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INTERGENERATIONAL TRANSMISSION OF NON COMMUNICABLE CHRONIC DISEASES

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Abstract

We introduce a theoretical framework that contributes to the understanding of non-communicable chronic diseases' (NCDs) epidemics: even if NCDs are non-infectious diseases, they may spread due to the social 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, 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.[1] Moreover, besides death, NCDs also lead to substantial disability. Examples of NCDs are cardiovascular diseases (mainly heart disease and stroke), cancers, respiratory diseases, diabetes, and musculoskeletal disorders like osteoarthritis.

In this paper, we provide an economic modeling 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.[2] 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 en-

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[1]The World Health Organization (WHO) points out that, even if NCDs have been commonly associated with the elderly of wealthy countries being responsible for 87% of deaths in these 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.

[2]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).

vironment. 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). Our analysis does not consider genetics nor population aging, but focus instead on the contribution of modifiable risk factors to the NCDs epidemics. In reality, while there is a common agreement regarding the genetic propensity to develop some NCDs, the recent increase in NCDs prevalence—as for example the double of obesity in the US (Cutler et al., 2003) and its increase by threefold in many European countries (WHO, 2009) in the last 30 years—cannot be supported by a similar genetic change (Hill and Peters, 1998). In turn, it is estimated that 80% of the premature deaths due to heart disease, stroke and diabetes can be avoided with appropriate behavior regarding modifiable risk factors and pharmaceuticals (Daar et al., 2007). The novelty of our approach is to give emphasis to the role of modifiable risk factors in preventing NCDs and to consider its intergenerational transmission, which contributes to explain the epidemic dimension of NCDs.

In particular, 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 are 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, secondhand smoking, 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 one assumes that children inherit their parents' health capital that is affected by their parents' choices of consumption

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of unhealthy and prevention goods. The second considers that a agent's probability of suffering from a NCD when old depends on her adulthood health capital, which is affected by both her own choices and the inherited health capital. 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 analyze how policy instruments such as a tax on the unhealthy goods, 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 (informational programs) on the level of health capital, NCDs prevalence rates, and how it affects the optimal policy. From a public policy perspective and given the health and socioeconomic costs associated to NCDs, it is important to understand the mechanisms behind its wide and fast spread. We highlight how the social intergenerational transmission mechanism of the modifiable risk factors can lead to the spread of NCDs and how policy instruments can be used to limit the prevalence of these diseases.

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 welfare implications of NCDs and, in Section 5, we provide further analytical results by focusing on the long term effects of early life conditions. Finally, Section 6 concludes.

2   Evidence on NCDs' social transmission

By definition, NCDs are non-infectious and, as already argued above, the genetic component cannot be responsible for NCDs' prevalence increase. Consequently, the NCDs epidemics is mainly due to population aging and to the social transmission of unhealthy behaviors regarding modifiable risk factors. Social transmission can either occur within peers, family ties and, in particular, between parents and children, and take, for instance, the form of imitation behavior or peer effects. Additionally, it can be affected by socioeconomic factors such as wealth, education, race, age as well as sex or others.

We abstract from any biological determinant of NCDs and focus exclusively on one of the possible forms of social transmission of behaviors regarding modifiable risk factors. In point of fact, we analyze the intergenerational trans-

mission occurring between parents and children concentrating on the effect of parents' decision upon their offsprings' childhood consumption of modifiable risk factors such as unhealthy diet, physical inactivity or secondhand smoking. We therefore do not consider peer effects nor imitation, nor add to the problem any socioeconomic factors.

Additionally, our economic modeling of NCDs' social transmission is based on two main assumptions. First, since modifiable risk factors have a negative impact on health capital, parents' choices regarding these affect their children's health capital. Second, the accumulation of health capital decreases the probability of suffering from NCDs at old age and, therefore, parents' choices regarding modifiable risk factors also affect their children's probability of NCDs. In this section we review the literature that provides evidence on these assumptions.

First, 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; and 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. Finally, using data of individuals exposed to the Chinese famine of 1959-61, Chen and Zhou (2007) and Meng and Qian (2006) conclude that early life malnutrition decreases adult height. 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, the recent 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%).

Second, 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 Lindeboom et al (2010) 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 $U_t(c_t, v_t, h_{t+1}, \pi_t)$. Agents care about consumption c_t and unhealthy activities v_t, which encompass the modifiable risk factors. 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. Moreover, we consider that $U_t(\cdot)$ is a strictly increasing function of c_t, v_t, and h_{t+1}, but decreasing in π_t.[3] In particular, as in Blackburn and Cipriani (2002), we can consider the following function in order to get closed-form solutions:

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

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[3]We also assume that $\partial^2 U_t(\cdot)/\partial c_t^2, \partial^2 U_t(\cdot)/\partial v_t^2, \partial^2 U_t(\cdot)/\partial h_{t+1}^2 < 0$, $\lim_{c_t \to 0} \partial U_t(\cdot)/\partial c_t$, $\lim_{v_t \to 0} \partial U_t(\cdot)/\partial v_t$, $\lim_{h_{t+1} \to 0} \partial U_t(\cdot)/\partial h_{t+1} = +\infty$ (see, for instance, Acemoglu, 2009).

where $\mu, \lambda > 0$ 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.[4]

Consistently with the extensive medical literature on NCDs reviewed in Section 2, we assume that the probability of suffering from a NCD is a decreasing function of agent's adulthood health capital, i.e., $\pi_t = \pi(h_{t+1})$, such that $\partial\pi(h_{t+1})/\partial h_{t+1} < 0$, $\lim_{h_{t+1}\to 0} \pi(h_{t+1}) = \pi_H$ and $\lim_{h_{t+1}\to\infty} \pi(h_{t+1}) = \pi_L$, 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 (1).[5]

Equation (3) considers that health capital at the old age h_{t+1} is a function of the inherited health capital h_t. However, agents may modify their health capital through health investments and unhealthy activities during adulthood. But 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* effect, since parents' choices have a direct impact on their children's inherited health capital (h_{t+1}), and an *indirect* effect, since parents' choices also affect their children's probability of

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

[5]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 (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.

developing a NCD at the old age ($\pi(h_{t+2})$).[6] Therefore, if individuals do not internalize these effects NCDs will spread to future generations. Thus, still acknowledging that NCDs are non-infectious diseases, we model NCDs' epidemics based on a intergenerational transmission mechanism related to modifiable risk factors.

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 of NCDs, health-risks have mainly long-term consequences, limited perception of health effects frequently arises.[7]

4   Welfare implications of NCDs

In this section we show that a major consequence of the social transmission of NCDs is that individual choices are socially non-optimal. We establish this result for a general utility function $U_t(.)$, under the conditions introduced in Section 3. In addition, Section 4.3 considers the golden rule problem, which allows us to provide further analytical results.

4.1   Decentralized solution vs. social optimum

Let us first study the decentralized solution. Individuals choose consumption, unhealthy activities and health investments that maximize their utility $U_t(c_t, v_t, h_{t+1}^p, \pi_t)$ subject to their budget constraint (2), the "perceived" law of motion of health capital (4), and $c_t, v_t, m_t, h_t > 0$, where w_t and h_t are taken as given. For a general utility function, the corresponding first order conditions

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

[7]For a theoretical contribution in this regard see, for instance, Cremer et al. (2010). For the empirical evidence see for example Davison et al. (1991), Frankel et al. (1991), and Brownell et al. (2009).

(FOCs) are summarized in the following Euler equation:[8]

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

Let us now characterize the social optimum by means of considering a full-fledge forward-looking planner, which maximizes the 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), and $c_t, v_t, m_t, h_t > 0$, where w_t and h_0 (initial condition) are taken as given, and $\beta \in (0,1)$ represents the inter-temporal discount rate. For this problem the Lagrangian is provided by

$$\mathcal{L} = \beta^{-1}U_{-1} + \sum_{t=0}^{\infty} \beta^t \left[U_t (c_t, v_t, h_{t+1}, \pi_t) + \xi_{t+1}\Omega_t \right], \tag{6}$$

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

$$\frac{\partial U_t}{\partial v_t} = \frac{\partial U_t}{\partial c_t} + \alpha \left[\frac{\partial U_t}{\partial h_{t+1}} + \frac{\partial U_t}{\partial \pi_t} \frac{\partial \pi_t}{\partial h_{t+1}} + \beta\xi_{t+2}(1-\delta) \right]. \tag{7}$$

Comparing this expression with (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. Actually, agents do not consider the *direct* effect of their individual choices on future health conditions, $\alpha\beta\xi_{t+2}(1-\delta)$. Second, because of the limited perception of the consequences of their unhealthy consumption (ϵ), they do not completely account for the *indirect* effect of their individual behaviour on the future generation through the probability of suffering from a NCD, $\alpha\frac{\partial U_t}{\partial \pi_t}\frac{\partial \pi_t}{\partial h_{t+1}}$. In fact, if there is no (social) transmission of NCDs ($\delta \to 1$) and agents have a high level of health information ($\epsilon \to 1$), (5) and (7) coincide and the decentralized solution is thus socially optimal. Moreover, in the absence of misperception ($\epsilon \to 1$) the decentralized solution is still non-optimal, thus justifying public intervention grounded on the intergenerational transmission of NCDs and not just because the policy maker is better informed than individuals are.

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[8]Notice that knowing the optimal choices, the dynamics of the economy are completely characterized by taking the "true" law of motion of health capital (3). Moreover, since agents inherit their current health conditions, our results would not change if h_t is also introduced in the utility function.

4.2 Implementing the social optimum

A natural question to raise is how to implement the social optimum. In this paper we study the case of a tax on unhealthy activities. Real world examples encompass the typical tobacco and alcohol taxation, and the much debated *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 being discussed in several countries.[9] Not surprisingly, 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 and soft drinks does not involve negative externalities as secondhand smoking and drunk driving accidents (Rudd, 2009). Nevertheless, authors as Brownell et al. (2009) and Finkelstein et al. (2009) identify 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, we assume that the corresponding tax revenues are used to finance healthy activities.[10]

Let us consider the decentralized problem with a tax (τ_t) on the 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

$$w = c_t + (1 - s_t)m_t + (1 + \tau_t)v_t, \tag{8}$$

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[9]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).

[10]For an analytical dynamic setup of income distribution under epidemics, see Boucekkine and Laffargue (2010).

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

$$\frac{\partial U_t}{\partial v_t} = \frac{\partial U_t}{\partial c_t} + \left(\sigma \frac{\tau_t}{1 - s_t} + \epsilon\alpha \right) \left(\frac{\partial U_t}{\partial h_{t+1}^p} + \frac{\partial U_t}{\partial \pi_t} \frac{\partial \pi_t}{\partial h_{t+1}^p} \right). \tag{9}$$

Since at the social optimum $h_{t+1} = h_{t+1}^p$, we get the trajectory for the optimal policy by equating (7) and (9):

$$\frac{\tau_t}{1 - s_t} = \frac{\alpha}{\sigma} \left[(1 - \epsilon) + \beta(1 - \delta) \left(\frac{\xi_{t+2}}{\frac{\partial U_t}{\partial h_{t+1}} + \frac{\partial U_t}{\partial \pi_t} \frac{\partial \pi_t}{\partial h_{t+1}}} \right) \right]. \tag{10}$$

Clearly this expression shows that the optimal policy takes into account the two sources of inefficiency described before, i.e., the intergenerational externality and the limited perception of the agents. Indeed, as one can expect, in the absence of transmission mechanism ($\delta \to 1$) and misperception ($\epsilon \to 1$) the optimal tax and subsidy vanish.[11]

4.3 Golden rule

Let us now consider the golden rule defined in Chichilnisky et al. (1995). As in John and Peccenino (1994), 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. 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, and $c, v, m, h > 0$. The corresponding FOC is provided by

$$\frac{\partial U}{\partial v} = \frac{\partial U}{\partial c} + \frac{\alpha}{\delta} \left(\frac{\partial U}{\partial h} + \frac{\partial U}{\partial \pi} \frac{\partial \pi}{\partial h} \right). \tag{11}$$

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 (11) 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 (9) at the

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[11]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 $s_t = 0$.

steady state with (11):

$$\hat{\tau} \equiv \frac{\tau}{1-s} = \frac{\alpha}{\sigma}\left(\frac{1}{\delta} - \epsilon\right).$$

(12)

As it is clear from (11) and (12), under a high level of health information ($\epsilon \to 1$) and no intergenerational transmission ($\delta \to 1$) both FOCs coincide and, therefore, tax and subsidy become zero (notice that, at the equilibrium, $sm = \tau v$). Moreover, in contrast to (10), now we can see that the greater the misperception and the intergenerational transmission (i.e., the lower ϵ and δ, respectively) the higher $\hat{\tau}$ ($\partial\hat{\tau}/\partial\epsilon, \partial\hat{\tau}/\partial\delta < 0$).[12]

5   NCDs and early life conditions

Up to now we have assumed that parents affect their children's probability to develop NCDs through their impact on the inherited health capital. However, individuals could counterbalance this effect by investing in health m_t and decreasing unhealthy activities v_t, with respectively positive and negative impacts on the accumulation of health capital and, consequently, lower own probability of NCDs. To give emphasis to the fact that parents choices affect their children's probability of developing NCDs we now analyze the extreme case in which individuals cannot affect their own probability and therefore consider $\pi_t = \pi(h_t)$. Moreover, besides being consistent with the literature revised below, this case has as well the advantage of improving tractability from an analytical point of view leading to further and more intuitive results.

Many studies have found a positive correlation between small birth weight and risk of coronary heart diseases in later life.[13] Small birth weight can nevertheless be associated to genetics and other socioeconomic determinants affecting as well morbidity at older ages. To circumvent this issue Ravelli et al. (1998) and Rosebom et al. (2001) analyze data on individuals exposed to the 1944-45 Dutch famine in early life, considered "a natural experiment" and therefore uncorrelated with other individual characteristics. They conclude that there is a casual effect of early life conditions on the propensity to develop NCDs at older ages. In particular, individuals exposed to the 1944-45 Dutch famine in early life have reduced glucose tolerance and, additionally, Rosebom et al. (2001) find that these individuals have higher BMI, higher risk

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[12]For the case of a lump-sum transfer, $\hat{\tau} = \tau$ (see Footnote 11) and, therefore, the optimal tax will increase with misperception and intergenerational transmission.

[13]See for instance Lithell et al. (1996), Stein et al. (1996), Rich-Edwards et al. (1997), Forsen et al. (1999), Huxley et al. (2000), and Osmond and Backer (2000) and the references therein.

of coronary heart diseases and high blood pressure later in life. Also, Lindeboom et al (2010) find evidence that early life exposure to the Dutch 1846-47 famine results in lower survival rates at old ages for men. The effect that parents affect children's propensity to develop NCDs at old age is therefore well documented in the literature, at least for early life.[14]

5.1 Individual behaviour

Let us consider the decentralized problem of Section 4.1 with $\pi_t = \pi(h_t)$.[15] In this case, the Euler equation is provided by

$$\frac{\partial U_t}{\partial v_t} = \frac{\partial U_t}{\partial c_t} + \epsilon\alpha\frac{\partial U_t}{\partial h_{t+1}^p}. \tag{13}$$

Comparing this expression with (5) one can observe that agents do not consider now the *indirect* effect of individual choices on the probability of suffering a NCD because π_t is just affected by early life conditions. Indeed, for c_t and h_{t+1}^p given, individuals choose more unhealthy activities than in the previous case. Moreover, since $\pi_t = \pi(h_t)$ and individuals take h_t as given we can provide the optimal choices' closed-forms for the utility function (1):

$$m_t = \frac{\sigma[\gamma(\sigma + \epsilon\gamma)(1 - \phi\pi_t) + \epsilon\lambda\alpha]w_t - (1 - \delta)[(\lambda + \mu)\sigma + \epsilon\mu\delta]h_t}{\sigma(\sigma + \epsilon\alpha)[\lambda + \mu + \gamma(1 - \phi\pi_t)]}, \tag{14}$$

$$v_t = \frac{\lambda[(1 - \delta)h_t + \sigma w_t]}{(\sigma + \epsilon\alpha)[\lambda + \mu + \gamma(1 - \phi\pi_t)]} \tag{15}$$

and

$$c_t = \frac{\mu[(1 - \delta)h_t + \sigma w_t]}{\sigma[\lambda + \mu + \gamma(1 - \phi\pi_t)]}. \tag{16}$$

As in Blackburn and Cipriani (2002), before completing the equilibrium examination (see Section 5.2), a comparative statics analysis allows us to provide an interpretation of the individual optimal choices.[16] From (14)-(16) we can observe that, all other things being equal: first, income (w_t) has a positive effect on consumption, unhealthy activities and health investment. This is an expected result due to the preferences considered in this paper (see Section 3)

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[14]Obviously, that appropriate data allowing to analyze the effect of parents' on full-length childhood conditions and its subsequent impact on the occurrence of NCD later in life is even more scarce, or non existent, what may explain the absence, to our knowledge, of such studies.

[15]As in Section 3, we also assume 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 $0 < \pi_L < \pi_H < 1$.

[16]Notice that π_t is endogenously determined at the equilibrium. Actually, the initial condition $h_0 > 0$ (parameter of the model) allows us to determine m_0, v_0, and c_0. Taking the transition function (17) and h_t, we get h_{t+1} and, thus, m_{t+1}, v_{t+1}, and c_{t+1}.

and the absence of mechanisms such as educational choices.[17] Second, for a given probability of NCDs, greater inherited health conditions (h_t) will increase consumption and unhealthy activities, but will decrease health investment: if inherited health conditions improve, for a given π_t, investment in health capital is less needed. Third, the greater the probability probability of suffering from a NCD the lower the value of old age (this is equivalent to a reduction of the discount rate). Therefore, π_t has positive effect on consumption and unhealthy activities, but a negative one on health investment (h_{t+1} affects the utility at the old age). Fourth, similarly, a greater disutility of NCD (ϕ) or a lower concern about future health capital (γ) 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.[18]

5.2 Equilibrium

The dynamics of our economy are completely characterized by the evolution of health capital, as given by the "true" law of motion. By substituting (14)-(16) into (3), we get the corresponding transition function:

$$h_{t+1} = \frac{[\gamma(\sigma + \epsilon\alpha)(1 - \phi\pi(h_t)) - (1 - \epsilon)\lambda\alpha][(1 - \delta)h_t + \sigma w]}{(\sigma + \epsilon\alpha)[\lambda + \mu + \gamma(1 - \phi\pi(h_t))]} \equiv \varphi(h_t), \quad (17)$$

where income is assumed to be constant ($w_t = w$) for the sake of simplicity. Since we are interested in positive interior solutions, let us first establish a sufficient condition for $h_{t+1} > 0$:

Proposition 1 *If NCDs' morbidity is low enough ($\phi < \tilde{\phi}$, where $\tilde{\phi} = 1 - \lambda\alpha/\gamma\sigma$) then $h_{t+1} > 0$. However, if NCDs' morbidity is high ($\phi > \tilde{\phi}$) 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 enough concerned about their future health conditions ($\gamma > \lambda\alpha/\sigma$) to ensure positive health capital at old age.*

Proof. From (17) we find that $\varphi(h_t) > 0$ iff $\pi_t < \tilde{\pi}$, where $\tilde{\pi} = \frac{1}{\phi}[1 - \frac{(1-\epsilon)\lambda\alpha}{\gamma(\sigma+\epsilon\alpha)}]$. Consequently, $\tilde{\pi} > 1 \Rightarrow h_{t+1} > 0$ because $0 < \pi_t < 1$. Therefore, let us consider the case $\tilde{\pi} > 1$. This sufficient condition can be rewritten as $\epsilon > \tilde{\epsilon}$, where $\tilde{\epsilon}$ is defined as in Proposition 1. Since $\epsilon > 0$, it is easy to see from the

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[17]Higher income may induce greater education and, thus, greater concern about health. For income and educational choices see, for instance, Case and Paxson (2008), and Acemoglu (2009). As we have observed in Section 2, our paper does not focus on income effects. However, Section 6 provides further discussion in this regard.

[18]Notice that, due to the assumption of additive separable preferences (1), ϵ does not directly affect c_t. However, it will do it in equilibrium (see Section 5.2).

definition of $\tilde{\epsilon}$ that the condition $\lambda\alpha - (1-\phi)\gamma\sigma < 0$ implies $\tilde{\epsilon} < 0$ and, thus, $\epsilon > \tilde{\epsilon}$. Note also that $\lambda\alpha - (1-\phi)\gamma\sigma < 0 \Leftrightarrow \phi < \tilde{\phi}$, where $\tilde{\phi}$ is defined as in Proposition 1. Hence, $\phi < \tilde{\phi}$ 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 $\tilde{\phi}$) $\blacksquare$

Proposition 1 just says that we focus on positive interior solutions. Still we are able to provide intuition for the intervals imposed on the parameters. As we have observed in Section 5.1, a high damage (morbidity) of the disease reduces the value of old age and, thus, health capital. However, agents will still invest in health if they are (i) sufficiently informed about the effect of unhealthy activities, i.e., they "understand" the negative health impact of v_t and, thus, are willing to invest more in health; and (ii) enough concerned about their future, otherwise, if they do not value future they would not invest in it.[19] In contrast, health capital is always positive if NCDs' morbidity is low because the expected utility in the second period is sufficiently high.

In this paper we focus on the steady-state equilibrium h^*, which is defined as a fixed point of the transition function, i.e., $h^* = \varphi(h^*)$. One can easily verify that h^* is stable (unstable) iff $\varphi'(h^*) < 1 (> 1)$. As in Azariadis (1996), and Azariadis and Stachurski (2005), we can assume the following step function for the probability of disease in order to get further analytical results:

$$\pi(h_t) = \begin{cases} \pi_H & \text{if} \quad h_t < h^c \\ \pi_L & \text{if} \quad h_t \geq h^c, \end{cases} \tag{18}$$

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.[20] Moreover h^c, together with π_H and π_L, may also account for regional differences such as, for instance, medical technology.

Taking (18), the corresponding transition function is therefore composed by two branches given by

$$\varphi(h_t) = \begin{cases} \frac{[\gamma(\sigma+\epsilon\alpha)(1-\phi\pi_H)-(1-\epsilon)\lambda\alpha][(1-\delta)h_t+\sigma w]}{(\sigma+\epsilon\alpha)[\lambda+\mu+\gamma(1-\phi\pi_H)]} \equiv \varphi_{\pi_H}(h_t) & \text{if} \quad h_t < h^c \\ \frac{[\gamma(\sigma+\epsilon\alpha)(1-\phi\pi_L)-(1-\epsilon)\lambda\alpha][(1-\delta)h_t+\sigma w]}{(\sigma+\epsilon\alpha)[\lambda+\mu+\gamma(1-\phi\pi_L)]} \equiv \varphi_{\pi_L}(h_t) & \text{if} \quad h_t \geq h^c. \end{cases} \tag{19}$$

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[19]See the comparative statics analysis for ϵ and γ in Section 5.1.

[20]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.

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

Proposition 2 *Assuming the functional form (18) and the conditions established in Proposition 1, let us define $h^*_{\pi_i}$ as*

$$h^*_{\pi_i} = \frac{[\sigma\psi_{\pi_i} - (1-\epsilon)\lambda\alpha]w}{\gamma\delta\psi_{\pi_i} + \sigma(\lambda+\mu) + \alpha[\lambda(1-\delta) + \epsilon(\mu+\lambda\delta)]},$$

where $\psi_{\pi_i} = \gamma(\sigma + \epsilon\alpha)(1 - \phi\pi_i)$, $i = \{H, L\}$, and $h^*_{\pi_H} < h^*_{\pi_L}$. If $0 < h^*_{\pi_H} < h^c < h^*_{\pi_L}$, there exist two steady-states given by $h^*_{\pi_H}$ and $h^*_{\pi_L}$. Instead, if either $0 < h^*_{\pi_H} < h^*_{\pi_L} < h^c$ or $0 < h^c < h^*_{\pi_H} < h^*_{\pi_L}$, there is a unique steady-state given by $h^*_{\pi_H}$ and $h^*_{\pi_L}$, respectively. Moreover, all the steady-states are stable.

Proof. Under Proposition 1, $0 < \varphi'(h_t) < 1$ for all $h_t > 0$. Then all possible steady-states are stable. Assuming (18), there exist two 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 $\partial\varphi(h_t)/\partial\pi_t < 0$). Moreover, the corresponding closed-forms are provided by computing the fixed points of (19). Additionally one can easily check that if the health capital threshold is high (low) enough so that $0 < h^*_{\pi_H} < h^*_{\pi_L} < h^c$ ($0 < h^c < h^*_{\pi_H} < h^*_{\pi_L}$), the economy admits only one steady-state given by $h^*_{\pi_H}$ ($h^*_{\pi_L}$). ∎

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.[21]

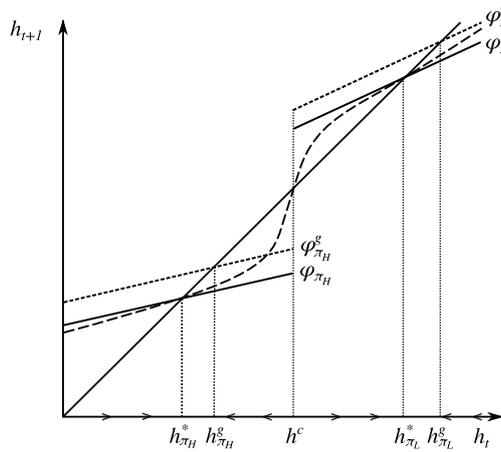Figure 1: Dynamics

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[21]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 (17) that h^* is positively affected by w, σ, γ, and ϵ. Moreover, the steady-state value is negatively affected by ϕ too.

Figure 1 represents the two possible steady-states, assuming $0 < h^*_{\pi_H} < h^c < h^*_{\pi_L}$. In the figure, the intersection between the 45-degree line and φ_{π_H} and φ_{π_L} define the steady-states (for the moment we abstract from the dashed functions to be used later). 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.[22]

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.

5.3 Welfare analysis

In this section we study the welfare implications of NCDs when $\pi_t = \pi(h_t)$, taking advantage of the analytical tractability of this case with respect to Section 4.

5.3.1 Social optimum

Let us consider the social optimum problem of Section 4.1 with $\pi_t = \pi(h_t)$. The corresponding FOCs are summarized by

$$\frac{\partial U_t}{\partial v_t} = \frac{\partial U_t}{\partial c_t} + \alpha \left\{ \frac{\partial U_t}{\partial h_{t+1}} + \beta \left[\frac{\partial U_{t+1}}{\partial \pi_{t+1}} \frac{\partial \pi_{t+1}}{\partial h_{t+1}} + \iota_{t+2}(1-\delta) \right] \right\}. \qquad (20)$$

Comparing this expression with (13), we can see that the individual choices are socially non-optimal and, for c_t and $h_{t+1} = h^p_{t+1}$ given, agents choose too

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[22]As pointed out in this section, we have assumed the step function (18) in order to provide closed-form solutions (see sections 5.2 and 5.3.2). Indeed, this functional form may be considered as a discretized version of a concave-convex π_t (in Figure 1, the sigmoid dashed curve corresponds to the transition function of a concave-convex π_t). However, a step-simplification is not possible for $\pi_t = \pi(h_{t+1})$: this function must be differentiable in its whole domain. In this case, we may directly consider a concave-convex function like, for instance, $\pi(h_{t+1}) = (\pi_L - \pi_H)h^2_{t+1}/(1 + h^2_{t+1}) + \pi_H$. Nevertheless, the analysis would be restricted to numerical results that we leave for future research.

much unhealthy activities than what is socially optimal. Indeed, individuals do not account neither for the *direct* nor for the *indirect* effect. The difference with respect to Section 4.1 is that even under low misperception ($\epsilon \to 1$) the *indirect* effect $\beta \frac{\partial U_{t+1}}{\partial \pi_{t+1}} \frac{\partial \pi_{t+1}}{\partial h_{t+1}}$ does not vanish.

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

$$\frac{\partial U_t}{\partial v_t} = \frac{\partial U_t}{\partial c_t} + \left(\sigma \frac{\tau_t}{1 - s_t} + \epsilon \alpha \right) \frac{\partial U_t}{\partial h_{t+1}^p}, \tag{21}$$

and, taking $h_{t+1} = h_{t+1}^p$, the optimal policy is provided by equating (20) and (21):

$$\frac{\tau_t}{1 - s_t} = \frac{\alpha}{\sigma} \left\{ (1 - \epsilon) + \beta \left[\frac{\frac{\partial U_{t+1}}{\partial \pi_{t+1}} \frac{\partial \pi_{t+1}}{\partial h_{t+1}} + (1 - \delta) \iota_{t+2}}{\frac{\partial U_t}{\partial h_{t+1}}} \right] \right\}. \tag{22}$$

As expected the optimal tax accounts for the *direct* and *indirect* effects, and for misperception. Let us compare this expression with (10). In the previous case the *indirect* effect was not fully internalized due to misperception. Assuming instead $\pi_t = \pi(h_t)$ reinforces the intergenerational externality, even in the absence of misperception, and consequently the optimal policy increases with respect to (10) (for instance, a higher fat tax in the case of a lump-sum transfer). Similarly to the previous case, (13) and (20) coincide under a high level of health information ($\epsilon \to 1$) and absence of transmission mechanism of NCDs ($\delta \to 1$ and $\frac{\partial \pi_{t+1}}{\partial h_{t+1}} = 0$). Then, the corresponding tax and subsidy vanish. Finally, as in Section 4, the decentralized solution is socially non-optimal even without misperception. Therefore, public intervention would be again justified due to the intergenerational transmission of NCDs and not just because of information asymeties.

5.3.2 Golden rule

Let us consider the golden rule. Since it maximizes the aggregate surplus at the steady state, one can easily verify that this social planner problem is identical to the one of Section 4.3, with the corresponding Euler equation provided by (11). The difference between this section and Section 4.3 relies on the individual behaviour and, in particular, on the assumption $\pi_t = \pi(h_t)$. Clearly, due to both the intergeneration transmission of NCDs and the misperception problem, condition (11) does not coincide with the FOC of the decentralized economy (21) at the steady-state. However, 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 $h_{t+1} = h^p_{t+1}$, the golden rule policy is provided by equating (11) with (21) at the steady state:

$$\frac{\tau}{1-s} = \frac{\alpha}{\sigma}\left[\left(\frac{1}{\delta} - \epsilon\right) + \frac{1}{\delta}\frac{\frac{\partial U}{\partial \pi}\frac{\partial \pi}{\partial h}}{\frac{\partial U}{\partial h}}\right]. \tag{23}$$

By comparing (12) and (23) we confirm that, due to the presence of the *indirect* effect, the optimal policy has to be higher than before. Moreover, as one can also expect, in the absence of both misperception problem ($\epsilon \to 1$) and intergenerational transmission ($\delta \to 1$ and $\frac{\partial \pi}{\partial h} = 0$) the FOCs coincide and, consequently, tax and subsidy vanish.

Considering the functional forms (1) and (18) allow us to characterize further analytical results. Taking (2) and (3) at the steady-state, and (11), the golden rule values for health capital and unhealthy activities are respectively:

$$h^g_{\pi_i} = \frac{\gamma\sigma(1-\phi\pi_i)w}{\delta[(\lambda+\mu)+\gamma(1-\phi\pi_i)]} \tag{24}$$

and

$$v^g_{\pi_i} = \frac{\lambda\sigma w}{(\sigma+\alpha)[(\lambda+\mu)+\gamma(1-\phi\pi_i)]}, \tag{25}$$

for $i = \{H,L\}$.[23] As it is clear from Proposition 2, the individual choices are different from 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 (15) at the steady state and (25), it is easy to see that $v^*_{\pi_i} > v^g_{\pi_i}$ since $\epsilon < 1$ and, from Proposition 2, $h^*_{\pi_i} > 0$, for $i = \{H,L\}$. Let us show that $h^*_{\pi_i} < h^g_{\pi_i}$, for $i = \{H,L\}$. It is sufficient to prove that $\varphi_{\pi_i}(h_t) < \varphi^g_{\pi_i}(h_t)$, for all $h_t > 0$, where $\varphi^g_{\pi_i}(h_t) = (1-\delta)h_t + \sigma m^g_{\pi_i} - \alpha v^g_{\pi_i}$ (the corresponding π_i-branch for the golden rule problem). Using (2) and (3) in the golden rule problem, we get $m^g_{\pi_i} = \frac{[\lambda\alpha+\gamma(\sigma+\alpha)(1-\phi\pi_i)]w}{(\sigma+\alpha)[(\lambda+\mu)+\gamma(1-\phi\pi_i)]} > 0$. Therefore, taking (25), $\varphi^g_{\pi_i}(h_t) = \frac{[(\lambda+\mu)+\gamma(1-\phi\pi_i)](1-\delta)h_t+\gamma\sigma(1-\phi\pi_i)w}{(\lambda+\mu)+\gamma(1-\phi\pi_i)}$. Rearranging terms in (19) we get $\varphi_{\pi_i}(h_t) =$

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

$\frac{[\gamma(1-\phi\pi_i)-(\frac{1-\epsilon}{\sigma+\epsilon\alpha})\lambda\alpha][(1-\delta)h_t+\sigma w]}{(\lambda+\mu)+\gamma(1-\phi\pi_i)}$. Finally, by comparing these two expressions, we conclude that $\varphi_{\pi_i}(h_t) < \varphi_{\pi_i}^g(h_t)$, for all $h_t > 0$ $\blacksquare$

Furthermore, from (2) we can also verify that agents choose too much consumption ($c_{\pi_i}^* > c_{\pi_i}^g$) and too little health investment ($m_{\pi_i}^* < m_{\pi_i}^g$) at the steady-state. From (23) and assuming the step function (18), the corresponding policy to implement the golden rule is now given by

$$\frac{\tau_{\pi_i}}{1 - s_{\pi_i}} = \frac{\alpha}{\sigma}\left(\frac{1}{\delta} - \epsilon\right). \tag{26}$$

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*

$$\tau_{\pi_i} = \frac{\tilde{\tau}}{1 + \frac{v_{\pi_i}^g}{m_{\pi_i}^g}\tilde{\tau}} \tag{27}$$

and

$$s_{\pi_i} = \tau_{\pi_i}\frac{v_{\pi_i}^g}{m_{\pi_i}^g}, \tag{28}$$

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 (26) 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 (27) and (28). Finally, from (25) and the formula of $m_{\pi_i}^g$ (see the proof of Proposition 3) we obtain the expression for $v_{\pi_i}^g/m_{\pi_i}^g$ $\blacksquare$

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.[24] 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) predict 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) conclude 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), show 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

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[24]Notice that a greater the intergenerational transmission (i.e., a lower δ) also increases both tax and subsidy ($\partial\tau_{\pi_i}/\partial\delta, \partial s_{\pi_i}/\partial\delta < 0$).

reduce obesity and overweight.[25] For further empirical results in the same direction see, for instance, Mytton et al. (2007) and Epstein et al. (2007).

6   Concluding remarks

Our contribution highlights how the social intergenerational transmission of the modifiable risk factors can lead to the spread of NCDs and how policy instruments can be used to limit the prevalence of these diseases. Public intervention is grounded on the existence of a social intergenerational transmission mechanism and not due to information asymmetries between the policy maker and the individuals (even though we also analyze this aspect).

Several remarks can be made with regard to our modeling. First, genetics and aging are also causes for the NCDs epidemics, as well as other forms of social transmission of modifiable risk factors (see Introduction). Concerning genetics, one could be misled into thinking that in our setup social and genetic transmission are almost equivalent when $\pi_t = \pi(h_t)$ because h_t is taken as given by the agent. However, an important difference is that social transmission assumes that even if children receive "mechanically" the probability $\pi_t = \pi(h_t)$ parents do not transfer it "mechanically". In contrast, π_t depends on parents' rational choices regarding unhealthy activities and health investments. Actually, the individuals' choices v_t and m_t allow them to affect their health capital at old age and therefore enjoy more/less utility. In turn, if the transmission was exclusively genetical individuals could not affect the transfer of their stock of health capital h_{t+1} to their children. Still, in the present model the parameters h_0, h^c, π_H and π_L may also capture population genetics. Nevertheless, it remains to be analyzed the effect of inherited genetics heterogeneity across individuals on the propensity to develop NCDs. Another aspect of interest would be to develop a model able to capture population aging and allowing for the probability of NCDs to increase in age. Moreover, other forms of social transmission of modifiable risk factors, as network or peer effects, deserve to be analyzed. However, we believe that microeconomic inspections would be more suitable in this regard.

Second, since our paper focuses on the intergenerational transmission of NCDs, we have considered a simple setup that incorporates a social transmission mechanism of the disease. However, we could extend our framework

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[25]Among other things, they find 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 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%).

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 setup 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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