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Smoking, health, risk, and perception☆

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Abstract

We provide a description of health-related incentives faced by a rational smoker by considering the role of perception in both immediate quality-of-life effects of smoking and future risk of mortality. A person who adapts psychologically to a lowered health state, smokes more early in life and shifts demands for health investments and health-complementary activities later in life. He also smokes more in total. Someone aware of the full mortality consequences of smoking, smokes less and demands less medical care than someone who believes that these effects are highly reversible. The impacts of new information on mortality risk are most valuable early in life. Lastly, someone endowed with a longer life expectancy smokes more in the first part of life but conditional on access to medical care.

JEL classification: C61; D91; I12

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1. Introduction

Smoking is hazardous to your health. This fact features prominently both in discussions of public policy towards smokers and tobacco products, and in discussions of rational

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Nomenclature

Endogenous variables

A	addictive stock
C	consumption of cigarettes
D	smoking stock
H	objective health stock
K	subjective health stock
M	medical care
R	relaxation produced by cigarette consumption
U	utility
W	wealth
Z	consumption of non-addictive good
λ	shadow price of addictive stock
π	shadow price of money
γ	the value of a statistical life (in negative term)
η	shadow price of objective health
σ	shadow price of subjective health
ϕ	shadow price of smoking stock

Function symbols

F	health investment function
g,h	hazard functions
LU	lifetime utility
S	survivor function
V	current value Hamiltonian function
y	discount rate due to stochastic life time

Exogenous variables

m	price of medical care
p	price of cigarettes
q	price of non-addictive good
r	market interest rate
Y	income
β	parameter representing the degree of health adaptation
δ	depreciation rate of health
μ_A	depreciation rate of the addictive stock
μ_D	depreciation rate of the smoking stock
ρ	time preference rate

addiction (Becker and Murphy, 1988). But whereas rational addicts themselves are assumed to have precise, quantitative beliefs about the effects of smoking, rational addiction theories commonly bundle health effects into a reduced form along with all social, psychological and other effects.[1] This makes it unclear what we actually assume about tobacco's effects on the rational smoker's health and welfare.

This study focuses on three aspects of the relationship between smoking and health. First, we introduce the possibility of health investments in a rational addiction model. The specification, inspired by Grossman (1972a,b), allows us to explicitly study the dynamics of health damages from smoking and how they shape incentives to invest in health at different times in life. While aspects of the relationship between smoking and health have been studied in rational addiction models such as Suranovic et al. (1999), Goldbaum (2000) and Adda and Lechene (2001), none of these studies has focused on the ability of consumers to offset health damages of smoking by investing in health.

Our model also recognizes that individuals perceive two very different types of health effects—quality-of-life effects and death risk effects. These effects operate on the individual's well being through different channels and on different time scales.

The short-run, quality-of-life effects (such as compromised cardiovascular fitness and coughing) affect the individual's present well being directly by combining with smoking and other consumption in its instantaneous utility function. We focus in particular on whether it is the *level* of their general health that matters for the quality-of-life effects or the *transitions* between higher and lower health states. This is the second contribution of our analysis. Recent studies, e.g., Groot (2000), suggest that people have the ability to psychologically adapt to even very large changes in health. If so, these health effects are, in a sense, transitory and will have different implications for rational smoking and health investment trajectories. To our knowledge, no one has studied what this means for the choices that smokers make. Our framework is inspired by Gjerde et al. (2001), which presents a formal model of health adaptation based on Grossman (1972a).

Unlike quality-of-life effects, it is more difficult for individuals to know the increased mortality risks associated with their smoking habit.[2] Although one's current health state certainly provides some information about the likelihood of future health outcomes, the risk of getting lung cancer may also depend on other factors such as smoking history, which are more or less independent of one's observable health state. In fact, a frustrating

[1] Becker and Murphy (1988) assumed a known length of life and thus did not treat the effects of tobacco on death risks. However, they assumed a direct, negative effect on present utility of past consumption, which they interpret as tolerance, but this effect could also capture lagged health harms from tobacco (see Chaloupka, 1991; Skog, 1999; Suranovic et al., 1999).

[2] The first treatment of death risks in a rational addiction context was Goldbaum (2000), who subtracted a fixed number of minutes from the known, finite life of the rational smoker for each cigarette he smoked. This isolates the endowment effect, but the implication that smoking levels rise with potential lifetime lacked empirical support (see, e.g., Sterling and Weinkam, 1990; Smith and Shipley, 1991). A different approach was to introduce health effects in a model with stochastic lifetime (Adda and Lechene, 2001, and the appendix to Suranovic et al., 1999). In this way, smoking today raises the probability of dying at all points in the future. The longer you expect to live, the more life you can expect to lose from smoking, which allows us to bring the results back in line with empirical observations.

feature of many types of cancer is that they do not produce symptoms that would prompt someone to see a doctor until they are advanced beyond the stage at which they can be easily treated.

Instead, smokers rely on other signals such as government campaigns and epidemiological studies to warn them of these dangers. But even those who pay attention to these messages may remain uncertain about how exactly these effects function. Campaigns discouraging smoking typically say little about the *reversibility* of the long-term effects of smoking (i.e., how fast the risk of death will converge towards the risk of a non-smoker.) If death risk is believed to be highly reversible, then smokers might feel that they can safely continue to smoke as long as they quit "before it's too late." Smith et al. (2001) note this sort of ineffectiveness in anti-smoking campaigns. Far more significant, their empirical results suggest, are the changes in beliefs that a smoker undergoes in response to serious health shocks such as heart attacks. The third contribution of our paper is to model this process and consider the implications of receiving new information about the reversibility of smoking-related death risk.[3]

One of our more surprising results is that, along with the more obvious, negative health consequences of smoking, individuals may get health benefits in the sense that the act of quitting smoking serves as an investment in health as their smoking stock depreciates over time. This tends to give smokers an incentive to build their smoking stock up earlier in life and then quit in order to reap the benefits of recovery.

We find that smokers who are able to psychologically adapt to changes in health, smoke more overall because they are better able to accommodate the lowered health state that the habit implies. They demand fewer investments in health early in life but more than the equivalent non-adaptive smoker in later years. Complementarity between health and other consumption also plays role in shifting smoking activity earlier and demands for health and health-complements later in life. Because of this, the positive health benefits of smoking mentioned above may be particularly compelling for adaptive smokers.

Naturally, someone who believes that the death risk associated with smoking is irreversible, smokes less than one who thinks he can quit after an extended period of smoking and fully recover. Knowledge that risks are irreversible also tends to shift smoking toward the end of one's life in order to minimise the death risk consequences. New information about the health risks of smoking is most valuable early in life, as someone who has smoked heavily for much of his life has less to gain by quitting.

Lastly, we find that someone endowed with a longer life expectancy smokes more in the first part of life. However, this result rests on the ability of the individual to offset the effects of smoking through medical care, raising interesting questions about smoking and access to health care.

[3] One could formalise this source of uncertainty by establishing a probability distribution over different states of reversibility. In one extreme, smoking is completely reversible and does not depend on past smoking at all. In the other, every cigarette consumed counts equally towards determining one's accumulated death risk, regardless of how long ago it was consumed. In the numerical results that follow, we simply look at the consequences of discovering that the death risk is more or less reversible than expected, rather than explicitly trying to model this dimension of the expected utility problem.

Section 2 presents the model and analytical results. To illustrate the effects of health, risk, and perception more concretely, Section 3 describes a series of simulations from a numerical version of the model. Section 4 concludes.

2. The model[4]

The individual designs a consumption plan involving three market commodities – regular goods, cigarettes and medical care – to maximise expected lifetime utility from a stable instantaneous utility function defined over three goods – regular goods, relaxation and subjective health. Relaxation and subjective health are produced goods whose values depend on both past and present consumption choices.

2.1. The instantaneous utility function

Assume a rational agent with the following time-separable utility function:

$$U(t) = U(Z(t), R(t), K(t)) \tag{1}$$

Z is the non-addictive good (regular goods), R the relaxation or pleasure produced from cigarettes and K is the subjective (perceived) health, which will be explained below. The utility function is strictly concave in Z, R and K jointly. Since relaxation and subjective health are produced by the agent, they require further specification, as does a third magnitude – objective (measurable) health (H) – whose past and present values determine present subjective health.

2.1.1. Relaxation

As in Chaloupka (1991), relaxation is a function of current cigarette consumption, C, and past smoking captured by a stock variable, A[5]

$$R(t) = R(C(t), A(t)), \quad R'_C > 0, \quad R''_C < 0, \quad R'_A < 0, \quad R''_A < 0, \quad R''_{CA} > 0 \tag{2}$$

This specification identifies the common features of rational addiction:

- *Tolerance* in the sense of "reduced relaxation from a given amount of drug"; $R'_A < 0$.
- *Reinforcement*. The effect of cigarettes increases with past consumption ($R''_{CA} > 0$), a necessary condition for "adjacent complementarity", where higher future and past smoking increase present smoking (see Becker and Murphy, 1988).
- *Withdrawal effect*. Withdrawal has been interpreted as[6]

[4] A list of all symbols used is provided in the nomenclature.

[5] The relaxation function introduces weak separability: The marginal rate of substitution between C and A is now independent of Z, unlike the more general utility function of the Becker–Murphy model.

[6] Only the last of these interpretations is not explicitly included in our model.

- o utility reduction from reduced consumption (in Chaloupka, 1991); $U'_R > 0$ and $R'_C > 0$;
- o larger utility reduction from reduced consumption the larger the consumption stock (in Orphanides and Zervos, 1995); $U'_R > 0$ and $R''_{CA} > 0$;
- o adjustment costs (in Suranovic et al., 1999; Jones, 1999).

The addictive stock accumulates following (3), where a dot above a variable represents its derivative with respect to time, and μ_A is the constant depreciation rate:

$$\dot{A}(t) = C(t) - \mu_A A(t) \tag{3}$$

2.1.2. The development of objective health

We distinguish between an *objective measure* of the health status, *H*, and a *subjective measure* of the quality of health, *K*, following Groot (2000). The first is health as measured, for example, by a general physician. The second measure, *K*, is the individual's perception of the impact of health on his quality-of-life as specified in (1).

Objective health is influenced by medical care, cigarette smoking and age. It does not, however, include the unobservable effects of tobacco on mortality risk. These effects are described in Section 2.2.1.

The direct investments in health are captured by a continuous, twice differentiable household production function $F(M, \dot{D}; t)$ that is falling in $\dot{D}$, i.e., changes in aggregated smoking, and increasing in medical care, $M \geq 0$, a proxy for all market goods that improve health. The function is concave in both M and $\dot{D}$ jointly.[7] The investments are also a function of time, capturing the fact that damages done by cigarettes become more intensive with age (see Appendix B).

The effect of smoking on health has usually been combined with the addictive aspects of smoking, despite the fact that the effects of tobacco on health surely take place over a longer time horizon. We define a separate depreciation rate on the lagged health effects, μ_D, and lagged taste effects (relaxation) of smoking, μ_A. As in Adda and Lechene (2001), we assume $\mu_D < \mu_A$. We follow Suranovic et al. (1999) and Adda and Lechene (2001) in defining a smoking stock, *D*, whose change follows:

$$\dot{D}(t) = C(t) - \mu_D D(t) \tag{4}$$

As specified in the health investment function, we assume that it is *changes* in the smoking stock that affect health. Increasing smoking activity will gradually reduce health. However, this also captures the positive health effects of reducing smoking. Reduced smoking from a steady-state level is an investment in health; when the smoking stock shrinks, health is improved.

In addition to the two influences on health that are subject to the individual's control, the objective health stock also depreciates with age at depreciation rate $\delta(t)$, where $\dot{\delta}(t) > 0$ for

[7] As opposed to Grossman (1972a), we do not consider time as an input factor of health investments or consumption commodities. As in most of the rational addiction literature, we simplify and only consider the "pure consumption version" of Grossman's model (see Grossman, 1972b, Chapter 3).

all t. Thus, the development in H is as follows:

$$\dot{H}(t) = F(M(t), \dot{D}(t); t) - \delta(t)H(t) \tag{5}$$

2.1.3. The subjective health measure

Menzel et al. (2002) conceive of health adaptation as the alteration of activities, desires, goals and values in response to changes in health states, and distinguishes adaptation processes both from overcoming initial shocks and horror related to health state changes and the increased knowledge that arises from experiencing adverse health conditions. Though it has not been studied in the context of smoking, adaptation has been observed empirically following successful transplants (Adang, 1997), heart conditions (Wu, 2001), elderly persons' ability to manage stressful events (Cassileth et al., 1984) and in the evaluation of impaired health states by the general public compared to by patients (Sackett and Torrance, 1978).[8]

As in theories of habit formation (see, e.g., Boyer, 1982), an adaptable individual is more concerned about changes in the level of health than the absolute health state. A psychological recovery from a setback (Heyink, 1993) occurs as times passes. How long this recovery process takes depends on how the individual weights previous health changes when evaluating its subjective health state. Thus, following Gjerde et al. (2001), we model the subjective health variable K as:

$$K(t) = \frac{H_0}{1+\beta} + (1+\beta)\int_0^t e^{-\beta(t-s)}\dot{H}(s)\mathrm{d}s, \quad \beta \geq 0 \tag{6}$$

where $H_0 > 0$ is the initial health endowment. Thus, a higher $H(t)$, either through initial health or health improvements, has a positive impact on $K(t)$. To find a solution for the decision problem specified in Section 2.3 below, we need the derivative of K with respect to time:

$$\dot{K}(t) = \frac{\beta}{1+\beta}H_0 - \beta K(t) + (1+\beta)\dot{H}(t) \tag{7}$$

Our specification allows us to consider various degrees of adaptation to poor health. This degree is captured by β. To see this, consider the following cases as shown in Hoel and Isaksen (1994):

$$K(t) = H_0 + \int_0^t \dot{H}(s)\mathrm{d}s = H(t), \quad \text{for } \beta = 0 \tag{8}$$

$$\lim_{\beta\to\infty} K(t) = \dot{H}(t) \tag{9}$$

[8] As seen from these examples, adaptation has been studied in problems that may be defined as negative exogenous health consequences, and not following a decline in health due to a choice factor such as smoking. However, many changes in health including heart conditions and amputation (smoking leg) are due to life style, which is a matter of choice. Thus, in studying adaptation, it may not matter how the negative health change happened. The individual may adapt to negative health consequences of smoking, even if he knew in advance that smoking would be harmful.

There are three possible cases, see Fig. 1 taken from Gjerde et al. (2001):

Non-adapter: With $\beta = 0$ subjective health coincides with objective health, see (8). A shock to the health state permanently shifts the perception of health.

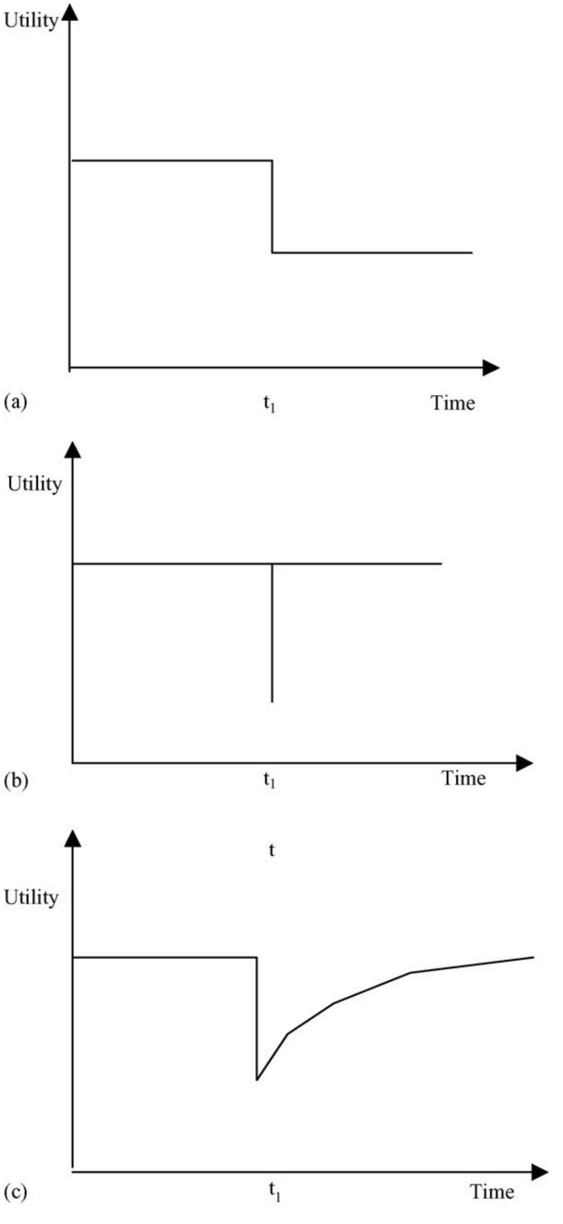

Fig. 1. Adaptation processes for different values of β: (a) $\beta = 0$, (b) $\beta \to \infty$, (c) $0 < \beta < \infty$.

Partial adapter: With $0 < \beta < \infty$ the impact of an innovation in the consumer's health on the agents perceived health dissipate over time, i.e., shocks to the health state have a persistent but depreciating effect on subjective health.
Perfect adapter: As $\beta \to \infty$ subjective health becomes equal to the current changes in objective health, see (9), and the individual suffers a loss only at the time of the negative health change.

2.2. Expected lifetime utility

The rational agent is forward-looking and aims to construct an optimal lifetime consumption plan with respect to his expected lifetime utility. This introduces three additional factors in his decision problem: time discounting, death risk and wealth accumulation. Time discounting is exponential, ensuring time-consistent plans.

2.2.1. Death risk

Our agent sees the death risk at any time as determined by the objective health at that time (H) and by past smoking (D) directly. It is this second effect that captures the unobservable component of death risks due to lung cancer and cardiovascular disease. These effects are so named because they are not directly observable by, for instance, a general physician in a routine doctor's visit.[9] Formally, the death risk is modelled using the hazard rate approach (conditional probabilities). The individual's beliefs about the probability of death at an arbitrary instant of time are represented by a hazard function $h = h(H(t), D(t))$, such that $h'_H < 0$. At high levels of H, $h''_{HH} > 0$, and at low levels, $h''_{HH} < 0$; a given reduction in health has little impact on the probability of dying when the health stock is very high, but has a larger impact when it is somewhat lower, and then decreases again as probability of death approaches certainty. Similarly, $h'_D > 0$. At low levels of D, $h''_{DD} > 0$, and at high levels, $h''_{DD} < 0$.

The hazard function is related to a survivor function, S (see Kiefer, 1988);

$$S(t) = \mathrm{e}^{-\int_0^t h(H(s), D(s))\mathrm{d}s} \tag{10}$$

The survivor function is the probability of living at time t seen from the initial date. It is a function of all hazard functions from the initial time and until t. Now define

$$y(t) = -\ln S(t) \tag{11}$$

This gives

$$\dot{y}(t) = -\frac{\dot{S}(t)}{S(t)} = h(H(t), D(t)) \tag{12}$$

[9] Note that "unobservable" is not meant to imply that the agent fails to take these effects into account in his optimal plan. It simply means that this aspect of the death risk cannot be known by observing one's current health state.

Thus, the hazard function can be interpreted as the probability of dying at the next point of time given that the individual is alive.

Moore (1996) summarises studies on smoking and health, and concludes “in most cases, the mortality ratios for former smokers approach those of never smokers after ten to fifteen years” (p. 417).[10] However, different beliefs about death risk may be reflected by the epidemiological literature or from public information campaigns. To stop people from taking up smoking, the dangers are often stressed in a way that makes the effects seem irreversible (e.g., “each cigarette shortens your life by 10 minutes”). Other campaigns focus on benefits of quitting, noting reductions in cancer risk etc., suggesting that health effects are highly reversible.

We model the different informational regimes that an individual might find himself in by changing the specification of the hazard function. Whereas (12) described the “true” process by which health and smoking stocks are assumed to contribute to the probability of death, we also consider a scenario in which the smoking stock, D, is omitted. The resulting change in the model structure is shown in (12′).

$$\dot{y}(t) = -\frac{\dot{S}(t)}{S(t)} = g(H(t)) \qquad (12')$$

In the numerical simulations, we interpret the scenarios that include the assumption described by (12) as those in which the agent has full information regarding these unobservable effects, and we call this assumption the “irreversible” case.[11] The “reversible” cases use the assumption described by (12′). This assumption represents one extreme with respect to an individual’s potential beliefs about the unobservable risks of smoking – he believes there are none. In this case, the individual sees only the health effects that are captured by the health investment function. The individual still pays a price of past smoking through the degree to which it has reduced past investment in the objective health stock. But if he were to quit today, his accumulated smoking stock would not continue to reduce his probability of survival as it would in the irreversible case.[12]

Inserting (12) into (10), we find

$$S(t) = \mathrm{e}^{-\int_0^t \dot{y}(s)\mathrm{d}s} = \mathrm{e}^{-y(t)} \qquad (13)$$

[10] More recently, while intensity of smoking is related to risks (Leffondré et al., 2002), Godtfredsen et al. (2002) show that reducing the intensity does not seem to alter risks. Adda and Lechene (2001) refer to studies showing irreversible effects on the lung function associated with an increased mortality risk, and also to other studies finding that risks faced by ex-smokers converge over time to those of non-smokers.

[11] The stock D depreciates over time, and in this sense the changes are reversible. They are also reversible in the sense that the agent can *compensate* for these risks by investing in objective health and reducing other risks of death. Unlike effects operating through the objective health stock, though, they are *irreversible* in the sense that the agent cannot choose to reduce them more quickly by *undoing* the effects. He simply has to wait for the smoking stock to depreciate naturally through time.

[12] A referee points out that an equally valid comparison would be between an individual who knows (12) and one who has beliefs that represent an *overestimate* of the death risk consequences of smoking. This would produce symmetric results to those we present in Section 3. Our point is not to suggest that people always underestimate the consequences of smoking, but to simply characterise the changes in the time paths of smoking and medical care that would result from a change in beliefs.

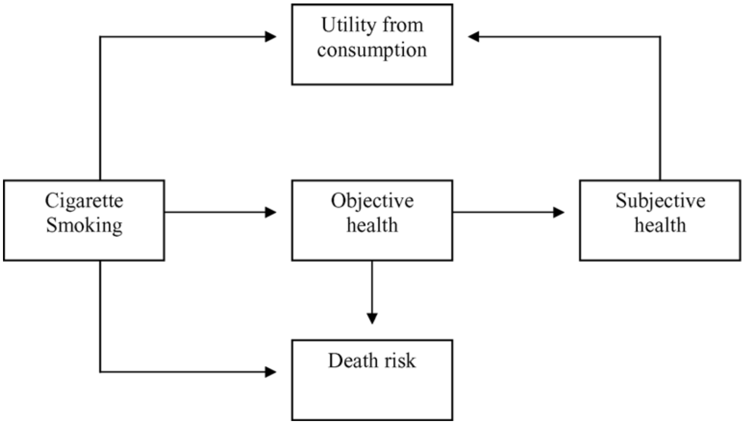

Fig. 2. Direct and indirect effects of smoking on utility and death risk.

Define death as instantaneous utility equal to zero in all future periods. Using (13), we are now able to express the expected lifetime utility (LU) of the individual, where ρ is the time preference rate, and τ is the time of death, which is stochastic:[13]

$$E(\mathrm{LU}) \equiv E\left\{\int_0^{\tau} \mathrm{e}^{-\rho t} U[Z(t), R(t), K(t)]\mathrm{d}t\right\}$$
$$= \int_0^{\infty} \mathrm{e}^{-\rho t} U\,[Z(t), R(t), K(t)]\,S(t)\mathrm{d}t + \int_0^{\infty} 0\cdot(1-S(t))\mathrm{d}t$$
$$= \int_0^{\infty} \mathrm{e}^{-(\rho t + y(t))} U(Z(t), R(t), K(t))\mathrm{d}t \tag{14}$$

Here, y becomes a state variable and a *risk premium* that is added to the time preference rate;[14] the individual knows that he might die before the future is reached.

Fig. 2 summarises the discussion so far by showing all the effects of smoking including death risk, where the death risk is based on (12).

2.2.2. Wealth

The individual's asset accumulation is given by the following equation:

$$\dot{W}(t) = rW(t) + Y(t) - m(t)M(t) - p(t)C(t) - q(t)Z(t) \tag{15}$$

where W is the wealth, r the market interest rate, Y the income, and m, p and q are the prices of medical care (M), cigarettes (C) and non-addictive consumption (Z), respectively. We restrict the wealth to be non-negative in all periods, i.e., $W(t) \geq 0$.

[13] E(LU) is the discounted expected utility over an infinite period of time. However, as the risk of dying increases with lower health and aggregated smoking, the possibility of living infinitely can be ignored.

[14] Kamien and Schwartz (1971) were the first to introduce this framework in the economic literature. For later applications, see Clarke and Reed (1994) and Gjerde et al. (1999, 2001).

2.3. Optimal plans

Optimal paths of medical care, cigarettes and non-addictive consumption are found by maximising the expected lifetime utility in (14) subject to the developments in the state variables A, D, H, K, y and W (see (3), (4), (5), (7), (12) and (15)). The current value Hamiltonian, as well as the sufficient conditions are given in Appendix A. Below are the shadow prices for the state variables, which have very intuitive interpretations.

$$\pi(t) = \pi_0 \mathrm{e}^{-(r-\rho)t} > 0 \tag{16}$$

$$\gamma(t) = -E\left\{\int_t^{\tau} U\mathrm{e}^{-(v-t)\rho}\mathrm{d}v\right\} = -\int_t^{\infty} \mathrm{e}^{-(v-t)\rho-y(v)}U\mathrm{d}v < 0 \tag{17}$$

$$\lambda(t) = E\left\{\int_t^{\tau} \mathrm{e}^{-(\rho+\mu_A)(v-t)}U'_R R'_A\,\mathrm{d}v\right\} = \int_t^{\infty} \mathrm{e}^{-(\rho+\mu_A)(v-t)-y(v)}U'_R R'_A\,\mathrm{d}v < 0 \tag{18}$$

$$\sigma(t) = E\left\{\int_t^{\tau} \mathrm{e}^{-(\rho+\beta)(v-t)}U'_K\,\mathrm{d}v\right\} = \int_{\mathrm{t}}^{\infty} \mathrm{e}^{-(\rho+\beta)(v-t)-y(v)}U'_K\,\mathrm{d}v > 0 \tag{19}$$

$$\eta(t) = \int_t^{\infty} \mathrm{e}^{-\left[\rho(v-t)+\int_t^v \delta(s)\mathrm{d}s\right]}\left[\gamma(v)h'_H - \sigma(v)(1+\beta)\delta(v)\right]\mathrm{d}v \tag{20}$$

$$\phi(t) = \int_t^{\infty} \mathrm{e}^{-(\rho+\mu_D)(v-t)}\left[\gamma(v)h'_D - F'_D\mu_D(\eta(v)+\sigma(v)(1+\beta))\right]\mathrm{d}v \tag{21}$$

π is the *shadow price of money*, which is positive and represents the utility benefit of an increase in W. It is decreasing over time for $r > \rho$. γ is the expected lifetime utility at time t measured in negative value. It represents the loss to the individual if he dies in period t, and can be interpreted as the negative of the *value of a statistical life*. Thus, $\gamma(t) < 0$. Further, λ is the *shadow price of the addictive consumption capital*. It is the present value of the tolerance effect, and is therefore, negative.

More central to our analysis are Eqs. (19)–(21). The *shadow price of the subjective health stock*, σ, represents the discounted expected marginal utility of an increase in K. As utility is increasing in K, $\sigma(t) > 0$. The *shadow price of the objective health stock*, η, which is the expected utility effects of an increase in H, discounted by the time preference rate as well as the health depreciation rate, is determined by several effects, see (20). The first term within the parentheses represents the positive value of H on life extension. Further, a high H has an impact on the subjective stock of health, which again has utility implications. For $\beta = 0$, we know from (8) that $K = H$, and this effect is positive. However, for all other values of β, a high H will involves costs in terms of future health depreciation, see (5). This effect is given by the second term within the parenthesis, which is negative.[15] Thus, η can be positive or negative. This means that the value of a high objective health, H, is lower for an adapter than for a non-adapter. One who is able to adapt to a lower health level, would presumably try to avoid a high H early in life that would give high costs in terms of a large fall in H later in life.

Finally, ϕ is the *shadow price of the smoking stock*. It reflects the present value of aggregated smoking on expected lifetime utility. The first part in the brackets of (21) is the effect on the death probability, which is negative. The second part reflects the effect on health investments; the higher D, the higher is the depreciation of this stock, which increases health investments. As for the shadow price of H, these effects have an ambiguous impact on expected lifetime utility, and the shadow price of the smoking stock can, therefore, be positive or negative. Thus, an increase in the smoking stock may under certain conditions increase expected lifetime utility, even if smoking is harmful.

The *flow conditions* for optimal consumption of cigarettes and medical care presented in Appendix A can be rewritten as follows:[16]

$$p(t) = \frac{U'_R R'_C \mathrm{e}^{-y(t)} + \lambda(t) + \phi(t) + F'_{\dot{D}}\,(\eta(t) + \sigma(t)(1+\beta))}{\pi(t)} \tag{22}$$

$$m(t) = \frac{F'_M(\eta(t) + \sigma(t)(1+\beta))}{\pi(t)} \tag{23}$$

The expression for the *optimal smoking path* in (22) is relatively complex. The unit price of a cigarette is the money equivalent of the expected net gains from smoking a cigarette. These gains consist of expected immediate utility effects plus the tolerance effect and health effects. Health effects enter both in ϕ and in the last term in the numerator, which accounts for the negative effects smoking a cigarette have on health investments. This term could be either positive or negative because reduced health may have ambiguous effects on utility as mentioned above. A similar term is found in the condition for the optimal path of *medical care*, see (23). For positive consumption of M, the term $(\eta(t) + \sigma(t)(1+\beta))$ is positive, implying that the last term in the numerator in (22) is negative, and the health effects become an additional cost of cigarette smoking.

3. Numerical results

To examine the importance of various assumptions on health effects, information and lifetime, we specify the model numerically. Model specification and parameter values are given in Appendix B. Though similar in many ways to the model in Gjerde et al. (2001), our utility function differs by treating non-addictive consumption and health as

[15] Note that the optimisation problems looks different in the cases where $\beta = 0$ and $\beta \to \infty$, see Appendix A.

[16] The flow condition for non-addictive consumption can be deduced from Appendix A.

complementary goods in a Cobb–Douglas bundle, whereas the utility function in Gjerde et al. (2001) is additively separable in consumption and health. This means that our agents derive more utility from regular consumption the better their health, whereas utility from the relaxation term is independent of both health and regular consumption.

The model is calibrated in order to attain realistic paths for the endogenous variables such as consumption and health variables. Most parameter values are taken from Gjerde et al. (2001), whose calibration of the benchmark scenario sought realistic consumption paths and death probabilities. Our parameter for the health effect of cigarettes, cig_1 (see Appendix B) is calibrated against empirical estimates of minutes lost per cigarette. In our central simulation case, each cigarette reduces life by 11.4 min, an estimate that is consistent with the literature (see Adda and Lechene, 2001).

In addition to the results presented here, a number of robustness tests were conducted with respect to key model parameters such as the effectiveness of medical care, utility functional form, and income trajectory. The qualitative results presented here were found to be stable to these tests. For completeness, we summarise the changes in model variables induced by these test in Table 1.

3.1. Four cases

We examine decision problems involving perfect adaptation and no adaptation, and different beliefs about the reversibility of the unobservable effects of cigarettes on death risks. As described in Section 2.2.1, we compare the case where the smoking stock enters the death risk hazard function, see (12) – called the *irreversible* case – with the case where the individual believes that all death risk is embedded in the objective health stock, see (12′) – the *reversible* case. We also consider two different assumptions about adaptation, namely the two special cases where $K = H$ (the *no-adapt* case) and $K = \dot{H}$ (the *adapt* case). This gives the four cases shown and labelled in Table 2.

Table 1
Sensitivity analyses

	Shift direction	Effect on smoking	Effect on health investments
Health investment (med_1)	+	+/−	−/+
	−	−/+	+/−
Health investment (med_2)	+	+/−	+
	−	−/+	−/−
Health effects of smoking (cig_1)	+	−	ε
	−	+	ε
Time preference rate (ρ)	+	+	+/−
	−	−	−/+
Utility effects of smoking (χ)	+	+	−
	−	−	+
Income each period	250 for $t < 105$	−	+

Table 2
Different cases

	Death risk	Subjective health
Reversible/adapt	$\dot{y}(t) = h(H(t))$	$K(t) = \dot{H}(t)$
Irreversible/adapt	$\dot{y}(t) = h(H(t), D(t))$	$K(t) = \dot{H}(t)$
Reversible/no-adapt	$\dot{y}(t) = h(H(t))$	$K(t) = H(t)$
Irreversible/no-adapt	$\dot{y}(t) = h(H(t), D(t))$	$K(t) = H(t)$

3.2. Adaptation and reversibility effects

Comparisons between the four cases involve Figs. 3 and 4 and Table 3. The first two columns of Table 3 summarise the changes in key variables when adaptability and irre-

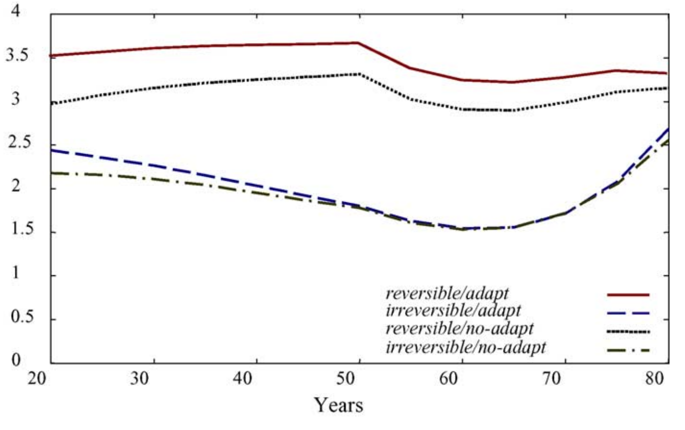

Fig. 3. Smoking levels by adaptability and death risk reversibility (cigarettes per day).

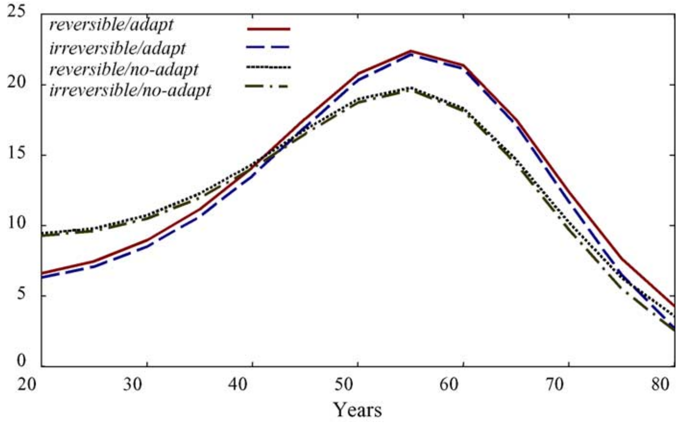

Fig. 4. Medical care levels by adaptability and death risk reversibility (amount spent on medical care per year).

Table 3
Changes in key model variables by adaptation, reversibility and life length

	Adaptable	Irreversible	Long life
Smoking	+	–	+/–
Medical care	–/+	–	–/+
Objective health	–	+	–
Other consumption	–/+	+	–/+

All changes calculated from a base of no adaptation and reversible death risks.

versibility are introduced.[17] Hence, the “+” entry for {*Smoking*, *Adaptable*} denotes an increase in the use of cigarettes when we move from a non-adaptable individual to an adaptable one. Similarly, {*Smoking*, *Irreversible*} indicates the change in the demand for smoking when we move from an individual with reversible beliefs regarding death risk to one with irreversible beliefs, everything else equal. When the sign of the effect changes over time, we use a “/” to signify the regime change, with the sign preceding it signifying the effect early in life and the sign following it showing the effect late in life.

When health effects are assumed reversible, changes in smoking level become a way of investing and disinvesting in health. By reducing smoking, the gross investment in the smoking stock can become smaller than the depreciated amount, leading to a falling stock. This incentive for reducing smoking, however, is counteracted by the complementary relationship between health and regular consumption, which – as noted – makes cigarettes relatively more desirable as the health stock falls.

With irreversible health effects, each cigarette increases death risks directly in addition to its other consequences for objective health – the costs of smoking are higher – and smoking is consequently reduced. As you get older, the number of years left to live falls, decreasing the expected life lost from smoking. Consequently, the smoking level starts to increase and move towards the level of those who do not assume irreversible effects at the end of life.[18]

While medical care could act as either a complement or substitute to smoking, we find that demand for medical care is, in general, lower under the irreversible death risk specification (see Fig. 4) because they are unable to alter the component of death risk that depends on past smoking (D). Reducing smoking is a more effective technology for reducing death risk than increasing medical expenditures because it impacts both the health and smoking stock component of the hazard function.

Adapters smoke more for two reasons. First, the subjective health stock is lower than objective health in the first half of life and higher in the second half since it does not accumulate the health investments, smoking injuries and aging effects that objective health is subject to. Since regular consumption is a complement to health, lower subjective health makes regular consumption less attractive and cigarettes relatively more attractive. This tends to bias unhealthy activities earlier and health-complementary activities later in life. The complementarity of health and regular consumption also explains why adapters increase

[17] As suggested by the results presented in Figs. 3 and 4, we do not find any notable interactions between reversibility and adaptation. Hence, the entries in Table 2 hold for all combinations of model assumptions.

[18] The rational smoker in our model is not representative of real smokers in all regards as he lightly smokes (less than five cigarettes a day), though this depends on parameters and the utility function specified.

demand for medical care in the last part of life compared to non-adapters (Fig. 4). Unlike in Gjerde et al. (2001), our agents also invest in health in order to increase their utility from regular consumption, so to the extent that regular consumption will be later in life, it is optimal to save complementary health investments for that time period as well. At our calibration of the model, an adaptable person lives 0.6–0.7 years shorter than a non-adapter, due to the lower objective health stock implied by less early investment in health (Table 3).

3.3. Health information and changes in belief

Above we assumed that agents do not revise beliefs about the reversibility of health effects from smoking throughout the planning horizon. What happens to smoking paths if agents who assume reversible effects are informed about the irreversible effects? New information on reversibility may have different effects depending on whether agents smoke or are considering starting. A focus on irreversibility increases expected cost of smoking, but it also reduces expected benefits from reducing smoking. The first effect probably counts more when young, while the second effect may count more when old. Thus, we simulate the effects of new information both at ages 30 and 60 years. As the results are qualitatively similar for adapters and non-adapters, we only present the first case.

Fig. 5 shows the effect on the smoking paths. New information about irreversible effects reduces smoking for both age groups. Thus, the increased damage from smoking has a higher effect than the reduced benefit from smoking reduction even at age 60. As expected, the effects are somewhat smaller for people at age 60 than at 30.

Smith et al. (2001) finds evidence that serious health shocks provide long-time smokers with a credible signal about the mortality risks of their habit—having a heart attack provides specific information on the consequences of your past choices. From a policy perspective, the problem is that the signal to quit comes too late. The simulations here model the informational content contained in such an event. Despite that fact that the impacts of new

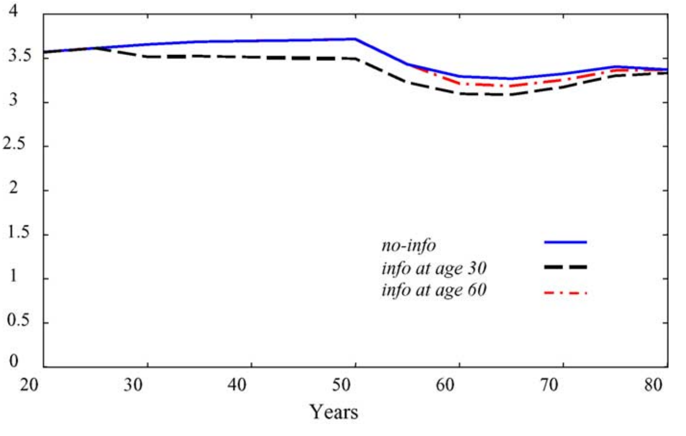

Fig. 5. Smoking levels by information shock scenarios, adaptation case (cigarettes per day).

information are not very high in our simulations, they show that new information is most valuable early in life.[19]

3.4. Potential lifetime effects

Life expectancy varies with socio-economic status, and agents may use family history to refine beliefs about their own life expectancy. How do estimates of potential life expectancy influence the smoking decision of rational agents? There are two main incentives at work, operating in different directions: The first is the *endowment effect*; the more life you have, the more you can afford to spend on, e.g., cigarettes. The second is the *cost effect*, which says that the longer life you expect to have, the more you stand to lose from dying young because of smoking.

A longer expected life is induced in the model by changing a parameter in the hazard function (par_1, see Appendix B), so that expected lifetime increases by approximately 3 years. The final column of Table 3 shows the effects of this increase. In contrast to Adda and Lechene (2001), we find that endowing the individual with a longer life expectancy causes him to smoke more up to age 50 years, and less in second part of life.[20] The endowment effect dominates when young, while the cost effect dominates at older ages. With longer potential lifetime, he will also invest less in health through medical care.

Adda and Lechene (2001) assume that individuals cannot invest in health. Another run of our model simulates this case by removing medical care as a choice variable. This generates less smoking when potential life is longer, apart from the first few years. When investments in health are not an option, smoking becomes more expensive since you cannot counteract its health effects. This reinforces the cost effect mentioned above, and explains the differences in results. As Adda and Lechene note, their results are more in line with the empirical regularities found in the epidemiological literature. However, our results raise interesting questions regarding the interaction between life expectancy, access to medical care, and smoking.

4. Discussion and conclusions

The explicit analysis of health investment, mortality and adaptation effects yields a number of new insights. When the agent assumes that the mortality risks of smoking are highly reversible, reducing smoking becomes a way of investing in health. How important this incentive is for real smokers remains an open empirical question, however. More irreversible effects cause smokers to reduce their smoking early on. They also smoke more in old age, when the cost of reduced life expectation is small.

[19] An alternative interpretation of the information provided by a negative health event would be that individuals are uncertain about whether they are at higher or lower risk than the averages presented in epidemiological studies. A heart attach for example, would then serve to update their priors regarding this personalised risk. We do not model such a process.

[20] In the sensitivity analysis, we found that the endowment effect dominates later into life when a flat income profile is assumed.

By assuming complementarity between health and non-addictive consumption, investing in health is also a way of investing in the ability to enjoy consumption goods. Adaptation, the ability to psychologically absorb negative health shocks, makes it less important to have a high level of objective health and more desirable to smoothen the health stock path. This tends to push smoking toward the beginning of life and health investment and health-complementary activities toward the end of life. We also find that adaptive smokers smoke more overall.

An informational shock that makes the individual fully aware of the mortality risks of long-term smoking, uniformly results in reduced smoking. However, the impact is more pronounced if the information is revealed earlier in life. One aspect not studied here is that information shocks may also change an individual's perception of the more observable health effects of smoking. In our framework, one could imagine such an effect taking place through the adaptation parameter.

Finally, we find that individuals endowed with longer life expectancy smoke more in the first part of life, but this result rests on their ability to invest in their own health. With no access to medical care, a short life expectancy actually makes smoking more attractive. If socio-economic status can be regarded as an indicator of "life potential", then this behaviour could be important to a number of policy questions. In a sense, those most likely to smoke are also those least likely to have the medical care, which in our model is needed to discourage the habit. Given the policy ramifications, this dimension of the analysis warrants further study.

Appendix A. Solving the model

A.1. The general case

The current value Hamiltonian function to the optimisation problem in Section 2.3 is:

$$\begin{aligned} V(t) = {} & U(Z(t), R(C(t), A(t)), K(t))\mathrm{e}^{-y(t)} + \lambda(t)(C(t) - \mu_A A(t)) \\ & + \pi(t)(rW(t) + Y(t) - m(t)M(t) - p(t)C(t) - q(t)Z(t)) + \gamma(t)h(H(t), D(t)) \\ & + \eta(t)(F(M(t), C(t) - \mu_D D(t); t) - \delta(t)H(t)) \\ & + \sigma(t)\left(\frac{\beta}{1+\beta}H_0 - \beta K(t) + (1+\beta)(F(M(t), C(t) - \mu_D D(t); t) - \delta(t)H(t)\right) \\ & + \phi(t)(C(t) - \mu_D D(t))x \end{aligned} \quad \text{(A.1)}$$

where λ, π, γ, η, σ and ϕ are the costate variables in current values, and represent shadow prices of the stock variables A, W, y, H, K and D respectively. Since the Hamiltonian is strictly concave in Z, C and M jointly, the sufficient conditions assuming interior optimal solutions become (Seierstad and Sydsæter, 1987);

$$\frac{\partial V(t)}{\partial Z(t)} = U'_Z \mathrm{e}^{-y(t)} - \pi(t)q(t) = 0 \quad \text{(A.2)}$$

$$\frac{\partial V(t)}{\partial C(t)} = U'_R R'_C \mathrm{e}^{-y(t)} + \lambda(t) - \pi(t)p(t) + \eta(t)F'_{\dot{D}} + \sigma(t)(1+\beta)F'_{\dot{D}} + \phi(t) = 0 \tag{A.3}$$

$$\frac{\partial V(t)}{\partial M(t)} = -\pi(t)m(t) + \eta(t)F'_M + \sigma(t)(1+\beta)F'_M = 0 \tag{A.4}$$

$$\dot{\lambda}(t) - \rho\lambda(t) = -\frac{\partial V(t)}{\partial A(t)} = -[U'_R R'_A \mathrm{e}^{-y(t)} - \lambda(t)\mu_A] \tag{A.5}$$

$$\dot{\pi}(t) - \rho\pi(t) = -\frac{\partial V(t)}{\partial W(t)} = -\pi(t)r \tag{A.6}$$

$$\dot{\gamma}(t) - \rho\gamma(t) = -\frac{\partial V(t)}{\partial y(t)} = U(Z(t), R(t), K(t))\mathrm{e}^{-y(t)} \tag{A.7}$$

$$\dot{\eta}(t) - \rho\eta(t) = -\frac{\partial V(t)}{\partial H(t)} = -\left[\gamma(t)h'_H - \eta(t)\delta(t) - \sigma(t)(1+\beta)\delta(t)\right] \tag{A.8}$$

$$\dot{\sigma}(t) - \rho\sigma(t) = -\frac{\partial V(t)}{\partial K(t)} = -[U'_K \mathrm{e}^{-y(t)} - \sigma(t)\beta] \tag{A.9}$$

$$\dot{\phi}(t) - \rho\phi(t) = -\frac{\partial V(t)}{\partial D(t)} = -[\gamma(t)h'_D - \eta(t)F'_{\dot{D}}\mu_D - \sigma(t)(1+\beta)F'_{\dot{D}}\mu_D - \phi(t)\mu_D] \tag{A.10}$$

with the following transversality conditions:

$$\lim_{t\to\infty} \mathrm{e}^{-\rho t}\lambda(t)A(t) = 0; \quad \lim_{t\to\infty} \mathrm{e}^{-\rho t}\pi(t)W(t) = 0; \quad \lim_{t\to\infty} \mathrm{e}^{-\rho t}\gamma(t)y(t) = 0;$$
$$\lim_{t\to\infty} \mathrm{e}^{-\rho t}\eta(t)H(t) = 0; \quad \lim_{t\to\infty} \mathrm{e}^{-\rho t}\sigma(t)K(t) = 0; \quad \lim_{t\to\infty} \mathrm{e}^{-\rho t}\phi(t)D(t) = 0 \tag{A.11}$$

A.2. The no-adapt case (β = 0 and K = H)

In this case, the current value Hamiltonian becomes:

$$\begin{aligned} V(t)_{\beta=0} = {} & U(Z(t), R(C(t), A(t)), H(t))\mathrm{e}^{-y(t)} + \lambda(t)(C(t) - \mu_A A(t)) \\ & + \pi(t)(rW(t) + Y(t) - m(t)M(t) - p(t)C(t) - q(t)Z(t)) \\ & + \gamma(t)h(H(t), D(t)) + \eta(t)(F(M(t), C(t) - \mu_D D(t); t) - \delta(t)H(t)) \\ & + \phi(t)(C(t) - \mu_D D(t)) \end{aligned} \tag{A.12}$$

The sufficient conditions can be outlined as above. The transversality conditions follow from (A.11).

A.3. *The adapt case ($\beta = \infty$ and $K = \dot{H}$)*

The current value Hamiltonian becomes:

$$\begin{aligned} V(t)_{\beta=\infty} = {} & U(Z(t), R(C(t), A(t)), F(M(t), C(t) - \mu_D D(t); t) - \delta(t)H(t))\mathrm{e}^{-y(t)} \\ & + \lambda(t)(C(t) - \mu_A A(t)) + \pi(t)(rW(t) + Y(t) - m(t)M(t) - p(t)C(t) - q(t)Z(t)) \\ & + \gamma(t)h(H(t), D(t)) + \eta(t)(F(M(t), C(t) \\ & - \mu_D D(t); t) - \delta(t)H(t)) + \phi(t)(C(t) - \mu_D D(t)) \end{aligned} \tag{A.13}$$

The sufficient conditions can be outlined as above. The transversality conditions follow from (A.11).

Appendix B. Specifications and parameter values in the numerical model

Below, the functional forms are written in discrete time to match with the GAMS code, which is available from authors upon request. The agent is constrained from smoking before age 20, and the model is optimised for a horizon of 140 years, in order to avoid horizon effects (though probability of living past 100 were small in all simulations), using 5-year periods. Simulations were carried out using the GAMS/CONOPT system (Brooke et al., 1998).

The *utility function* is additive in consumption of addictive and non-addictive goods, while non-addictive consumption and health are complementary:

$$U(Z_t, R_t, K_t) = Z_t^{\alpha}(K_t + H_0)^{\gamma} + R_t^{\chi} \tag{B.1}$$

Initial endowment of health, H_0, is set equal to 50, and is included to allow for a negative K-value and to balance Z and K. $\alpha = 0.35$, $\gamma = 0.35$ and $\chi = 0.35$.

The endogenous *hazard function* is specified as follows:

$$y_t = y_{t-1} + 5\,\mathrm{par}_1\,\mathrm{par}_2\left(\frac{1}{\left[(H_t - \mathrm{cig}_1\mathrm{e}^{D_t/2})/\mathrm{critical}\right]^3 + 1}\right)^{\mathrm{par}_2 - 1} \tag{B.2}$$

$\mathrm{par}_1 = 0.02$, $\mathrm{par}_2 = 2.5$ and critical = 37 are taken from Gjerde et al. (2001). cig_1 is calibrated to reach a realistic lifetime loss from smoking, and is set equal to 0.03.[21] Eq. (B.2) corresponds to (12) in the description of the general analytical model. If we were to omit the

[21] To calculate this, we assume that the price of a pack, i.e., 20 cigarettes is US$ 3.50, and that the average income is US$ 33,000. Thus, smoking a pack a day costs 3.9% of income. For income equal to 250 as in our model, a pack a day costs 9.68, and a cigarette a day costs 0.48. Using these assumptions, each cigarette reduces life by 11.4 min in the adapt_reverse case, and 13.7 min in the no-adapt_reverse case.

term ($\text{cig}_1 e^{\frac{D_t}{2}}$) from the denominator, we would arrive at the functional form described in (12′).

The *health investment function* is of the following type;

$$F(M_t, D_t - D_{t-1}; t) = \text{med}_1 \cdot M_{\text{t}}^{\text{med}_2} - \text{cig}_1\ \text{fragility}_t\ (2e^{D_t - D_{t-1}} - 2) \tag{B.3}$$

$\text{med}_1 = 2$ and $\text{med}_2 = 0.1$ are set to reproduce similar results as in Gjerde et al. (2001) when smoking is set equal to zero in all periods. $\text{fragility}_t = 1$ for $t \leq 55$, and $(t - 50)/5$ for $t > 55$. This parameter is included to capture the fact that damage done by cigarettes becomes more intensive with age.

The *relaxation function* is specified as:

$$R_t = r_c C_t + r_a A_t + r_{c2} C_t^2 + r_{a2} A_t^2 + r_{ac} C_t A_t \tag{B.4}$$

with $r_c = 20$, $r_a = -2$, $r_{c2} = -0.1$, $r_{a2} = -1$ and $r_{ac} = 1$. With these parameters, the properties of the relaxation function are satisfied, see Section 2.1.

Initial endowment of wealth, W_0, is set equal to 0. The market interest rate, r, is set at 4% p.a. the utility discount factor, ρ, is assumed to be 3% p.a., while the unit prices of non-addictive consumption (q), addictive consumption (p) and medical care (m) are constant and equal to 1 in all periods. Annual income, Y_t, increases from 60 to 100 for the first 20 years, is equal 250 for $20 < t < 70$, and 50 for $70 \leq t \leq 100$, and 0 thereafter ($t > 100$). The health depreciation rate, δ, varies over time and equals 1% p.a. for the two first decades, for then to increase with 1 percentage point every fifth year. As in Suranovic et al. (1999), the addiction stock depreciation rate, μ_A, is 10% p.a., while the smoking stock depreciation rate, μ_D, is 1% p.a.

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