDs ($\delta \to 1$). Therefore, the corresponding tax and subsidy vanish.[12]

6   Concluding remarks

We have developed a model that rationalizes the "social" transmission mechanism behind NCDs' epidemics: the transmission of modifiable risk factors results in a intergenerational externality. Such externality arises through two different effects: a direct effect because parents affect the inherited health capital of their children, and an indirect effect because they affect the probability of their children suffering from a chronic disease when old. Parents are not fully altruistic and when they make their choices they do not entirely account for the impact of their choices on their children's health. Consequently, the negative externality results in a lower health capital level and a higher level of unhealthy activities at the descentralized equilibrium, which contrasted to the optimal choice. Taxes on unhealthy activities, where the corresponding revenues are used to subsidized health investments (Cremer et al., 2010), can be used to restore socially optimal health capital and unhealthy activities' levels. Only when the two effects are neutralized is the externality absent. Still, even in the absence of externality there is room for public intervention if individuals

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[12]Notice that, when $\pi_t = \pi(h_t)$, we have assumed a step function (10) in order to provide closed-form solutions (see sections 4.1.1 and 5.2.1). Indeed, this functional form may be considered as a discretized version of a convex-concave $\pi(h_t)$ (in Figure 1, the convex-concave broken line corresponds to a convex-concave $\psi(h_t)$). However, a step-simplification is not possible if $\pi_t = \pi(h_{t+1})$: the function must be differentiable in its whole domain. In this case, we may directly consider a convex-concave function like $\pi(h_t) = (\pi_L - \pi_H)\frac{h_{t+1}^2}{1+h_{t+1}^2} + \pi_H$. Nevertheless, the analysis would be restricted to implicit functions and numerical results.

are not well informed about unhealthy activities health effects. Finally, we also show that our model is consistent with the existence of international/regional asymmetries regarding the prevalence of obesity and NCDs.

Several remarks can be made with regard to our modelization. Since our paper focuses on the intergenerational transmission of NCDs, we have considered a simple set-up that incorporates a social transmission mechanism of the disease. However, we could extend our framework by adding other effects behind epidemics. Among them, would be an endogenous income effect. Indeed, Boucekkine et al. (2009) have already studied the interaction between epidemics and income within the context of communicable diseases such as HIV/AIDS and malaria. In particular, they empirically found a significant effect of this kind of epidemics on educational choices and wages. Following de la Croix and Doepke (2003), one could endogenize income in our set-up as being a function of human capital $w(\tilde{h}_t)$: NCDs would affect income through agent's educational choices. Finally, another simplification of our framework is the absence of savings: since in our model health is already an inter-temporal choice variable we did not include physical capital (savings) for simplicity. Therefore, one could incorporate savings as a technical extension of our paper. In this regard the approach introduced by Mariani et al. (2010) might provide a fruitful possibility.

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