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Health Investments:**

**Impacts of Health Adaptation
and Damage Reversibility**

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Smoking and Health Investments: Impacts of Health Adaptation and Damage Reversibility*

by

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Abstract

In the present paper we examine how different sets of beliefs about the health effects of smoking would influence a rational smoker. By embedding the rational addiction theory in a Grossman model of health investment modified to take account of psychological adaptation effects, we present a model of a rational addict that allows us to explicitly specify beliefs about a direct and indirect effect on both death risk and utility. This allows us to study how a rational addict would smoke with different beliefs of cancer risks, and with or without the well-documented ability to adapt to health changes. Numerical simulation results illustrate a number of different incentives that influence the smoking paths and health investments under the various beliefs, and suggests that beliefs have different impacts at different ages, providing a richer set of dynamics than might initially be expected.

1. Introduction

The major reason smoking is controversial, is that it causes death and disease, knowledge that was established successfully only in the middle of the last century. Despite massive efforts to spread this knowledge, a sizeable number of people persist in smoking. How does this cohere with the idea of the smoker being rational?

In the rational addiction literature, smokers have precise, quantitative beliefs about the effects of smoking, but the health effects are bundled into a reduced form along with all social, psychological and other effects.¹ Since health effects are a major issue with tobacco smoking with potentially profound dynamic consequences for an individual, we believe the health beliefs of the rational addict should be studied explicitly: What, specifically, are these beliefs, and how important are they for the smoking decision?

One issue is whether health damage from tobacco is reversible or irreversible.² On the one hand, public information campaigns focus on the dangers of smoking in order to stop people from taking up the habit. The dangers are stressed in a way that makes the effects seem irreversible (e.g., “each cigarette shortens your life by 10 minutes”). Since some start in spite of this, other campaigns focus on the benefits of quitting, noting reductions in cancer risk etc. This suggests that many health effects are reversible. What impact do changes in beliefs along this dimension have for a rational addict’s smoking career?

A second topic concerns the well-documented fact that “objective” health is less important for experienced well-being than “subjective” health -- people adapt to loss of

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, and Suranovic et al., 1999).

² The epidemiological literature is not clear whether all health damages from smoking are reversible or irreversible. Intensity of smoking is related to risks (Leffondré et al., 2002), yet reducing your intensity of smoking does not seem to alter your risks (Godtfredsen et al., 2002). Some studies show irreversible effects on the lung function which is associated with an increased mortality risk, while other studies find that the risks faced by ex-smokers converge over time to those of non-smokers (see, e.g., references and discussion in Adda and Lechene, 2001).

health.³ This could be seen as caring more about recent *changes* in their overall level of health than about the level itself. Does this make tobacco more desirable for a rational smoker since he can adapt to the harm? Or does it make it less desirable to smoke because smoking continually weakens his health, leading to a series of undesirable health changes?

Finally, a central aspect of the analysis deals with the idea that a rational smoker would take into account not only the more visceral effects of smoking on one's quality of life, but also its consequences for lifespan. For instance, rational smokers with different expected lifespans should take these future consequences into account in different ways.⁴ This intimate relationship between smoking and health makes it natural to combine the rational addiction model with a model in which the ability to invest in health is explicit. Would for instance a smoker compensate the negative health effects of smoking by buying more medical care?

The issues above highlight a neglected area of research in the rational addiction literature: How *should* the smoking of rational addicts vary with their beliefs about health consequences? Answers to these questions have varied: The drop in smoking that coincided with health campaigns was originally used to support rational addiction theory in Becker and Murphy's (1988) original article. More recently, Sloan et al. (2002) has suggested that price in a rational addiction model is *sufficient* to explain changes in smoking levels over the 20th century, and that health information had little if any impact.

Decisions of both smoking and health involve dynamic reasoning, which can be modelled as stock decisions. Thus, to analyse the importance of health beliefs for

³ Though it has not been studied in the context of smoking, adaptation has been observed empirically following successful transplants (Adang, 1997), following heart conditions (Wu, 2001), in the context of elderly persons' ability to manage stressful events (Cassilleth et al., 1984), as well as in the evaluation of impaired health states by the general public compared to by patients (Sackett and Torrance, 1978).

⁴ Effects on death risks were first treated by Goldbaum (2000), who subtracts a fixed number of minutes from the known, finite life of the rational smoker for each cigarette he smokes. Those with a long potential lifetime are more able to "afford" this life-span cost, and are predicted to smoke more than those with a short potential lifetime. The lack of empirical support for this prediction (see, e.g., Sterling and Weinkam, 1990, and Smith and Shipley, 1991), led to the introduction of health effects in a model with stochastic lifetime (Adda and Lechene, 2001, see also the appendix to Suranovic et al., 1999). In this model, smoking today irreversibly increases the probability of dying at all points in the future. Individuals with a long potential lifetime, therefore, risk losing more life years from smoking than similar individuals with short potential lifetimes, bringing the results back in line with empirical observations.

smoking behaviour we embed the rational addiction model in a formal model of health investment with adaptation effects (Gjerde et al., 2001): The individual can invest in health by buying medical care, and disinvest in health by smoking. The framework allows us to explicitly model four separate ways smoking affects the rational smoker: A direct and indirect effect on utility, and a direct and indirect effect on health (see Figure 1):

- Tobacco has a direct effect on utility as a consumption good.
- Tobacco has a direct effect on the death hazard (e.g., cancer risk).
- Tobacco has indirect effects on utility and death hazard through its effects on the objective health. This indirect influence arises because
 - The objective health stock influences the death risk.
 - The objective health stock influences the subjective health stock. The subjective health stock is the experienced well-being from health, i.e., health as a consumption good.

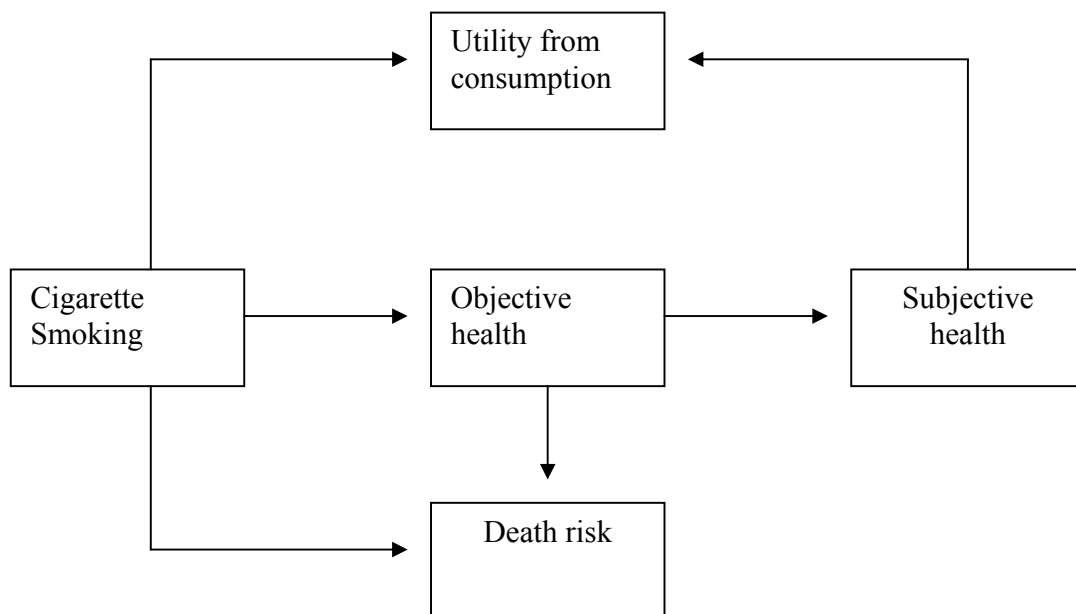


Figure 1: Direct and indirect effects of smoking on utility and death risk

The direct effect on utility requires no comment. The direct effect on the death hazard captures effects on health that are “hidden.” For instance, the individual is unable to directly perceive how past and present smoking influences his risk of cancer. This is an effect that only becomes apparent when the cancer strikes, and health information based on population studies is required to form beliefs about this beforehand.

The indirect effect on the death hazard operates through the stock of health, which is known to the individual. Smoking reduces objective health in a variety of perceivable ways such as reducing cardiovascular fitness and inducing coughing fits. This kind of health effect can plausibly be seen as known: Health hazards of smoking were discussed long before the epidemiological research of the 20th century on cancer risk and smoking.⁵

Finally, the indirect effect of smoking on utility operates through the objective and subjective health stock. The subjective health stock is the subjective experience of health, which differs from objective health due to adaptation. Menzel et al. (2002) perceive 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.

The paper is organised as follows. Section 2 presents the model and analytical results, while the different cases studied are presented in Section 3. To illustrate the effects of adaptation and health damage reversibility, the model is solved numerically for a representative consumer. The results of the numerical analysis are given in Section 4, while Section 5 concludes.

⁵ Walton (2000) collects excerpts from a variety of sources, and shows that smoking was denounced as both immoral and unhealthy already in the 1600s, most famously when King James I in 1604 anonymously published his “A Counterblaste to Tobacco.” Roughly two and a half centuries later, the medical journal *Lancet* carried a heated exchange of letters on the subject of tobacco’s health effects, and in 1898 a statement of the Supreme Court of Tennessee upholding the State’s anti-cigarette legislation asked:

“Are cigarettes legitimate articles of commerce? We think they are not because they are wholly noxious and deleterious to health. Their use is always harmful; never beneficial. [...] Beyond any question, their every tendency is toward the impairment of physical health and mental vigor.”

2. The model⁶

2.1 The utility function

Assume an individual with the following time separable utility function:

$$(1) U(t) = U(Z(t), R(t), K(t))$$

Z is a non-addictive good, R the relaxation or pleasure “produced” from cigarettes, and K is health as a consumption good. The utility function is strictly concave in Z , R and K jointly.

Relaxation is a function of current cigarette consumption, C , and past smoking captured by a stock variable, A . This follows Chaloupka (1991).⁷

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

Three features of addictions can now be reproduced:

- *Tolerance.* While higher past consumption of cigarettes does not produce tolerance in the sense of “reduced effect from given amount of drug,” it does produce tolerance in the sense of “reduced relaxation from given amount of drug.” The *effect* of the drug is actually increased (since $R''_{CA} > 0$), but this is outweighed by a reduction in the “baseline” relaxation level (since $R'_A < 0$)
- *Reinforcement.* The effect of cigarettes increases with past consumption ($R''_{CA} > 0$), a necessary condition for “adjacent complementarity” of cigarettes, where higher past consumption increases present consumption, and where higher planned future consumption increases present consumption.
- *Withdrawal effect.* Three interpretations have been used for withdrawal, two of which are captured by our model: Chaloupka (1991) argues that withdrawal implies reduced utility from reduced consumption. Since less smoking produces less of the desirable good relaxation, this is fulfilled. Orphanides and Zervos (1995) interpret withdrawal as a larger utility loss from reduced smoking the higher the consumption stock is, i.e.,

⁶ A list of all symbols used is provided in Appendix 1.

$U''_{CA} > 0$. This, too, is captured in our model. Finally, withdrawal has been seen as adjustment costs; see, e.g., Suranovic et al. (1999) and Jones (1999). This aspect is not explicitly included in our model.

The addictive stock accumulates in the following way:

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

A dot above a variable represents its derivative with respect to time. This means that the effect of past consumption diminishes over time due to the constant depreciation rate μ_A .

2.2 The subjective health measure

“Health as a consumption good” (K in our model) may differ from “health as an objective state” (H in our model) since the individual is able to adapt to poor health. Here, adaptation does not mean a change in preferences. Rather, it captures the idea that an individual might place differential weights on past changes in health. Adaptation as a psychological recovery from a setback (Heyink, 1993) occurs as time passes. At one extreme are people who only care about present *level* of objective health, at the other extreme those who only care about current *changes* in objective health. Following Gjerde et al. (2001), we model the subjective health variable K as

$$(4) K(t) = \frac{H_0}{1 + \beta} + (1 + \beta) \int_0^t e^{-\beta(t-s)} \dot{H}(s) ds, \quad \beta \geq 0$$

H is here objective health as measured by, e.g., a general physician, and does not include “invisible” effects of tobacco on cancer risks etc. To find a solution for the decision problem specified in Section 2.6 below, we need the derivative of (4) with respect to time:

$$(5) \dot{K}(t) = \frac{\beta}{1 + \beta} H_0 - \beta K(t) + (1 + \beta) \dot{H}(t)$$

⁷ 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.

In equation (4), the degree of adaptation is captured by β , which weights the initial health endowment $H_0 > 0$ and changes in health that occurred at different times. The two extremes of adaptation are found by letting $\beta \rightarrow \infty$ and setting $\beta = 0$. It follows from (4) that⁸

$$(6) \quad K(t) = H_0 + \int_0^t \dot{H}(s) ds = H(t), \quad \text{for } \beta = 0$$

$$(7) \quad \lim_{\beta \rightarrow \infty} K(t) = \dot{H}(t)$$

We see that $\beta = 0$ captures the case where subjective health coincides with objective health. For $\beta \rightarrow \infty$, however, subjective health equals the current change in the objective health state only, and the individual may be called a *perfect adapter*: He suffers only at the point of time where the health deteriorates.

The tree panels in Figure 2 (from Gjerde et al. (2001)) illustrate different degrees of adaptation to an abrupt permanent change in health. Other consumption is held constant throughout the period, and the health state is assumed fixed apart from the negative shock at t_1 . For the non-adapter ($\beta = 0$), utility is permanently lowered (panel a)). The partial adapter in panel b) ($0 < \beta < \infty$) suffers a drop in utility when hit by the health shock, but gradually adapts to the permanent lowering of objective health. Over time, his utility approaches its previous level. Finally, in panel c), the perfect adapter ($\beta \rightarrow \infty$) suffers a loss only at time t_1 as the shock hits him, and immediately adjusts and adapts, accepting the lowered health with no later loss of welfare.

⁸ For a proof of equation (7), see Hoel and Isaksen (1994).

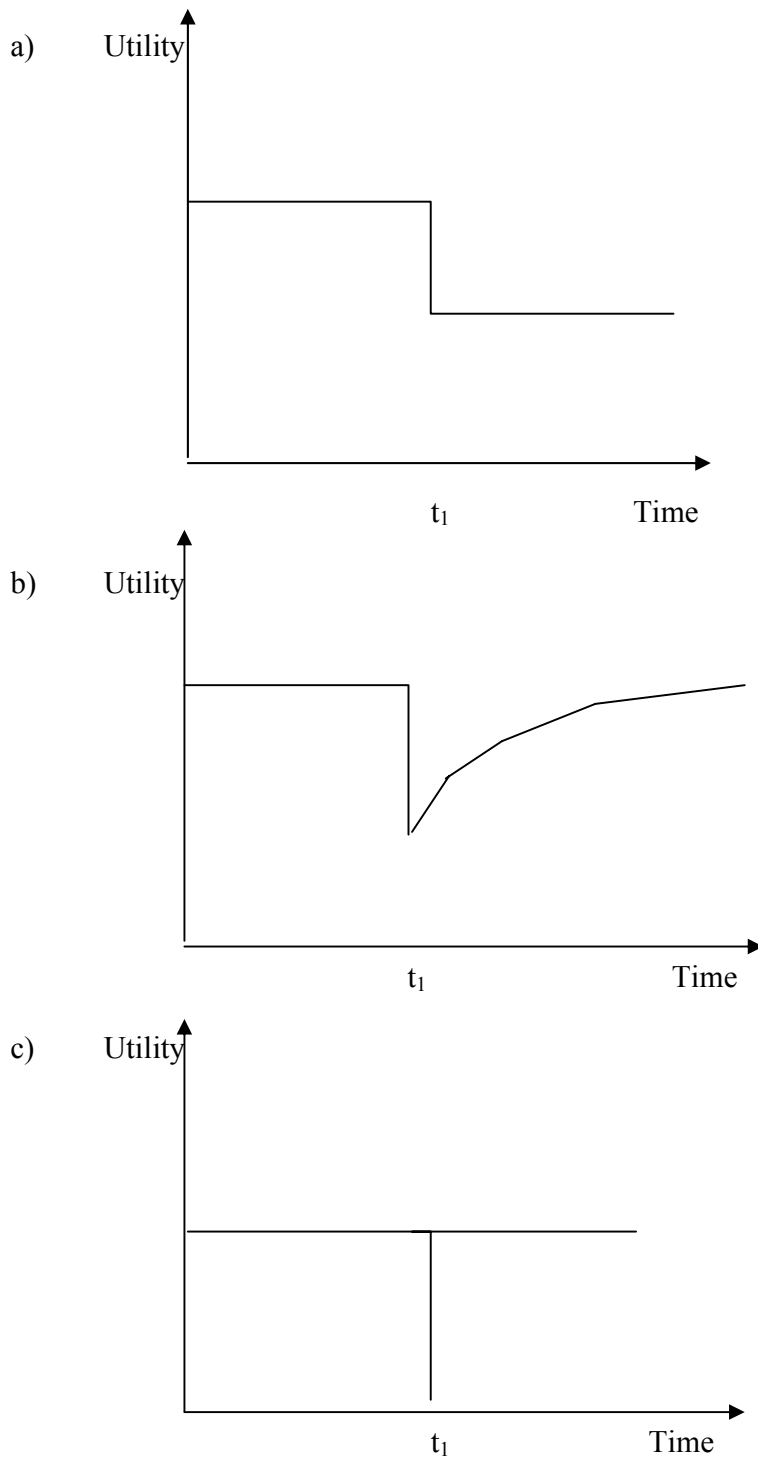


Figure 2: Adaptation processes for different values of β .
a) $\beta = 0$, b) $0 < \beta < \infty$, c) $\beta \rightarrow \infty$.

2.3 The development of objective health and reversible health effects

The individual influences his objective health in two ways: By investing in health directly, or by altering his smoking level. The direct investment in health involves purchasing medical care, $M \geq 0$, where medical care is used as a proxy for all services and goods that can be bought to improve health.

The effects of tobacco on health may be longer lasting than the effects of tobacco on the production of relaxation. The “addiction stock” (A), which captures the effect on relaxation production, is therefore insufficient to capture health effects. Following Suranovic et al. (1999) and Adda and Lechene (2001), we also define a smoking stock, D , which develops over time in the following way:

$$(8) \dot{D}(t) = C(t) - \mu_D D(t)$$

The reason for using two stocks is that past smoking may have longer lasting effects on health than it has on relaxation production, i.e., the depreciation rate may differ. As in Adda and Lechene (2001), $\mu_D < \mu_A$ so that the smoking stock is higher than the addiction stock (see equation (3)).

The function that captures how the individual can influence his objective health is a continuous, twice differentiable function $F(M, \dot{D})$ that is falling in \dot{D} , increasing in medical care, and concave in both M and \dot{D} jointly.⁹ Note that it is *changes* in the smoking stock that affect health. Reducing your smoking level from a steady state will therefore function as an investment in health: As the reduced smoking makes the smoking stock shrink, health is improved.¹⁰

⁹ 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), ch. 3.

¹⁰ As seen from equation (9) below, the perceived health effects may not be totally reversible. This requires that the health stock path for an individual who quits smoking approaches the same health stock path as if he were not smoking, every thing else equal. As the health depreciation rate increases over time, this will among other things depend on the time of quitting.

In addition to investments in health, the health stock will depreciate due to the natural process of aging, with the depreciation rate set equal to $\delta(t)$, and $\dot{\delta}(t) > 0$ for all t . Thus, the development in H is as follows:

$$(9) \dot{H}(t) = F(M(t), \dot{D}(t)) - \delta(t)H(t)$$

2.4 Uncertain lifetime and irreversible health effects

Lifetime is stochastic, and the individual has subjective beliefs about future death risks (see Gjerde et al. (2001)). The individual is aware of how the probability of death is related to the *objective* health stock, and how he can influence the health stock through health investments. Death is defined by a utility equal to zero in all future periods.

In addition to this, we consider a case where smoking has a direct effect on the death hazard, in that “hidden” death risk due to e.g. lung cancer and cardiovascular disease increases in the smoking stock, D . Thus, the death risk due to smoking is relatively irreversible.

To model the death risk, we use the hazard rate approach (conditional probabilities). The individual’s beliefs about the probability of the occurrence of death at an arbitrary instant of time are represented by a hazard function; h . As mentioned above, we assume that the hazard function is related to the objective health state (H) and the smoking stock (D), i.e., $h = h(H(t), D(t))$, such that $h_H' < 0$ and $h_{HH}'' > 0$; a given reduction in health has little impact on the probability of dying when the health stock is high, but has a high impact when the health stock is low. Also, $h_D' > 0$ and $h_{DD}'' > 0$.

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

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

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

$$(11) y(t) = -\ln S(t)$$

This gives

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

Thus, \dot{y} equals the hazard function. From (12) we see that the hazard function can be interpreted as the probability of dying at the next point of time given that the individual is alive. Inserting (12) into (10), we find

$$(13) \quad S(t) = e^{-\int_0^t \dot{y}(s) ds} = e^{-y(t)}$$

We are now able to express the expected lifetime utility (LU) of the individual, where ρ is the time preference rate, τ is the time of death, and the utility at death is zero:¹¹

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

With this framework, y becomes a state variable and a *risk premium* that is added to the time preference rate.¹² The individual does not value future utility as highly as present, and in addition he knows that he might die before the future is reached.

2.5 Asset accumulation

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

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

where W is wealth, r is the market interest rate, Y is income, and m , p and q are prices of medical care (M), cigarettes (C) and non-addictive consumption (Z) respectively.¹³ We restrict the wealth to be non-negative in all periods, i.e., $W(t) \geq 0$.

¹¹ $E(LU)$ 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 over an infinite period of time can be ignored.

¹² 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).

2.6 Analytical results

The 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 , K , D , H , y and W (see equations (3), (5), (8), (9), (12) and (15)). The current value Hamiltonian to this optimisation problem, as well as the sufficient conditions are given in Appendix 2.

The shadow prices for the stock variables in this model turn out to have very intuitive interpretations.

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

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

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

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

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

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

π is the *shadow price of money*, which is positive for $t < \infty$ 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

¹³ Alternatively, income could be a function of the time spent working as in Grossman (1972a). However, as in most rational addiction models, we have omitted the time budget here, see also footnote 9. Another alternative could be to model income as a function of consumption capital (aggregated smoking) as in Becker and Murphy (1988).

if he dies in period t (shadow price of y), and can be interpreted as the negative of the *value of a statistical life*. Thus, $\gamma(t) < 0$, for $t < \infty$, see (17). Further, λ is the *shadow price of the addictive consumption capital*. This shadow price is the present value of the tolerance effect, and is, therefore negative.

More central to our analysis are equations (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 , $\sigma(t) > 0$ for $t < \infty$. 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 depreciation rate, is determined by several effects (see (20)). The first term within the parentheses represents the positive value an increase in H has on life extension. Further, a higher health stock has an impact on the subjective stock of health, which again has implications for utility. For $\beta = 0$, we know from (6) that $K = H$, and this effect is positive. However, for all other values β , a high value of H will involve costs in terms of additional future health depreciation. This is seen from equation (9). This effect is given by the second term within the parenthesis, which is negative.¹⁴ Thus, η can be positive or negative. The implication of this is that the value of a high objective health stock, H , is lower for an adapter than for a person who does not adapt. A person who is able to adapt to a lower health level, would presumably try to avoid a high H in an early stage of life that would give high costs in terms of a large fall in H in later periods.

Finally, ϕ is the *shadow price of the smoking stock*. It reflects the present value of the effects of aggregated smoking on expected lifetime utility. The first part in the brackets is the effect on the probability of dying, which is negative. The second part reflects the effect on health investments; the higher this stock is, the higher is the depreciation of the smoking stock, which increases health investments. As for the shadow price of objective health, these effects have an ambiguous impact on the 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 the expected lifetime utility, even if smoking is harmful.

The *flow conditions* for optimal consumption of cigarettes and medical care presented in Appendix 2 can be rewritten as follows;¹⁵

$$(22) \quad p(t) = \frac{U'_R \cdot R'_C e^{-y(t)} + \lambda(t) + \phi(t) + F'_D \cdot (\eta(t) + \sigma(t)(1 + \beta))}{\pi(t)}$$

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

The expression for the *optimal smoking path* in (22) relatively complex. Here, the unit price of a cigarette is to equal the money equivalent of the expected net gains from smoking a cigarette. These consist of the expected immediate utility effects of smoking plus the tolerance effect and the health effects of smoking. The effects on health enter both in the shadow price of the smoking stock, ϕ , and in the last term in the numerator, which accounts for the negative effects smoking a cigarette has on health investments and therefore on health. The sign of this term could be either positive or negative as a reduction in health due to cigarette smoking may have ambiguous effects on utility as mentioned above. However, a similar effect is found in the condition for the optimal path of *medical care*, see equation (23). For an internal solution (a positive consumption of medical care), the terms $(\eta(t) + \sigma(t)(1 + \beta))$ that evaluate the health effects of health investments are positive, implying that in this case the last term in the numerator in equation (22) is also negative, and the health effects, represented by this term, become an additional cost of cigarette smoking.

3. Four Cases

To study the effects of health adaptation and reversibility of death risk due to smoking, we consider four different cases, which combine the different assumptions as shown in Table 1.

¹⁴ Note that the optimisation problems looks different in the cases where $\beta = 0$ and $\beta \rightarrow \infty$, see Appendix 2.

¹⁵ The flow condition for non-addictive consumption is straightforward and can be deduced from Appendix 2.

Table 1: Different cases

	Irreversible death risk	Reversible death risk
No Adaptation	<i>no-adapt_irreverse</i>	<i>no-adapt_reverse</i>
Adaptation	<i>adapt_irreverse</i>	<i>adapt_reverse</i>

We consider two cases to see the effects of different beliefs about *reversibility* concerning the death risk of smoking. In the first case, the individual believes that all health effects are *reversible* as captured by the health investment function. This means that the hazard function is specified as a function of the objective health stock only:

$$(24) \quad \dot{y}(t) = -\frac{\dot{S}(t)}{S(t)} = g(H(t))$$

Smoking still affects the probability of dying, but only through the effects on objective health. This case is named *reverse*.

The second case is the one as specified in Section 2.5, where in addition to the reversible health effects of smoking, we also has a direct effect on the probability of dying, which is not perceivable, i.e., a stronger hidden effect. This case is named *irreverse*.

We also consider two different assumptions about *adaptation*, namely the two special cases where $\beta = 0$ and $\beta = \infty$. For $\beta = 0$, $K = H$, and there is no adaptation. This is the *no-adapt* case. In the *adapt* case, we set $\beta = \infty$, i.e., $K = \dot{H}$, and the individual is a perfect adapter.¹⁶

4. Numerical Results

There are several difficulties in characterising optimal paths and conducting comparative dynamics in a continuous time model with six stock variables. For this reason, we employ a numerical version of the model. As we want to simulate a

representative consumer, the model is calibrated in order to attain realistic paths for the endogenous variables such as consumption and health variables.

The model specification and parameter values are given in Appendix 3. However, a few important assumptions are worth mentioning at the outset. While the utility function in Gjerde et al. (2001) was additively separable in consumption and health, non-addictive consumption and health are complementary goods in our specification, represented by a Cobb-Douglas bundle. This means that a certain level of consumption is more valuable to the consumer the better the health is. However, the utility function is additively separable across this Cobb-Douglas bundle and the addictive good, implying that the marginal utility of smoking is independent of the health state.

Most of the parameters are taken from Gjerde et al. (2001). In the calibration of the benchmark scenario, Gjerde et al. (2001) sought to reach realistic age profiles for consumption of non-addictive goods and the contingent probabilities for the occurrence of death, based on empirical evidence from Attanasio and Banks (1998) and SSB (2000).¹⁷ The health stock is supposed to reach its maximum around age 30.

To calibrate the parameter of the health effects of cigarettes,¹⁸ we search the parameter space until we arrive at a realistic number of minutes lost per cigarettes. In the *adapt_reverse* case, each cigarette reduces life by 11.4 minutes, an estimate that is consistent with the literature; see, e.g., Adda and Lechene (2001). We constrain the individuals so that they are not allowed to smoke before the age of 20.

To avoid horizon effects, the model is optimised for a horizon of 140 years, using 5-year periods. However, we restrict our graphical presentations to the years from 20 to

¹⁶ An appendix with the shadow prices and the flow condition for cigarette consumption for the four different cases is available from the authors.

¹⁷ The consumption path for non-addictive consumption is calibrated to reach its maximal value at mid-phase of life (age 50), which is based on empirical evidence from the US and the UK (Attanasio and Banks, 1998). To reach a realistic development in the contingent probabilities for the occurrence of death, we calibrate the model to reproduce an expected length of life estimate for an individual living in an industrialised country; see, e.g., SSB (2000). In a simulation where smoking is not allowed, the expected lifetime for an adaptive person is 78.04 years, while it is 78,48 for a non-adaptive person.

¹⁸ cig_t , see Appendix 3.

80 only, since the probability of living past 100 is very small in all of our experiments. Simulations were carried out using the GAMS/CONOPT system (Brooke et al., 1998).¹⁹

4.1 Adaptation and Reversibility Effects

Figure 3 explores the effects on smoking of the different adaptability and reversibility assumptions discussed above.²⁰ The paths are labelled as described in Table 1. The last dimension of the labels concerns an assumption about the individual's expected life length. We consider changes in this last dimension in Section 4.3.

The rational smoking paths under different beliefs vary in ways that can be understood by noting the different investment incentives the beliefs involve and the different consumption incentives the utility function involves.

In Figure 3, we see that the *belief dimension* with the largest effect on smoking paths is reversibility. The difference between these is that someone who believes in the irreversible effects is aware of the direct effect of smoking on the death hazard in addition to the indirect effect. Smoking the same lifetime quantity of cigarettes is much more damaging to the individual's health, *ceteris paribus*, due to the effects on expected lifetime. Based on equations (21) and (22), we might expect the individual to smoke less if they assume effects to be irreversible, which is confirmed by Figure 3.

Smokers only aware of the indirect, *reversible effects* of smoking on the death hazard use changes in smoking level as a way of investing and disinvesting in health. By smoking at low levels, the (gross) investment you make in the smoking stock is not enough to outweigh the depreciation. Your smoking capital shrinks, and this results in positive health effects.

The “reducing smoking to invest in health” incentive is not strong enough to result in strong falls in the smoking path in Figure 3. The reason is that (non-addictive)

¹⁹ The GAMS code is available from authors upon request.

²⁰ As all prices are set equal to 1, the numbers on the vertical axis represents the amount spent on the goods per year, and is dependent on the values chosen for income. However, assuming that smoking a pack a day costs 3.9% of income as we used in the calibration of the model, see Appendix 3, a cigarette a day costs 0.48. Thus, the individual in our model is not a heavy smoker. A smoking level of about 1.7 as in the *adapt_reverse* case, corresponds roughly to 3.5 cigarettes a day. See footnote 23 below.

consumption goods and health are complements in the utility function. Since the objective health stock falls from age 30 (Figure 4) non-addictive consumption (Figure 5) becomes less and less effective at producing utility. Cigarettes become more and more desirable *relative* to consumption goods as a result, and especially as the smoker ages and his health stock becomes low, cigarettes become the consumption good of choice. This effect counteracts the “reducing smoking to invest in health” incentive.

With *irreversible effects*, i.e., an additional direct effect of smoking on the death hazard, a third incentive enters: Each cigarette now imposes a greater cost in terms of future death risk. The rational smoker consequently reduces his smoking. This cost of increased death risk obviously varies with expected remaining lifetime, and as the rational smoker ages and his expected lifetime shortens, this cost is reduced, leading to increased smoking towards the end of his life. The health investment incentive from reducing smoking is also less in the irreversible cases, especially not at the end of life where the expected lifetime is very short. Thus, the rational smoker decreases his smoking at low ages (to avoid the costly decrease in expected lifetime) and increases his smoking when old, giving his profile a marked up-turn towards the end of his life.

The belief in irreversible, direct effects also has a different implication: Medical care can no longer directly offset the effects of smoking. As long as all health effects of tobacco work through the health stock, investing in the health stock through medical care purchases (Figure 6) could potentially neutralise all health effects. With direct, irreversible effects, the death hazard is influenced permanently. Since increased death hazard in this model is tantamount to increased impatience (why care for the future if there is a good probability you will die?), there is less investment in health. It is more difficult to raise the expected lifetime, and there is less chance of surviving to reap the increased welfare from non-addictive consumption that a higher health stock would give.

Figure 3:

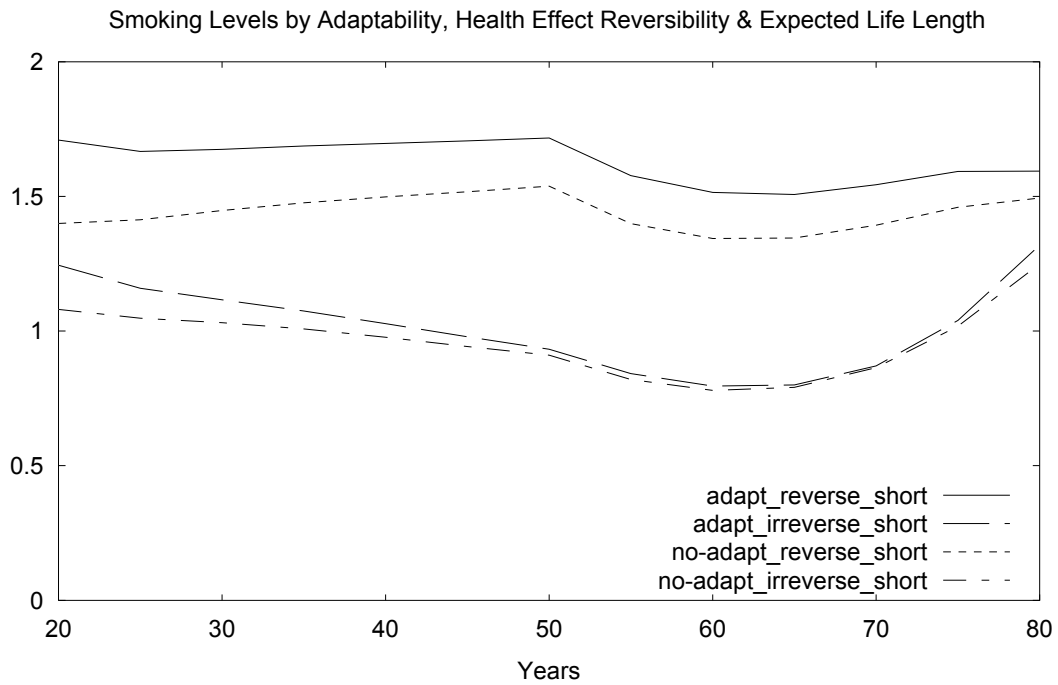


Figure 4:

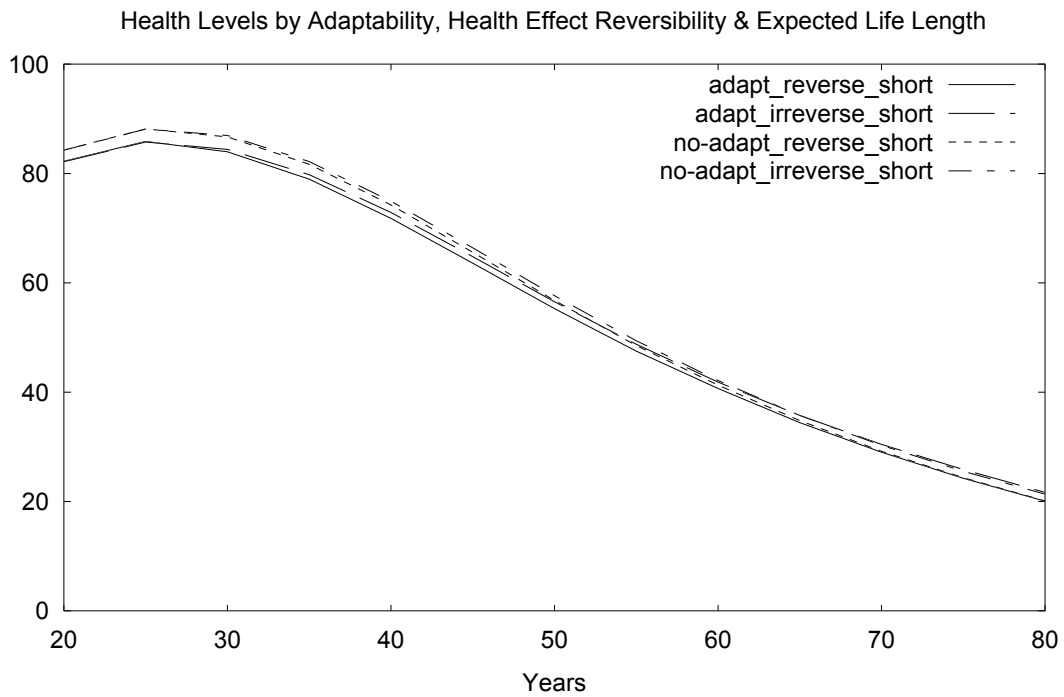
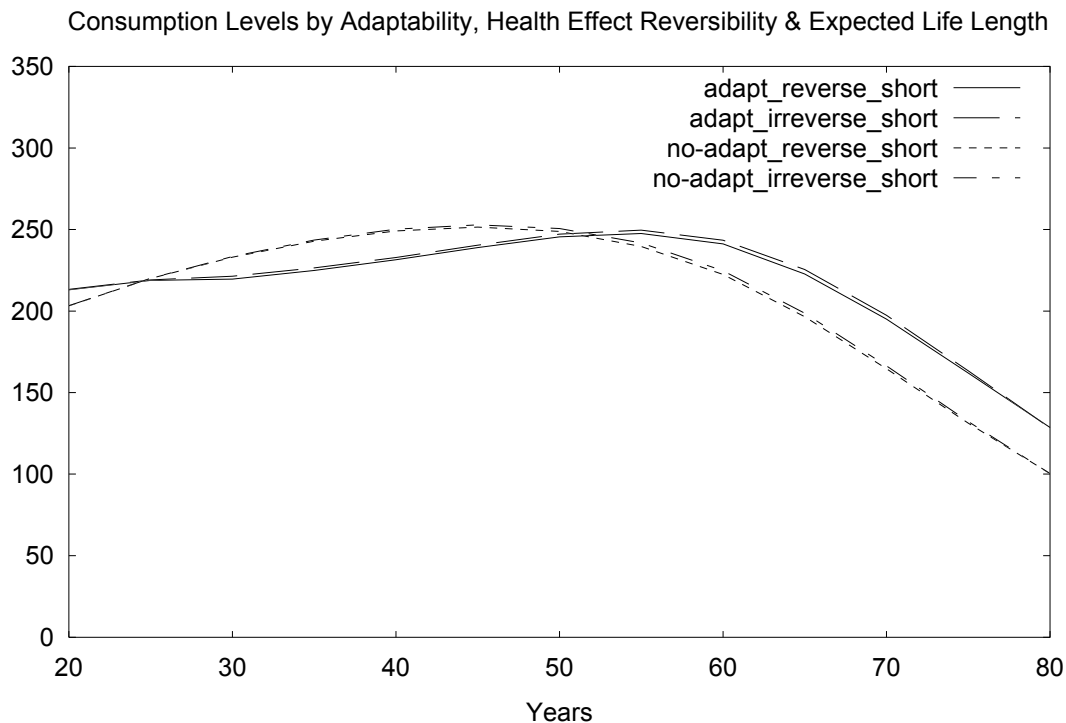


Figure 5:

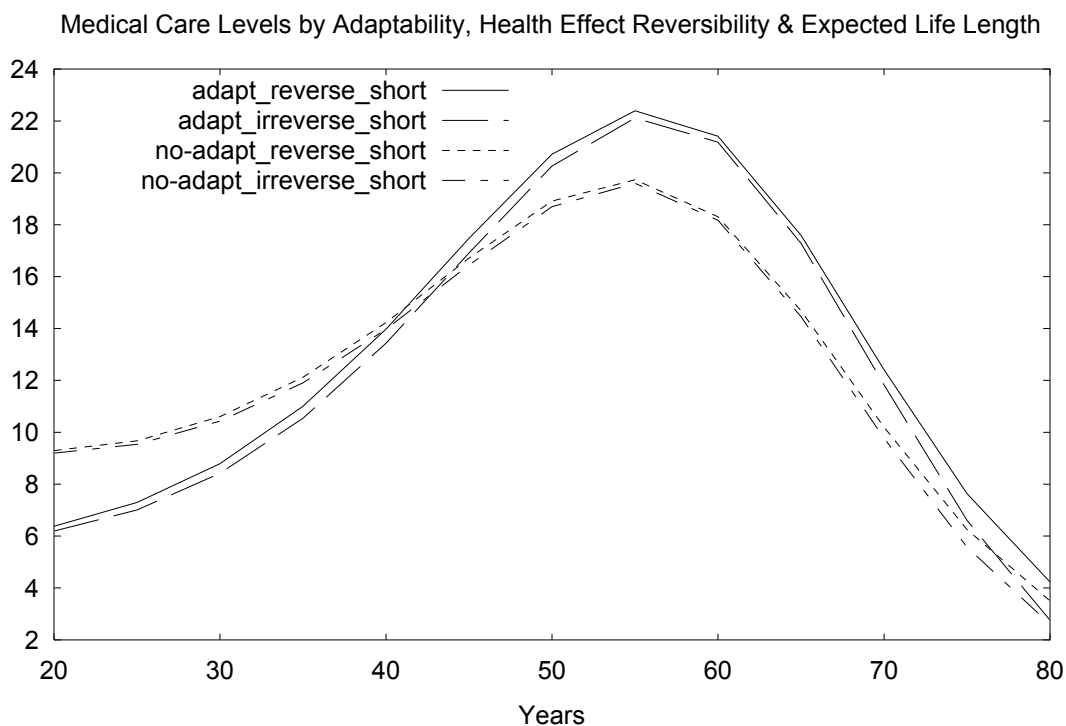


The *adaptation* dimension has similar effects for a smoker whether or not he believes in irreversible effects: He smokes more across the entire time horizon. This is because the adaptive individual's immediate utility levels may not be effected by a low health stock – as long as the rate of change in health is slow enough, the immediate health effects on utility are small. Higher smoking levels also come from the fact that the level of the subjective health stock is much lower when it embodies only changes in the stock. Since the lower health stock also makes non-addictive consumption less desirable, the smoker also chooses a lower level of non-addictive consumption up to age 50, substituting into cigarettes. Thus, for both reversible and irreversible health effects, adapters desire less health and smoke more compared to non-adapters.

Unlike the results in Gjerde et al. (2001), we find that adaptation increases the demand for medical care in the last part of life (Figure 6). In both models, adaptation gives an incentive to smooth the trajectory of the objective health stock so as to minimise the magnitude of the health stock depreciation from period to period, see Figure 4. However, in our model health and non-addictive consumption goods are complements

and not substitutes as in Gjerde et al. (2001). While lower health leads to higher consumption in their model, in our model investing in health is partly a means of investing in one's ability to consume non-addictive goods. This added incentive for investing in health explains the difference. In both models the major incentive for investing in health is prolonging life, an incentive that is weakened at older ages when the health stock depreciates so rapidly that the effect of medical purchases is shortened considerably. The inverted U path with peak at age 55 coincides with the results in Cropper (1977).

Figure 6:



As seen above, both reversibility belief and adaptation give an incentive to smoke more. However the trade-off between smoking and medical care is different in the two cases. While an adaptive person smokes more cigarettes and demands less medical care, at least in the first half of life, than a person who is not adaptive, the relationship between cigarettes and medical care is different when comparing the reversibility assumptions. A person, who believes in reversible smoking damages, consumes more medical care at the

same time as he also consumes more cigarettes than a person who believes in irreversibilities; there is greater potential for changing the probability of death late in life.

4.2 Changes in belief

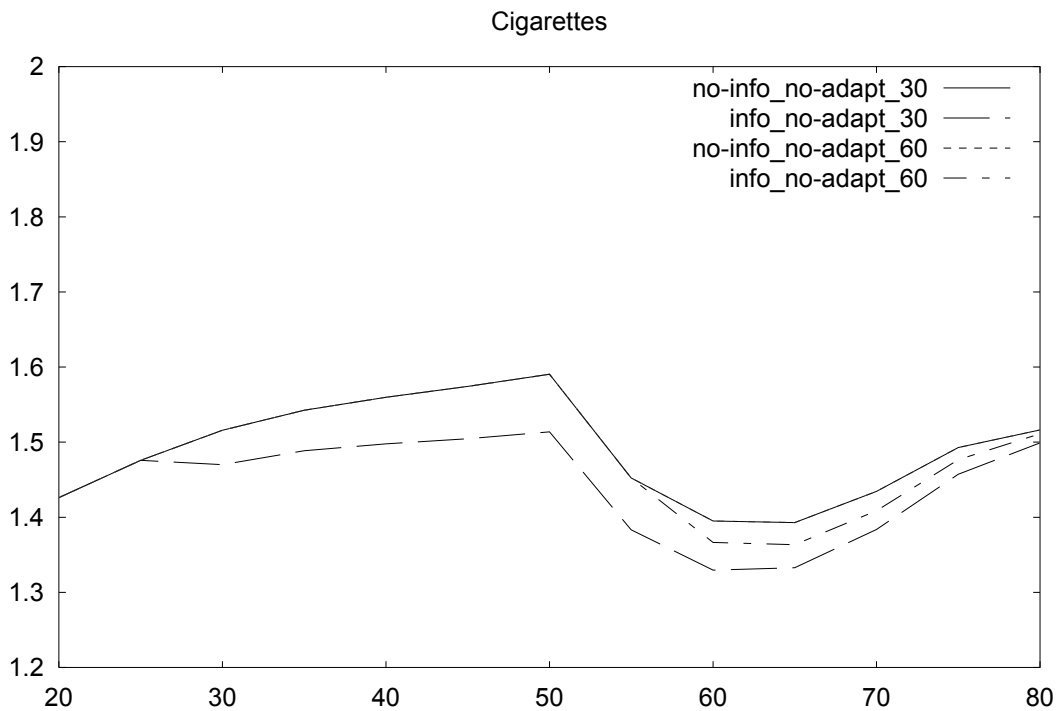
Above, we assumed that the individual does not revise his beliefs about the reversibility of health effects from smoking throughout the planning horizon. There may be reasons to believe that new information about reversibility may have different effects on individuals, depending on whether individuals already smoke or are considering taking up smoking. A focus on irreversible health effects increases the expected cost of smoking, but it also reduces the expected benefits from quitting or reducing smoking. Thus, a person who has been smoking for a long time may have a higher expected benefit from quitting or reducing smoking if he believes that the damages are reversible. A focus on reversibility of health effects would, however, also reduce the expected costs of smoking, and, therefore, induce more smoking in the earlier phase of life. Reducing smoking in a later phase of life may then be a way of investing in health.

Here, we study the effect of introducing information on irreversible health effects. The individual assumes the death risk to be reversible (see equation (24)), but gets new information about irreversible death risk (equation (12)). As mentioned above, in addition to increase the damage from smoking, introducing irreversibility gives a smaller incentive to reduce smoking as the effects of reduction on the probability of dying is less. The first effect probably counts more for a young person, while the second effect may count more for an old person. Therefore, we simulate the effects of new information at age 30 and 60.

Figures 7 and 8 show the effect on the smoking paths of new information for a non-adaptable as well as an adaptable person. This makes 4 new scenarios: *no-adapt_30* and *adapt_30* refer to the scenarios where new information is introduced when the individual is 30, while in *adapt_60* and *no-adapt_60*, information is introduced at age 60. 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 in our simulations. The effects are relatively small, but they are somewhat smaller for people at age 60. The introduction of health adaptation

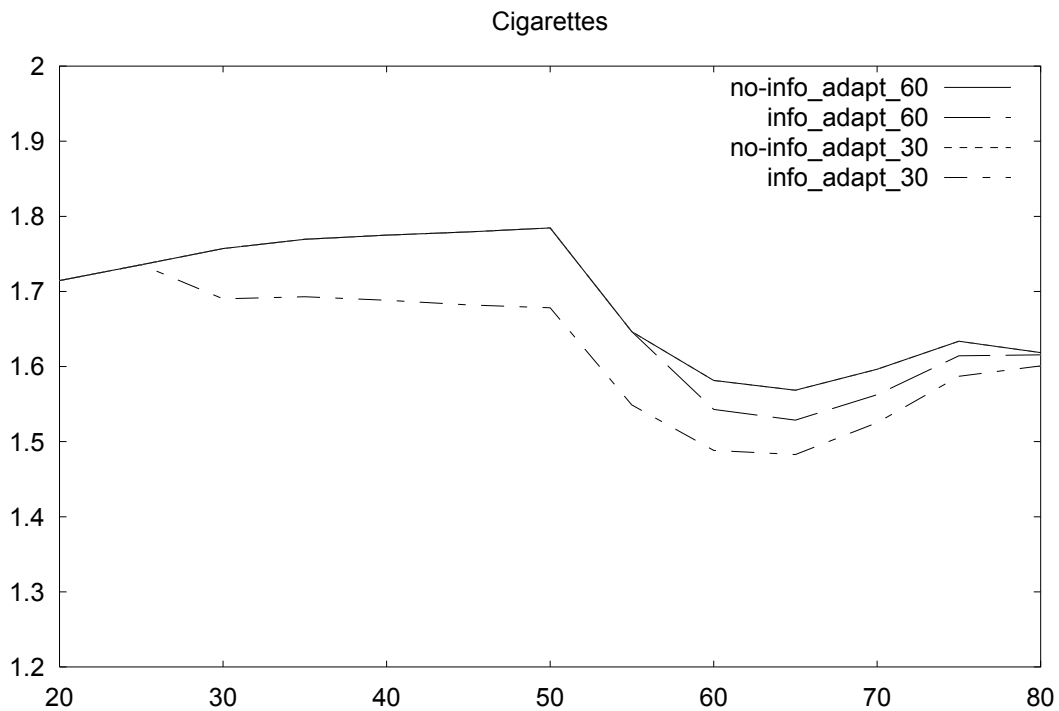
(see Figure 8) does not change the results. We also made simulations in the opposite case, i.e., using irreversible damage as the starting point and introduce reversible damage, but the results were symmetric.

Figure 7:



Our simulations show that the effects of new health information are relatively small. Similar results are found in other studies, however, in different settings. According to Viscusi (1990), information about health consequences of tobacco smoking seems to have reached consumers to the extent that even smokers apparently overestimate the death risk from tobacco. The impact of this information on smoking levels, though, may have been less marked than commonly believed. Also, according to Sloan et al. (2002), the impact of health information on smoking levels may have been less marked than commonly believed; price changes had a higher impact than health information.

Figure 8:

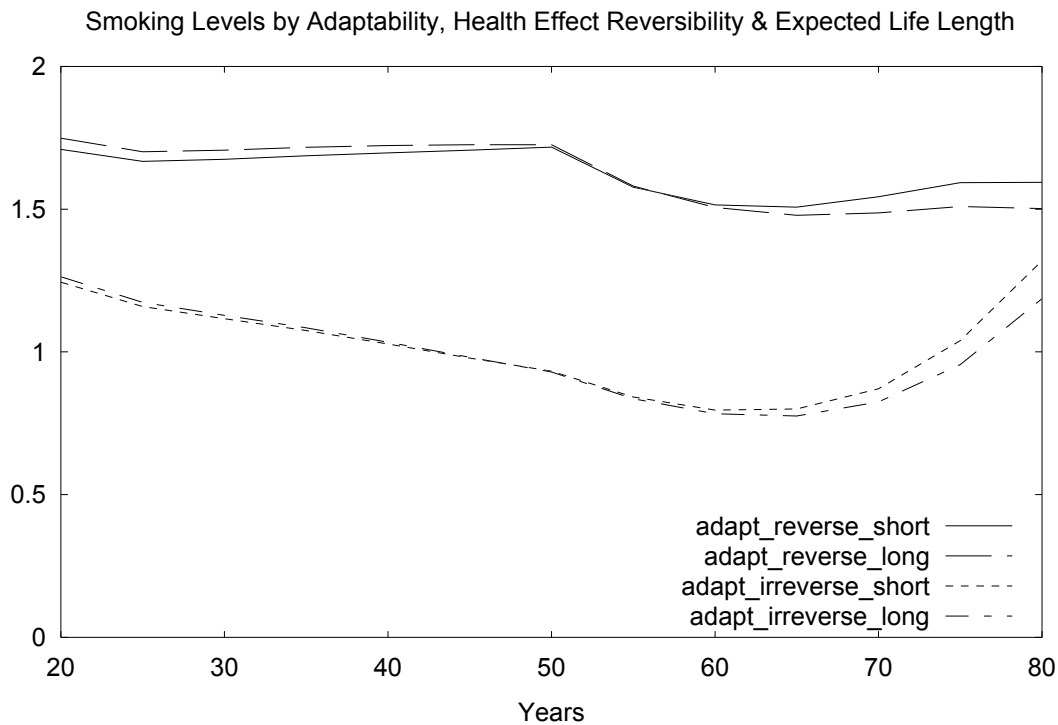


4.3 Potential Lifetime Effects

There is evidence from several developed countries that people in lower socio-economic groups have a shorter life expectancy. Also, people may have made some predictions of life expectancy based on their family history. Should we expect people endowed with a longer potential lifetime to smoke more or less? On one hand, if an individual can reasonably expect to live longer, then depreciating the health stock with smoking might be less costly, inducing him to smoke more (endowment effect). Alternatively, a longer potential life could imply that more future utility is at stake for each unit of health lost. Individuals with especially long lives have more to lose by increasing the probability of death at any point in time. We might reasonably expect this effect to lead to less smoking (substitution effect). Adda and Lechene (2001), using a similar, probabilistic approach to the problem, find that people smoke less as the latter argument dominates. The major difference between their model and ours is that our model adds a consumption value of health as well as the option to buy medical care. Any

overturn of their results, therefore, would have to focus on these additions. We want to investigate the effect of potential lifetime further by also identifying how the beliefs about health damage reversibility and health adaptation affect the result.

Figure 9:



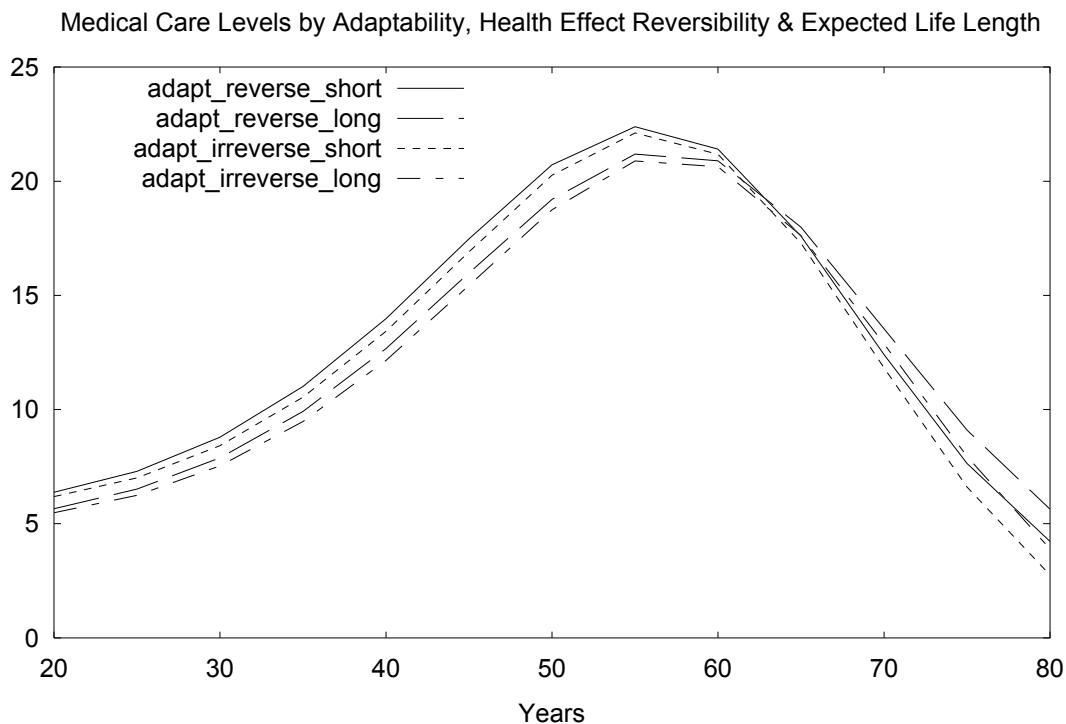
The experiment is carried out by changing the value of a parameter in the individual's hazard function, so that the survival probability associated with a certain health stock goes up or down.²¹ Figure 9 shows the effects of different assumptions on the length of the individual's potential lifetime in the *adaptation* case. We also run simulations for the no-adaptation case, but the results were similar. Even if the impacts are relatively small, endowing the individual with a longer potential lifetime causes him to smoke more up to age 50, and less in second part of life.²² Thus, while the individual is young, the depreciation effect dominates, while the effect that more future utility is at stake for an individual with a long potential lifetime (substitution effect), is the dominant

²¹ The key model parameter underlying this experiment is par_1 , which is listed in Appendix 3.

²² In the sensitivity analyse, see Section 4.4, we found that the endowment effect is dominating for a longer period if there is a flat income.

effect when the individual gets older. When the individual is endowed with more life, the shadow price on the health stock drops in the first phase of life, because the fact that life length is less scarce is the dominant effect. As a result, the individual substitutes into smoking until the margins on these two goods are once again equal. Further, as seen from Figure 10, he will also invest less in his health through medical care. More smoking and reduced consumption of medical care are ways to reduce health investments. However, while a person with a long potential life smoke less in the second half of life, he would consume less medical care for the whole life.

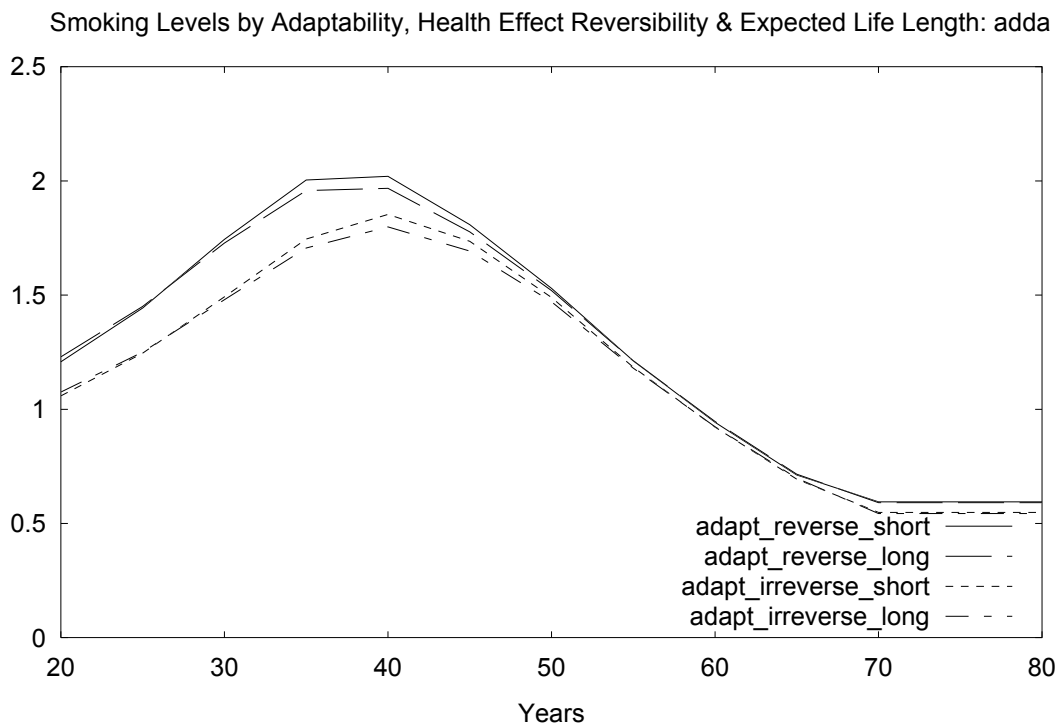
Figure 10:



The result is contrary to the findings in Adda and Lechene (2001), in which a longer potential life always leads to *less* smoking. How can we explain the different results? As seen from Figures 9 and 10, potential life length does not have any interesting interactions with the reversibility assumption. Thus, the difference in results does not lie in the reversibility or adaptability assumptions. Also, having a health good in the utility function as in our model, would seem to increase the value of extending life – not only would the individual get extra utility by living longer, but his health stock would also be

higher, which would tend to add utility through its effect on period utility. Thus, this cannot explain the different results.

Figure 11:



The other possibility is the introduction of medical care investments. Figure 11 shows a simulation where medical care is absent. As seen, we are able to get our model to exhibit reduced smoking when potential life is longer, under this new assumption, apart from the first few years. The reason is that the absence of medical care makes smoking more expensive, which reinforces the substitution effect mentioned above. One interpretation of this is that people, who have no access to medical care because they are poor, and also are endowed with a short potential life, would smoke more.

Finally, Table 2 shows the expected life length of the individual under the different cases and assumptions studied. The rows list the individual's adaptability, the columns list reversibility, and the two sub-tables apply to the different potential life assumptions.

Table 2:

Expected Life Length by Adaptation, Reversibility, & Potential Life

	Irreversible death risk	Reversible death risk
Short Potential Life		
No Adaptation	76.51	76.50
Adaptation	77.17	77.08
Long Potential Life		
No Adaptation	79.67	79.70
Adaptation	80.38	80.33

The first thing we learn from the table is that, at our calibration of the model, adaptation seems to have the largest effects on life length, with an adaptable person living 0.6-0.7 years shorter than a non-adaptable person. This is because the individual no longer cares about the level of his health stock in his utility function. While the life extension imperative is still there, it is no longer reinforced by this second channel.

Also noteworthy is the effect of reversibility of health damages on life expectancy. As seen from the table, the reversibility assumptions have only marginal impacts on this. Reversible damages lead always to increased smoking and increased medical care, while the opposite is true for irreversible damages. However, the net effect is actually a higher health stock in the irreversible case, see Figure 4. When it comes to life expectancy, this is outweighed by a higher death probability.

4.4 Sensitivity analyses

A numerical model as the one presented in this paper, includes a lot of parameters, which are hard to quantify. Therefore, sensitivity analyses are needed to test the robustness of the results. The parameters chosen are the parameters in the health

investment function (*med1* and *med2*), the health effects of smoking (*cig1*), time preference rate (ρ), and the utility effects of smoking (χ). We also simulate the case in which the income path is flat, i.e., Y is equal to 150 for $t < 150$, which is the average income for the core model. Finally, we made some simulations for different utility functions. Below, we will focus on the results of these sensitivity analyses for smoking and medical care paths under the four cases.

A change in the parameters will have an effect on the *development of the paths*. We studied this by doing runs with parameter values 20% higher and lower than in the base case. Using the *no-adapt_irreverse* case as the default case, we found that more effective medical care will increase the demand for medical care late in life, and move the bulk of smoking towards the first half of life. In general, smoking will increase with lower health damages, and with no damages from smoking at all, the level of smoking will increase over the lifetime. A lower time preference rate, i.e., if the individual cares more about the future, will reduce the level of smoking and increase the demand for medical care. Increasing the utility effects of smoking will also increase the smoking levels, but the health effects is somewhat outweighed by higher demand for medical care up to age 60. A flat income as specified above, will have minor impacts on smoking and medical care levels, while increasing income to 250 in all periods, will actually increase demand for medical care and reduce the smoking level.

To study the effects of changing parameters on *adaptability* and *reversibility*, we reduced all parameter values by 20 %. The results above are rather robust to these changes. The only changes are that with a lower time preference rate, medical care demand will be higher with irreversible health effects late in life in the no-adaptation case. In addition, a flat income makes medical care demand higher in the no-adaptation cases than in the adaptation cases after age 70.

As noted above, the additive *utility function* in Gjerde et al. (2001) gave some different results than the utility function in our analysis. Therefore, we made some runs with two other utility functions; one where all goods (Z , C , K) are complimentary (Cobb-Douglas bundle), and one where all goods are substitutes (additive utility function). The additive utility function gives the same qualitative results as in our analyses, apart from

the fact that medical care paths do not cross for the adaptation and no-adaptation cases, i.e., a similar result as in Gjerde et al. (2001). However, when all the goods are complementary as for the Cobb-Douglas utility function, the results are qualitatively different. The individual may demand more medical care in the irreversible case in the first half of life, and may actually also smoke more in the no-adaptation cases than in the adaptation cases in the mid phase of life. These results can be explained by the facts that all goods are complements, e.g., higher health also gives a higher marginal utility of smoking. As H has a higher absolute value than \dot{H} , the marginal utility of smoking will also be higher in the first case. However, this type of utility function does not seem to be standard in the addiction literature. Several papers use a quadratic form, which means that consumption and smoking are substitutes, see, e.g., Becker and Murphy (1988).

To sum up, while the developments of the paths are sensitive to changes in the different parameter values, the qualitative results from our analysis are quite robust. The results are, however, sensitive to the choice of utility function.

5. Discussion and conclusions

Smoking is harmful to your health. Beliefs about these harms differ, and public campaigns have tried to influence these beliefs. In this paper, we analyse the smoking decision of a rational addict in a framework where the health effects are explicitly modelled and the consequences of various beliefs are contrasted. The analysis reveals a number of non-obvious incentives influencing the rational smoker: With reversible effects, reducing smoking becomes a way of investing in your health. With irreversible effects, rational smokers reduce their smoking early on, but increase it in old age when the cost in terms of reduced life expectation is small. With complementarity between health and non-addictive consumption, investing in health is also a way of investing in your ability to enjoy consumption goods. Adaptation, getting used to poor health so that negative health shocks have only transitory effects on instantaneous utility, makes it less important to have a high level of objective health and more desirable to smoothen the health stock path. As a result, rational smokers smoke more. The same incentive influences the medical care levels: Adapters invest less in health when young and more

when old. Their health stock thus rises less when young, and falls less rapidly when old, which also results in a different consumption pattern.

The results imply that altering the beliefs of rational smokers along these dimensions, for instance due to an information campaign, might change the smoking paths rather than cause the smoker to quit. The effects are also relatively small. At the same time, it should be noted that the rational smoker in our model is not representative of real world smokers in all regards: As noted earlier, he smokes more lightly than most (less than five cigarettes a day). This, however, also depends on parameters and the utility function specified. The main point of the model is to make the different types of health beliefs and their consequences explicit and to integrate the health effects of smoking into a Grossman-style model of health investment. In this way, we also bring out the trade-off between smoking and investment in medical care. A person who smokes more because of adaptive preferences would not offset the health effects of smoking by demanding medical care in first half of life, but may do this in the last phase of life. Adaptation lowers the incentives to invest in health, and both smoking and lower demand for medical care are ways to reduce investments in health. On the other hand, a person who smokes more because he believes that health effects are reversible, would partly offset some of the immediate health effects by demanding medical care, especially in the latter half of life. The reason is that there is a greater potential for changing the probability of death late in life, compared to the case where the death risk is assumed to be irreversible.

Interestingly, the assumption of reversibility of health effects plays an almost insignificant role when it comes to expected lifetime. The reason is that even if a person assuming reversible health effects smokes more than a person assuming irreversible health effects, his investments in health will also be higher; he buys more medical care, and his reduction in smoking levels in the latter half of his life is also a way of investing in health. We do not find that a longer potential life leads to less smoking early in life as suggested by Adda and Lechene (2001). The reason for this lies in the specification of the model. While potential life length does not have any interesting interactions with the adaptability and reversibility assumptions, the effect of potential lifetime on smoking levels depends on access to medical care. With no access to medical care, the results by Adda and Lechene (2001) can be reproduced. One interpretation of this is that people,

who have no access to medical care because they are poor, and also are endowed with a short potential life, would smoke more.

The present paper can be seen as part of a line of research attempting to add realism to the decision problem in rational addiction models. However, many aspects of adaptation and health information are yet to be studied. The subject of addiction and risky behaviour is an interesting subject for economists to study, and introducing aspects from psychology may give new and important insight.

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APPENDIX 1: Symbols from the text

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

LU = lifetime utility

S = survivor function

V = current value Hamiltonian function

g, h = hazard functions

Exogenous variables

Y = income

m = price of medical care

p = price of cigarettes

q = price of non-addictive good

r = market interest rate

y = discount rate due to stochastic life time

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

Appendix 2: Solving the model

The general case

The current value Hamiltonian function to the optimisation problem in Section 2.6 is:

$$\begin{aligned}
 V(t) = & U(Z(t), R(C(t), A(t)), K(t))e^{-y(t)} \\
 & + \lambda(t)(C(t) - \mu_A A(t)) \\
 & + \pi(t)(rW(t) + Y(t) - m(t)M(t) - p(t)C(t) - q(t)Z(t)) \\
 (A1) \quad & + \gamma(t)h(H(t), D(t)) \\
 & + \eta(t)(F(M(t), C(t) - \mu_D D(t)) - \delta(t)H(t)) \\
 & + \sigma(t) \left(\frac{\beta}{1+\beta} H_0 - \beta K(t) + (1+\beta)(F(M(t), C(t) - \mu_D D(t)) - \delta(t)H(t)) \right) \\
 & + \phi(t)(C(t) - \mu_D D(t))
 \end{aligned}$$

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);

$$(A2) \quad \frac{\partial V(t)}{\partial Z(t)} = U'_Z e^{-y(t)} - \pi(t)q(t) = 0$$

$$(A3) \quad \frac{\partial V(t)}{\partial C(t)} = U'_R R'_C e^{-y(t)} + \lambda(t) - \pi(t)p(t) + \eta(t)F'_D + \sigma(t)(1+\beta)F'_D + \phi(t) = 0$$

$$(A4) \quad \frac{\partial V(t)}{\partial M(t)} = -\pi(t)m(t) + \eta(t)F'_M + \sigma(t)(1+\beta)F'_M = 0$$

$$(A5) \quad \dot{\lambda}(t) - \rho\lambda(t) = -\frac{\partial V(t)}{\partial A(t)} = -[U'_R R'_A e^{-y(t)} - \lambda(t)\mu_A]$$

$$(A6) \quad \dot{\pi}(t) - \rho\pi(t) = -\frac{\partial V(t)}{\partial W(t)} = -\pi r$$

$$(A7) \quad \dot{\gamma}(t) - \rho\gamma(t) = -\frac{\partial V(t)}{\partial y(t)} = U(Z(t), R(t), K(t))e^{-y(t)}$$

$$(A8) \quad \dot{\eta}(t) - \rho\eta(t) = -\frac{\partial V(t)}{\partial H(t)} = -[\gamma(t)h'_H - \eta(t)\delta(t) - \sigma(t)(1 + \beta)\delta(t)]$$

$$(A9) \quad \dot{\sigma}(t) - \rho\sigma(t) = -\frac{\partial V(t)}{\partial K(t)} = -[U'_K e^{-y(t)} - \sigma(t)\beta]$$

$$(A10) \quad \dot{\phi}(t) - \rho\phi(t) = -\frac{\partial V(t)}{\partial D(t)} = -[\gamma(t)h'_D - \eta(t)F'_D \mu_D - \sigma(t)(1 + \beta)F'_D \mu_D - \phi(t)\mu_D]$$

with the following transversality conditions:

$$(A11) \quad \lim_{t \rightarrow \infty} e^{-\rho t} \lambda(t)A(t) = 0; \quad \lim_{t \rightarrow \infty} e^{-\rho t} \pi(t)W(t) = 0; \quad \lim_{t \rightarrow \infty} e^{-\rho t} \gamma(t)y(t) = 0;$$

$$\lim_{t \rightarrow \infty} e^{-\rho t} \eta(t)H(t) = 0; \quad \lim_{t \rightarrow \infty} e^{-\rho t} \sigma(t)K(t) = 0; \quad \lim_{t \rightarrow \infty} e^{-\rho t} \phi(t)D(t) = 0$$

The *no-adapt* case ($\beta = 0$ and $K = H$)

In this case, the current value Hamiltonian becomes:

$$(A12) \quad \begin{aligned} V(t)_{\beta=0} &= U(Z(t), R(C(t), A(t)), H(t))e^{-y(t)} \\ &+ \lambda(t)(C(t) - \mu_A A(t)) \\ &+ \pi(t)(rW(t) + Y(t) - m(t)M(t) - p(t)C(t) - q(t)Z(t)) \\ &+ \gamma(t)h(H(t), D(t)) + \eta(t)(F(M(t), C(t) - \mu_D D(t)) - \delta(t)H(t)) \\ &+ \phi(t)(C(t) - \mu_D D(t)) \end{aligned}$$

The sufficient conditions can be outlined as above. The transversality conditions follow from (A11).

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

The current value Hamiltonian becomes:

$$\begin{aligned}
 (A13) \quad V(t)_{\beta=\infty} &= U(Z(t), R(C(t), A(t)), F(M(t), C(t) - \mu_D D(t)) - \delta(t)H(t)) e^{-\gamma(t)} \\
 &+ \lambda(t)(C(t) - \mu_A A(t)) \\
 &+ \pi(t)(rW(t) + Y(t) - m(t)M(t) - p(t)C(t) - q(t)Z(t)) \\
 &+ \gamma(t)h(H(t), D(t)) \\
 &+ \eta(t)(F(M(t), C(t) - \mu_D D(t)) - \delta(t)H(t)) \\
 &+ \phi(t)(C(t) - \mu_D D(t))
 \end{aligned}$$

The sufficient conditions can be outlined as above. The transversality conditions follow from (A11).

APPENDIX 3: 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 *utility function* is additive in consumption of addictive and non-addictive goods, while non-addictive consumption and health are complementary goods:

$$(A14) U(Z_t, R_t, K_t) = Z_t^\alpha (K_t + H_0)^\gamma + R_t^\chi$$

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;

$$(A15) y_t = y_{t-1} + 5 \cdot par_1 \cdot par_2 \left(\frac{1}{\left[\frac{H_t - cig_1 e^{\frac{D_t}{2}}}{critical} \right]^3 + 1} \right)^{par_2 - 1}$$

$par_1 = 0.02$, $par_2 = 2.5$, 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 0.03.²³

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

$$(A16) F(M_t, D_t - D_{t-1}) = med_1 \cdot M_t^{med_2} - cig_1 \cdot fragility_t \cdot (2e^{D_t - D_{t-1}} - 2)$$

²³ To calculate this, we assume that the price of a pack, i.e., 20, cigarettes is \$3,50, and that the average income is \$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 minutes in the *adapt_reverse* case, and 13.7 minutes in the *no-adapt_reverse* case.

$med_1 = 2$ and $med_2 = 0.1$ are set to reproduce similar results as in Gjerde et al. (2001) when smoking is set equal to zero in all periods. $fragilty_t = 1$ 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:

$$(A17) R_t = r_c C_t + r_a A_t + r_{c2} C_t^2 + r_{a2} A_t^2 + r_{ac} C_t A_t$$

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..