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SMOKING AND ENDOGENOUS MORTALITY: DOES HETEROGENEITY IN LIFE EXPECTANCY EXPLAIN DIFFERENCES IN SMOKING BEHAVIOR?

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Smoking and Endogenous Mortality:
Does Heterogeneity in Life Expectancy Explain Differences in Smoking Behavior?

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Abstract

This paper proposes a joint model of tobacco consumption and mortality over the life-cycle. The decision to smoke is a trade off between current utility derived from smoking and a mortality risk increasing with age. Individuals with a longer potential life expectancy have more incentive to cut back on smoking and thus self select out of smoking. Using detailed data on mortality, morbidity and smoking we are able to identify this selection effect. We empirically evaluate its importance in explaining heterogeneity in smoking behaviour among adults. We find that heterogeneity in potential life expectancy explains part of the heterogeneity in smoking behaviour, even when conditioning on sex, education and occupation and information on other risky behaviour. When we embed heterogenous potential life expectancies within a rational addiction model of smoking, we find that the model is able to match the life cycle profiles of smoking.

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1 Introduction

In developed countries, tobacco use is not uniform across the population. It has been persistently associated with differences in education, sex, occupation or race. For instance, low educated individuals, men or blue collar workers are more likely to smoke. It has also been documented that *non* smokers belonging to these groups have shorter life expectancies. In other words, variables which predict smoking are also found to predict mortality for non smokers. [1]

In this paper, we construct a model of smoking and endogenous life expectancy which can account for these facts. We develop a model of smoking where rational and forward looking agents perceive the future mortality risks due to smoking. In this setting the agents face a trade-off between current cigarette consumption and a subsequent increase in their mortality rate. A key variable in this trade-off is potential life expectancy, defined as the life expectancy of the same individual as a non smoker. Under the assumption that the detrimental effect of tobacco on health is greater for individuals with long potential life expectancy, these individuals are more likely to quit or to reduce their cigarette consumption to avoid a premature death. We discuss the identification of selection into or out of smoking and we demonstrate empirically that trade-offs between smoking and longevity are important to understand heterogeneity in smoking behavior among adults.

We extend the model of rational addiction by Becker and Murphy (1988) to account for endogenous mortality. Models with endogenous longevity have been derived by Grossman (1972), Cropper (1977), Ehrlich and Chuma (1990), Orphanides and Zervos (1998), Suranovic et al. (1999), Ehrlich (2000) and Choo (2001). Becker and Mulligan (1997) also investigate a theoretical model where agents have an endogenous discount rate.

Heterogeneity in smoking behaviour has been studied by Farrell and Fuchs (1982) in the case of teenagers. They point out that smoking does not appear to be caused by education, but rather that both outcomes are caused by a "third variable". The authors discuss how differences in discount rates can explain differences in both human capital and health investment. In the context of intertemporal choice, the weight individuals

[1] See Smith and Shipley (1991) or Sterling and Weinkam (1990) for instance.

put on future events depends on the pure rate of time preference, but also on the length of the life horizon. Although the effect of the life horizon on smoking decision is likely to be small for teenagers, older individuals with a long potential life expectancy might have greater incentives to curb their tobacco consumption, as smoking related diseases may jeopardize many years of expected life. On the contrary, individuals with a short potential life expectancy might realize that the benefit of quitting smoking are smaller. Irrespective of smoking, individuals differ in their life expectancy. For instance, men, blue collar workers or less educated individuals have a lower life expectancy. Hence, these differences might explain partly the differences in smoking, and be part of the "third variable" discussed by Farrell and Fuchs (1982).

The evidence in the literature suggests a selection into and out of smoking. Observed mortality is obviously affected by smoking. We conjecture that smoking decisions are in turn partially determined by mortality. In such a context, identification within the framework of an intertemporal choice model can be achieved only through the use of restrictive identifying assumptions on unobserved heterogeneity. With only data on consumption and mortality, we show that the selection effect is identified in two rather unappealing cases; either consumption heterogeneity derives from heterogeneity in life expectancy alone, or the joint distribution of all heterogeneity terms in the model is known. However, we show that these restrictive assumptions can be relaxed if one also observes repeated information on the health of the individual.

We use an extensive panel data set following Swedish individuals for up to eighteen years, recording their smoking behavior, mortality, cause of death, a range of morbidity indicators and information on individual and family characteristics such as education, occupation and family income. Using this data set, together with epidemiological results, we separate the selection effect from the causal effect of tobacco. We take into account heterogeneity in potential life expectancy, and we find that the cost of smoking in terms of life expectancy is higher for individuals with longer potential life expectancy. We also find evidence of selection as predicted by our model, as individuals with longer potential life expectancy smoke less and quit earlier. This is true when we condition on social or demographical variables such as sex, education level, occupation or region of living. The result still holds when we control for an extensive set of variables capturing other risky consumption or behaviors. At young ages, potential life expectancy has

little effect on smoking. But as individuals age, the effect becomes as strong as other more "classic" predictors of smoking such as sex, education or occupation.

Our methodology enable us to explain the selection into and, more importantly, out of tobacco. At young ages, a large fraction of individuals would experiment with cigarettes. This is because the marginal impact of smoking on health is low for beginners and because the consequences on health are far away in time from adolescence. A large part of the differences in smoking arises around mid age as only part of the smokers quit. In our model, individuals quit to limit the effect of cigarettes on future mortality. These individuals might quit even before any serious health problems occur. The empirical literature has found mixed results on the effect of health and health shocks on quitting (Jones (1994)). From our model, this is not necessarily an indication that health plays no role in smoking decisions, but that smokers are forward looking.

Finally, we investigate the ability of the model to fit observed life cycle profiles by life expectancy groups. We evaluate how much of the difference in observed smoking behavior across groups of different potential life expectancy, can be explained by a measurable heterogeneity in life expectancy. In particular, can a simple model where agents have identical preferences and the same pure rate of time preference, but a different potential life expectancy come close to the observed data? We find the parameters describing preferences and the pure rate of time preference (common across all agents), that bring the predicted smoking behavior for each type of individuals as close as possible to the observed one. We show that a non trivial part of the variance between groups can be explained by a model where agents trade-off current utility of smoking against an increased mortality risk later on in life. Failing to take into account the incentives provided by differences in potential life expectancy would lead to overstate differences in taste or in the pure rate of time preference across agents.

The literature has attempted to explain the heterogeneity in smoking, appealing to differences in the understanding of the consequences of smoking on health, to differences in the rate of time preference across education, occupation and possibly sex, to social norms correlated both with smoking and life expectancy and to heterogeneity in addiction or in taste across demographic or socio-economic groups.

Smoking is a complex phenomenon, and some of these explanations may be perti-

nent to understand behavior. However, Kenkel (1991) shows that conditioning on the medical knowledge about the consequences of smoking explains little of the variance in smoking behavior. Meara (2001) also confirms this point. Viscusi (1990) shows that, far from ignoring health related risks, smokers overstate the probability of death due to smoking. [2] Therefore, differences in medical knowledge do not seem a promising route to pursue. Moreover, it can hardly explain the gender difference. While in theory, differences in discount rates are usually associated with differences in education, there is very little empirical evidence of this, mainly because discount rates are very difficult to identify separately from preferences. Using the PSID, Lawrence (1991) finds evidence of differences in discount rates across education groups, but whether these differences stem from differences in life expectancy or in the pure rate of time preference is not clear. Finally, whether the pure rate of time preference is sex specific remains an open question.

The implicit assumption in most of the models in the literature is that differences in smoking behavior stem from heterogeneity in preferences or in the pure rate of time preference. In this paper, we provide empirical evidence which shows that potential life expectancy explains smoking behavior independently of education, occupation, sex and other risky choices. We conjecture that it plays the same role in determining other risky behaviors as drinking or substance abuse. Potential life expectancy may also influence other life cycle choices, such as education, consumption and savings for retirement. Davies (1981) and Hurd (1989) show the effect of mortality on savings. Part of the heterogeneity in savings behavior could stem from heterogeneity in potential life expectancy.

We begin in Section 2 with a presentation of the evidence on smoking across age and socio-demographic groups. Section 3 presents the structural model of smoking and endogenous longevity. We estimate individual specific life expectancies, conditional on smoking and potential life expectancies in section 4. These results are imbedded into the behavioral model to produce life cycle profiles of smoking in section 4.3. In section 5, we evaluate how much the heterogeneity in potential life expectancy explains the heterogeneity in smoking behaviour. Finally, the structural consumption model is estimated in section 6. Section 7 takes an informal view on other developed countries

[2] Further evidence is given in Viscusi (1992) and Antoanzas et al. (2000).

to evaluate how the Swedish results can be extrapolated.

2 Socio-economic Evidence on Smoking and Death

After a presentation of the data, we document differences in prevalence and quantities smoked for different socio-economic groups, as well as mortality for non smokers.

2.1 The Data

We use data from ULF, the Swedish Survey of Living Condition. Approximately 6000 individuals are surveyed each year. The ULF reports information on quantities smoked, smoking history, education, occupation, family composition, income as well as several health measures. The data set has been merged with the Record of Deaths in 1999, so we observe whether a given individual is alive up to the end of 1998, and if not, the date and cause of death. We use the 1980-81, 1988-89 and 1996-97 cross sections, as in these years the survey has a special section on health. In total, the data set includes 38986 individuals.

Of these 38986 individuals, approximately 3000 are actually present at three dates, in 1980, 88 and 96, or in 1981, 89 and 97, which provides a panel. This panel is obviously not representative of the Swedish population, as it oversamples young and healthy individuals who have survived for 16 years.

Some individuals, interviewed in 1980-81 have also been interviewed in 1988-89, but died before the third interview. Finally, a third category of individuals were surveyed in 1980-81, but died before the second interview. For the purpose of this study, we constructed a data set in a way that it is representative of the Swedish population in 1980 for an extensive set of characteristics. This was done by incorporating different types of observations. First, we used the individuals who were surveyed three times, a group which is on average younger. We then brought in individuals who were surveyed in 1980, but who died before the second or the third interview, in such a manner that the characteristics for the constructed data set matches the characteristics of the Swedish population in 1980. More precisely, from the whole cross-section, we first computed the fraction of Swedish individuals alive in 1980 but dying in year $1980 + i$, $i = 1, \ldots, 16$. We then drew randomly from the 1980 survey a given number of individuals who were

dying in year $1980 + i$, such that the share of these individuals in the panel data set were similar to the one in the whole cross-section. With this procedure, we obtained a panel data set which matched the Swedish population in terms of mortality over the period 1980-1998.

We repeated the random draws with replacement until both data sets were similar along other characteristics as well. Table A in the appendix displays the characteristics for our data set and of the Swedish population. Our sample is representative in terms of the mean and variance of age, sex, household income, education, marital status, occupation, health, smoking status and smoking prevalence. [3]

The resulting data set follows 3962 individuals from 1980 to 1998, with information at up to three dates in 1980-81, 1988-89 and in 1996-97. We also matched the data set with monthly prices of tobacco, relative to other consumption goods, provided by Statistic Sweden.

2.2 Evidence on Smoking and Mortality

With an average smoking prevalence of about 26% over the period 1980-1997, Sweden is below the average of European and developed countries (at about 30%). The decrease in prevalence from 33% to 20% over that period compares with similar countries. This decrease in prevalence is accompanied by a large increase in the relative price of tobacco of about 7% per year over the same period, after a long period of stability or even decrease in real terms. The relative price went from 1 in 1990 to 1.6 in 1998, together with a host of measures destined to reduce prevalence and quantities smoked in Sweden.

2.2.1 Socio-Economic Determinants of Quantities Smoked and Duration to Quitting

Table 1 shows the determinants of the quantities smoked, by regressing the quantities on socio-economic variables. Sex, education and being in a white collar job are all

[3] We also checked the representativeness of each of the three categories of individuals we merged together. Individuals who have been surveyed three times have similar characteristics to Swedish individuals who survive at least up to 1999. Individuals who are surveyed twice are close to individuals who survive at most up to 1996, and individuals who are surveyed just once are representative of those who survive at most up to 1988 (results available upon request).

important determinants. Age has a strong effect, as young and older individuals smoke less. Prices have an elasticity of -0.25, which is comparable to other countries (see for instance, Chaloupka and Warner (2000)). Family income explains independently quantities smoked with an elasticity of about 0.03. This elasticity is positive but very low. The significant relationship between income and smoking levels is due to the presence of young individuals in the survey. When performing the regression on individuals older than 25, the elasticity is divided by a factor of ten and is not significant any more. This might be due to the fact that the younger individuals in the data (16 years old) face liquidity constraints which prevent them from buying cigarettes.

Table 1: Determinants of Quantities Smoked and Quitting

	All		Current Smoker		Quitting	
Variable	Estimate	t-stat	Estimate	t-stat	Odds Ratio	t-stat
Log Price	-2.97	-18.6	-2.35	-5.8		
Log Current Income	-.16	-3.2	.10	0.9	1.03	0.9
Male	.75	10.3	2.27	13.8	1.3	5.2
Low Education	1.76	13.3	.94	3.2	0.58	-7.4
Middle Education	1.30	11.1	.66	2.3	0.79	-4.1
Blue collar	.84	6.4	-.34	-1.2	1.14	1.2
White Collar	-.41	-2.6	-.47	-1.3	1.16	1.2
Self-employed	.55	2.4	1.14	2.4	1.13	0.9
household size	-.63	-15.5	-4.45	-5.5	1.2	10.7
Age	0.22	18.2	0.42	13.5		
Age Square	-0.003	-23.1	-0.005	-15.4		
Number of Obs.	38464		9653		3593	

Notes: Heteroskedastic corrected standard errors were computed. For duration to quit, retired, students and individuals younger than 25 were excluded from the regression.

The last two columns displays the results of a proportional hazard model of duration to quitting. Individuals with a higher education as well as white collars are more likely to quit smoking at all durations.

2.2.2 Death and Socio-Economic Characteristics among Non-Smokers

We estimate a logit model for the probability of dying within five years for *non* smokers, to establish that less educated individuals, men, and blue collar workers are more likely to die. In the data, there are no details of previous occupation for retired individuals. Retired individuals are more likely to die, so when they are included in the regression, they scale down the effect of occupation. We therefore present the results with and without retired individuals.

Table 2: Probability of Death within 5 Years, Non Smokers

	All		Excluding Retired	
Variable	Odds Ratio	Std Error	Odds Ratio	Std Error
Men	1.94	0.15	1.74	0.42
Middle Education	0.67	0.07	0.83	0.21
High Education	0.63	0.12	0.72	0.34
Blue Collar	0.57	0.13	1.3	0.47
White Collar	0.40	0.13	0.90	0.46
Self Employed	0.53	0.16	0.89	0.44

Note: Regression included age and year dummies. All regression excluded farmers and students. Robust standard errors were computed.

In Sweden, as in most developed countries, socio-demographic characteristics such as sex, occupation or education predict both smoking and the likelihood of death for non smokers. We present in next section a model that can account for these facts.

3 A Structural Model of Smoking and Life Expectancy

We assume that the agent is rational and forward looking. His decision about cigarette smoking is at each date a trade off between an immediate pleasure of smoking and an increased mortality risk in the future. The elements of the trade-off vary through time, as by smoking, the agent increases smoking related stocks which modify both the immediate pleasure from smoking and the future mortality risks.

3.1 Addiction and Health Effects of Smoking

The medical and epidemiological literature point to two distinct effects of smoking through the life cycle. One has to do with addiction, and the other has to do with the health effect of smoking. The addictive effect of cigarette consumption is analysed by economists within the framework of the rational addiction model, following Becker and Murphy (1988), by introducing past cigarette consumption as an argument of utility. The other effect of tobacco consumption is an effect on health, both an instantaneous and a long term effect. We will capture these two aspects of cigarette consumption by introducing two stocks in the model, denoted S^A and S^M, affecting respectively utility and the mortality risk. The stock S^A captures mainly the effect of nicotine, while S^M captures the effect of tar and carcinogens. The stocks depreciate respectively at rates δ_A and δ_M, which are thought to be different. According to the medical and epidemiological literature, the addictive stock depreciates relatively fast, at least from a life cycle perspective, and the stock affecting mortality depreciates very slowly, if at all. [4]

3.2 Preferences

The agent can consume two types of goods: a quantity q_t of cigarettes and a consumption bundle c_t. The agent derives utility from the two goods and from the stock of addiction:

[4] The epidemiological literature gives mixed results on the depreciation of the stock of cigarettes and its effect on health. For instance, clinical studies show that the respiratory capacity of smokers is lowered permanently, and impaired lung function has been associated with an increased mortality risk. Peto et al. (2000) show that the risk of lung cancer of quitters never gets quite as low as for life-long non smokers. On the contrary, other studies such as Doll et al. (1994) show that life table estimates of quitters converge to those of non smokers. The latter could suggest that either the stock depreciates, or that, longer life expectancy individuals quit earlier so that we observe individuals who have quit for a long time being in better health. This explanation does not rely on a depreciation of the stock, but suppose a selection out of tobacco based on life expectancy. Our model implies such a selection and is thus able, at least qualitatively, to generate similar results. Even if we do not model a depreciation of the stock S^M, simulated data from our model on smoking and survival would "appear" to support a depreciation of the stock.

$$u(q_t, c_t, S_t^A) = \nu_1 q_t^2 + \nu_2 c_t^2 + \nu_3 S_t^A q_t + \nu_4 q_t + \nu_5 c_t + \nu_6 \quad (1)$$

q_t is the number of cigarettes smoked per day and can take discrete values between 0 and Q. This utility function is similar to the one used in previous studies, such as Becker et al. (1994). [5]

3.3 Mortality

We assume that the agent has an uncertain life time, although the agent knows at all time the distribution of the age at death. The agent updates its life expectancy every period by observing that he is still alive. As a consequence, the uncertainty about the age at death decreases as the individual ages. We also assume that the agent has a perfect knowledge of the consequences of smoking. [6]

At each period in time, the agent faces a probability of surviving from one period to the next, which depends on age and on the quantity of tobacco smoked up to that period, through the stock S^M. This probability also depends on an agent specific characteristic ε which is known to the agent but unobserved by the econometrician. ε captures the influence of genetic differences (sex or parental health), of differences in-utero exposure (Barker (1992)) or because of psycho-social effects (Marmot et al. (1991)). We consider all of these causes as fixed and beyond the control of the individual. Let's denote $\pi(age, S^M, \varepsilon)$ the probability of surviving from one period to the next (conditional on being alive at a given age). We assume that $\pi(age, S^M, \varepsilon)$ is decreasing in age and bounded between 0 and 1. $\pi(age, S^M, \varepsilon)$ is also decreasing in S^M. Epidemiological studies suggest that the effect of tobacco is non linear and increasing in the stock(see Doll et al. (1994)). This can be captured by allowing a non zero second derivative with respect to S^M. As the function π depends on age, we also allow for an age specific effect of tobacco on mortality. In the empirical section, we use

[5] Given the intertemporal trade-off described below and especially the endogenous discount rate, the constant in the utility function is actually identified up to a multiplicative constant.

[6] A number of studies show that individuals are able to forecast their life expectancy, see for instance Hurd and McGarry (1995), Hurd et al. (1999) or Smith et al. (2000). Antoanzas et al. (2000) using Spanish data, shows that smokers correctly evaluate the loss of life due to smoking, while Viscusi (1990) shows that individuals overestimate the risks from smoking.

a flexible specification and we allow for type specific differences in the effect of tobacco on overall mortality.

3.4 Optimal Behavior

Let $V(S^A, S^M, age, p)$ be the value (i.e. the life time utility flow) of an agent with a stock of addiction S^A, a stock affecting mortality S^M, given the state of prices p. The value evolves as:

$$V(S^A, S^M, age, p) = \max_q \left[u(q, y - pq, S^A) + \beta\pi(S^M, age, \varepsilon)E_{p'/p}V(S^{A\prime}, S^{M\prime}, age + 1, p')\right] \tag{2}$$

$$\text{with} \quad \begin{array}{l} S^{A\prime} = (1 - \delta_A)S^A + q \\ S^{M\prime} = (1 - \delta_M)S^M + q \end{array}$$

Let p be the relative price of cigarettes. We assume the following budget constraint:

$$pq + c = y(age, \varepsilon) \tag{3}$$

where $y(age, \varepsilon)$ is income which depends on the age and on the specific mortality characteristic of the agent. This capture both the effect of health on earning capabilities and the different investments in human capital which might arise because of differences in life expectancies. The path of income is taken as deterministic for simplicity, but can vary between agents. For simplicity, we assume that the agent cannot save. Another interpretation is that the saving decision is taken in a first step and $y(age, \varepsilon)$ is the total expenditure to be allocated between the two goods in a second step.

In (2), the agent chooses a cigarette consumption q and has a probability $\pi(S^M, age, \varepsilon)$ of surviving into next period. β is the pure discount factor. [7] The relative price of tobacco is taken to be stochastic and we assume rational expectation over future prices. Next period, both stocks are updated, and the agent re-optimizes.

The trade-off between smoking and an increased life expectancy depends on several factors and can vary across individuals and across time. First there might be heterogeneity in taste (i.e. in the utility function), with some individuals having a higher

[7] In this model, the behavior of the agent is time consistent, although the weight on the future flow of utility is time varying. See Gruber and Koszegi (2001) for time inconsistent behaviors. Orphanides and Zervos (1998) also presents a model with an endogenous discount rate, where consumption of cigarettes increases myopic behaviors.

taste for tobacco. Second, there might be differences in the pure discount rate, with some individuals being more patient. Finally, a natural source of heterogeneity is in potential life expectancy, the life expectancy the individual would have had without smoking. This is captured by ε. If the detrimental effect of tobacco on health is higher for individuals with longer potential life expectancy then smoking is less desirable for these individuals, whereas individuals who would have died early anyway would have low incentives to curb smoking. Thus our model is able to explain part of the heterogeneity in smoking without resorting to differences in taste or in the pure rate of time preference. Individuals with low education might (partly) smoke more because they also have a shorter potential life expectancy rather than because they have a higher taste for cigarettes. We empirically evaluate the importance of this effect in section 5.

The model does not have an analytical solution, even for very simple utility functions. The model has to be solved numerically, using the Bellman equation (2). The optimal smoking path depends on the shape of the survival probability and on the effect of tobacco on health, which is characterized by the probability $\pi(age, S, \varepsilon)$. As it is important to have an accurate characterization of this function, we delay presentation of optimal consumption rules until section 4.3.

3.5 Old Age

As the model is set up, individuals close to their expected age of death- mainly in their late seventies in the developed world- would appear to be extremely myopic, as their probability of surviving gets very low. It would therefore be rational for smokers to increase their consumption and for ex-smokers to take up smoking again. [8] Obviously, there is no such evidence in the data. This can arise for a number of reasons. First, smoking could be less attractive in old age, either because taste changes over time or because health problems are magnified by smoking when old. This latter effect probably exists. However it might be difficult to estimate, as smokers who would experience such health problems are certainly encouraged to quit before their realization. [9] Second,

[8] Note that the problem would be similar in the rational addiction model with a finite horizon. As the agent approaches the end of life, there is less and less reasons to care for the future effect of cigarettes.

[9] We also tried a survival model where smoking at an old age had an effect on mortality, independent from the stock of cigarettes. We found this effect to be very minor.

individuals could be less myopic than the model would suggest. The pure rate of time preference may be decreasing with age.

Given that this problem arises only at old age, we do not put too much emphasis on this point. First, most of the interesting action takes place years before, from adolescence until the mid sixties with different patterns of consumption across groups. Second, the optimal consumption rules may be increasing after seventy, but given that mortality is higher around that age, not all the individuals would have a chance to display such features. Third, the model could easily incorporate a change in taste for elderly people. However, with time specific utility functions, any pattern could be explained, even at young ages, so we do not find this approach very attractive.

The problem is somehow similar to the saving puzzle during retirement. Elderly people have much higher savings than standard models would predict, as if their life horizon was much longer than it actually is.

3.6 Peer Effects and Multiple Risky Choices

In (2), we have not modelled any influence of smoking by other agents through the utility function, mainly because we do not observe in our data set the smoking status of other members in the household or at work. This correlation is often interpreted as peer-pressure. There are two reasons why our model would generate such a correlation, even without any interaction through the utility function. First, there could be assortative matching if individuals with similar potential life expectancy tend to associate together. Within a household, it might be difficult to agree on intertemporal trade-offs if the members have very different life horizons. If this is the case, then smokers are more likely to associate with smokers. Second, to the extent that passive smoking has an effect on mortality, there is an indirect effect in our model through the survival probability. An agent who is a passive smoker realizes that her potential life expectancy is lower and would hence be more likely to smoke.

In the above model, the agent can only choose to consume one type of risky good. The theoretical model could be extended in a straightforward way to incorporate more choices, say tobacco and alcohol, both affecting mortality through a specific stock. Through the effect on mortality both goods would tend to be complements even if the instantaneous utility function displays no complementarity. We discuss in detail later

the effect of ignoring other risky choices on the estimation and how we control for this.

3.7 Selection and Identification

It is commonly observed that smokers die on average younger than non smokers. There are three possible sources (non exclusive) to this observed correlation: one is that tobacco directly causes death and some individuals randomly smoke, for instance because they have a higher exogenous taste for tobacco. The second is that both death and smoking are caused by some underlying factor (such as occupation for instance). Finally it is possible that because tobacco causes death, individuals with a longer life expectancy net of smoking select out of smoking. Whilst we have shown within a theoretical framework the mechanisms at play under the latter hypothesis, there remains the question of whether this can be identified on data on smoking through the life cycle. What assumptions do we need to make to be able, on the basis of the data we can observe, to disentangle between the selection and the causal effect? In other words, can we test for the selection hypothesis?

To fix ideas, we simplify the problem and consider a linear model. Denote T_i the age at death of individual i and q_i the quantity of cigarettes. We assume a relationship such as:

$$T_i = \alpha_0 - \alpha_1 q_i(\varepsilon_i) - \varepsilon_i + u_i \tag{4}$$

where ε_i is, as before, an individual characteristic known to the agent which affects life expectancy, and which is potentially correlated with q_i. u_i is a random shock, uncorrelated with smoking and unobserved by the agent. Regressing age at death on quantities smoked does not identify the effect of smoking on mortality because of the selection. If individuals were dying repeatedly, one could "first differentiate" the relationship to get rid of ε_i, and consistently estimate α_1, provided that there were some variations in q_i over different lives. As this is clearly infeasible, one has to impose more structure on the problem.

To solve the problem we can augment the first relationship with a model which links smoking to the unobserved heterogeneity ε_i. The optimal model of behavior described in (2) would produce such a relationship. To simplify, suppose that smoking can be expressed as:

$$q_i = \beta + \varepsilon_i$$

In this formulation, the only heterogeneity in smoking comes from heterogeneity in potential life expectancy ε_i. In this case, we can invert this relationship to obtain a measure for ε_i. We can then turn to the mortality equation to separate the effect of tobacco from selection. Hence, under the assumption that there is only one source of heterogeneity, we can identify the true effect of tobacco. However, there is no way to test for the selection, as this is the identifying restriction.

If there is an additional source of heterogeneity, as taste differences, we could write this relationship as $q_i = \beta + \varepsilon_i + \nu_i$, where ν_i is a taste shock. In that case, observing a given cigarette consumption is not very informative about ε_i. Agents smoking similar quantities could have different ε_i if they have different tastes. In this more general case, the "structural" model is useless to achieve identification, unless one assume that the variance and covariance of ε_i and v_i are known, which is rather unappealing.

Another route altogether is to use more data related to mortality, such as the cause of death, or repeated information on health and impose some structure on the evolution of health. [10] Death is usually preceded by poor health. Individuals with a high ε_i should also be observed in poorer shape at a given age. Hence repeated information on health can help to identify the characteristic ε_i. Obviously, one has to disentangle the effect of tobacco from selection on health. This is easier than in (4) as one can obtain repeated information on health for a given individual.

Observing the cause of death is useful for testing for the selection effect. The medical literature provides classification of deaths into tobacco or non tobacco related death. Using this information, one can empirically evaluate whether smokers are more prone to die of a non tobacco related death than non smokers.

Our approach is to use both repeated information on the health of the individual and data on the cause of death to identify the survival probabilities independently from parameters describing preferences. Because the survival probabilities can be identified separately from the complete structural model, we do not have to impose a particular relationship between q and ε at that stage. The identification of ε and $\pi(S^M, age, \varepsilon)$ do not rely on a specific form for the utility function, nor on the assumption of inter-temporal optimisation by forward looking rational agents. This also reduces significantly the computational burden by breaking up the estimation into two

[10] A related identification scheme can be found in Van den Berg et al. (2001).

parts.

In the next section, we estimate a one factor model of duration to death with competing risks, jointly with a model of health evolution.

4 Identifying Selection based on Potential Life Expectancy

In order to demonstrate selection effects, we need to identify potential life expectancies, net of the causal effect of tobacco. As we have argued in the previous section, this can be done by estimating a joint model of mortality and morbidity.

The epidemiology literature provides estimates of life expectancies and the effect of smoking. However, these estimates are usually computed by sex, education level or occupation. Using these results would limit the scope of our analysis as predicted life expectancy would be collinear with these observed characteristics. We would not be able to distinguish the effect of life expectancy per se, from socio-demographic factors influencing smoking. Moreover, the results concerning smoking usually distinguish between smokers and non smokers only and not particular quantities. To solve our model, we need information on how each cigarettes at a given age affects mortality.

In this section, we identify and estimate the survival probabilities, conditional on age, quantities smoked and unobserved heterogeneity. The probabilities are used to construct potential life expectancies.

4.1 Estimation Method and Identification

4.1.1 General Overview

Let T_i be a random variable indicating the age at death of individual i. Denote by T_{0i} and T_{1i} the age at death from a non tobacco related cause and from a tobacco related cause respectively. We model age at death and the cause of death, I_i, in a competing

risk framework:

$$\begin{cases} T_i = \min\left[T_{0i}(\varepsilon_i, u_{0i}), T_{1i}(S_i^M(\varepsilon_i), u_{1i})\right] \\ \\ I_i = 0 \text{ if } T_{0i}(\varepsilon_i, u_{0i}) < T_{1i}(S_i^M(\varepsilon_i), u_{1i}) \\ \\ I_i = 1 \text{ if } T_{0i}(\varepsilon_i, u_{0i}) \geq T_{1i}(S_i^M(\varepsilon_i), u_{1i}) \end{cases} \tag{5}$$

Time to death for a non smoker depends on two unobserved components ε_i and u_{0i}. The former, although unobserved by the econometrician, has a distribution which is known by the agent, as discussed in section 3. The latter is truly unobserved. It captures the randomness in duration to death for a non smoker. Time to a tobacco related death depends on the stock of cigarettes affecting mortality $S_i^M(\varepsilon_i)$, itself a function of ε_i through the choice of smoking. Time to a tobacco related death also depends on a random shock u_{1i}, supposed to be independent from u_{0i}. The stock of cigarettes affecting mortality, $S_i^M(\varepsilon_i)$, may also depend on other forms of heterogeneity such that differences in preferences or in prices (hence the i subscript). Individuals are supposed to differ with respect to their life horizon (different ε_i), even abstracting from the effect of smoking. We classify the nature of the death using the information on the cause of death provided in the data set and epidemiological results.

In general, a model of competing risk is not identified (Cox (1962), Tsiatis (1975)). For any joint distribution function of (T_{0i}, T_{1i}) there exists a joint distribution with independent variables which produces the same results. Heckman and Honoré (1989) show that the model is identified under certain conditions, when introducing regressors into the competing risk model. We propose to identify the model by considering a one factor model of both mortality, cause of death and health. To this end, we augment (5) with a model of health evolution:

$$M_{it} = f(\varepsilon_i, t, S_{it}^M(\varepsilon_i)) \tag{6}$$

where M_{it} is individual's i health status in period t. Under appropriate assumptions on $f(.)$, we are able to identify ε_i from variations in the individual's health through time. The idea is that individuals who are likely to die prematurely are also likely to be observed in poorer health while alive. As in Heckman and Singer (1984), Eckstein and Wolpin (1989) or Eckstein and Wolpin (1998), we introduce heterogeneity by

considering different types of agents. Each type of agent has a specific distribution for (T_{0i}, T_{1i}).

4.1.2 Mortality

We decompose the conditional survival probability $\pi(age, S^M, \varepsilon)$ into the product of two components: [11]

$$\pi(age, S^M, \varepsilon) = \pi_0(age, \varepsilon)\ \pi_1(age, S^M) \quad \text{with} \quad \pi_1(age, 0) = 1 \tag{7}$$

hence, a non-smoker has a conditional survival probability of $\pi_0(age, \varepsilon)$. We assume that $\pi_0(age, \varepsilon)$ and $\pi_1(age, S^M)$ are decreasing in age and bounded between 0 and 1, and that $\pi_1(age, S^M)$ is decreasing in S^M. Let J be the number of types existing in the population. Each type j has a specific survival probability $\pi_0^j(age)$. Let $\lambda^j,\ j \in \{1, \ldots, J\}$ be the probability associated with each type. We estimate these probabilities jointly with the other parameters of the model. Note that with this method we make no prior assumption on the distribution of the heterogeneity, nor do we directly specify life expectancy by sex or education levels.

Our results in this section depends on the possibility of classifying diseases into tobacco or non tobacco related deaths. This is where the epidemiological results are important. We rely here on the results of Peto et al. (1992) which separate the effect of tobacco from other sources. [12] Some deaths are almost entirely due to smoking (such as lung cancers) but some are only partially caused by smoking (such as cardio-vascular diseases). Peto et al. (1992) provide estimates of the likelihood that a particular death is caused by tobacco. For instance, 94% of lung cancers before age 70 are caused by tobacco, and so are 81% of chronic obstructive pulmonary diseases. We use our model to match up with these estimates. Given the indirect computation method used by

[11] This is a standard two destination duration model where the hazard of dying can be expressed as the hazard of dying from a non tobacco related death plus the hazard of dying from a tobacco related death. We express the model in terms of survival rather than hazards to be consistent with the specification of our behavioral model in section 3.

[12] This study has an advantage over more conventional studies as it uses an indirect method to assess the effect of tobacco on health based on lung cancers which are almost exclusively tobacco related. More traditional approaches consist in regressing age at death on quantities smoked, but are subject to endogeneity biases.

the authors, these numbers represent the effect of tobacco on mortality and are free of confounding effects. [13]

We use the information in our data set to compute the stock for each smoker using information on duration and quantities. We assume that the stock affecting mortality does not depreciate. We display the likelihood for the duration model as well as the particular functional forms in appendix C. We provide in section 4.2.3 a comparison of our estimated results with previous results in the literature, to assess the validity of our estimation strategy.

Using data on mortality alone causes a problem when we consider a non parametric unobserved heterogeneity as in Heckman and Singer (1984). Suppose we consider a healthy individual with an overall long life expectancy at birth, but who happens to die early, say by an unfortunate infectious disease. The estimation would tend to consider this individual as belonging to a group of individuals with a short life expectancy, as the contribution to the likelihood in a long life expectancy group would be very small. The estimation results would tend to group individuals who die early together, while those more likely to belong to a long life expectancy group would appear to face almost no risk of dying at an early age. It is unlikely that the individual in our example knew from birth that life would be so short. When planning consumption over the life cycle, this individual would probably have expected a longer life expectancy with a small but non zero probability of dying early, as accidents can always happen.

To solve this problem, we have to incorporate more information. One way is to characterize mortality outcomes as accidental or not. While car or boat accidents are easily distinguishable from other causes of death, it is much more difficult and sometime impossible to classify any other cause of death as accidental or not, for a given individual. A second way is to incorporate panel data on morbidity over an extended time before death and estimate jointly a model of duration to death and of health evolution. The idea is that if the individual in our example appeared very fit five or ten years prior to death, his or her expected life expectancy is probably much longer than an individual dying at the same age, but diagnosed, for example, with hypertension ten years earlier and heart problems five years prior to death.

[13] Hence, the function π_1 should capture the true effect of tobacco. If the smoker is also more prone to drink, this should be captured by the heterogeneity in π_0 as discussed in section 4.1.4 below.

Incorporating data on health evolution is also useful as we only observe age at death for 30% of the surveyed individuals. For older individuals, survival does provide information on the type of life expectancy. The problem arises for the identification of the type of young individuals as it is difficult to classify them into potential mortality groups, based on this information alone. We now turn to the specification of our model for the evolution of health (6).

4.1.3 Modelling Morbidity

In total, we have more than 35 variables describing the individual's health. (Table B in the appendix gives a list of these variables.) Instead of looking at each variable individually, we try to summarize the information into one index. We regress a dummy equal to one if the individual dies within 5 years of the interview on all morbidity variables, using a probit regression and we use the predicted index as a measure of health. In doing so, we get a morbidity index which is a weighted sum of health outcomes and which predicts mortality optimally. The weights are larger for more serious conditions. In our data, malignant neoplasm, heart problems, diabetes and poor self assessed health are very significant predictors of death and have the most important weights. [14]

This ill health index is increasing with age. Its variance is also increasing with age (see Figure 6 in the appendix). However, there is considerable heterogeneity even at young ages. From the panel dimension, health appears to be very persistent through time. Individuals in poorer health in one period are very likely to be in poor health eight years later. [15]

Let M_{tj} be the morbidity index for an individual of age t with type j. We conjecture an evolution of this index as:

$$M_{tj} = \mu_j + \delta_{01} t + \delta_{02} t^2 + \delta_1 S_{tj}^M + \delta_2 S_{tj}^{M2} + \eta_{tj} + u_{tj} \quad \text{with} \quad \eta_{tj} = \eta_{t-1,j} + v_{tj} \qquad (8)$$

Health evolves as a random walk with a drift and depends on two shocks, one permanent, v_{tj}, and one transitory, u_{tj}. The permanent shock allows for an increase of health inequalities with age. The transitory shock takes into account possible fluctuations in health which are not persistent (a flu, for instance).

[14] The morbidity index is linked to the probability of death through the probit structure.

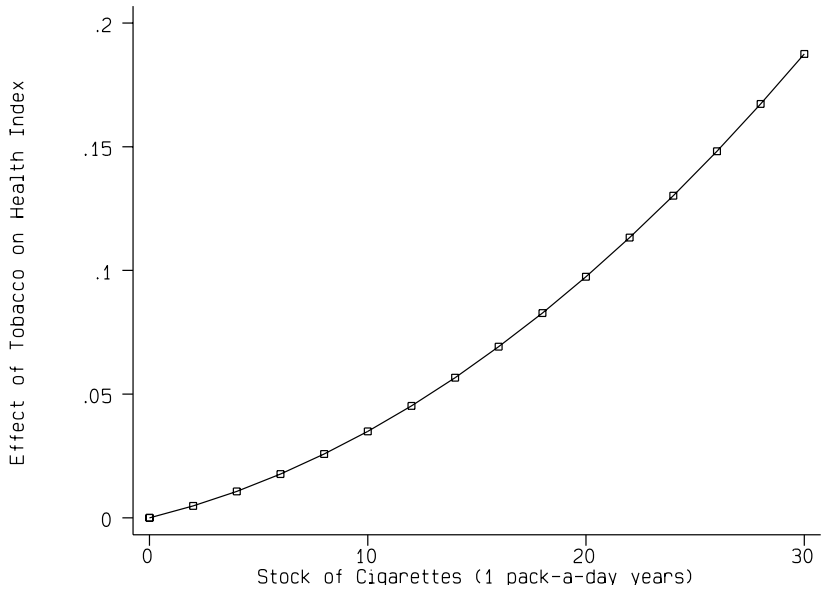
[15] In fact, health appears to be a random walk at the individual level.

The morbidity index depends on an initial condition, μ_j, specific to the individual. We allow for heterogeneity in initial conditions, as from the data, there is a large heterogeneity even at young ages. These differences in initial conditions will help identify those who are in systematic better or poorer health, indicating different life expectancy. The morbidity equation (8) is linked with the competing risk model by imposing that all individuals of type j have the same ε_j and μ_j. In other words, we assume that $\varepsilon_j = g(\mu_j)$, where $g(.)$ is a function to be estimated.

Health depends non linearly on the stock of cigarettes smoked up to that point. The quadratic function allows cigarettes to have an effect on health with some delays. [16]

As we have argued that the decision to smoke depends on life expectancy, there is a correlation between the fixed effect μ_j and the accumulated stock S_{tj}, which potentially biases the estimation of δ_1 and δ_2 in the level equation. Given the structure of the model, these parameters can be estimated from the changes in health in a first step. The estimation of the unobserved fixed effect is done in a second step.

Figure 1: Effect of Tobacco on Morbidity



[16] An alternative method would have been to compute a health index with only health problems not related to tobacco, leaving out for instance all lung cancers or chronic obstructive diseases. While some health outcomes are straightforwardly related to tobacco, it would be less clear what to do with, for instance, self-assessed health, body mass index or hospital admissions, and indeed with most of the health variables.

Figure 1 displays the effect of tobacco (measured as a cumulated stock of cigarettes over time) on the ill health index. Note that the effect of the stock is non linear. This is in line with epidemiological results such as Doll et al. (1994). The regression was also instrumented by lagged values to control for attenuation biases due to measurement errors. [17]

Appendix C provides details of the computation of the likelihood for the joint model of mortality and health.

4.1.4 Multiple Risky Choices

In (7), we have only modelled the effect of tobacco on health through π_1. Suppose the agent can also consume alcohol, which increases the overall mortality risk. We can then decompose the survival probability into $\pi_t = \pi_{0t}\pi_{1t}\pi_{2t}$, where π_{2t} measures the effect of alcohol. Given data on causes of death, epidemiological results linking alcohol to specific causes of death and data on alcohol consumption over the life cycle, we could identify the survival probability net of tobacco and alcohol as well as the contribution of the two goods to mortality. Unfortunately, such precise data is not available in our data set. By ignoring the effect of alcohol, we estimate in fact $\tilde{\pi}_{0t} = \pi_{0t}\pi_{2t}$, i.e. the survival probability net of tobacco, but not of alcohol. Given that we have allowed for unobserved heterogeneity in π_0, this is not a problem so far. Further on, we have to be cautious in interpreting the correlation between the potential life expectancy we are estimating and smoking. We are estimating the life expectancy net of tobacco, but not necessarily the potential life expectancy net of all risky choices that individuals make. We deal with this problem later on by bringing in additional information on other risky behaviors we have in the data set.

[17] Obviously, the estimates of potential life expectancy will be sensitive to a bias in the estimated effect of tobacco on the morbidity index. For sensitivity analysis, we computed an upper bound on the effect of tobacco by estimating equation (8) in levels. If individuals in poorer health are more prone to smoke, a regression in levels would overstate the effect of tobacco. For a level of stock below ten years at a pack-a-day, whether the effect of tobacco on health is estimated from a regression in levels or in first difference does not make much of a difference. Approximately 50% of the smokers have a stock lower than this level. For young smokers, quitters or light smokers, we expect to have a good measure of the effect of tobacco. If we want to relate smoking to health types, most of the information will come from young or middle aged individuals as they have the highest smoking prevalence.

4.2 Estimation Results for the Survival Probabilities

4.2.1 Potential Life Expectancy

We selected a number of types equal to three. The likelihood only slightly improved when we increased the number of types to four. [18] The estimated parameters can be found in Table C in the appendix.

Each type of agent is characterized by a distribution of age at death, had the individual not smoked. In particular, each type of agent has a different potential life expectancy at birth, which is calculated as:

$$PLE(\varepsilon) = \sum_{s=1}^{\infty} s \left(\prod_{l=1}^{s} \pi(l, 0, \varepsilon) \right) \ (1 - \pi(l+1, 0, \varepsilon))$$

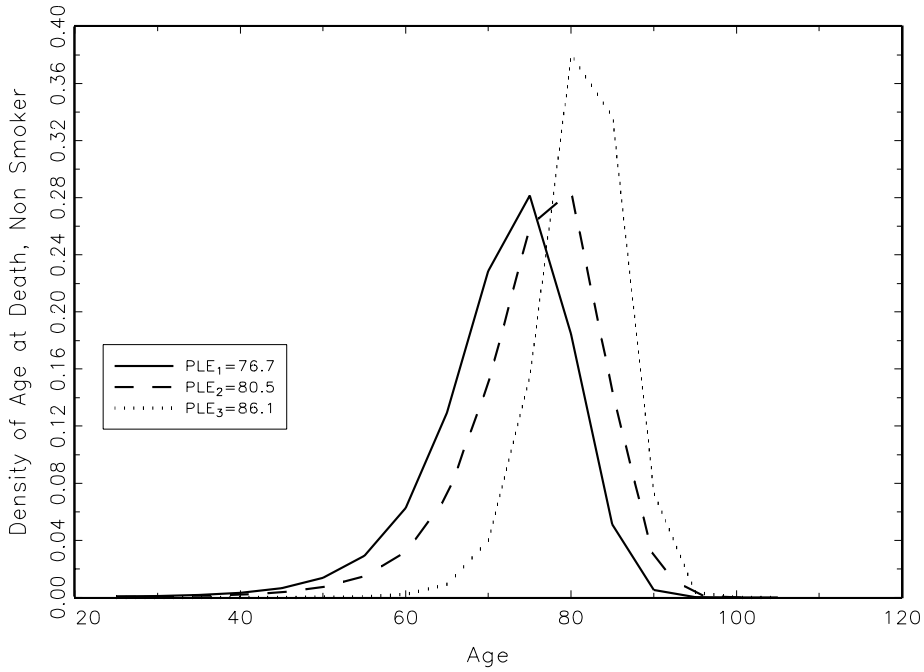
which is the life expectancy if the individual never consumes any cigarettes. These potential life expectancies are equal to 76.7 (Type 1) for the least healthy group, 80.5 for the middle group (Type 2), and 86.1 for the healthiest group (Type 3). The average *observed* life expectancy varies from 75.8 to 84.1. Net of tobacco, the first group has a probability of about 0.5 of surviving more than 75 years. Type 2 individuals have a probability of about 0.7, while for Type 3 this probability is 0.95. However, the probability of surviving beyond age 90 is respectively 0, 0.03 and 0.07.

Figure 2 displays the estimated densities of age at death for three types of individuals. Note also that the distribution for the less fit is more skewed to the left. Those individuals have not only a lower life expectancy, but also have a disproportional chance of dying at a young age.

Table D in the appendix gives summary statistics for each type, by sex, age, education and occupation. Men, low educated individuals, ever smoker as well as self-employed and retired are more represented in the low life expectancy group (Type 1). For instance, 50% of high educated individuals belong to Type 3, whereas only 31% of the low educated do. In Type 1, 72% of individuals are men and 68% have a low education. Within Type 3, the share of men is only 9% and 33% of individuals have a low education.These results are hardly surprising. Several explanations can be thought of. First, genetic differences could partly explain the differences between men and women. Differences across sex, education and occupation might also stem from

[18] The log-likelihood went from -4259 to -4123.

Figure 2: Density of Age at Death of Non Smoker, by Type



occupational stress (Marmot et al. (1998)), job accidents or behavior other than smoking. The distribution of smokers and non smokers across types indicates that there is a self-selection into smoking as individuals with low life expectancy, net of tobacco are more likely to have smoked at some time.

4.2.2 Effect of Tobacco on Life Expectancy

The effect of tobacco on life expectancy can be seen in Table 3. The first column displays the observed life expectancy, which varies from 75.8 to 84.1. The second column displays the life expectancy for non smokers in each category. One can compare the first and second column to evaluate the effect of tobacco on health. This could be misleading as different categories actually smoke different quantities. Column 3 displays the life expectancy if everybody smoked one pack a day over their entire life. This is computed from our estimated health and mortality model. Column 4 displays the loss of life. The loss of life is bigger for individuals belonging to longer life expectancy groups. It is more than twice as costly to smoke, in terms of expected years of life, for individuals belonging to a long versus a short life expectancy group. This might provide an incentive for individuals with long life expectancy to refrain from smoking or to smoke less.

Table 3: Effect of Tobacco on Life Expectancy

	Life Expectancy			Loss of
Type	Observed	Potential	1 pack-a-Day	Life
	(1)	(2)	(3)	(3) - (2)
Type 1	75.8	76.7	74.0	2.7
Type 2	79.2	80.5	76.6	3.9
Type 3	84.1	86.1	80.1	6.0

Note: Column 3: all individuals are set to smoke 1 pack a day over entire life. Column 4 measure the average number of years of life lost when all types smoke 1 pack a day.

4.2.3 Comparison with Epidemiological Studies

Epidemiological studies do not break up life expectancy by potential life expectancy types. However, given our results, we can compute the average life expectancy for

men and women and compare them with published results. From our estimation, the average life expectancy is 79.1 for men and 83.4 for women. According to Statistic Sweden, in 1999, the life expectancy for males is 77.0 at birth, 79.1 at age 50 and 81.4 at age 65. For women, it is 81.9 at birth, 83.2 at age 50 and 84.9 at age 65. Given that we are computing the life expectancy for a population that is on average 50 years old in our data set, the numbers we get are comparable to the one published elsewhere. [19]

We now turn to the effect of tobacco on health. Again, the results in the literature are not broken down by mortality type. Doll et al. (1994) and Shaw et al. (2000) analyze mortality in relation to smoking for a cohort of British male doctors surveyed from 1951 onwards. This study is interesting as it focuses on a relatively homogenous group, and one might think that selection is less likely to be important than in other epidemiological studies. However, it is not clear how to compare these British doctors with the Swedish life expectancy groups. On one hand, these doctors are highly educated, and presumably have a long potential life expectancy. However, being male and being born in the nineteen twenties at the latest, their overall life expectancy should be much less than the average high educated Swedish individual in our data set. It is thus not obvious to which of the three types we should compare them. We conjecture that they are most comparable to the Type 2 group.

Shaw et al. (2000) calculate that for this population, each cigarette reduces life by (very roughly) 11 minutes. From our data, each cigarettes reduces life expectancy by 13.7 minutes on average. For Type 2 individuals, the loss is about 12 minutes. Here again we find that our results are in line with known epidemiological results. This is an indication that our identification strategy discussed in section 4 appears to be valid.

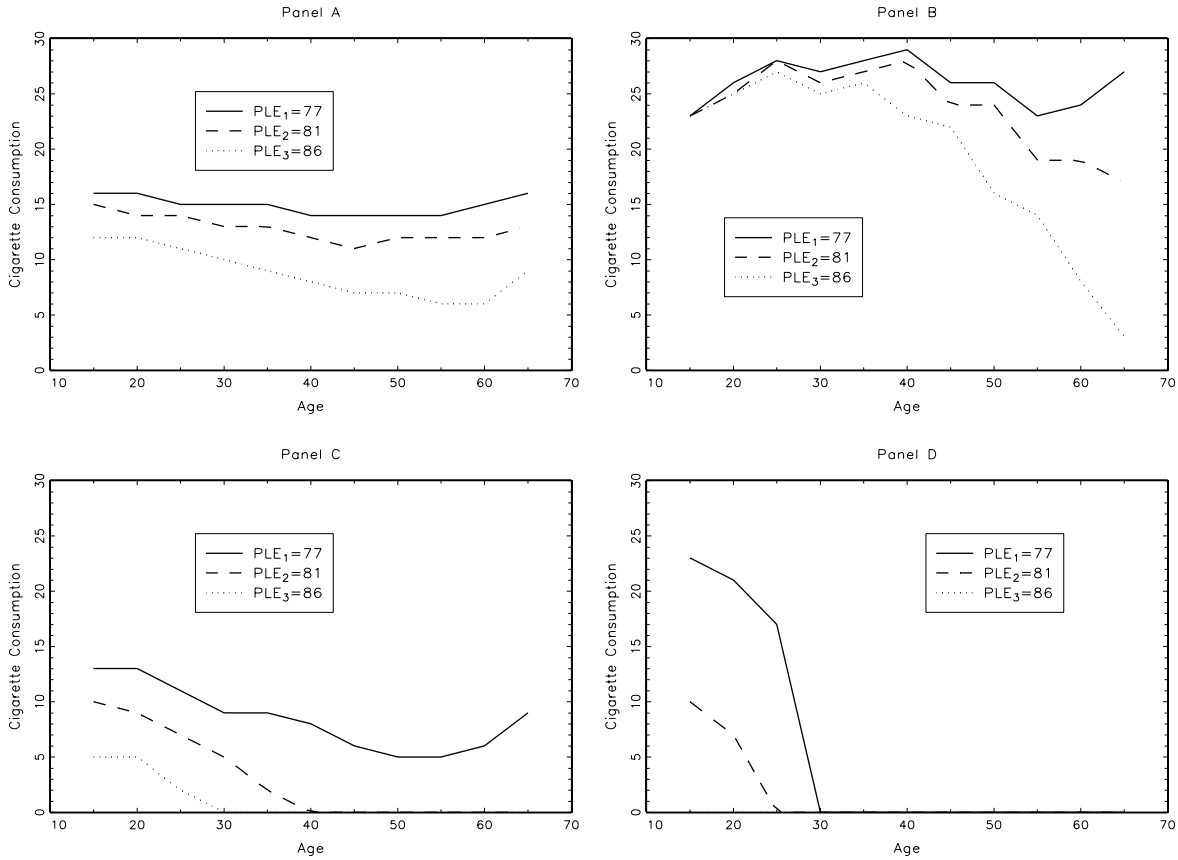
4.3 Simulation of Life Cycle Profiles of Smoking

Given the estimated survival probabilities, conditional on age and quantities smoked, we are now able to investigate the predictions of our behavioral model of smoking. Here, we vary the parameters of the utility function to generate different life cycle profiles. In this computation, we keep income and prices constant over the life cycle, as not to generate any trivial smoking profiles only based on income or price variability.

[19] Broken down by education level, our results predict a life expectancy of 78.7 for low educated males, 79.2 for middle education and 79.7 for high educated males.

We select different parameters for the preferences and solve for the optimal life cycle profile using (2), for each life expectancy type. Figure 3 displays four different graphs, computed using four different set of parameters describing preferences. Within each graph, we display the life cycle profile of three types of agents with identical preferences and pure rate of time preferences, but with different potential life expectancies. [20]

Figure 3: Example of Life Cycle Profiles of Smoking



In panel A, consumption is almost flat for all agents. Although they have the same preferences, individuals with a longer potential life expectancy smoke on average 6 cigarettes a day less than those with the shortest life expectancy. In panel B, the consumption profiles are more hump-shaped, with a peak in smoking around age 40. As the discount rate is set at a rather low 0.9 per year, young individuals smoke a similar number of cigarettes, regardless of their life expectancy. As they age, those with the

[20] The exact parameterization is the following: ν_1 =-2.5,-0.14,-0.5,-1; ν_2=-0.01; ν_3=0,-0.06,9,-0.01;ν_4=-40*ν_1;ν_5=-60*ν_2;ν_6/1e3=30,16,20,51; β=0.98, 0.90, 0.97, 0.95

longest life expectancies have a much more pronounced decrease in their consumption of cigarettes.

In panel C, the parameterization induces two of the three types of agents to quit. Those with an expected life time of 86.1 quit at age 30, while those with a life expectation of 80.5 quit at age 40. Those with the shortest life expectancy never quit. Here quitting, when it occurs, takes place after a gradual decline in smoking. The model is also able to generate abrupt quitting. We display such profiles in panel D. Individuals with the shortest life expectancy smoke about a pack a-day up to age 25 and quit at age 30. Similarly, individuals with a potential life expectancy of 80.5 smoke around half a pack a day at age 15 and 20 and quit at age 25. The model is therefore able to generate "cold turkey" behavior as in Becker and Murphy (1988). Note however, that here quitting is endogenous to the model. Individuals with the longest life expectancy choose never to smoke given their taste for tobacco. Had tobacco not been dangerous to life, the model predicts that all individuals would have chosen to smoke three packs a day.

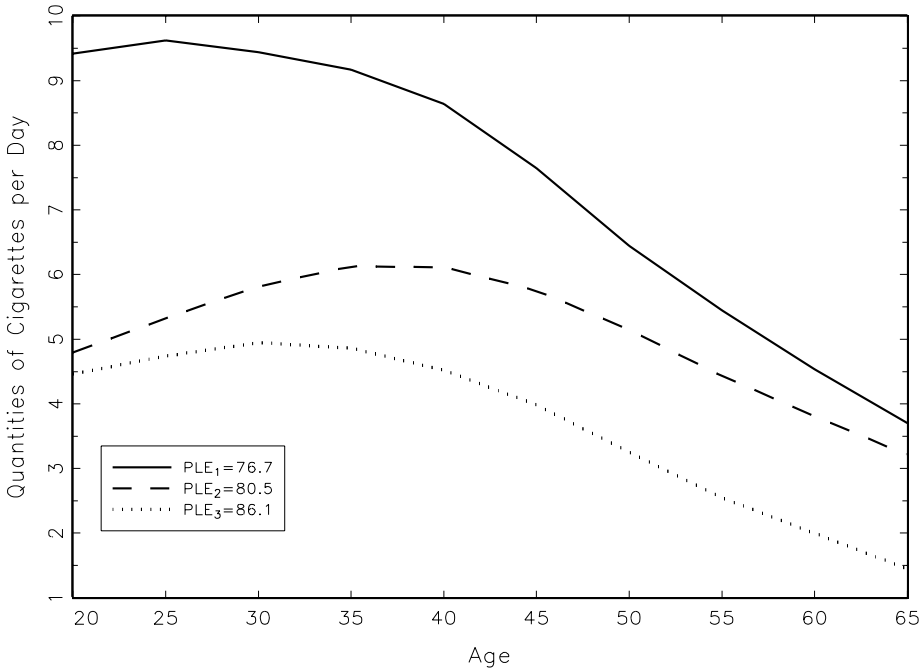
Given the estimated survival probabilities, the model is able to generate a number of realistic smoking profiles. An additional implication of the model is that individuals with longer potential life expectancies are smoking fewer quantities and quitting earlier. This is a testable implication, which we evaluate in next section.

5 Does Heterogeneity in Life Expectancy Explain Differences in Smoking Behavior?

We now analyse the smoking behavior in the data by different life expectancy groups, net of tobacco. We compute the average quantity of cigarettes smoked, by type, at a given age. Figure 4 displays the result. The profiles were obtained by a non-parametric regression of quantities on age, for each type. Individuals coming from short life expectancy groups (net of tobacco) smoke more than individuals with a long potential life expectancy. This is particularly true between age 30 and 50. This correlation between smoking and potential life expectancy has been documented by Adda and Lechene (2001), using other identification schemes. This pattern of smoking is consistent with a trade-off between smoking and longevity. Those who face the highest cost of smoking

are those who effectively smoke less. However, smoking is often seen as determined predominantly by social norms (within gender, education or occupation groups), which are also correlated with life expectancy. It might be the case that the correlation between smoking and life expectancy net of tobacco is only due to the fact that we have not conditioned on any variables characterizing social class. To check this, we regress smoking behavior on sex, education levels, occupation and regional dummies, together with potential life expectancy dummies. The regression is performed on the pooled cross-sections for 1980-81, 1988-89 and 1996-97. The results are displayed in Table 4.

Figure 4: Average Quantity of Cigarettes by Health Types



Even when we condition on variables linked with social norms, the effect of the life expectancy net of tobacco is significant. For instance, individuals belonging to the shortest potential life expectancy group smoke on average 2.3 cigarettes more than those belonging to the longest group. Note that the difference in smoking behavior between life expectancy group is as large as the difference within education group or between blue and white collar occupation.

In section 4.1.4 we analyzed the consequences of ignoring other risky behaviors on our estimation results. We return to this issue here. Part of the results in Table 4 could be due to the fact that the life expectancy net of tobacco we are estimating contains the

results of other risky choices which are in turn correlated with tobacco consumption. For instance drinking and smoking are usually correlated, and provided that alcohol has a significant effect on health, we would observe a correlation between smoking and the health type of the agent. This would arise because drinking affects health (our estimate of π_0 would be lower), but not because the potential life expectancy is shorter.

To capture these effects, we use additional information in our data set on other risky behaviors. In 1988-89 and 1996-97, information on weekly alcohol consumption was reported. We construct three dummies for the amount of alcohol consumed, low (less than 0.1 liter of pure alcohol per week), moderate (0.1 to 0.35 liters) and high (in excess of 0.35 liter). The data set also reports information on whether the job of the individual is risky in 1980-81 and in 1988-89. In all periods, information on whether the individual is using moist oral snuff is provided. Finally, we also condition on a number of characteristics which might capture the attitude towards risks such as marital status, number of children, taxable wealth and household income. The results are also reported in Table 4. Even when conditioning on a number of variables pertaining to risky behavior, the effect of the life expectancy is still significant. Not surprisingly, smoking is correlated with drinking and marginally to job risk. Smoking is however negatively related to moist snuff, which is often used as a replacement for cigarettes in Sweden. [21]

Interestingly, when we perform the regression on young individuals only, the effect of life expectancy type is reduced. From our model, there are two reasons why the selection based on potential life expectancy would be small for young individuals. First, from a teenager's perspective, even with a standard discount rate, the future increases in the mortality risk would be small. Second, the effect of cigarettes on health is non linear, so the health costs of experimenting with cigarettes is low.

Potential life expectancy types contributes approximately 6.8% of the overall explained variance in the data when we consider current smokers. In comparison, education explains 1.3%, occupation 3% and sex 15.6%. From this we see that many factors influence smoking intensity, with an important role for socio-demographic vari-

[21] We investigated further with other variables present only in the last wave, such as father's or mother's education or whether the individual has not taken up an insurance policy. The results did not change when including these additional variables.

Table 4: Determinants of Smoking Behavior and Quitting

	Quantities			Quitting		
	Current Smokers			Current and Former Smokers		
	Quantities of Cigarettes (Std. Err.)			Hazard Ratios (Std. Err.)		
	(1)	(2)	(3)	(4)	(5)	(6)
Type 1, PLE=76.7	4.39 (0.43)	2.29 (0.47)	3.00 (0.68)	0.65 (0.04)	0.70 (0.05)	0.76 (0.05)
Type 2, PLE=80.5	2.31 (0.20)	0.40 (0.25)	1.81 (0.30)	0.91 (0.03)	0.90 (0.04)	0.89 (0.04)
Relative price	-2.17 (0.89)	-1.89 (0.90)	-0.38 (0.90)			
Male		2.43 (0.27)	0.67 (0.32)		1.11 (0.04)	0.94 (0.04)
Low Education		0.81 (0.36)	0.37 (0.46)		0.35 (0.02)	0.47 (0.3)
Middle Education		0.56 (0.33)	0.32 (0.39)		0.78 (0.04)	0.78 (0.04)
Blue collar		0.81 (0.37)	2.1 (1.4)		1.15 (0.05)	1.22 (0.06)
White collar		0.71 (0.45)	1.37 (1.43)		1.29 (0.07)	1.39 (0.08)
Self-employed		1.96 (0.54)	2.92 (1.55)		1.32 (0.10)	1.21 (0.09)
Alcohol Low			-2.8 (1.11)			0.87 (0.08)
Alcohol Moderate			-2.7 (1.13)			1.18 (0.12)
Risky Job			0.40 (0.54)			0 (-)
Moist Snuff			-2.37 (0.60)			2.58 (0.13)
Number of obs.	6016	6016	3453	12239	12239	8158

Note: For quantities, regressions performed on all individuals aged 25 to 65, excluding students, farmers and retired individuals. Regressions also included a polynomial in age and year and regional dummies. For quits, a proportional hazard model was fitted to the data. No standard errors were reported for individuals with a risky occupation as none of them quitted.
Columns 3 and 6 also control for income, wealth, household size, number of children and marital status. Robust standard errors are displayed.

ables as the literature on smoking has emphasized. However, considerations about life expectancy do play an important role also.

Finally, we investigate the effect of potential life expectancy on quitting. We estimate a proportional hazard model for quitting. We also control for education, sex and occupation, as well as year effects. The results are displayed in rows 4 to 6 in Table 4.

Here again we find that the effect of potential life expectancy is significant and of the same magnitude as the effect of education, sex or occupation. An individual belonging to the shortest life expectancy group is 0.76 times less likely to quit at any duration compared to an individual with a long life expectancy.

The arguments presented here demonstrate that there is a negative *correlation* between potential life expectancy and smoking. To demonstrate causality, one would need to measure the response of smoking of a given individual to unexpected changes in her potential life expectancy. [22] There are several reasons why this is not likely to be an easy task. First, any negative health shock caused by tobacco, such as cancers or cardiovascular diseases would not qualify, as these shocks could be anticipated. Any family diseases would not qualify either, as the individual would take this into account when forecasting his or her life expectancy. Also, information on family diseases are usually not reported in data sets. Second, when individuals face serious illnesses it is likely that they also experience a change in preferences that our model cannot describe. However, most health problems would probably not be serious enough to affect the decision of smoking. Especially, a number of individuals stop smoking in their thirties or forties, before any serious health problems develop. This is consistent with our model, but would make it difficult to investigate empirically the effect of health outcomes on smoking. For instance, Jones (1994) or Clark and Etilé (2001) finds mixed results for the effect of health on quitting.

Alternatively, one could rely on positive shocks to health which extend potential life expectancy. The discovery of new drugs might constitute such positive shocks. One could then find individuals with a specific disease or at risk of this disease and see how it affects smoking behavior. Positive health shocks do happen when comparing different birth cohorts. Younger cohorts have longer life expectancy than older ones.

[22] Adda (2000) uses the "Mad Cow" crisis as a natural experiment to study changes in behavior towards risks.

Interestingly, young cohorts smoke less than older ones, even when accounting for prices.

6 Matching Observed and Predicted Life Cycle Profiles

In the previous sections, we have found that a model with a trade-off between smoking and longevity is able to deliver realistic smoking profiles. We have also tested an empirical prediction of this model. Can our model really match up with the data?

We take here an extreme approach and assume that there is *no* heterogeneity in preferences or in the pure rate of time preference across individuals. The only heterogeneity we allow is observable heterogeneity in life expectancy. How far can such a simple model fare with the data? We use a method of moments approach to estimate the parameters of the utility function and the pure rate of time preference. We match the observed life cycle profiles of smoking in 1980-81, by life expectancy groups. We compute the average number of cigarettes smoked by individuals at ages between 20 and 65. In total, we have 30 moments and 8 parameters.

Figure 5: Match of the Model

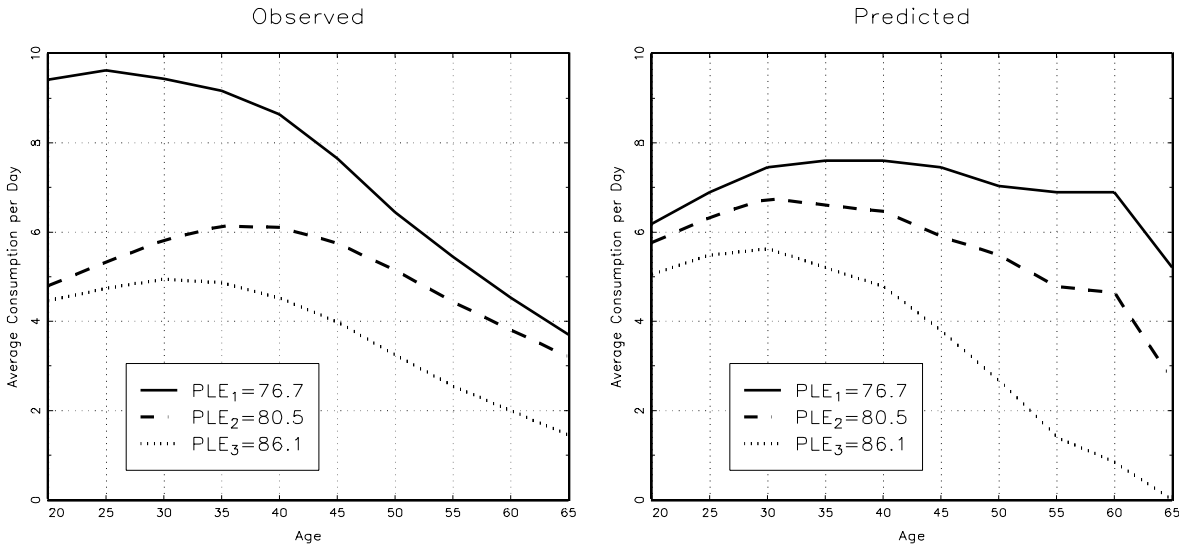


Figure 5 displays the observed and predicted profiles. Although the model is so

simple, it is able to match the data in two important dimensions. First, the shape of the predicted consumption profiles are close to the observed one. Overall, the correlation between the observed and the predicted consumption is 0.82. Second, the model predicts, at least qualitatively, the difference in smoking intensities between groups. The model has some problems to predict a high enough smoking profile for the lowest life expectancy group. However, this is a small share of the population and this profile has also the highest variance. In our estimation, the observed smoking profiles are conditional on type, but unconditional on socio-demographic variables. If we condition on sex and education, the life cycle profile of smoking is much lower and more consistent with our estimates. The importance of socio-demographic variables indicate that smoking is also influenced by other factors than potential life expectancy, as shown in the previous section. For instance, social norms or peer pressure would magnify smoking behaviors.

However, over all types, the R^2 is equal to 0.7. A simple model of rational choice and a trade-off between smoking and longevity, with only heterogeneity in life expectancy, is able to explain a substantial part of the *between* group variance. In addition, the estimated model gives plausible life-cycle profiles of smoking. Hence, to model smoking and the selection in or out of tobacco consumption, we do not have to rely on complex interaction between agent through peer effects nor do we have to appeal to time inconsistent preferences. Observable differences in life expectancy appear to explain the between group behavior quite well.

7 International Comparisons

The study has been carried out for Sweden mainly for the availability of an extensive data set on behavior, health and mortality. Are the results presented here specific to this country or can they be used to understand smoking behavior in other countries? The goal of this section is not to replicate the methodology on other countries (assuming adequate data were available), but to informally present evidence which would be in line with the model. For instance, is there a correlation between potential life expectancy and smoking in other countries as well?

The epidemiological literature has been concerned about these issues. In the UK,

Smith and Shipley (1991) shows that smokers are drawn predominantly from groups of individuals who have a short life expectancy irrespective of smoking. The study uses a panel data of civil servants and show that non smokers in clerical occupation have a much higher mortality risk than administratives. At the same time, the prevalence of smoking is higher for clerical worker than for administratives. [23] Sterling and Weinkam (1990) show similar results for the US, studying occupation and smoking. The results in Rogers and Powell-Griner (1991) for the US can be used to show that there is a correlation between life expectancy of non smokers and prevalence within gender groups.

So *within* developed countries, the observed smoking patterns seem to be in line with the prediction of the model. Across countries, the evidence is more mixed. For instance, both the life expectancy and the quantities smoked are larger in the US than in the UK. This is not per se a contradiction of our model, as it indicates that the taste for tobacco might be higher in the US than in the UK. Similarly, Japan has the highest life expectancy in the world together with a very high prevalence of smoking. This does not imply that within the Japanese population smoking is unrelated to potential life expectancy. Part of the high consumption in Japan can also be explained by the very low relative price of cigarettes, which is a third of the Swedish one.

Our model can also explain the pattern of tobacco consumption for a country through time. Although we do not have evidence through time for one country, we illustrate the point by calling on the evidence of different but similar countries. We start with a poor country with low life expectancy. In this country, the life expectancy is too short for individuals to worry about the consequences of tobacco, but most individuals are not rich enough to afford smoking. The prevalence would be low, with only richer individuals smoking. An example could be Peru, where the prevalence is only 10% for non educated individuals and 62% among those with a university degree. As growth takes place, income increases and a larger share of the population would smoke. An example could be Mexico or Brazil, where the prevalence is roughly the same across education groups. Eventually, the potential life expectancy would increase such that smoking would appear as undesirable, at least for those who have the longest

[23] Of course, given these results, the correlation is not necessary causal. An interpretation of the results could be that smoking is only due to the occupation and not to the life expectancy.

life expectancy, usually those who are well off. Hence, the correlation between smoking and socio-economic status can be reversed through time. Finally, as potential life expectancy rises, the overall quantity smoked would tend to decrease, as more and more people are discouraged to smoke.

In a number of countries, social norms have an overwhelming effect excluding some groups from cigarette consumption. This is often the case for women in third world countries, and used to be the case in the past in developed countries. If this is the case, our model should accommodate a specific taste shock.

8 Conclusion

This paper develops and tests a model of smoking and endogenous longevity and provide empirical evidence of the importance of differences in potential life expectancy when it comes to understand smoking behavior. Agents have a trade-off between the utility of smoking and an increase in future mortality risks. Hence, part of the heterogeneity in smoking behavior can originate from differences in potential life expectancy, because individuals have incentives to self-select into or out of smoking based on their potential life expectancy. Identification of this selection effect can be achieved when the individual's health is observed repeatedly. Using an extensive panel data, we show that differences in potential life expectancy explain partly the heterogeneity in smoking behavior.

Differences in smoking behavior is often seen as the result of different social norms among occupation or education groups. Our model could provide a partial rationalization of these social norms. Farrell and Fuchs (1982) note that the differences in smoking behavior among different socio-economic groups have increased since information on the effect of tobacco on health has been released in the fifties. This is actually consistent with our model, in so far that new information on the effect of tobacco on health has spurned a selection into or out of smoking based on heterogeneity in potential life expectancy.

We have approached the problem of smoking from a very long-run perspective, typically over the whole life cycle and its implication on mortality. Obviously, in the short-run our simple model could be at odds with the data. Smoking is a complex

phenomena and more is obviously needed to understand better the determinants of smoking at the individual level. Time inconsistent behaviors could explain certain aspect of the data such as the effect of expected price increases or the difficulty to stop smoking. A number of papers have investigated the smoking behavior of teens (Tauras and Chaloupka (1999), Gruber (2000) or DeCicca et al. (2001)), to understand the rise in prevalence among this group. We see our approach as complementary to these lines of research.

However, the methodology developed in this paper, as well as the concept of potential life expectancy can be useful to explain not only smoking behavior but also heterogeneity in other inter-temporal choices. Differences in consumption and savings profile could result from differences in potential life expectancy, as individuals may have different incentives to accumulate savings towards retirement.

A Descriptive Statistics

Table A: Comparing the Panel Data with Characteristics of Swedish Population in 1980

Variables	Mean	Variance	Mean	Variance
	All Sample		Panel Data	
age	47.99	20.26	47.57	19.51
sex	.4854	.4998	.4829	.4997
Number of cigarettes (per day)	4.072	7.758	4.091	7.390
Number of cigarettes if smoking	13.97	8.25	13.69	7.161
prevalence	.2914	.4544	.2987	.457
ever smoked	.5148	.4997	.5258	.4994
income	376.9	269.1	381.6	252.5
Low Education	.5521	.4973	.5247	.4994
Middle Education	.3097	.4620	.3304	.4704
High Education	.1381	.3457	.1447	.3519
blue collar	.0604	.2382	.0659	.2481
white collar	.3721	.4833	.3864	.4870
self-employed	.1438	.3509	.1504	.357
farmers	.0434	.2038	.0454	.2082
students	.024	.1552	.0287	.167
retired	.067	.2504	.0599	.2374
other	.2881	.4529	.2630	.4403
number of children in household	.6597	1.00	.6917	1.015
self assessed health good	.7154	.4512	.7252	.4464
self assessed health fair	.2111	.4081	.2083	.4062
self assessed health bad	.0733	.2607	.0663	.2488
body-mass index	23.78	3.470	23.89	3.522
neoplasm	.0095	.0972	.0094	.0968
hypertention	.0928	.2902	.1031	.3042
asthma	.0173	.1304	.018	.1351
ischemic heart disease	.0205	.1419	.0211	.1438
cerebrovascular disease	.0020	.0454	.0031	.0560
chronic obstructive disease	.0229	.1498	.0233	.1510
stroke	.0488	.2155	.0466	.2110
Back pain	.0525	.2232	.0529	.2240
multiple sclerosis	.0014	.0374	.0009	.0307
epilepsy	.0038	.0616	.0035	.058
Proportion still alive in 1998	70.70		69.87	

B Morbidity Index

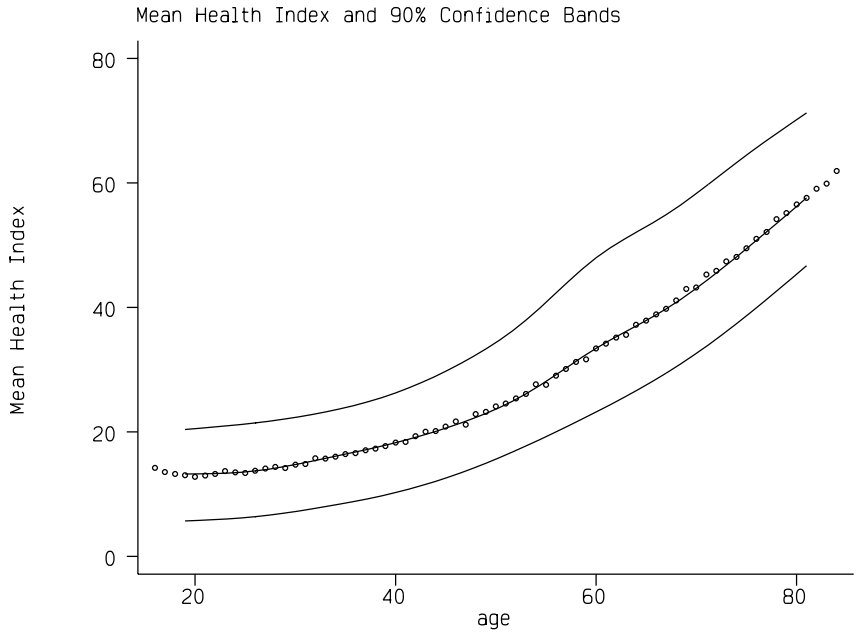
This section describes how we construct a morbidity index, which is used along in the likelihood function to identify health groups. As the data set contains many variables describing the individual's health, we try to summarize the relevant information into one single index, which weights different health outcomes. The weights are computed by estimating a probit model for the probability of death within five years. The explanatory variables are age dummies, sex, self assessed health, functionality (ability to walk a mile, climb stairs, run or board a bus), body mass index and a list of illnesses such as diabetes, malignant neoplasm, ischemic heart diseases, other heart problems, chronic obstructive pulmonary disease, allergy, hypertension, epilepsy, diseases of the circulatory systems, depression... For diabetes, the age of first diagnoses was also included to distinguish between diabetes of type 1 and type 2. The explanatory variables accounted for roughly 40% of the variance of the dependent variable.

Table B: Variables used in Ill Health Index

Self assessed health, poor, fair or good.	
Self assessed health relative to same age group, poor, fair or good	
Body mass index, <20, $[20,30]$,>30.	
Ability to climb stairs.	Ability to board a bus.
Ability to walk a mile.	Ability to run 100 meters.
In hospital for more than 2 weeks, last 12 months.	In hospital last 3 months.
Medication for cough.	Medication for sleep.
Medication for anxiety.	Taking antibiotics.
Medication for pain (with prescription).	
Diabetes.	Age of start of diabetes
Epilepsy	Allergy
Malignant neoplasm.	Ischemic heart disease.
Other heart problem.	Cerebrovascular disease.
Chronic obstructive pulmonary disease	Hypertension.
Asthma.	Multiple sclerosis.
Back pain.	Disease of digestive system.
Disease of arteries.	Disease of veins and lymphatics.
Hernia of abdominal cavity.	Disease of the skin.
Migraine.	Deafness.
Disease of esophagus, stomach, and duodenum.	

From the regression results, we compute the linear prediction and we use it as a morbidity index. Without any loss of generality, we rescaled the index between 0 and 100. A zero value corresponds to the fittest individual in the dataset and 100 to the worst off. The morbidity index is plotted against age in Figure 6. The morbidity index is linearly increasing in age and evidence not shown here shows that it is higher for men than for women and linked to education level, with the low educated cumulating more diseases at a given age. The variance of the morbidity index is also increasing with age, at least until age 70, when it decreases, probably due to a selection of the fittest.

Figure 6: Morbidity Index, Mean and Standard Deviation



C Likelihood and Functional Form Specification

C.1 Likelihood

We derive here the likelihood of the "health" model, where we estimate jointly a competing risk duration model and the model of health evolution. Let T a random variable indicating life duration.

Given the specification of the conditional survival probability, the survival probability at a given age is:

$$P(T \geq age) = P^j_{Surv}(age) = \prod_{l=1}^{age} \pi(l, S^M(l, \varepsilon_j), \varepsilon_j)$$

The probability of dying (irrespective of the cause) at a given age is:

$$P^j_D(age) = P^j_{Surv}(age-1)(1 - \pi(l, S^M(l, \varepsilon_j), \varepsilon_j)) = P^j_{Surv}(age-1) - P^j_{Surv}(age)$$

Peto et al. (1992) provide estimation of the fraction of individuals dying from a particular cause at different ages. From our specification in terms of competing risks, this fraction is:

$$\frac{\pi_0(age)\left(1 - \pi_1(age, S^M)\right)}{1 - \pi(age, S^M)}$$

We compute the likelihood of observing a tobacco related death for a smoker. If the probability that the death is caused by tobacco is p, the likelihood of observing such a cause of death is:

$$L_C(age; j) = \phi((\frac{\pi_0(age; j)[1 - \pi_1(S_{age}, age)]}{1 - \pi_0(age; j)\pi_1(S_{age}, age)} - p)/\omega_C)/\omega_C$$

where ω_C is penalty, proportional to the precision of the computation, to account for the error in the computation of the weights.

The likelihood of observing a morbidity index of m at a given age is:

$$P_M(age, m; j) = \phi(\frac{m - \mu_j - \delta_{01}age - \delta_{02}age^2 - \delta_1 S_{age} - \delta_2 S^2_{age}}{\sqrt{age\ \sigma^2_\varepsilon + \sigma^2_u}})/(\sqrt{age\ \sigma^2_\varepsilon + \sigma^2_u})$$

where δ_1 and δ_2 have been already estimated in the previous section.

We now proceed to compute the likelihood of observing the data for a given individual. Let age^h_{Death} be the age at death of individual h, if relevant. Let Death^h be a dummy equal to one if death occurs any time before 1999. Let Death^h_l, $l = \{80, 88, 96\}$ be three dummies equal to one if the individual is dead at the time of the interview. Let age^h_{98} be the age in 1998 if alive at the end of the sample. Let S^h be a dummy equal to one if the individual has been or is a smoker. For any individual h with type j, the likelihood of observing the data is

$$\begin{aligned} L^{hj} \quad = \quad & P_D(age^h_{\text{Death}}; j)^{\text{Death}^h} P_{Surv}(age^h_{98}; j)^{(1-\text{Death}^h)} L_C(i; j)^{\text{Death}^h S^h} \\ & \prod_{l=\{80,88,96\}} P_M(age_l, m_l; j)^{(1-\text{Death}^h_l)} \end{aligned}$$

The likelihood of the entire sample is:

$$\mathcal{L}_{health} = \prod_h (\sum_{j=1}^J L^{hj}\lambda_j)$$

where λ_j is a weight associated to type j, to be estimated.

C.2 Functional Form Specification

$\pi_0(age)$ is assumed to be decreasing in age and bounded between zero and one. Moreover, we know that the probability of surviving at young ages is constant and almost one, at least up to age 40. After that age the probability of surviving declines at an increasing rate.

$$\pi_0(age) = \frac{1}{2}\left[1 - \tanh(\pi_{01} + \pi_{02} age + \pi_{03} age^2)\right]$$

$\pi_1(age, S^M)$ is assumed to be bounded between 0 and 1 and decreasing with both arguments when $S^M > 0$. For $S^M = 0$, $\pi_1(age, 0) = 1$:

$$\pi_1(age, S^M) = 1 + \frac{S^M}{\bar{S}^M}\left(\frac{\tanh(\pi_{11} + \pi_{12} age^{\pi_{13}})}{\tanh(\pi_{11} + \pi_{12})} - 1\right)$$

where $\bar{S}^M$ is a constant representing a maximum stock accumulated during the life cycle. In the estimation, we fix this number to be equal to fifty years of smoking 3 packs a day. The number of parameters in each of these functions were settled after some experimentation. In particular, a quadratic term in age in π_0 improved the fit.

D Estimation Results

Table C: Estimation Results

Health						
	Type 1 PLE=76.7		Type 2 PLE=80.5		Type3 PLE=86.1	
Parameter	Est	S.E.	Est	S.E.	Est	S.E.
π_{01}	-4.87	1.51	-4.47	0.63	-12.25	3.6
π_{02}	0.021	0.05	5.3e-5	0.02	0.13	0.09
π_{03}	4.9e-4	3.5e-4	6.4e-4	1.6e-4	1.5e-4	5.9e-4
π_{11}	1.16	0.4	1.16	0.4	1.16	0.4
π_{12}	-3.2e-10	3.7e-10	-3.2e-10	3.7e-10	-3.2e-10	3.7e-10
π_{13}	5.01	0.3	5.01	0.3	5.01	0.3
μ_j	-1.49	0.02	-2.11	0.02	-2.53	0.02
λ_j	0.42	0.014	0.51	0.013	0.07	0.006
δ_{01}	-0.04	0.001	-0.04	0.001	-0.04	0.001
δ_{02}	7.8e-4	1e-5	7.8e-4	1e-5	7.8e-4	1e-5
δ_1	8.5e-4	0.02	8.5e-4	0.02	8.5e-4	0.02
δ_2	2.2e-5	1.7e-5	2.2e-5	1.7e-5	2.2e-5	1.7e-5
σ_u	0.0025	0.2	0.0025	0.2	0.0025	0.2
σ_ε	0.032	0.0005	0.032	0.0005	0.032	0.0005

Preferences		
Parameter	Est	S.E.
β	0.952	0.003
ν_1	-6.79	0.31
ν_2	-0.18	0.02
ν_3	0.13	0.01
ν_4	258.1	4.0
ν_5	110.1	4.0
ν_6	1.6e5	3.7e4

Note: Asymptotic standard errors were computed.

Table D: Descriptive Statistics, by Health Type

Var	Type 1 PLE=76.7	Type 2 PLE=80.5	Type 3 PLE=86.1	Male	Female	Low Educ	Middle Educ.	High Educ	Blue Collar	White Collar	Self Employed	Retired	Ever Smoker	Non Smoker
Type 1	1	0	0	.14	.05	.16	.05	.03	.04	.03	.06	.24	.11	.07
Type 2	0	1	0	.78	.23	.52	.50	.45	.52	.53	.65	.43	.53	.47
Type 3	0	0	1	.07	.70	.31	.43	.50	.42	.43	.28	.32	.35	.44
Male	.72	.75	.09	1	0	.46	.50	.50	.48	.56	.72	.43	.56	.41
Low Ed.	.68	.43	.33	.40	.44	1	0	0	.34	.08	.32	.70	.39	.45
Middle Ed.	.23	.39	.42	.39	.37	0	1	0	.58	.27	.46	.22	.42	.34
High Ed.	.07	.17	.24	.19	.18	0	0	1	.07	.63	.20	.06	.17	.20
Blue collar	.19	.39	.40	.36	.36	.29	.55	.15	1	0	0	0	.43	.29
White collar	.05	.18	.19	.19	.14	.03	.12	.57	0	1	0	0	.17	.17
Self-employed	.03	.06	.03	.07	.02	.03	.05	.05	0	0	1	0	.05	.04
Retired	.64	.22	.20	.24	.29	.44	.16	.09	0	0	0	1	.23	.30
Ever Smoker	.60	.53	.45	.58	.42	.46	.55	.46	.59	.50	.57	.43	1	0
Age	61.2	44.6	44.9	45.9	47.7	54.0	41.5	41.3	38.2	41.5	44.9	70.4	46.4	47.2
# of Cig	3.8	3.9	2.9	3.9	3	3.3	4.1	2.5	5.0	3	4.5	2.1	6.9	0
# of Cig if>0	14.1	14.4	12.5	14.7	12.6	13.3	14.0	13.8	14.3	14.3	16.2	11.9	13.7	0

Note: Statistics on 38405 individuals, in all years.

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