Longevity, Genes and Efforts: An Optimal Taxation Approach to Prevention*

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Abstract

This paper applies the analytical tools of optimal taxation theory to the design of the optimal subsidy on preventive behaviors, in an economy where longevity varies across agents, and depends on preventive expenditures and on longevity genes. Public intervention can be here justified on three grounds: corrections for misperceptions of the survival process and for externalities related to individual preventive behavior, and redistribution across both earnings and genetic dimensions. The optimal subsidy on preventive expenditures is shown to depend on the combined impacts of misperception, externalities and self-selection. It is generally optimal to subsidize preventive efforts to an extent depending on the degree of individual myopia, on how productivity and genes are correlated, and on the complementarity of genes and preventive efforts in the survival function.

Keywords: genetic background, heterogeneity, misperception, optimal taxation, prevention, survival prospects.

JEL codes: H21, I10

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1 Introduction

As shown by demographers, there exist significant longevity inequalities within a given cohort. Although all cohort members belong to the same country (at birth) and epoch (and the associated technology), some of them turn out to be short-lived, whereas others enjoy a longer existence. This fact is illustrated by Figure 1, which shows the distribution of the age at death for the cohort born in 1900 in Sweden.[1] Whatever we consider males or females, there is a significant variance in the age at death. Moreover, women enjoy, on average, a longer life than men.

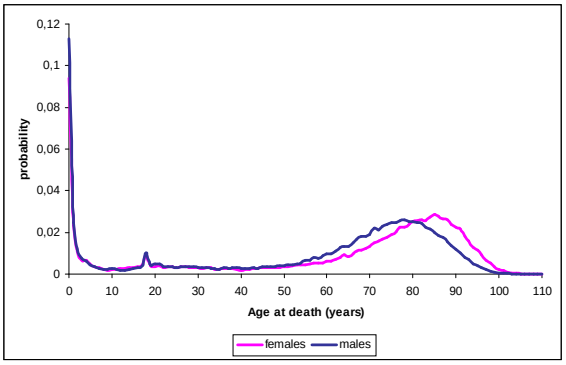


Figure 1: Distribution of the age at death (Sweden, 1900 cohort)

Longevity inequalities are due to various factors. In short, we can classify the determinants of longevity in two broad classes: on the one hand, "external" factors, on which agents, as individuals, have little control; on the other hand, "behavioral" factors, through which agents can influence their survival prospects.

Factors of the first kind include the genetic background of individuals. As emphasized by Christensen *et al* (2006), genetic factors account for not less than a quarter of the variance in adult human longevity. That result was obtained from the comparison of longevity prospects of twins of the two kinds: monozygotic (sharing - nearly - the same genetic background) and dizygotic (having different genetic backgrounds). The variance of the age at death is smaller among monozygotic twins, revealing the role of genes as a determinant of longevity inequalities (Herskind *et al*, 1996). But genes are not the unique determinant on which agents have individually no control. Environmental quality is another determinant of longevity on which individuals have, on their own, no control.[2]

[1] Sources: The Human Mortality Database (2010).

[2] Environmental determinants of longevity include the quality of lands (Kjellström, 1986), of waters (Sartor and Rodia, 1983), and of the air (Kinney and Ozkanyak, 1991).

Among the determinants controlled by man, demographic studies emphasize the crucial role played by lifestyles.[3] Individuals can make preventive efforts of various types. A first, major kind of preventive behavior consists of regular physical activity. The longitudinal study by Kaplan *et al* (1987) highlighted that individuals above age 60 who had little leisure-time physical activity in 1965 faced, during the next 17 years, an overall mortality risk that is 1.38 times the mortality risk of those who had regular physical exercise. Another example of preventive effort consists of a healthy diet. As shown by Kaplan *et al* (1987), individuals above age 60 who did not eat breakfast regularly in 1965 faced, during the next 17 years, a mortality risk that is 1.40 times the risk of those eating regular breakfast.[4]

Preventive efforts can also take other forms, which are related to particular diseases. In the case of influenza, preventive efforts include hygiene habits such as frequent hand washing, as well as wearing a face mask when being with the sick. Moreover, as shown by Fireman *et al* (2009), influenza vaccination can also reduce mortality significantly, even when controlling for selection biases. Note, however, that not all vaccinations are relevant examples of individual preventive efforts. Children immunisations, for instance, are not chosen by the persons who directly benefit from these, and, thus, can hardly be regarded as individual preventive efforts. The same is true for mandatory vaccinations. Hence, only non-mandatory immunisations chosen by adults are relevant instances of preventive efforts. Such adult immunisations include vaccinations against numerous diseases, such as influenza, tetanus, hepatitis B virus, whooping cough and meningitis. Finally, note that, besides those immunisations, preventive efforts include also, but to a smaller extent, a regular attendance to medical checkup (see Franks *et al*, 1996).[5]

Longevity inequalities due to external and behavioral factors raise complex problems for policy-making. What should governments do in front of such inequalities? In particular, should governments subsidize preventive behaviors, and, if so, to what extent? The goal of this paper is precisely to apply the analytical tools of optimal taxation theory to the design of the optimal subsidy on preventive behaviors, in an economy where longevity is endogenous, and depends on "external" and "behavioral" factors.[6] For that purpose, we will adopt a standard utilitarian approach, and design the social welfare maximizing tax policy. Note that there are two major difficulties faced by governments in our particular context.

First, external factors affecting longevity are, in general, hardly observable. This is particularly true for what scientists call "longevity genes". As discussed

[3] The impact of lifestyles is also confirmed by studies taking unobserved heterogeneity into account, such as Contoyannis and Jones (2004) and Balia and Jones (2008).

[4] On the impact of inadequate or excessive eating on survival, see also Bender *et al* (1998).

[5] Note that the effectiveness of medical checkups seems to vary with the gender. For women, the overmortality risk factor due to no checkup equals 1.64, against 1.07 for men.

[6] As such, this complements Bommier *et al* (2009, 2010) where longevity is exogenous.

by Christensen *et al* (2006), it is extremely difficult for scientists to isolate and identify longevity-improving genes. Therefore, governments could hardly observe longevity genes, and base their policy on these. Note, however, that individuals may have more information on their health than authorities (e.g. past occurrence of diseases in the family, etc.). If so, the design of the optimal policy can then be regarded as a problem of optimal policy-making under asymmetric information.

Second, the design of the optimal policy is also made difficult by the imperfect nature of agents' preventive effort decisions. As argued by Besley (1989), there exist various behavioral imperfections in health-related choices. For instance, myopic agents misperceiving the survival process may underestimate the effect of their behavior on future longevity, and adopt suboptimal behaviors, which they will regret *ex post*.[7] In this case, the government should intervene to make agents make the right decisions from the start, and these would be, *ex post*, grateful to the government for its intervention. But there can be imperfections of other natures. Individuals may, when choosing preventive efforts, ignore their impact on the survival of others. Standard externalities may arise, for instance, in the case of vaccinations against infectious diseases.[8] Such externalities invite a Pigouvian subsidy aimed at inducing the optimal level of prevention.

This paper aims at studying the welfare-maximizing taxation policy under endogenous longevity while doing justice to those difficulties.[9] What we propose here is an optimal taxation approach to prevention against death. For that purpose, we develop a two-period model where the probability of survival to the old age (second period) is a function of three inputs: (1) the preventive effort chosen by the individual; (2) the overall preventive effort made by the population as a whole; (3) the genetic background of the individual. Factor (1) is purely "behavioral" and factor (3) is purely "external".[10] However, factor (2) is mixed, as each individual can only have a minor effect on the total preventive effort, while his survival is affected by the aggregate level of prevention.

To account for the heterogeneity of populations, we examine the optimal policy in an economy where agents differ in three characteristics: their productivity, their genetic background and their (more or less correct) perception of the survival process. Those characteristics lead, either directly (for genes), or indirectly (for productivity and misperception), to longevity differentials. Note that preventive

[7] There exists a large empirical literature on the occurence of regrets in health-related choices. For instance, studies by Slovic (2001), Jarvis *et al* (2002) and Fong *et al* (2004) showed that about 85-90 % of smokers would not start smoking if they had to face that choice again.

[8] Note, however, that not all immunisations involve externalities on the health of others. For instance, immunisation against the tetanus involves private benefits, but no externalities.

[9] As such, this paper is complementary to Leroux *et al* (2010), which considers optimal linear taxation under endogenous longevity, without externalities.

[10] See Jouvet *et al* (2010) on optimal policy when longevity depends on the environment.

choices are imperfect: myopia makes decisions non optimal, and the same is true for externalities, as agents ignore the effect of their preventive effort on others' survival.

In order to design the optimal subsidy on preventive behaviors in that context, we will make some simplifying assumptions. Firstly, we will concentrate on preventive efforts, and abstract here from curative expenditures.[11] Secondly, the present study will, for simplicity, focus on preventive efforts chosen by individuals, and, thus, will ignore mandatory public preventive programs. Thirdly, we will ignore here the effect of health on old-age welfare, and consider that preventive expenditures affect the agent's lifetime only through better survival prospects.

Anticipating our results, the main contribution of this paper is to highlight some major - but often neglected - determinants of the optimal policy under endogenous longevity. At the first-best optimum (i.e. under perfect information), the social planner does not suffer from any myopia (unlike individuals), and internalizes all externalities. Hence, if agents underestimate their survival prospects, and if preventive expenditures have positive externalities on others' survival (as for some vaccinations), the decentralization of the first-best requires preventive efforts to be subsidized, to induce the optimal preventive behavior among agents.

However, in the second-best problem, where neither genes nor productivities are observable by the social planner, a taxation of preventive expenditures can, under some conditions, serve as an indirect way to achieve social welfare maximization. Actually, if *(high productivity, good genes)* agents invest more in prevention, it can be socially optimal to tax preventive expenditures of *(low productivity, bad genes)* agents in such a way as to relax the incentive compatibility constraint. Note that, whereas taxing preventive efforts to relax the self-selection constraint can be perceived as shocking, the justification is to help agents disadvantaged by Nature to get more benefits from the society and, thus, at the end of the day, to live better.[12] Furthermore, as we show in the paper, myopia and externalities more than offset that incentive-based taxation motive, so that it remains true that preventive efforts should be subsidized rather than taxed.

This paper is organized as follows. Section 2 presents the model and describes the laissez-faire equilibrium. Section 3 characterizes the social optimum, and studies its decentralization. The second-best problem is examined in Section 4. A numerical discussion of the optimal policy is provided in Section 5. Section 6 discusses the robustness of our results. Section 7 concludes.

[11] On the curative *versus* preventive expenditures, see Cremer *et al* (2010).

[12] Note also that, in other settings, one could recommend taxing services such as education or car insurance, for the same reasons, and this would sound less shocking.

2 The model

2.1 Basic assumptions

We consider a two-period model, where all agents live a first period (of length normalized to one) with certainty, and enjoy a second period of life (also of length one), but with a probability π.

The population is heterogeneous in three characteristics:

- a longevity-related characteristic (e.g. genes), denoted by ε_i;
- a productivity at work, denoted by w_i;
- a perception parameter, denoted by α_i, reflecting the agent's (more or less accurate) knowledge of the survival process.

Each of those characteristics influences longevity directly or indirectly. The genetic background is directly responsible for about one quarter of longevity differentials within a given cohort.[13] More productive agents benefit from a larger purchasing power, and can thus invest more in their health, leading to better longevity prospects.[14] Finally, the more or less accurate perception of the survival process affects also longevity indirectly, and its influence can take various forms. The misperception can consist of an underestimation of the effect of preventive efforts on longevity (implying an underinvestment in prevention), or of an overestimation of that effect (leading to an overinvestment in prevention).

Throughout this section, we describe, for simplicity, the laissez-faire in general terms, i.e. by considering the decisions made by an agent of type i, who exhibits particular characteristics ε_i, w_i and α_i, and examine how those characteristics affect his decisions and longevity prospects.

The survival probability π_i of an agent of type i depends on the (first-period) preventive expenditure e_i, on the total preventive expenditure in the population $\bar{e}$, and on a genetic characteristic ε_i, according to the survival function:

$$\pi_i = \pi\left(e_i, \bar{e}, \varepsilon_i\right) \tag{1}$$

where $\bar{e} = \Sigma_i n_i e_i$ denotes the total preventive expenditure in the population, n_i being the proportion of agents of type i, namely with characteristics ε_i, w_i and α_i.[15] We assume that the first and second derivatives are such that $\pi_e > 0$,

[13] See the study of Herskind *et al* (1996).

[14] On the impact of health spending on longevity, see Poikolainen and Eskola (1986), who show that large longevity gains result from merely consuming more health services.

[15] This specification accounts for the fact that individual survival prospects depend on overall health conditions (which we proxy here by the total preventive expenditure $\bar{e}$) of the society he lives in. Each agent contributes to the overall health conditions by his preventive efforts.

$\pi_{ee} < 0$, $\pi_{\bar{e}} > 0$, $\pi_{\bar{e}\bar{e}} < 0$, $\pi_{\varepsilon} > 0$, and $\pi_{\varepsilon\varepsilon} < 0$. We also assume that genetics and preventive effort (individual and total) are complements, so that the impact of preventive expenditures is higher when the agent has better genes (i.e. $\pi_{e\varepsilon} > 0$ and $\pi_{\bar{e}\varepsilon} > 0$).[16]

Throughout this paper, we assume that agents do not necessarily have a correct perception of their actual survival probability. They make their saving and preventive effort decisions on the basis of a *perceived* survival function $\hat{\pi}_i$ defined as:[17]

$$\hat{\pi}_i = \alpha_i \pi \left(e_i, \bar{e}, \varepsilon_i \right) \tag{2}$$

where the parameter α_i, which lies between 0 and $1/\pi(\cdot)$, stands for the agent's subjective perception of the survival law. It is only under α_i equal to 1 that the objective and subjective valuations of one's survival prospects coincide. Otherwise, if α_i differs from 1, there is a disconnection between how the agent perceives his survival prospects and what those survival prospects really are.[18] When $\alpha_i \in [0, 1[$, agents tend to underestimate their survival probability, and to underestimate the impact of their preventive effort on survival possibilities. We will, in this paper, refer to this case as 'pessimism'. On the contrary, when $1 < \alpha_i < 1/\pi(\cdot)$, agents overestimate their survival prospects, and exaggerate the impact of preventive effort on survival prospects: that alternative case is here referred to as 'optimism'.[19]

Lifetime welfare is assumed to be additive over time, and temporal welfare depends on consumption and on labor time during the period. Setting the discount and interest rates equal to zero and assuming that the utility of being dead is normalized to zero, the expected lifetime utility of an agent of type i is given by:[20]

$$U \left(c_i, d_i, l_i \right) = u \left(c_i \right) + \alpha_i \pi \left(e_i, \bar{e}, \varepsilon_i \right) u \left(d_i \right) - v \left(l_i \right) \tag{3}$$

where c_i and d_i denote the consumption in the first and second periods respectively, and l_i is the labor supply in the first period. Per period utility of consumption, $u(\cdot)$, is such that $u'(\cdot) > 0$ and $u''(\cdot) < 0$. The disutility of labor is increasing and strictly convex: $v'(\cdot) > 0$ and $v''(\cdot) > 0$.

[16] The robustness of our results to this assumption is examined in Section 6.2 below.

[17] Besley (1989, p. 136) distinguishes between four behavioral imperfections: probabilistic misperception, objective misperception (e.g. health technology), subjective misperception (i.e. latent preferences) and evaluator relativity (imperfect knowledge of latent selves). Our model assumes both a probabilistic misperception and a subjective misperception of survival prospects.

[18] On the gap between objective and subjective life expectancies, see Hamermersh (1985) and Ludwig and Zimper (2007).

[19] The terms 'pessimism' and 'optimism' are used here for the simplicity of presentation.

[20] That expression presupposes no pure time preference, but the survival probability can be interpreted as a natural discount factor.

2.2 The laissez-faire

Agents invest all their savings on a perfect annuity market, which yields an actuarially fair return for each risk class.[21] Hence, the return on saving for an agent of type i, which is denoted by R_i, is equal to $1/\pi\left(e_i, \bar{e}, \varepsilon_i\right)$ when the interest rate is zero, and depends on the actual survival rate (and not on the perceived one).

An agent of type i chooses his savings s_i, his labor supply l_i and his preventive expenditure e_i by maximizing his expected lifetime utility subject to the budget constraint.[22] When solving that problem, agents do not care about the effect of their preventive investment on the survival of others, but only consider the impact it has on their probability of survival and on the return on savings. Thus the agent's problem can be written as:

$$\max_{c_i,d_i,e_i,l_i} \; u\left(c_i\right) + \alpha_i \pi\left(e_i, \bar{e}, \varepsilon_i\right) u\left(d_i\right) - v\left(l_i\right)$$
$$\text{s.t.} \left\{ \begin{array}{l} c_i = w_i l_i - s_i - e_i \\ d_i = s_i R_i = \frac{s_i}{\pi(e_i, \bar{e}, \varepsilon_i)} \end{array} \right.$$

where $\bar{e}$ is taken as given, i.e. as unaffected by the agent's effort e_i.

From the first-order conditions (FOCs), we obtain the marginal rates of substitution:

$$MRS^{LF}_{l_i,c_i} \equiv \frac{v'\left(l_i\right)}{u'(c_i)} = w_i \tag{4}$$

$$MRS^{LF}_{d_i,c_i} \equiv \frac{u'(d_i)}{u'(c_i)} = \frac{1}{\alpha_i} \tag{5}$$

$$MRS^{LF}_{e_i,c_i} \equiv \frac{\pi_e\left(e_i, \bar{e}, \varepsilon_i\right) u\left(d_i\right)}{u'\left(c_i\right)} = \frac{1 + \pi_e\left(e_i, \bar{e}, \varepsilon_i\right) d_i}{\alpha_i} \tag{6}$$

Equation (4) is standard: the marginal rate of substitution between labor and consumption is equal to the wage rate. Equation (5) defines the optimal level of savings. If the agent is perfectly rational (i.e. $\alpha_i = 1$), consumption is smoothed over time (i.e. $c_i = d_i$); on the contrary, for any $\alpha_i < 1$ (resp. $\alpha_i > 1$), first-period consumption is higher (resp. lower) than second-period consumption.

To explain the determinants of the optimal preventive expenditure, the FOC (6) for an interior optimum e_i can be written as

$$u'\left(c_i\right) = \alpha_i \pi_e\left(e_i, \bar{e}, \varepsilon_i\right)\left[u\left(d_i\right) - u'\left(d_i\right) d_i\right]$$

The LHS is the marginal utility loss due to foregone consumption, while the RHS is the net utility gain from a larger prevention. That net utility gain includes

[21] The assumption of perfect annuity markets for each risk class is a simplification made for conveniency (see Brown, 2007). See Cremer *et al* (2010) for the study of collective annuitization.

[22] To reduce the number of parameters in our model, the price of e_i is normalized to 1.

two parts. On the one hand, a higher e_i directly increases the probability of enjoying second-period consumption through survival, which yields an additional utility $u(d_i)$, but, on the other hand, it decreases second-period consumption, through a lower return of annuitized savings, as captured by $-u'(d_i)\,d_i$.[23] The marginal net utility gain from preventive effort depends also on the kind and degree of misperception: e_i decreases with α_i, so that a pessimistic agent (i.e. with $\alpha_i < 1$) invests less in prevention than an optimistic one (i.e. with $\alpha_i > 1$).

Finally, remind that agents, when choosing their preventive expenditure e_i, take $\bar{e}$ as given, and, thus, do not take into account that, by investing an amount e_i, they modify also the survival prospects of *all other* agents through $\bar{e}$. By considering themselves as being one agent among a multitude of agents, they believe that their prevention has no impact on others, whereas it actually does. This is a source of externality, as we shall discuss in the next section.

3 Paternalistic optimum

In this study, the social planner is assumed to be utilitarian and paternalistic.

By a 'utilitarian' planner, we mean that the planner pursues the standard Benthamite goal of the maximization of the sum of individual utilities. That ethical framework suffers from two particular weaknesses in our context. On the one hand, its reliance on consequentialism makes it hard to account for issues of responsibility (as only outcomes matter from a consequentialist point of view). On the other hand, as this is well-known in the population ethics literature, utilitarianism in its classical form exhibits limitations in the context of varying longevity: this regards any additional life-period with a positive (even extremely low) utility level as desirable (see Broome, 2004). Thus, classical utilitarianism should only be regarded as an - analytically attractive - starting point for the issue at stake.

By 'paternalistic' planner, we mean that the planner uses, in his maximization program, the *true* survival probability, and not the one perceived by individuals (i.e. he sets $\alpha_i = 1\ \forall i$ in his planning problem). The reason why the social planner does so is that agents misperceiving the survival process (i.e. with $\alpha_i \neq 1$) tend, *ex post*, to regret their first-period choices, that is, to regard their past choices as inadequate. The existence of such regrets legitimates the paternalistic approach

[23] Here, we assume that the agent perfectly anticipates the impact of preventive efforts on the return of annuitized savings. Another approach would consist in assuming that the annuity return is taken *as given* by the agent, so that he does not internalize the impact of e_i on the annuity return (see Becker and Philipson, 1998). In this case, the laissez-faire level of preventive effort is typically not optimal. Given that no empirical study has yet been able to provide empirical evidence on this phenomenon, we assume that such a behavioral imperfection does not take place here. See Leroux *et al* (2010) on the impact of that imperfection on optimal taxation.

followed here by the planner. The social planner's motivation is standard in self-control problems: the planner knows that individuals will be grateful to him to have forced them to behave according to their true survival probability.

3.1 First-best solution

In the first-best setting, the social planner observes the types of individuals, i.e. their productivity and genes, and ignores any misperception of the survival process (i.e. $\alpha_i = 1 \; \forall i$). His problem is thus:

$$\max_{c_i, d_i, e_i, l_i \forall i} \sum n_i \left[u\left(c_i\right) + \pi\left(e_i, \bar{e}, \varepsilon_i\right) u\left(d_i\right) - v\left(l_i\right) \right]$$

subject to the resource constraint of the economy

$$\sum n_i \left(c_i + e_i + \pi\left(e_i, \bar{e}, \varepsilon_i\right) d_i - w_i l_i \right) = 0 \tag{7}$$

The FOCs of this problem can be rearranged as

$$\begin{aligned} v'\left(l_i\right) &= \mu w_i & (8) \\ u'(c_i) = u'(d_i) &= \mu & (9) \\ \pi_e\left(e_i, \bar{e}, \varepsilon_i\right) u\left(d_i\right) + \Sigma_j n_j \pi_{\bar{e}}\left(e_j, \bar{e}, \varepsilon_j\right) u\left(d_j\right) &= \mu \left[1 + \pi_e\left(e_i, \bar{e}, \varepsilon_i\right) d_i \right. \\ & \left. + \Sigma_j n_j \pi_{\bar{e}}\left(e_j, \bar{e}, \varepsilon_j\right) d_j \right] & (10) \end{aligned}$$

where μ is the Lagrange multiplier associated with the resource constraint and $d\bar{e}/de_i = n_i$. From equation (8), it follows that agents with a higher productivity should supply more labor l_i than agents with a lower productivity. Equation (9) shows that consumption should be equalized for all types and across all periods, $c_i = d_i = \bar{c} \; \forall i$. That result is different from what we have at the laissez-faire, where the relation between c_i and d_i depends on α_i. This equalization of all consumptions at all periods is a direct implication of paternalistic utilitarianism and of additivity across periods in individual lifetime utility. From the above FOCs, we have:

$$\begin{aligned} MRS^{FB}_{l_i, c_i} &= w_i & (11) \\ MRS^{FB}_{d_i, c_i} &= 1 & (12) \\ MRS^{FB}_{e_i, c_i} &= 1 + \pi_e\left(e_i, \bar{e}, \varepsilon_i\right) d_i - \frac{\Delta}{u'\left(c_i\right)} & (13) \end{aligned}$$

where $\Delta = \Sigma_j n_j \pi_{\bar{e}}\left(e_j, \bar{e}, \varepsilon_j\right) \left[u\left(d_j\right) - \mu d_j \right]$ is the external effect of preventive expenditures on longevity.

Let us compare (13) with its laissez-faire counterpart (6). These conditions differ on two grounds. Firstly, in the absence of misperception (i.e. $\alpha_i = 1$), the

first-best MRS differs from the laissez-faire MRS by a term $\Delta/u'(c_i)$. This reflects the impact of the preventive effort of agent i on the survival probability of all other j agents through the argument $\bar{e}$ in the survival function. The agent, by investing in his longevity, creates a positive externality on the survival probability of all other agents. This externality is taken into account by the social planner, while, in the laissez-faire, agents did not take it into account. The first-best level of preventive effort for each agent is thus larger than the laissez-faire one. Secondly, the first-best expression also differs from the laissez-faire by α_i. At the social optimum, there is no misperception of the survival process, so that the first-best e_i is larger (resp. lower) than the laissez-faire level if α_i is smaller (resp. larger) than 1, i.e. if agents under- (resp. over-) estimate their survival prospects.

Regarding the combination of those two effects, it should be stressed that, when $\alpha_i \leq 1$, both effects (externality and misperception) go in the same direction, so that the first-best level of e_i is unambiguously larger than the laissez-faire one. On the contrary, if $\alpha_i \geq 1$, the two effects go in opposite directions, and whether the first-best level of e_i is superior or inferior to its laissez-faire level is not clear.

Note also that, in the first-best, preventive expenditures are differentiated according to the genetic endowment ε_i, but not with respect to the perception parameter α_i. Clearly, according to (13), the differential value of e_i depends only on ε_i. It is even straightforward to see that $\varepsilon_i > \varepsilon_j$ implies $e_i > e_j$ if $\pi_{\varepsilon e} > 0$, that is, if both arguments are complements. Thus, if private preventive effort and genes are complementary, more should be invested on agents who have, *ceteris paribus*, better genes, as this is what maximizes social welfare.

Finally, one can notice that assuming, in a paternalistic way, that $\alpha_i = 1\ \forall i$ leads the social planner to redistribute only according to individual genes and productivity, but not according to the misperception (i.e. the level of α_i).

3.2 Decentralization

Let us now consider how the above paternalistic optimum can be decentralized. In the following, we assume that the instruments available to the social planner are: individualized linear taxes or subsidies on labor τ_i, on health θ_i, and on savings σ_i, as well as individualized lump-sum transfers T_i. We still assume that the annuity market is actuarially fair, so that $R_i = 1/\pi(e_i, \bar{e}, \varepsilon_i)$ prevails at the equilibrium. The individual's problem is thus to maximize:

$$u(w_i l_i(1-\tau_i) - s_i(1+\sigma_i) - e_i(1+\theta_i) + T_i) - v(l_i) + \alpha_i \pi(e_i, \bar{e}, \varepsilon_i)\, u(R_i s_i)$$

Solving this problem, we obtain the following marginal rates of substitution,

$$MRS^D_{l_i,c_i} = w_i(1-\tau_i) \quad (14)$$

$$MRS^D_{d_i,c_i} = \frac{1+\sigma_i}{\alpha_i} \quad (15)$$

$$MRS^D_{e_i,c_i} = \frac{1+\theta_i}{\alpha_i} + \frac{\pi_e(e_i,\bar{e},\varepsilon_i)\,u'(d_i)\,d_i}{u'(c_i)} \quad (16)$$

where D stands for decentralization. By comparing those FOCs with FOCs (11)-(13) of the first-best, we obtain that the social optimum can be decentralized with:

$$\begin{aligned}\tau_i &= 0\\ \sigma_i &= \alpha_i - 1\\ \theta_i &= \alpha_i - 1 - \alpha_i\frac{\Delta}{u'(c_i)}\end{aligned}$$

In addition, the decentralization of the first-best requires lump-sum transfers $T_i \lessgtr 0$, to be discussed below. As we can see, the social optimum can be reached with a zero tax on labor τ_i. However, the other tax instruments σ_i and θ_i are likely to differ from zero. Let us now focus on the determinants of those taxes.

Actually, it is optimal to subsidize savings (i.e. $\sigma_i < 0$) for any agent of type i who underestimates his survival chance (i.e. when $\alpha_i < 1$). This subsidy aims at correcting for individual misperception: since pessimistic agents do not save enough in the laissez-faire, it is optimal to subsidize their savings, so as to encourage them to save more. Alternatively, in the absence of misperception (i.e. $\alpha_i = 1$), the tax σ_i is equal to zero. However, if agents tend to overestimate their survival probabilities (i.e. $\alpha_i > 1$), they are saving more than what is optimal, so that the government should then tax their savings.[24]

Regarding preventive expenditures, the sign of the tax θ_i is ambiguous, as it depends on two countervailing effects. To see this, let us first assume that there is no misperception (i.e. $\alpha_i = 1$). In this case, we have $\theta_i = -\Delta/u'(d_i) < 0$, so that e_i are subsidized. This subsidy corrects for the under-spending in prevention in the laissez-faire, which is due to the fact that agents do not see the impact of their preventive efforts on the survival of other agents. Thus, without misperception, the internalization of the positive externality requires a subsidy on e_i.

Let us now assume that there is no externality (i.e. agents internalize the impact of e_i on others' survival). In that case, $\theta_i = \alpha_i - 1$, and it is optimal to subsidize (resp. tax) preventive efforts to offset pessimism (resp. optimism), which makes agents invest too little (resp. too much) in their health. Here the

[24] Here we consider that case for completeness, but there exists little empirical support for such an over-saving behaviour.

government intervenes in such a way as to make agents make the right decisions from the start, and these would be, *ex post*, grateful to the government for its intervention. For instance, if agents, because of ignorance or myopia, underestimate the deferred effect that preventive expenditure has on longevity (i.e. $\alpha_i < 1$), such an imperfection of behavior invites a subsidy aimed at avoiding underinvestment in prevention (i.e. $\theta_i < 0$). Alternatively, if agents, because of misinformation, overestimate the effect of preventive efforts on longevity (i.e. $\alpha_i > 1$), the government should tax preventive expenditures to avoid excessive e_i (i.e. $\theta_i > 0$). Thus individual misperception of the survival process can justify the subsidization or the taxation of preventive expenditures, in order to avoid *ex post* regrets.

In conclusion, the tax θ_i on health spending is positive or negative, depending on the magnitude of these two effects (externalities and misperception).

Table 1 summarizes the impact of misperception (MI) and the externality (EXT) on the subsidies on savings, preventive expenditures and labor.

Table 1: First-best taxation

Taxes	EXT	MI		Total effect	
		$\alpha_i < 1$	$\alpha_i > 1$	$\alpha_i < 1$	$\alpha_i > 1$
σ_i	0	-	+	-	+
θ_i	-	-	+	-	?
τ_i	0	0	0	0	0

The labour supply should not be distorted ($\tau_i = 0$), but savings is affected by a tax or a subsidy σ_i that may vary across agents, as it depends on α_i. As to preventive expenditures, the tax/subsidy rate θ_i depends on α_i and Δ, which is the same for all agents. Thus, σ_i and θ_i should be differentiated across agents only according to the misperception of the survival process (i.e. α_i), but not according to productivities w_i and genetic background ε_i. However, characteristics (w_i, ε_i) play a crucial role, as these affect the direction of lump-sum transfers.

Actually, to decentralize the first-best solution, we need, in addition to the above taxes or subsidies, appropriate lump-sum transfers, which are differentiated according to productivities and genetic background. To discuss this, let us, for simplicity, take two types of agents, with equal genetic backgrounds ε_i, for $i = 1, 2$. At the first-best, $c_i = d_i = \bar{c}$, and, with $\varepsilon_1 = \varepsilon_2$, equation (13) is the same across types, so that $e_1 = e_2$. Hence, if $w_2 > w_1$, the lump-sum tax redistributes from type-2 agents to type-1 agents, to allow them to make the same choices. Alternatively, unequal genes ε_i along with complementarity of e_i and ε_i in the survival function imply that, if $\varepsilon_2 > \varepsilon_1$, the redistribution goes from type-1 agents to type-2 agents (as $e_1 < e_2$). Which redistribution dominates is uncertain. To the extent that we focus on the case where type-2 agents are tempted to pretend

to be of type-1 (i.e. to "mimic" type-1), we shall, in the rest of the paper, adopt the characteristics' values yielding that outcome.

4 Second-best non linear taxation

4.1 Incentive constraints and the planner's problem

Let us now turn to the second-best problem, wherein the social planner cannot observe agents' types (w_i, ε_i). By assumption, the degree of misperception is still observable, and, for simplicity, we suppose it to be equal across types: $\alpha_1 = \alpha_2 = \alpha < 1$. In other words, all agents are assumed to be shortsighted. We consider two types of agents with characteristics (w_1, ε_1) and (w_2, ε_2). For simplicity, but without loss of generality, we focus here on the cases where type-2 agents are tempted to mimic type-1 agents. This possibility appears if

$$u(c_2) + \alpha\pi(e_2, \bar{e}, \varepsilon_2)\, u(d_2) - v(l_2) \leq u(c_1) + \alpha\pi(e_1, \bar{e}, \varepsilon_2)\, u(d_1) - v\left(\frac{y_1}{w_2}\right)$$

where the variable $y_i = w_i l_i$ denotes the gross income earned by an agent with productivity w_i, and where the values c_i, d_i, y_i and e_i are those of the first-best optimum, and parameters ε_2 and w_2 are not common knowledge. For such a mimicking to be possible, it is essential that type-2 agents have characteristics w_2 and ε_2 that, in combination, imply a social redistribution that they would prefer to avoid, so that they pretend to be of type 1. The first-best optimum implies transfers from individuals with the higher productivity towards the ones with the lower productivity, but, also, transfers from individuals with the worse genetic background towards the ones with the better genetic background. Hence, it is not obvious that type-2 agents are necessarily tempted to pretend to be type-1 agents. Under asymmetric information, whether type-1 agents mimic type-2 agents or the reverse depends on whether $w_1 \gtrless w_2$ and $\varepsilon_1 \lesseqgtr \varepsilon_2$. Four cases can be distinguished:[25]

- Case A: $w_2 \geqslant w_1$ and $\varepsilon_1 \geq \varepsilon_2$
- Case B: $w_2 \geqslant w_1$ and $\varepsilon_1 < \varepsilon_2$
- Case C: $w_2 < w_1$ and $\varepsilon_1 > \varepsilon_2$
- Case D: $w_2 < w_1$ and $\varepsilon_1 \leq \varepsilon_2$

[25] Note that each of those four cases can even be splitted into two subcases, if we compare the relative sizes of the productivity gap (w_1 *versus* w_2) and of the genetic gap (ε_1 *versus* ε_2).

Substituting the first-best allocation into the above inequality, it is straightforward to see that under asymmetric information, type-2 agents always mimic type-1 agents in Case A. Such a mimicking will also occur in Case B if the genetic gap is small relative to the productivity gap, and in Case C if the genetic gap is high relative to the productivity gap. In Case D, type-2 agents have never interest in mimicking type-1 agents. We will thus exclude Case D, and suppose that the most realistic case is Case B, where genes and productivities are positively correlated.

Hence, only considering the cases where type-2 agents mimic type-1 agents, the social planner's problem is the following:[26]

$$\max_{c_i,d_i,e_i,y_i} \sum_{i=1,2} n_i \left[u\left(c_i\right) + \pi\left(e_i, \bar{e}, \varepsilon_i\right) u\left(d_i\right) - v\left(\frac{y_i}{w_i}\right) \right]$$

$$\text{s.to} \begin{cases} \sum_{i=1,2} n_i \left(y_i - c_i - e_i - \pi\left(e_i, \bar{e}, \varepsilon_i\right) d_i\right) \geq 0 \\ u\left(c_2\right) + \alpha\pi\left(e_2, \bar{e}, \varepsilon_2\right) u\left(d_2\right) - v\left(\frac{y_2}{w_2}\right) \geq u\left(c_1\right) + \alpha\pi\left(e_1, \bar{e}, \varepsilon_2\right) u\left(d_1\right) - v\left(\frac{y_1}{w_2}\right) \end{cases}$$

[26] Note that one cannot, in general, rule out the case where both agents want to mimic each other. However, given that the survival function is *concave* in its arguments, while the disutility of labour is *convex*, it follows that if type-2 agents are tempted to pretend to be of type 1, it must also be the case that type-1 agents do not want to mimic type-2 agents. Indeed, under the first-best bundle,

$$\begin{aligned} \left[\pi\left(e_2, \bar{e}, \varepsilon_2\right) - \pi\left(e_2, \bar{e}, \varepsilon_1\right)\right] u\left(\bar{c}\right) &\leq v\left(l_2\right) - v\left(\frac{y_1}{w_2}\right) \\ \left[\pi\left(e_1, \bar{e}, \varepsilon_1\right) - \pi\left(e_2, \bar{e}, \varepsilon_1\right)\right] u\left(\bar{c}\right) &\leq v\left(l_1\right) - v\left(\frac{y_2}{w_1}\right) \end{aligned}$$

cannot be satisfied at the same time under Cases A and B. This is also true under Case C provided the genetic gap is much larger than the productivity gap.

In the Appendix, we show that the marginal rates of substitution are:

$$MRS^{SB}_{d_2,c_2} = \frac{1+\lambda/n_2}{1+\lambda\alpha/n_2} \tag{17}$$

$$MRS^{SB}_{d_1,c_1} = \frac{1-\lambda/n_1}{1-\lambda\frac{\alpha\pi(e_1,\bar{e},\varepsilon_2)}{n_1\pi(e_1,\bar{e},\varepsilon_1)}} \tag{18}$$

$$MRS^{SB}_{e_2,c_2} = \left[1+\pi_e\left(e_2,\bar{e},\varepsilon_2\right)d_2\right]\left[\frac{1+\lambda/n_2}{1+\lambda\alpha/n_2}\right] - \frac{\Delta}{u'\left(c_2\right)\left(1+\lambda\alpha/n_2\right)} \tag{19}$$

$$\begin{aligned} MRS^{SB}_{e_1,c_1} &= \left[1+\pi_e\left(e_1,\bar{e},\varepsilon_1\right)d_1\right]\left[\frac{1-\lambda/n_1}{1-\lambda\frac{\alpha\pi_e(e_1,\bar{e},\varepsilon_2)}{n_1\pi_e(e_1,\bar{e},\varepsilon_1)}}\right] \\ &\quad -\frac{\Delta}{u'\left(c_1\right)}\left[\frac{1}{1-\lambda\frac{\alpha\pi_e(e_1,\bar{e},\varepsilon_2)}{n_1\pi_e(e_1,\bar{e},\varepsilon_1)}}\right] \end{aligned} \tag{20}$$

$$MRS^{SB}_{l_2,c_2} = w_2 \tag{21}$$

$$MRS^{SB}_{l_1,c_1} = w_1\left(\frac{1-\lambda/n_1}{1-\frac{\lambda}{n_1}\frac{w_1}{w_2}\frac{v'(y_1/w_2)}{v'(y_1/w_1)}}\right) \tag{22}$$

where λ is the multiplier associated with the self-selection constraint.[27]

To interpret these expressions, we assume here $\alpha = 1$, and leave the complete problem with $\alpha \leq 1$ in the Appendix. Note that assuming $\alpha = 1$ makes the presentation of results more convenient, and does not fundamentally change the impact of the incentive constraint on the trade-offs, but only its size.

When $\lambda = 0$, we are in the first-best setting. In the absence of the externality, (19) and (20) would coincide with the laissez-faire conditions. Let us now reintroduce the self-selection constraint ($\lambda \neq 0$) and keep $\alpha = 1$. From equation (17), we find the usual result of no distortion at the top for type-2 agents, so that, for the mimicker, consumption should be smoothed across periods, as in the first-best. On the contrary, from (18), we have, under complementarity of ε_i and e_i:

$$MRS^{SB}_{d_1,c_1} = \frac{u'\left(d_1\right)}{u'\left(c_1\right)} \gtrless 1 \text{ depending on } \varepsilon_2 \gtrless \varepsilon_1$$

Hence, if, for example, the mimicker (type-2 agent) has better genes ($\varepsilon_2 > \varepsilon_1$), he has higher chances to live long than the mimicked agent (type-1 agent), and, thus, he would like to save more. It then makes sense to discourage savings by type-1 agents, so as to prevent type-2 agents from pretending to be of type 1.

[27] Note that the effect of e_i on $\bar{e}$ in the incentive constraint is not taken into account when computing the above trade-offs, as this effect is considered as being infinitely small.

We now analyze the trade-offs between preventive expenditure and first-period consumption. As the externality has the same impact in the first-best as in the second-best, i.e. to push towards higher preventive efforts, we assume here $\Delta = 0$, to better focus on the effect of the incentive constraint. Equation (19) becomes

$$MRS^{SB}_{e_2,c_2} = 1 + \pi_e\left(e_2, \bar{e}, \varepsilon_2\right) d_2 \tag{23}$$

In this case, the trade-off between preventive expenditure and first-period consumption is not distorted for type-2 agents, and is equal to the laissez-faire. As usual in these types of problem, the mimicker does not face additional distortions in the second-best. Hence, only misperception and externality effects have an impact on the optimal level of preventive expenditure, in the same way as in the first-best.

To see how the trade-off between preventive expenditure and consumption for type-1 agents is modified by the introduction of the incentive constraint, we proceed in the same way, and assume that $\Delta = 0$ (and $\alpha = 1$). Hence equation (20) is now

$$MRS^{SB}_{e_1,c_1} = \left(1 + \pi_e\left(e_1, \bar{e}, \varepsilon_1\right) d_1\right) \left[\frac{1 - \frac{\lambda}{n_1}}{1 - \frac{\lambda \pi_e(e_1, \bar{e}, \varepsilon_2)}{n_1 \pi_e(e_1, \bar{e}, \varepsilon_1)}}\right] \tag{24}$$

Under complementarity between genes and preventive effort, if $\varepsilon_2 > (<)\ \varepsilon_1$, the expression inside brackets is larger (resp. lower) than 1, which leads to lower (resp. higher) preventive expenditures by type-1 agents. For instance, if $\varepsilon_2 > \varepsilon_1$, the trade-off between preventive expenditure and consumption is distorted upwards, and it is optimal to decrease e_1. Indeed, as the mimicker has better genes than the mimickee, he would like to invest more in his prevention, and thus discouraging preventive effort by the mimickee relaxes the incentive constraint.[28]

Finally, concerning the choice of labor, there is no distortion for type-2 agents, and there is the standard downward distortion for type-1 agents.

4.2 Tax formulas

In the Appendix, we combine equations (17)-(22) and (11)-(13), and provide the full expressions of tax formulas. As in the first-best, the externality still supports the subsidization of preventive expenditures, and the underestimation of π_i requires a subsidy on savings and preventive expenditures. Given that those effects are still present in the second-best problem, but are the same as in the first-best case, we decided here, for ease of explanation, to focus only on the impact of incentive constraints on taxation, and thus to set $\Delta = 0$ and $\alpha = 1$. Under those

[28] In contrast, if $\varepsilon_2 < \varepsilon_1$, we have a downward distortion implying a higher e_1.

assumptions, we have

$$\begin{aligned}
\sigma_1 &= \left[\frac{1-\lambda/n_1}{1-\lambda\frac{\pi(e_1,\bar{e},\varepsilon_2)}{n_1\pi(e_1,\bar{e},\varepsilon_1)}}\right]-1\\
\theta_1 &= \left[\frac{1-\lambda/n_1}{1-\lambda\frac{\pi_e(e_1,\bar{e},\varepsilon_2)}{n_1\pi_e(e_1,\bar{e},\varepsilon_1)}}\right]-1\\
\tau_1 &= 1-\frac{1-\lambda/n_1}{1-\frac{\lambda}{n_1}\frac{w_1}{w_2}\frac{v'(y_1/w_2)}{v'(y_1/w_1)}}\\
\sigma_2 &= \theta_2=\tau_2=0
\end{aligned}$$

In the second-best, type-2 agents, being the mimickers, should not face any distortion. This is the usual "no distortion at the top" rule: as there is no externality ($\Delta = 0$) and no myopia ($\alpha = 1$) in this specific case, the taxes on savings, on preventive expenditures and on labour are null for that type of agents. On the contrary, type-1 agents, being the mimickees, will typically face non-zero taxes, as, in the second-best, the tax-subsidy instruments are used also to relax the incentive compatibility constraints, preventing type-2 agents from mimicking type-1 agents.[29] However, as it is clear from the above expressions for σ_1, θ_1 and τ_1, the level and the size of these taxes will depend on the distribution of the two characteristics, w_i and ε_i, as well as on the substitutability between e_i and ε_i in the survival function. For the time being, we keep on assuming that e_i and ε_i are complements.[30] Here are the three cases under which type-2 agents would mimic type-1 agents:

A. negative correlation between genetics and productivity;

B. positive correlation between genetics and productivity, but the productivity gap dominates the genetic gap;

C. positive correlation between genetics and productivity, but the productivity gap is dominated by the genetic gap.

In alternative A, type-2 agents dominate type-1 agents in productivity but not in genes. As it is clear from the above formula, the introduction of incentive compatibility (hereafter IC) constraints thus leads to a tax on earnings and a subsidy

[29] Note that the Atkinson-Stiglitz (1976) property applies to type-2 agents, but it does not apply to type-1 agents. According to the Atkinson-Stiglitz theorem, indirect taxes should be zero; in particular capital income taxation is not needed. Our violation of the Atkinson-Stiglitz theorem is due to the existence of two characteristics w_i and ε_i (see Cremer *et al*, 2004).

[30] As we show in Section 6.2, assuming that e_i and ε_i are substitutes only affects θ_1. Results on the impact of the incentive compatibility constraint are reversed.

on preventive expenditures and on saving for type-1 agents. The explanation goes as follows: because of their characteristics, type-2 agents are expected to work more, to spend less on prevention and to save less than type-1 agents. Hence, in order to prevent the formers from mimicking the latter, it is optimal to impose a tax on earnings and subsidies on preventive efforts and on savings of type-1 agents; this makes the allocation of type-1 agents less attractive to type-2 agents. In alternative B, type-2 agents dominate type-1 agents in both productivity and genes. The explanation is similar to the one provided previously. Type-2 agents are expected to work more, to spend more on prevention and on savings. Hence, to prevent these from mimicking type-1 agents, one has to tax earnings, preventive efforts and savings of type-1 agents. This is what we obtain from the above formula when $w_2 \geqslant w_1$ and $\varepsilon_1 < \varepsilon_2$. In alternative C, type-2 agents have worse genes and a lower productivity than type-1 agents. Hence the IC effect implies a subsidy on earnings, on savings and on preventive expenditures for type-1 agents. Those results are summarized in Table 2, in column IC. Table 2 recalls also the effects of externalities (EXT) in the survival process and of myopia (MI) ($\alpha < 1$) on the tax, along with a column for the final outcome.

second-best		EXT	IC	MI ($\alpha < 1$)	Total effect
Case A:	σ_1	0	-	-	-
$w_2 \geqslant w_1$	σ_2	0	0	-	-
and $\varepsilon_1 \geq \varepsilon_2$	θ_1	-	-	-	-
	θ_2	-	0	-	-
	τ_1	0	+	0	+
	τ_2	0	0	0	0
Case B:	σ_1	0	+	-	?
$w_2 \geqslant w_1$	σ_2	0	0	-	-
and $\varepsilon_1 < \varepsilon_2$	θ_1	-	+	-	?
	θ_2	-	0	-	-
	τ_1	0	+	0	+
	τ_2	0	0	0	0
Case C:	σ_1	0	-	-	-
$w_2 < w_1$	σ_2	0	0	-	-
and $\varepsilon_1 > \varepsilon_2$	θ_1	-	-	-	-
	θ_2	-	0	-	-
	τ_1	0	-	0	-
	τ_2	0	0	0	0

Table 2: Signs of taxes in the second-best

Let us focus on Case B, which seems most plausible. The optimal second-

best policy requires a subsidy on savings and on preventive efforts as well as zero taxation of labour for type-2 agents (i.e. with a high productivity and good genes). This tax scheme is equivalent to what we had in the first-best for any agent and aims at correcting for both myopia and health externalities. As already mentioned, type-2 agents, who are the mimickers, will not face additional taxation related to the imperfect observability of agents' types. On the opposite, the IC effect leads to additional taxation of type-1 agents (i.e. with a low productivity and bad longevity genes). The second-best policy now involves a tax on the earnings of type-1 agents. Contrary to what we had in the first-best, the subsidy on the savings for type-1 agents cannot be signed with certainty, as the IC effect and the myopia effect go in opposite directions. In the same way, while the misperception and the externality push for subsidizing preventive efforts, the IC effect supports a tax, so that one cannot say whether preventive expenditures should be taxed or subsidized for type-1 agents.

In sum, Table 2 illustrates that the introduction of asymmetric information tends to qualify some policy conclusions drawn in the first-best setting, in particular regarding the fiscal treatment of preventive expenditures. Whereas the subsidization of preventive efforts is, under $\alpha < 1$, definitely required in a first-best setting, the imperfect observability of agents' type introduces a motive for taxing preventive efforts, under the (plausible) assumptions of a positive correlation of productitivies and longevity genes, and of a complementarity between genes and preventive effort in the survival process. Hence, it is no longer obvious, in a second-best world, that preventive expenditures should be subsidized, despite the existence of myopia and of positive health externalities.

The signs of several taxation instruments being ambiguous, it is useful, in order to be able to draw more precise policy conclusions, to complement our analysis by numerical simulations under particular functional forms for individual utility and survival process. That task is carried out in the next section.

5 A numerical application

Let us now explore which optimal values policy instruments take under plausible functional forms for $\pi(\cdot)$, $u(\cdot)$ and $v(\cdot)$. This numerical section will also give us the opportunity to examine how the size of the various phenomena related to prevention, such as myopia and externalities, influence the optimal subsidy on preventive efforts.[31] For that purpose, we focus here on the standard case where preventive efforts and genes are complements in the production of longevity, and

[31] As it is stressed in Section 1, preventive efforts can take various forms, some of these giving rise to externalities, whereas others do not. Here we explore how those characteristics of preventive efforts affect the optimal subsidization of these.

where genes and productivities are positively correlated.[32]

5.1 Functional forms

To select a survival function $\pi \equiv \pi(e_i, \bar{e}, \varepsilon_i)$, note first that the individual and total levels of preventive expenditures, respectively e_i and $\bar{e}$, enter the survival function as standard inputs, for which it makes sense to talk about an elasticity of life expectancy $1 + \pi$ with respect e_i and $\bar{e}$, as these are easily observable. On the contrary, it does not seem to be equally meaningful to talk about an elasticity of life expectancy with respect to the genetic background, because a mere unit of measurement is not straightforward to establish for that variable. This lack of measurement unit for genetic background raises identification problems.[33] Therefore, to avoid identification problems, we will here treat ε_i as a scale parameter, which explains longevity inequalities between agents equal on all other aspects (i.e. for whom $e_1 = e_2$). Thus we shall, for simplicity, assume a survival function that has a constant elasticity with respect to individual preventive expenditure e_i and total preventive expenditure $\bar{e}$, while treating the genetic background as a scale parameter:

$$\pi(e_i, \bar{e}, \varepsilon_i) = \varepsilon_i A e_i^{\rho} \bar{e}^{\phi}$$

A is a scale parameter common to all agents (such that $0 \leq \pi_i < 1$), ρ is the elasticity of π with respect to e_i, and ϕ is the elasticity of π with respect to $\bar{e}$.[34]

Regarding $u(\cdot)$, we assume the following isoelastic form:

$$u(c_i) = \frac{c_i^{1-\kappa}}{1-\kappa} + \gamma$$

where κ is the coefficient of relative risk-aversion, or the inverse of the elasticity of intertemporal substitution, while γ is the pure welfare gain from survival.

Moreover, we assume also, for simplicity, a quadratic disutility from labour:

$$v(l_i) = \frac{l_i^2}{2}$$

Note that the chosen functional forms for $\pi(\cdot)$, $u(\cdot)$ and $v(\cdot)$ are not the unique possible ones, and that this section does not have the pretension to cover all possible cases. On the contrary, we just want to give an idea of what the optimal policy is under plausible survival functions and preferences.

[32] Discussions on other cases are left for Section 6.

[33] Actually, it is difficult to know whether longevity inequalities induced by unequal genetic backgrounds are due either to a tiny genetic differential with a large reactivity of survival to genes, or to a large genetic differential under a low reactivity of survival to genes.

[34] Note that the postulated survival function involves a complementarity of e_i and ε_i: $\pi_{e\varepsilon}(e_i, \bar{e}, \varepsilon_i) = A\rho e_i^{\rho-1}\bar{e}^{\phi} > 0$. We also have $\pi_{\bar{e}\varepsilon}(e_i, \bar{e}, \varepsilon_i) = A e_i^{\rho}\phi\bar{e}^{\phi-1} > 0$.

5.2 Calibration

In this subsection, we first calibrate $\pi(\cdot)$ on the basis of empirical work on genetic background as a determinant of longevity inequalities, and on the basis of plausible conjectures regarding the maximum life expectancy. Then, we shall use empirical material on the value of a statistical life (VSL) to calibrate $u(\cdot)$.

We shall consider here two types of agents, i.e. type-1 and type-2, with respectively a "bad" and a "good" genetic background (i.e. $\varepsilon_1 < \varepsilon_2$), and with low and high productivities (i.e. $w_1 < w_2$).[35] Following the works by Christensen *et al* (2006), we know that about 25 % of longevity inequalities within a given cohort can be explained by differences in genetic background. Thus, comparing the longevities of type-1 and type-2 agents, we have:

$$\frac{\pi_2}{\pi_1} = \frac{\varepsilon_2 A e_2^{\rho} \bar{e}^{\phi}}{\varepsilon_1 A e_1^{\rho} \bar{e}^{\phi}} = \left(\frac{e_2}{e_1}\right)^{\rho} \left(\frac{\varepsilon_2}{\varepsilon_1}\right) = 1 + k$$

where k is the relative inequality between the survival prospects of agents of types 2 and 1. Given that ε_i explains 25 % of longevity inequalities, we have[36]

$$\frac{\pi_2}{\pi_1} = \frac{\varepsilon_2 A e^{\rho} \bar{e}^{\phi}}{\varepsilon_1 A e^{\rho} \bar{e}^{\phi}} = \frac{\varepsilon_2}{\varepsilon_1} = 1 + \frac{1}{4}k$$

Normalizing ε_1 to 1 yields:

$$\varepsilon_2 = 1 + \frac{1}{4}k$$

The level of k depends on the groups under comparison. If, for instance, one compares men and women, then k depends on the differential in life expectancy at age 65 between those two groups. According to Cambois *et al* (2008), the life expectancy in France for men at age 65 equals 17.1 years, that is, in our model where a period lasts 40 years, we have $\pi_1 = 17.1/40 = 0.4275$ period. For women, life expectancy at age 65 equals $\pi_2 = 21.5/40 = 0.5375$ period. Hence we have $\frac{0.5375}{0.4275} = 1 + k$ so that $k = 0.2573$. Therefore, ε_2 should be here equal to 1.0643.

In order to calibrate A, ρ and ϕ, we consider the maximum life expectancy that can be reached, i.e. the level of life expectancy when the prevention investment variables take their maximum values. Note that the level of the maximum feasible life expectancy remains a matter of debates among demographers. Some of these, such as Olshansky and Carnes (2001), consider that life expectancy at birth will hardly exceed 88 years even in a distant future. On the contrary, Oeppen and Vaupel (2002) consider that future life expectancy may significantly exceed that level. Here we shall adopt an intermediate postulate. Assuming a maximum life

[35] We also assume that type-1 and type-2 groups have an equal size: $n_1 = n_2 = 0.5$.

[36] Here we assume $e_1 = e_2 = e$, as this is a *ceteris paribus* comparison.

expectancy at age 65 equal to 30 years for men, it follows, under a maximum individual income w_i normalized to 1 (so that $e^{\max} = \bar{e}^{\max} = 1$), that we have:

$$\frac{30}{40} = 0.75 = A1^{\rho}1^{\phi} \implies A = 0.75$$

As far as the elasticities ρ and ϕ are concerned, it is not obvious to have precise empirical estimates of the importance of individual efforts *versus* social, environmental variables on human longevity. As emphasized by demographers, the two kinds of factors are clearly at work. Note that there exists no precise estimate of the size of preventive expenditures, but only of total health spending. Given that total health spending represent about 10 % of the endowment per person in countries such as France or Germany (see OECD, 2009), we can use that piece of information as a proxy to define a range of plausible values for ρ and ϕ on the basis of current life expectancy levels:

$$0.4275 = 0.75\,(0.1)^{\rho+\phi} \implies \rho + \phi \simeq 0.25$$

We shall thus provide optimal policy results for various levels of those elasticities ρ and ϕ, with a sum equal to 0.25.

Regarding the calibration of κ and γ in $u\,(\cdot)$, we can first compute the VSL:[37]

$$VSL_i = \frac{\frac{\partial U_i}{\partial \hat{\pi}_i}}{\frac{\partial U_i}{\partial c_i}} = \frac{u(d_i) - d_i u'(d_i)}{u'(c_i)}$$

Under a VSL equal to about 150 times the annual income per head (see Miller, 2000), we have a VSL equal here to $\frac{150}{40} w_i = 3.75$. Hence, if c_i equals about 0.7, and d_i equals about 0.2, we have, after substituting for $u(\cdot)$:

$$3.75 = \frac{\frac{\kappa}{1-\kappa}(0.2)^{1-\kappa}}{(0.7)^{-\kappa}} + \frac{\gamma}{(0.7)^{-\kappa}}$$

From which we can derive κ and γ. For instance, under $\kappa = 0.83$ (Blundell *et al*, 1994), we have $\gamma = 1.329$. Using $\kappa = 0.83$ may be problematic given our periods of 40 years, so that we can, alternatively, use $\kappa = 0.50$, implying $\gamma = 4.038$. We will thus provide estimates for $(\kappa, \gamma) = (0.83, 1.33)$ and $(0.50, 4.04)$.

Finally, one should notice that our calibration was made independently from parameters α_i, so that we do not have so far any clue regarding the values of those

[37] Note that we consider here the marginal rate of substitution between individual consumption and subjective survival probability $\hat{\pi}_i$, as the agent bases his willingness-to-pay on his own knowledge of the survival process, and not on the actual one. This is the reason why the misperception parameter α_i does not enter that VSL formula.

parameters. It is likely, in the light of the widespread feelings of regrets in the field of health-affecting decisions (see Slovic, 2001, Jarvis *et al*, 2002, Fong *et al*, 2004), that α_i is lower than 1. This conjecture is confirmed by the literature on the gap between objective and subjective life expectancies, which shows that young agents underestimate their survival prospects (see Ludwig and Zimper, 2007). However, there is no clear evidence on the precise value of α_i, and this is why we shall provide policy estimates for a wide range of α_i, from 0 to 1. We shall also, for simplicity, assume here that all agents are equally myopic, i.e. $\alpha_1 = \alpha_2 = \alpha$.[38]

Table 3 summarizes the values for the parameters of our model.

A	ρ	ϕ	ε_1	ε_2	κ	γ	$\alpha_1 = \alpha_2$	w_2	w_1
0.75	$[0, 0.25]$	$[0, 0.25]$	1.00	1.06	$[0.50; 0.83]$	$[1.33; 4.04]$	$[0, 1.00]$	1.00	0.80

Table 3: Parameter values

Before considering our results, it should be stressed that our calibration is not the unique possible one, and that there exist other techniques by which one could assign numbers to our parameters on the basis of empirical data. Thus this numerical exercise is purely illustrative, and has no pretension to exhaustiveness.

5.3 Results

Let us first study the sensitivity of the optimal tax instruments to the degree of rationality of agents, under the benchmark assumptions $\phi = \rho = 0.125$ and $(\kappa, \gamma) = (0.83, 1.33)$. For that purpose, Table 4 shows the optimal taxes on saving, preventive expenditures and labour under different levels of α.

$\alpha_1 = \alpha_2$		σ_1	σ_2	θ_1	θ_1 (Δ=0)	θ_2	θ_2 (Δ=0)	τ_1	τ_2
1	FB	0.000	0.000	-0.500	0.000	-0.500	0.000	0.000	0.000
	SB	0.004	0.000	-0.450	0.004	-0.500	0.000	0.020	0.000
0.9	FB	-0.100	-0.100	-0.550	-0.100	-0.550	-0.100	0.000	0.000
	SB	-0.102	-0.095	-0.551	-0.102	-0.547	-0.095	0.022	0.000
0.5	FB	-0.500	-0.500	-0.750	-0.500	-0.750	-0.500	0.000	0.000
	SB	-0.518	-0.482	-0.759	-0.518	-0.741	-0.482	0.029	0.000

Table 4: Optimal taxes levels

Before going into the results, let us first explain the difference between the columns θ_i and $\theta_i\,(\Delta = 0)$. Column θ_i gives the level of the tax on preventive

[38] Ideally, one needs to consider the existence of a large heterogeneity on the level of α_i in the population. This is left for future research.

expenditures, while column $\theta_i\,(\Delta = 0)$ gives this level if the externality effect were to be zero. To see this, let us assume that there is no myopia (first two rows). In this case, the preventive expenditure tax / subsidy is only determined by the externality effect in the first-best, and by both the externality and the incentive effects in the second-best. Hence, in the first-best, the subsidy faced by type-1 agents is equal to 0.5, while, if there was no externality ($\Delta = 0$), the subsidy would be zero. In the second-best, θ_1 is equal to -0.450, while if there was no externality, we would have a tax equal to 0.004. These findings are in line with Case B results in Table 2. For any $\alpha_i < 1$, column $\theta_i\,(\Delta = 0)$ includes the effect of myopia in the first-best, as well as of the incentive constraint, in the second-best.

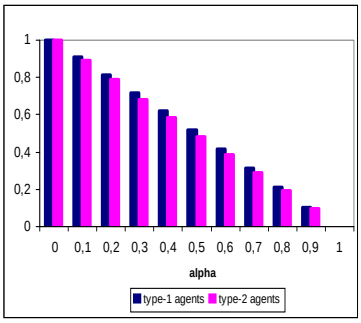
Table 4 shows that type-2 agents should face a subsidy on savings, so as to correct for myopia, as well as a subsidy on preventive expenditures, so as to correct for both the externality and for myopia. He faces no additional distortions in the second-best, as he is the mimicker.[39] Savings of type-1 agents should be subsidized, except when $\alpha = 1$. Hence, the myopia effect (which supports subsidization) always dominates the incentive effect (which supports taxation) in the second-best. Preventive efforts for type-1 agents are subsidized in the first-best, so as to correct for myopia and the externality effect. In the second-best, this is also the case, and the IC effect, which supports taxation, is not high enough to offset the myopia and the externality effects.[40] Labour is always taxed in the second-best, to solve the incentive problem. This illustrates the theoretical results of Table 2.

The figures below show the optimal saving subsidies, preventive expenditure subsidies and lump-sum transfers as a function of α, for agents of types 1 and 2, under asymmetric information. Saving and prevention subsidies are quite similar for all agents, and positive. Subsidies on savings and on preventive expenditures are always higher for type-1 agents than for type-2 agents, when $\alpha \in]0, 1[$.[41] Note that the optimal subsidies decrease in the degree of rationality α. This is due to the fact that, as agents become less myopic, laissez-faire preventive efforts and savings tend to their optimal levels, so that public intervention is less necessary. Note also that, when $\alpha \to 1$, the optimal preventive effort subsidy remains positive and high. Thus, the case for a large subsidization of preventive expenditures still holds under full rationality, because of externalities. In any case, the incentive constraint (which supports taxation) has only a second-order effect. Finally, regarding transfers, type-2 agents, who have better genes and a higher productivity, are, for any α, the beneficiaries of the redistribution in the second-best.

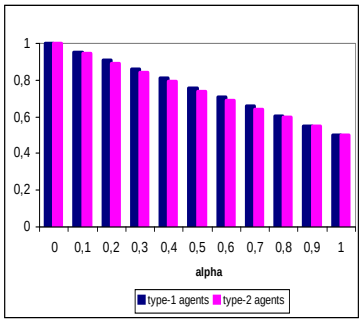
[39] The gap between first- and second-best σ_2 and θ_2 under $\alpha < 1$ is due to the fact that the incentive constraint includes α, unlike the planner's objective function. Hence expressions for second-best σ_2 and θ_2 include a term related to the incentive constraint (see Appendix).

[40] See the intersection of row $\alpha_i = 1$ with column $\theta_1\,(\Delta = 0)$ in the second-best.

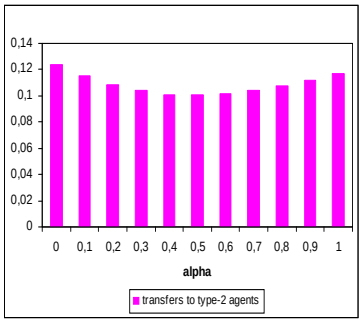
[41] Note that when $\alpha = 0$, $\sigma_i = \theta_i = 1\ \forall i$, while for $\alpha = 1$, $\sigma_1 = -0.004 < \sigma_2 = 0$ and $\theta_1 = -0.498 < \theta_2 = -0.500$.



Optimal saving subsidy



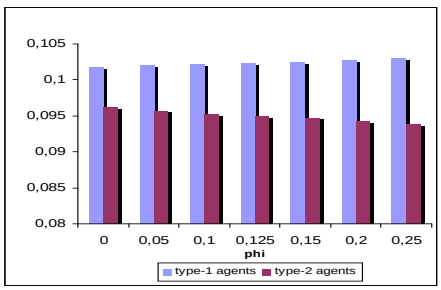
Optimal effort subsidy



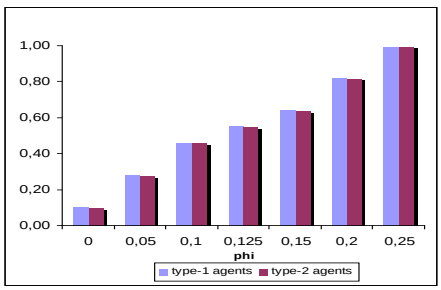
Optimal transfers

In the light of that analysis, policy instruments are found to be highly sensitive to the degree of myopia of agents. Nonetheless, to discuss the sensitivity of the optimal policy to other structural parameters of the economy, we will fix α to a single value. For that purpose, we will rely on the empirical evidence suggesting that agents' subjective life expectancy $1 + \hat{\pi}$ does not differ substantially from their actual life expectancy $1 + \pi$, and we will thus assume $\alpha = 0.9$.[42] That calibration is compatible with Ludwig and Zimper (2007), who show that agents tend to underestimate life expectancy.

Let us now focus on the sensitivity of the optimal policy to the elasticities of life expectancy with respect to private and average preventive expenditures. For that purpose, we still assume $\kappa = 0.83$ and $\gamma = 1.33$, and we make ϕ and ρ vary, in such a way as to have $\phi + \rho = 0.25$, in conformity with our calibrations.



Optimal savings subsidy



Optimal prevention subsidy

The higher ϕ is, the higher the savings subsidy for type-1 agents is, and the lower the savings subsidy for type-2 agents is. Moreover, the higher ϕ is, the higher

[42] On the objective and subjective life expectancies, see Hamermesh (1985).

the optimal preventive effort subsidies are, for all agents. Optimal preventive expenditure subsidies are also more sensitive to ϕ than savings subsidies.

Before going into the details, let us first show how the incentive effect varies when ϕ increases. We found, in unreported simulations, that, when ϕ increases, first-best consumption and labour supply remain constant, while e_1 increases, and e_2 decreases. Hence, the incentive constraint becomes more binding: type-2 agents would gain more from mimicking type-1 agents. The incentive effect in σ_1, θ_1 and τ_1 should then increase, and saving and prevention subsidies for type-1 should decrease, while his tax on labour should increase.[43] For type-2 agents, i.e. the mimickers, there should be no incentive effect.

When ϕ increases, the saving subsidy increases for type-1 agents and decreases for type-2 agents. The reason is that α appears in the incentive constraint (and leads to more subsidization), so that type-1 agents' optimal savings subsidy increases with ϕ when $\alpha < 1$.[44] Type-2 agents saving subsidy is also influenced by a term related to the incentive constraint. Hence, if $\alpha = 1$, σ_2 equals 0, and, in this case, a variation in ϕ has no impact, but, when $\alpha < 1$, the saving subsidy decreases with ϕ.

Regarding the preventive effort subsidy, an increase in ϕ raises the incentive effect in θ_1, so that we observe a decrease in the preventive effort subsidy for type 1. However, as ϕ increases, the externality effect, which pushes toward subsidization, also increases. This is due to the fact that the higher ϕ is, the higher the return of the total preventive effort for the society is, so that there is more need for correcting the externality effect. As we see from the graph, when all effects are taken into account, the externality effect, together with myopia, dominates the incentive effect, so that θ_1 is increasing in ϕ. For type-2 agents, when ϕ increases, θ_2 increases so as to correct for the increasing externality effect described above.

Finally, let us study the robustness of the optimal tax policy to preference parameters (κ, γ).[45] Table 5 shows that, in the first-best, the savings and the preventive expenditure subsidies are equal for any $(\kappa,\ \gamma)$. It is surely the case that optimal values of consumptions and preventive expenditures are different under the two specifications, but still the distortions due to myopia and the externality are of the same magnitude.[46] On the contrary, these are modified in the second-best,

[43] The tax on labour for type-1 agents (which is not reported here) is increasing with ϕ.

[44] When ϕ increases, the incentive constraint becomes more binding, so that the Lagrange multiplier associated with the incentive constraint, λ, increases, which, under $\alpha = 0.9$, leads to an increase in the subsidy on savings (equivalently σ_1 decreases). See the Appendix.

[45] As before, we keep the assumptions $\alpha_i < 1$ and $\phi = \rho = 0.125$.

[46] To see this, take the expressions for the first-best levels of σ_i and θ_i of Section 3.2. Under the two alternative specifications of $(\kappa,\ \gamma)$, the parameter α is the same, and it happens that $\Delta/u'(c_i)$ takes the same value. Moreover, the parameters $(\kappa,\ \gamma)$ do not directly enter the tax formulas, so that they have to be the same under the two specifications.

as a change in preferences affects second-best distortions through the incentive effect. Hence, for both specifications of (κ, γ), we should have $\sigma_2 = -0.10$ and $\theta_2 = -0.55$, both in the first-best and in the second-best, as type-2 agents (the mimickers) do not face distortions other than the ones related to MI and EXT in the second-best. As we already mentioned, the small differences between the two specifications are due to the presence of α in the incentive constraint, while it is absent from the objective function of the social planner. For this reason, subsidies on savings and on preventive efforts are increasing for type-2 when κ decreases. If α were equal to 1, σ_2 would be 0 and θ_2 would be equal to 0.5, both under symmetric and asymmetric information and for any (κ, γ).

Regarding σ_1 and θ_1, the incentive effect is modified following the variation of (κ, γ). To see this, first notice that, in the first-best, the differences between e_1 and e_2 are comparable under the two specifications, while the differences in labour are smaller for $\kappa = 0.50$ than for $\kappa = 0.83$. The incentive effect, which supports taxation of saving, preventive effort and labour of type-1 agents, is then smaller, and we obtain higher saving and prevention subsidies and smaller taxation of labour. Indeed, looking at Table 5, τ_1 is decreasing. Concerning σ_1 and θ_1, this is what we obtain under $\alpha = 1$. However, as we already discussed, if $\alpha < 1$, this may not be the case. Indeed, a less binding incentive constraint also implies that λ is smaller, and for $\alpha = 0.9$, σ_1 decreases with λ, so that the subsidy on savings is smaller (from -0.1023 to -0.1019). The same reasoning holds for θ_1, and this explains why the prevention subsidy is also smaller.

κ		σ_1	σ_1 (α=1)	σ_2	σ_2 (α=1)	θ_1	θ_1 (α=1)	θ_2	θ_2 (α=1)	τ_1	τ_2
0.83	FB	-0.1000	0.0000	-0.1000	0.0000	-0.5500	-0.5000	-0.5500	-0.5000	0.0000	0.0000
0.50		-0.1000	0.0000	-0.1000	0.0000	-0.5500	-0.5000	-0.5500	-0.5000	0.0000	0.0000
0.83	SB	-0.1023	0.0040	-0.0950	0.0000	-0.5508	-0.4980	-0.5470	-0.4990	0.0220	0.0000
0.50		-0.1019	0.0030	-0.0960	0.0000	-0.5507	-0.4980	-0.5480	-0.4990	0.0170	0.0000

Table 5: Sensitivity of policy instruments to the utility function parameters

All in all, those numerical simulations illustrate the sensitivity of the optimal policy to the fundamentals of the economy. In particular, the degree of rationality of agents plays an important role, both directly and indirectly (as it influences also the incentive effect).[47] This section has also shown the dependence of the optimal policy on survival and utility functions. However, it should be kept in mind that our analysis was made under the assumption of complementarity between preventive expenditures and genes, and considered only the case of positive correlation between genes and productivity. In the following section, we will discuss

[47] We found that it could even reverse the results related to the IC effect.

the implications of those assumptions, and of some others.

6 Discussions

Let us turn back to the three assumptions that play a key role in our model. The first crucial assumption concerns the distributions of w and ε in the population, the second assumption concerns the degree of substitutability between ε and e in the production of longevity, and the third assumption consists of the social criterion used (classical utilitarianism).

6.1 The productivity / genes correlation

Concerning the first assumption, there is little doubt that, in the real world, there exists a large number of types regarding the characteristics w and ε. Agents exhibiting a high productivity do not necessarily benefit from good longevity genes, and agents with bad longevity genes may nonetheless turn out to be highly productive. Hence, under two sources of heterogeneity, we should ideally consider four types of agents (leaving misperception aside).

Nonetheless, for reason of analytical treatment, we limited ourselves here to two types of agents, and, under this restriction, we argued that the most realistic case is, at first glance, the existence of a positive correlation between the characteristics ε and w (i.e. ruling out Case A). Further, to the extent we want type-2 agents to mimic type-1 agents, the most realistic case is the one with positive correlation between ε and w and the productivity gap larger than the genetic gap (with complementarity), that is, Case B.

While those restrictions - and the resulting emphasis on Case B - facilitate the exposition of the problem, it should be stressed here that assumptions on the relationship between productivity and longevity genes are not straightforward to make. The reason why those assumptions are somewhat fragile lies mainly in the difficulty to identify what we call the 'longevity genes'.[48] The genetic background is so large that it is not obvious to see what 'longevity genes' consist of, and the precise definition of those 'longevity genes' may affect the plausibility of the different cases. One cannot rule out *a priori* that some longevity genes are positively correlated with productivity, while others are negatively correlated with longevity, so that assumptions at the aggregate level should be made with caution.

Thus, even though Case B is, at first sight, the most plausible one, other cases should not be ignored, as these may arise in particular economic environments, and would thus involve a distinct optimal policy.

[48] On this difficulty, see Christensen *et al* (2006).

6.2 The production of longevity

Regarding the degree of substitutability between e and ε, we believe that the assumption of complementarity is the most realistic one. This means that having good genes implies that for the same increment of preventive expenditure, one increases one's survival probability more than someone with bad genes.

Having stressed this, it should be underlined that substitutability, although less plausible, is far from impossible. To illustrate this, take the example of two agents endowed with a different metabolism and striving to achieve an ideal weight. For the one with a favorable metabolism, a little effort allows him to reach the ideal weight. However, for the other agent, even with enormous efforts, it will be impossible. In the light of that example, the case for complementarity is not as strong as it may appear at first sight.[49] Given that the empirical testing of those two assumptions can hardly be made (because of the difficult identification of 'longevity genes'), it makes sense to explore the policy consequences of departing from complementarity of ε and e. For that purpose, Table 6 contrasts the tax rates under substitutability and complementarity for Case B (i.e. positive correlation of w and e and the productivity gap dominating the genetic gap).

Table 6: Complementarity versus substitutability

Case B: positive correlation (w_i, ε_i)		EXT	IC	MI $(\alpha < 1)$	Total effect
Complementarity	σ_1	0	+	-	?
	σ_2	0	0	-	-
	θ_1	-	+	-	?
	θ_2	-	0	-	-
	τ_1	0	+	0	+
	τ_2	0	0	0	0
Substitutability	σ_1	0	+	-	?
	σ_2	0	0	-	-
	θ_1	-	-	-	-
	θ_2	-	0	-	?
	τ_1	0	+	0	+
	τ_2	0	0	0	0

As one can see from Table 6, shifting from complementarity to substitutability only influences the IC effect for θ_1, as it only affects the ratio $\pi_e(e_1, \bar{e}, \varepsilon_1) / \pi_e(e_1, \bar{e}, \varepsilon_2)$.

[49] Actually, that example supports some substitutability of genes and effort in the production of longevity, for which a general expression is given by the following CES function: $\pi(e, \varepsilon) = (e^{\upsilon} + \varepsilon^{\upsilon})^{\chi/\upsilon}$, where $\upsilon \leqslant 1$ and $\chi < 1$, and where we assume away the externality factor $\bar{e}$.

Thus, we now have a negative sign for the influence of the IC on θ_1, whereas it was positive with complementarity. Hence, under substitutability between genes and preventive expenditures, the incentive constraint gives an additional reason for subsidizing preventive efforts of type-1 agents. Note that, in Cases A and C, under substitutability between genes and preventive expenditures, the incentive constraint now pushes towards taxation of preventive expenditures. All in all, taxation of preventive efforts may be second-best optimal under specific assumptions on the correlation between preventive efforts and genes and on the relation between productivity and genes.

6.3 Classical utilitarianism

Finally, there is a key assumption that we have made up to now, and which has some bearing on the optimal policy: it is the classical utilitarian objective function. When combined with the standard additive lifetime welfare assumption, classical utilitarianism imposes a redistribution from short-lived to long-lived individuals. Such a redistribution can be questioned, as it is not obvious to see why an individual should be penalized, in terms of total consumption, because he lives a shorter life than others.[50] Intuitively, one would prefer, on the contrary, to see short-lived agents 'compensated' for their short life, for which they are not responsible. Classical utilitarianism does not allow for such a compensation.[51]

In order to avoid the counter-intuitive redistributive bias in favor of long-lived agents, there is no other solution than to depart from the standard utilitarian optimization problem described above. This can be done in several manners. One solution is to modify the weighting of individual utilities.[52] Whereas classical utilitarianism weights equally the utility of all agents, and of all life-periods, one may depart from such a weighting of utilities in two ways. One can put a higher weight on the lifetime utility of the short-lived agents, or, alternatively, one can put a higher weight on the second-period utility of the short-lived agents.

For the sake of illustration, let us put a higher weight on the second-period

[50] For a criticism of that redistribution, see Bommier (2006) and Bommier *et al* (2009, 2010).

[51] The term 'compensation' presupposes the assumption that it is better, for a given amount of resources, to live a long life rather than a short life, so that short-lived agents are disadvantaged.

[52] Another solution, not explored in this paper, is to keep the standard utilitarian criterion, but do *as if* all agents had the same genetic background, i.e. fix $\varepsilon_i = \bar{\varepsilon}$ for all agents in the objective function, while keeping group-specific ε_i in incentive compatibility constraints. An alternative solution would consist of doing Maximin on lifetime utilities. This is left for future research.

utility of the short-lived agents.[53] Hence, at the first-best, the Lagrangian becomes:

$$\sum_{i=1,2} n_i \left[u\left(c_i\right) + \beta_i \pi\left(e_i, \bar{e}, \varepsilon_i\right) u\left(d_i\right) - v\left(l_i\right) \right.$$
$$\left. -\lambda\left(c_i + e_i + \pi\left(e_i, \bar{e}, \varepsilon_i\right) d_i - w_i l_i\right) \right]$$

where $\beta_2 = 1$ and $\beta_1 > 1$.

Under Case B, we have $c_1 = c_2$ and $l_2 > l_1$ as before. But now we have: $d_1 > d_2$. Type-1 agents consume more during the second period than type-2 agents. If we move to the second-best, those weights make the mimicking of type-1 by type-2 agents more attractive than without the modified weights. Therefore, introducing weights does not change the self-selection constraint, but reinforces it.

7 Conclusions

This paper proposed an optimal taxation approach to preventive expenditures. For that purpose, we studied the optimal non-linear taxation policy in a two-period economy where the probability of survival to the second period depends on preventive expenditures and on genetic background. Agents were heterogeneous on longevity genes, earnings capacity and the misperception of the survival process. The social planner was assumed to be of the classical utilitarian type and paternalistic (i.e. using the true - and not the perceived - survival probabilities).

When the social planner observes individual characteristics, the decentralization of the social optimum requires Pigouvian actions: a subsidy on preventive expenditures, to internalize positive health externalities, and to correct for the underestimation of survival prospects, and a subsidy on savings, also to correct for myopia.[54] Those subsidies should only be differentiated according to the degree of myopia of agents, but not according to individual productivities or genes.

In the second-best, beyond those Pigouvian actions, the non-observability of genes and productivity involves a self-selection constraint leading to an optimal tax or subsidy on preventive expenditures, savings and earnings for mimicked agents, depending on the relations between non-observable characteristics. The optimal subsidy on preventive expenditures is here dependent on individual productivities and genes (and not only on agents' misperception). As a consequence, the optimal second-best subsidy on preventive expenditures depends on the combined effects of misperception, external effects and self-selection. Take, for instance, the

[53] Here we rely on that approach for conveniency, even though it violates Pareto efficiency. However, with that approach, first-period consumption is equal across agents, and the impact of unequal weights is restricted to second-period consumptions, which is intuitive.

[54] In case of overestimation of survival prospects, a tax would be required instead.

benchmark case of a positive correlation with the productivity gap dominating the genetic gap, and of a complementarity between preventive effort and genetic endowment. Without misperception, the preventive expenditures of *(high productivity, good genes)* agents should be subsidized, whereas the ones of *(low productivity, bad genes)* agents should be either subsidized or taxed, depending on the strength of externalities with respect to the incentive constraint.[55] Moreover, we have a tax on earnings and on savings for *(low productivity, bad genes)* agents, but not for other agents. Finally, once myopia is introduced, there is an extra support for subsidizing preventive expenditures and savings of all agents.

All in all, a major contribution of our study is to show that the optimal subsidization of preventive expenditures consists of *one* aspect of the general optimal taxation problem, to be examined along with the optimal taxation of earnings and savings in a society of heterogeneous agents. More importantly, our analysis highlights determinants of the optimal tax policy that are usually absent from the taxation literature, such as the correlation between productivity and longevity-enhancing genes, and the degree of complementarity between genes and preventive expenditures in the production of longevity. Although generally unnoticed, those factors determine the optimal fiscal treatment of preventive expenditures in a world of imperfect observability of individual characteristics by governments.

While our results highlight how determinants of various kinds affect the optimal tax policy under endogenous longevity, this study had, for the sake of simplicity, to abstract from several other factors influencing or influenced by longevity. For instance, we abstracted here from education, which is a major determinant of longevity, and is also affected by it (see Cervellati and Sunde, 2005). Moreover, this study ignored fertility, whose level can be related to mortality (see de la Croix and Licandro, 2007). Furthermore, productivity differentials are taken here as exogenous, whereas productivity depends on health (see Bloom *et al*, 1998). In addition, this study relies on a static model, and leaves aside various dynamic arguments supporting subsidization of preventive expenditures.[56] Hence, our study should only be regarded as a first step, to be completed by others.

Finally, it cannot be overemphasized that our study, by relying on classical utilitarianism, suffers from the limits of that ethical framework in the context of varying longevity. The utilitarian bias in favor of agents with good genes is counter-intuitive. One would prefer agents with bad genes to be *compensated* for this handicap (for which they are not responsible). But utilitarianism, by relying on consequentialism, can hardly do justice to such responsibility-based intuitions. To account for that intuition, we considered an alternative social welfare function

[55] Note that numerical simulations suggest that the incentive constraint effect is of second order, so that preventive expenditures should be subsidized even for those agents.

[56] For dynamic studies on the optimal taxation under longevity changes, see Zhang *et al* (2006), Bloom *et al* (2007), Pestieau *et al* (2008) and Jouvet *et al* (2010).

in which more social weight is given to short-lived agents, which increases redistribution in their favor, but does not change the self-selection constraint. However, that slight departure from the Benthamite social welfare function invites a deeper exploration of the robustness of optimal policy to the postulated ethical framework. In the light of the well-known difficulties to accommodate utilitarianism with intuitions about responsibility and compensation, there can be no doubt that much work remains to be done in the future.

8 References

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9 Appendix

Second-best: centralised solution The Lagrangian is

$$\begin{aligned}\pounds &= \sum_{i=1,2} n_i \left[u(c_i) + \pi(e_i, \bar{e}, \varepsilon_i)\, u(d_i) - v\left(\frac{y_i}{w_i}\right) - \mu\left(c_i + e_i + \pi(e_i, \bar{e}, \varepsilon_i)\, d_i - y_i\right)\right] \\ &\quad + \lambda \left[u(c_2) + \alpha\pi(e_2, \bar{e}, \varepsilon_2)\, u(d_2) - v\left(\frac{y_2}{w_2}\right) - u(c_1) - \alpha\pi(e_1, \bar{e}, \varepsilon_2)\, u(d_1) + v\left(\frac{y_1}{w_2}\right)\right]\end{aligned}$$

where λ is the multiplier associated with the self-selection constraint.

The FOCs of this second-best problem are:

$$u'(c_1)\left[1-\frac{\lambda}{n_1}\right] = \mu \tag{25}$$

$$u'(c_2)\left[1+\frac{\lambda}{n_2}\right] = \mu \tag{26}$$

$$u'(d_1)\left[1-\frac{\alpha\pi(e_1,\bar{e},\varepsilon_2)}{n_1\pi(e_1,\bar{e},\varepsilon_1)}\lambda\right] = \mu \tag{27}$$

$$u'(d_2)\left[1+\frac{\lambda\alpha}{n_2}\right] = \mu \tag{28}$$

$$\begin{aligned}\pi_e(e_1,\bar{e},\varepsilon_1)\left[u(d_1)-\mu d_1\right] &= \mu+\frac{\lambda\alpha\pi_e(e_1,\bar{e},\varepsilon_2)\,u(d_1)}{n_1}\\ &\quad -\Sigma_j n_j\pi_{\bar{e}}(e_j,\bar{e},\varepsilon_j)\left[u(d_j)-\mu d_j\right]\end{aligned} \tag{29}$$

$$\begin{aligned}\pi_e(e_2,\bar{e},\varepsilon_2)\left[u(d_2)-\mu d_2\right] &= \mu-\frac{\lambda\alpha\pi_e(e_2,\bar{e},\varepsilon_2)\,u(d_2)}{n_2}\\ &\quad -\Sigma_j n_j\pi_{\bar{e}}(e_j,\bar{e},\varepsilon_j)\left[u(d_j)-\mu d_j\right]\end{aligned} \tag{30}$$

$$v'\left(\frac{y_1}{w_1}\right)\frac{1}{w_1}-\mu-\frac{\lambda}{n_1}v'\left(\frac{y_1}{w_2}\right)\frac{1}{w_2} = 0 \tag{31}$$

$$v'\left(\frac{y_2}{w_2}\right)\frac{1}{w_2}\left[1+\frac{\lambda}{n_2}\right]-\mu = 0 \tag{32}$$

Expressions (17)-(22) in Section 4 are deduced from those FOCs.

Second-best: tax formulas Equalizing (17) and (18) to (15), we obtain the following expressions for σ_1 and σ_2:

$$\sigma_1=\alpha\left[\frac{1-\lambda/n_1}{1-\lambda\frac{\alpha\pi(e_1,\bar{e},\varepsilon_2)}{n_1\pi(e_1,\bar{e},\varepsilon_1)}}\right]-1 \qquad \sigma_2=\alpha\left[\frac{1+\lambda/n_2}{1+\lambda\alpha/n_2}\right]-1$$

Equalizing (21) and (22) with (14), we obtain τ_1 and τ_2. Let now turn to the expressions of θ_1 and θ_2. Equalizing (16) with (19) and rearranging terms, we have

$$\begin{aligned}&\frac{1+\theta_2}{\alpha}+\frac{\pi_e(e_2,\bar{e},\varepsilon_2)\,u'(d_2)\,d_2}{u'(c_2)} = \left[1+\pi_e(e_2,\bar{e},\varepsilon_2)\,d_2\right]\left[\frac{1+\lambda/n_2}{1+\lambda\alpha/n_2}\right]\\ &-\frac{\Delta}{u'(c_2)(1+\lambda\alpha/n_2)}\end{aligned}$$

which yields

$$\begin{aligned}\theta_2 &= \alpha\left[1+\pi_e\left(e_2,\bar{e},\varepsilon_2\right)d_2\right]\left[\frac{1+\lambda/n_2}{1+\lambda\alpha/n_2}\right]-\frac{\Delta\alpha}{u'\left(c_2\right)\left(1+\lambda\alpha/n_2\right)}\\ &\quad -\alpha\frac{\pi_e\left(e_2,\bar{e},\varepsilon_2\right)u'\left(d_2\right)d_2}{u'\left(c_2\right)}-1\end{aligned}$$

Assuming that $\alpha=1$ and $\Delta=0$, one has

$$\theta_2=\pi_e\left(e_2,\bar{e},\varepsilon_2\right)d_2-\frac{\pi_e\left(e_2,\bar{e},\varepsilon_2\right)u'\left(d_2\right)d_2}{u'\left(c_2\right)}$$

and substituting for (26) and (28), $\theta_2=0$. Using the same procedure for θ_1, we equalize (16) with (20) such that

$$\begin{aligned}&\frac{1+\theta_1}{\alpha}+\frac{\pi_e\left(e_1,\bar{e},\varepsilon_1\right)u'\left(d_1\right)d_1}{u'\left(c_1\right)}=\left[1+\pi_e\left(e_1,\bar{e},\varepsilon_1\right)d_1\right]\left[\frac{1-\lambda/n_1}{1-\lambda\frac{\alpha\pi_e(e_1,\bar{e},\varepsilon_2)}{n_1\pi_e(e_1,\bar{e},\varepsilon_1)}}\right]\\ &-\frac{\Delta}{u'\left(c_1\right)}\left[\frac{1}{1-\lambda\frac{\alpha\pi_e(e_1,\bar{e},\varepsilon_2)}{n_1\pi_e(e_1,\bar{e},\varepsilon_1)}}\right]\end{aligned}$$

which yields

$$\begin{aligned}\theta_1 &= \alpha\left[1+\pi_e\left(e_1,\bar{e},\varepsilon_1\right)d_1\right]\left[\frac{1-\lambda/n_1}{1-\lambda\frac{\alpha\pi_e(e_1,\bar{e},\varepsilon_2)}{n_1\pi_e(e_1,\bar{e},\varepsilon_1)}}\right]-\frac{\Delta\alpha}{u'\left(c_1\right)}\left[\frac{1}{1-\lambda\frac{\alpha\pi_e(e_1,\bar{e},\varepsilon_2)}{n_1\pi_e(e_1,\bar{e},\varepsilon_1)}}\right]\\ &\quad -\frac{\alpha\pi_e\left(e_1,\bar{e},\varepsilon_1\right)u'\left(d_1\right)d_1}{u'\left(c_1\right)}-1\end{aligned}$$

Assuming that $\alpha=1$ and $\Delta=0$, and substituting for (25) and (27), we get

$$\theta_1=\left[\frac{1-\lambda/n_1}{1-\lambda\frac{\pi_e(e_1,\bar{e},\varepsilon_2)}{n_1\pi_e(e_1,\bar{e},\varepsilon_1)}}\right]-1$$