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Abstract

We consider how individuals' decisions on their health behaviours depend on the level of investment in their health provided by the State. We develop the model provided by Ehrlich and Becker (1972) and Peltzman (1975) and we show that higher levels of protection provided by the State (either through the increased availability or effectiveness of medical care) can crowd out or reinforce self-insurance. We apply this model to the smoking cessation decision made by individuals diagnosed with a cardiovascular disease in waves of the Health Survey of England between 1993 and 2006. There has been a considerable increase in the proportion of these individuals who receive prescriptions of statins from the State, a highly effective drug that reduces the probability of further heart attacks and premature death. We find that the probability of quitting smoking is increased by four percentage points amongst those individuals prescribed statins. This result is robust to allowing for the direct effects of smoking advice. When the potential endogeneity of doctors' decision to prescribe statins is dealt with using variables in national guidelines, we find that unobservable characteristics which make people more prone to stop smoking reduce the probability of receiving statins and the evidence of the complementarity between quitting smoking and prescription of statins is confirmed.

Keywords: Crowding-out; moral hazard; preventative behaviour; drugs

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prescription.
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1. Introduction

Individuals' health behaviours are known to be important in determining health status [Balia and Jones (2008), Contoyannis and Jones (2004)].

In recent years, the UK NHS has increased its expenditure both on the promotion of health behaviour change and on the supply of effective health care interventions. Although the effect of the latter may influence individual behaviour, most evaluations focus on the direct health effects of NHS interventions in terms of medical treatment or health promotion. In this paper we examine the indirect effects of health care investment on individual health behaviours. On the one hand, the State provides insurance for losses caused by ill-health. More comprehensive insurance may induce individuals to take more risks with their health and exert less effort on health production (the "crowding-out hypothesis"). On the other hand, greater investment by the State increases life expectancy, which, in turn, increases the returns to individual health investments. Individuals may therefore exert more effort in investing in their future health (the "complementarity hypothesis").

We develop a theoretical model in the framework of insurance contracts by Ehrlich and Becker (1972) and Peltzman (1975) where the individual has to choose the level of self-insurance (i.e. healthy behaviours) based on the level of protection/insurance by the State. We find that crowding out only occurs under specific assumptions on the relation between the volume and effectiveness of medical treatment and self-insurance.

We apply this model to the case of individuals with heart disease who face the decision of whether or not to quit smoking. According to the Department of Health, coronary heart disease (CHD), despite being preventable, kills more than 70,000 people each year. About 110,000 people have a heart attack in England every year and around two million people suffer from angina in the UK. Thus, CHD is a significant cause of morbidity and mortality.

The Department of Health published a "National Service Framework" for Coronary Heart Disease (CHD NSF) in 2001 which set out a ten-year plan to "transform the prevention, diagnosis, treatment and care of patients with heart disease" [DH, CHD NSF (2004)]. Part of this programme was more effective prescribing by family doctors, with prescription of statins alone intended to save 3,000 lives a year [DH, NHS Plan (2000)].

The CHD NSF proposed that people with diagnosed CHD should receive advice about how to stop smoking, personalised advice on how to reduce other

modifiable factors (including physical activity, diet, alcohol consumption and weight), advice and treatment to control blood pressure, daily aspirin, and statins and dietary advice to lower serum cholesterol concentrations. In addition, the CHD NSF placed an immediate priority on raising the proportion of people discharged from hospital following a heart attack receiving aspirin, beta-blockers and statins to 80-90% by April 2002.

Following the CHD NSF launch up to March 2004, prescription of statins increased by 30% every year [DH, CHD NSF (2004)].

Compared to ten years ago, smokers who now develop heart disease may respond to this better State-protection by either being more likely to quit (the returns are higher) or being less likely to quit (better protection induces moral hazard).

Whilst we find strong evidence of complementarity between prescription of statins[2] and quitting smoking, we highlight several methodological issues that may affect causal inference. First, the indirect effect of the prescription of statins needs to be disentangled from the direct effect of smoking cessation advice. Second, the timing of the decision to quit smoking should be after the diagnosis of cardiovascular problems. The results still hold after such robustness checks.

Potential biases may still arise if doctors - able to observe the underlying individual propensity to quit smoking - select patients, who, in their opinion, are more likely to benefit from the medical treatment. We estimate a recursive, simultaneous equation model with an endogenous dummy variable. Identification of "objectively" high risk patients is obtained through the use of variables contained in national guidelines which should be exogenous to doctors' decision to prescribe. We find that patients' willingness to quit smoking makes them less prone to be prescribed with statins (and vice versa). The prescription of statins exclusively based on observed risk factors reinforces the probability of quitting smoking. This is evidence that doctors use medical treatment to compensate for the patients' unobserved attitude toward health behaviour.

This paper is organized as follows. The next section presents the literature review. Section (3) solves a theoretical model. Then, section (4)

[2]Information on the prescription of lipid-lowering drugs in the Health Survey for England (HSE) was obtained from all prescriptions with a code of 2.12 in the *British National Formulary*. Actually, separate identification of statins is not possible in the HSE, but by 1998 statins comprised around 90% of all lipid-regulating drugs prescribed [ONS (1998)].

describes the data. Section (5) presents the results of the empirical models. Concluding remarks are offered in Section (6).

2. Literature review

The shift of attention away from health provision *per se* and towards how individual and societal behaviour can be changed to improve population health status, began to be the focus of the health policy in the late 20th century. The Lalonde's Report (1974) in Canada proposed to focus on the areas of lifestyle, environment and human biology rather than considering health care as the only determinant of health. The Report advocated that the most effective means to reduce mortality is the improvement of environmental and lifestyle factors.

Both the Acheson Report (1998) and the White Paper, Our Healthier Nation (1999) in the late 1990s emphasised that health depends on social and economic conditions, as well as the environment, lifestyle, access to services and genetic risk factors. The Reports gave recommendations on the promotion of healthier lifestyle; diet and nutrition; physical activity and smoking cessation.

At the beginning of the 21st century the Wanless Report (2004) used scenario planning to illustrate and quantify the benefits of investing in a long-term public health strategy that focuses on both health care provision and health behaviour. The Report illustrated the considerable difference in expected cost depending upon the extent to which the NHS services became more productive and people became fully engaged with their own health.

In arguing that the short-term benefits of engaging people in living healthier lives can involve a reduction of the demand for health services, the Report thus recognised that the demand for health care is derived from the demand for health.

Long before the Wanless Report (2004) was published, the seminal work by Grossman (1972) showed that health care is both a consumption good that yields utility, and an investment good, which yields satisfaction to consumers indirectly through increased productivity, fewer sick days, and higher wages. Investment in health is costly as consumers must trade-off time and resources devoted to self care with other goods such as leisure (i.e. the money and time prices of health as defined by Acton (1973)).

More recently, theoretical models have highlighted the defining feature of health as an uncertain good. As noted by Zweifel et al. (2009), theo-

ries of production can be applied to the choices of "healthy" inputs once uncertainty of the outcome and state-dependent preferences are taken into account. These models can be placed in the tradition of "state-contingent claims" as developed by Arrow (1973) and Debreu (1959).

Although unobservable factors affect health status, each individual, as producer of her own health, still has the ability to prevent ill-states or their negative effects should they occur. We can thus think of choices between "healthy" inputs and other goods in the framework of insurance contracts. Ehrlich and Becker (1972), make a distinction between the concept of insurance (where a redistribution of income toward hazardous states occurs) and self-protection (where the probability that hazardous states occur is reduced). They argue that complementarity between insurance and self-protection occurs whenever the assumption of independence between their prices is dropped. *"Not only are market insurance and self-protection complements in the sense that the availability of the former could increase the demand for the latter, but also in the sense that an increase in the productivity of self-protection or a decrease in the real cost of market insurance would increase the demand for both"*[Ehrlich and Becker (1972)].

Peltzman (1975) applied this concept to the effect of automobile safety regulation on road accidents. He demonstrated that mandatory installation of safety devices, designed to decrease the loss from an accident, has ambiguous effects on the total cost of accidents - the devices will lead to an increase in the probability of an accident, which may offset the reduction in the average loss per accident.

A recent paper by Chang and Trivedi (2009) has built a model of choice between self-medicating behaviour and professional care in developing countries. Under the assumption that self-medication is a low-quality and risky strategy in Vietnam, they showed that it is an inferior good (i.e. the demand for it declines with rising household incomes). After demonstrating that the demand for self-medication is decreasing in price, they imply that a decrease in the price of professional care lowers the demand for self-medication as its relative price increases. Their results seem to support crowding out of self medication, but rely on the strong assumption that the prices of self-medication and professional care do not depend on each other.

de Preux (2010) modifies Ehrlich and Becker (1972) model assuming that lifestyle benefits appear in the future and not immediately. Thus, focusing on the anticipatory effects of insurance she finds that as the individual approaches the period of Medicare coverage, moral hazard occurs and invest-

ments in healthy lifestyles are reduced.

There is little empirical literature that tests the effect of insurance on individuals' healthy behaviour. Courbage and de Coulon (2004) find that privately insured individuals are more likely to have breast screening and undertake healthier lifestyles than individuals covered by NHS. Stanciole (2007) uses a sequential model to look at the impact of insurance on lifestyle measures such as smoking and obesity. He finds evidence of moral hazard. de Preux (2010) uses the Health and Retirement Study and finds no evidence of moral hazard for the quitting smoking behaviour. But she finds weak evidence of moral hazard in drinking behaviour.

The empirical literature on statins is particularly focused on two aspects: a) the effectiveness of statins in increasing the survival of patients diagnosed with heart disease; and b) the issue of equitable treatment across patients.

Lipid-regulating drugs have been found to reduce mortality of patients with ischaemic heart disease (IHD) in observational data as well as randomised controlled trials. For instance, Hippisley-Cox and Coupland (2006) show that patients taking statins have higher survival rates than those who do not take statins. The six year survival rate was 89% for patients taking statins and 66% for those not taking statins. They find no significant evidence that the effectiveness of statins is related to age, sex or diabetes.

Statins have been around for 20 years. Their growth has been fuelled by trials that show they can cut deaths from heart disease by at least a quarter, and because they are relatively cheap and very safe. Indeed, since 1994 several large randomised clinical trials have established the effectiveness of lipid lowering drugs (particularly statins) in preventing deaths from coronary heart diseases through the lowering of cholesterol, both in the context of primary and secondary prevention.

Despite the effectiveness of statins being widely recognised, prescription rates vary across patients with the same risk factors, which raises question about the equity of treatment. Gill et al. (2004) use the Health Survey for England data in 1998 and 1999 and suggest that socioeconomic factors but not ethnicity affect the use of lipid-lowering drugs. In line with the results of Reid et al. (2002) and Ramsay et al. (2005), they find that the young and people with a history of myocardial infarction are those, amongst the patients with IHD, more likely to be taking lipid-regulating drugs. Undertreatment with statins amongst elderly and women has been higlighted by Tonstad et al. (2004), in the Oslo Health Study, and by Ma et al. (2005) in the National Ambulatory Medical Care Survey in U.S.

Reid et al. (2002) also suggest that smokers are at a disadvantage for receiving lipid lowering therapy. Large statin trials (as summarised by Isles and Norrie (2000)) have found that statins are equally effective in smokers and non-smokers. Nevertheless, the question is whether smokers are willing to improve the effect of lipid lowering drugs by giving up smoking and whether doctors compensate the negative health effects of smoking with medical treatment.

The difficulty of the clinician can be summarised as follows. On the one hand, as noted by Isles and Norrie (2000), doctors recognise that whilst the effect of pharmacological treatment is immediate, changes in lifestyles such as quitting smoking can be difficult particularly amongst poorly motivated people. Hence, they may over-supply treatment to compensate for the smokers' lower effort in healthier lifestyles. On the other hand, as suggested by Goddard and Smith (2001) and by the findings of Tonstad et al. (2004), doctors could under-supply medical treatment if they believe that the marginal benefit from the treatment to smokers is lower compared to non-smokers.

3. The theoretical model

In this section we develop a model that determines the effect of changes in the availability and effectiveness of State medical services on the care that individuals take of themselves. More specifically, the model aims at answering the following question: does State-protection (proxied by the provision of medical care) crowd out or reinforce self-insurance (defined as the individuals' healthy behaviour)?

Zweifel et al. (2009) have highlighted that the concept of inputs substitutability can be applied to medical and self-care in the health production function. This approach has been taken by Chang and Trivedi (2009) in a health production model of interaction between self-medication and professional care. However, closed form solutions of these models require the specification of utility functional forms and the health production function, thus casting doubts on the general implications of their results.

Our approach departs from the traditional health production function and adopts Ehrlich and Becker (1972) and Peltzman (1975) concepts of insurance and State-protection to health. We can think of the NHS as a benevolent State-provided organisation that both reduces the probability that the individual enters an ill-state and reduces the negative effects of a disease if and when it occurs. In addition, individuals can also reduce the effects of

an illness by adopting healthier lifestyles. But then the question we ask is whether an increase in the availability of State-protection is reinforced by self-care or not.

The model we set up can be thought of, in the terms of Ehrlich and Becker (1972), as the concomitant effects of "State protection" (whereas the quantity and effectiveness of medical services reduce the probability that illness occurs) and "State insurance" (whereas the quantity and effectiveness of medical services reduce the negative effects of illness) on self-insurance (whereas self-care reduces the negative effects of illness).

3.1. Setup

The traditional "state of the world" approach to insurance contracts assumes that the probability of a state is determined by "nature" and is independent of human actions. But this setup cannot be applied in the context of "health states" where the occurrence of an ill-state (partly) depends on individuals' behaviour.

Consider, for example, the probability that a heart attack occurs. Independent state probabilities for each episode cannot be obtained, since the probability of a heart attack occurring in a particular individual is not independent of the quantity and effectiveness of medical services. In this context, we define State-protection in health as the health interventions that reduce the probability of ill-states and raise the probability of better health states by shifting the distribution of health away from bad outcomes.

The State-provided NHS service can also reduce the negative effects of a heart attack, by prescribing statins for example, effectively acting as an "insurer" in a broad sense (so-called State-insurance). Individuals can also reduce the negative effects of a heart attack by complying to medical advice or adopting appropriate lifestyle (so-called self-insurance).

We consider the case where individual behaviour cannot affect the probability that ill-states occur. This can be considered a special but not unrealistic case, if we think of an individual who has already cardiovascular diseases or has had a first episode of heart attack. In these cases, the "endowed" probability of developing a more severe condition is so high that the effect of self-care can be considered to be negligible compared to medical care[3].

[3]This case corresponds to the sample of cardiovascular disease patients upon which the empirical analysis is based.

Assume that an individual is faced with only two states of the world $H \in \{0, 1\}$ with probabilities π and $(1 - \pi)$, respectively. In an attempt to abstract from functional forms of production functions, we look at an individual who wants to maximise her expected income in the healthy and ill-states [Peltzman (1975)].

In the "healthy state", H_0, she will enjoy the following income:

$$H_0 = I - rs - pm \tag{1}$$

where I =income; rs =expenditure on self-care and pm =expenditure on medical services (including time costs).

In the ill-state, H_1, she will incur in a cost of illness given by the loss of labour income:

$$H_1 = I - rs - pm - L \tag{2}$$

where L =loss of income due to illness and, as before, pm =expenditure on medical services. Should the individual be affected by a heart attack, she will incur an income loss from reduced number of working days, for example.

Assume also that:

$$\pi = \pi(\pi^e, \alpha, m) \tag{3}$$

whereby π, the probability of an ill state to occur, depends on π^e, the endowed probability[4] of ill-health, m, the quantity of medical services (i.e. number of visits to the doctor) and α, the effectiveness of such services (i.e. medical "technologies" such as statins). Note, $\frac{\partial \pi}{\partial m} \equiv \pi_m(\pi^e, \alpha, m) \leq 0$ such that an increase in the use of State-protection reduces the probability that the ill-state will occur. We distinguish between the volume and effectiveness of State intervention to highlight the potential effect of technological advances. We assume that, for example, frequent doctor visits can reduce the chances that a heart attack occurs. In addition, $\frac{\partial \pi}{\partial \alpha} \equiv \pi_\alpha(\pi^e, \alpha, m) \leq 0$ indicates that an improvement in medical technologies can make State volume more effective and further reduce the probability that ill-states occur.

[4]This can be thought of as the probability of having a heart attack given a family history of heart diseases. It is the "pre-determined" probability that an ill-state occurs no matter how much State-protection has been used. Of course, one can then specify several functional forms that allow for self-care and the pre-determined probability to interact with one another. We hereby assume that the endowed probability is high enough that the effect of self-care on reducing the probability of illness can be neglected.

In the next two sub-sections we first analyse the case where the income loss due to illness only depends on self-care and then we consider the case where the quantity and effectiveness of medical services can also affect the income loss.

3.2. State-protection and self-insurance

Assume that whilst self-care can reduce the income loss from illness, the quantity and effectiveness of health interventions reduce the probability that ill-states occur. Hence, we can think of this model as an analysis of the concomitant effects of State-protection and self-insurance on the choice of self-care and medical services.

We can thus let the loss function depend on self-care such that:

$$L = L(s) \tag{4}$$

where $L^{'}(s) < 0$.

By combining equations (1-4):

$$EI = [1 - \pi(\pi^e, \alpha, m)]\,(I - rs - pm) + \\ + \pi(\pi^e, \alpha, m)\,[I - rs - pm - L(s)] \tag{5}$$

An individual then chooses a combination of self-care, s, and medical services, m, to maximise expected income. After rearranging equation (5), the maximisation problem can be written as follows:

$$\max_{m,s} EI = I - rs - pm - \pi(\pi^e, \alpha, m)L(s) \tag{6}$$

$$\frac{\partial EI^*}{\partial m} \equiv EI_m = 0 \Rightarrow -p - \pi_m(\pi^e, \alpha, m)L(s) = 0 \tag{6a}$$

$$\frac{\partial EI^*}{\partial s} \equiv EI_s = 0 \Rightarrow -r - \pi(\pi^e, \alpha, m)L^{'}(s) = 0 \tag{6b}$$

Rearranging (6a) and (6b):

$$-\pi_m(\pi^e, \alpha, m) = \frac{p}{L} \tag{7}$$

$$-L^{'}(s) = \frac{r}{\pi(\pi^e, \alpha, m)} \tag{8}$$

Necessary second order conditions are:

$$EI_{mm} = -\pi_{mm}(\pi^e, \alpha, m)L(s) < 0 \tag{9}$$

$$EI_{ss} = -\pi(\pi^e, \alpha, m)L''(s) < 0 \tag{10}$$

These will hold if there are diminishing returns to State-protection and self-care loss reduction (i.e. $\pi_{mm}(\pi^e, \alpha, m), L''(s) > 0$).

Equations (7-10) have the following implications: 1) an increase in the price of medical services, p, or a reduction in the loss of income due to illness, L, reduces the use of State-protection and hence increases the risk of a heart attack; and 2) an increase in the price of self-care, r, or a reduction in the probability that a heart attack occurs, reduces the patient's use of self-care thus increasing the loss of income from illness.

3.2.1. Effect of change in the effectiveness of medical services

The direction of changes in the effectiveness of medical services on m and s depends on the numerator of equations (A.1) and (A.2) as derived in Appendix A.

As $EI_{s\alpha}$ and EI_{sm} are both unambiguously negative[5], the effect of a change in the effectiveness of medical services depends on the relation between quantity and effectiveness of medical services.

From the first order conditions in equations (6a-6b):

$$EI_{m\alpha} = -\pi_{m\alpha}(\pi^e, \alpha, m)L(s) \tag{11}$$

the sign of which is ambiguous depending on $\pi_{m\alpha}(\pi^e, \alpha, m)$.

Crowding-out of self-care (i.e. substitution of self-care with medical services) takes place whenever increased effectiveness enhances the effect of medical services in reducing the likelihood of an illness. More formally, the moral hazard hypothesis occurs if $\pi_{m\alpha} < 0$, which, in turn, implies that $EI_{m\alpha} > 0$ and hence $\frac{\partial m}{\partial \alpha} > 0$ and $\frac{\partial s}{\partial \alpha} < 0$.

Note that this result depends on the assumption that the quantity and effectiveness of medical services, m and α, do not affect the income loss from a heart attack but only the probability of its occurrence. In the next sub-

[5]This result arises from the following assumptions: a) the likelihood of an illness declines as either the effectiveness or the quantity of medical services increase; and b) the income loss declines as self-care is increased.

section we will drop this assumption to test whether our result still holds.

3.3. State-protection, self-insurance and State-insurance

A more realistic approach allows health interventions to reduce both the probability that an ill-state occurs and the income loss from an illness. Hence, we can think of this model as an analysis of the concomitant effects of State-protection, self-insurance and State-insurance on the choice of self-care and medical services.

We can thus let the loss function depend on self-care, quantity and effectiveness of medical services such that:

$$L = L(s, \alpha, m) \tag{12}$$

where $L_k(s, \alpha, m) < 0 \quad \forall \quad k = s, \alpha, m.$

The maximisation problem can be written as follows:

$$\max_{m,s} EI = I - rs - pm - \pi(\pi^e, \alpha, m)L(s, \alpha, m) \tag{13}$$

$$\begin{aligned} \frac{\partial EI^*}{\partial m} \equiv EI_m = 0 \Rightarrow & -p - \pi_m(\pi^e, \alpha, m)L(s, \alpha, m) - \\ & - \pi(\pi^e, \alpha, m)L_m(s, \alpha, m) = 0 \end{aligned} \tag{13a}$$

$$\frac{\partial EI^*}{\partial s} \equiv EI_s = 0 \Rightarrow -r - \pi(\pi^e, \alpha, m)L_s(s, \alpha, m) = 0 \tag{13b}$$

Necessary second order conditions are:

$$\begin{aligned} EI_{mm} = & - \pi_{mm}(\pi^e, \alpha, m)L(s, \alpha, m) - \\ & - 2\pi_m(\pi^e, \alpha, m)L_m(s, \alpha, m) - \\ & - \pi(\pi^e, \alpha, m)L_{mm}(s, \alpha, m) < 0 \end{aligned} \tag{14}$$

$$EI_{ss} = - \pi(\pi^e, \alpha, m)L_{ss}(s, \alpha, m) < 0 \tag{15}$$

These will hold if there are diminishing returns to State-protection and self-care loss reduction (i.e. $\pi_{mm}(\pi^e, \alpha, m), L_{kk}(s, \alpha, m) > 0 \quad \forall \quad k = m, s$).

The effect of changes in the effectiveness of medical services on m and s has been derived as before in Appendix A. The direction of such effects are even more ambiguous than before as it depends on the combined effect of self-care, quantity and effectiveness of medical services on the income loss and the likelihood of an illness.

Crowding-out of self-care (i.e. substitution of self-care with medical services) takes place whenever increased effectiveness enhances the effect of medical services either in reducing the likelihood of an illness or in reducing the income loss (or both). The income loss from illness, however, is not reduced by the combined effect of self-care and quantity or effectiveness of medical services.

More formally, the moral hazard hypothesis hinges upon the following conditions: a) $\pi_{m\alpha} < 0$ and/or $L_{m\alpha} < 0$ which, under certain conditions[6], imply that $EI_{m\alpha} > 0$; and b) $L_{s\alpha}, L_{sm} > 0$ which, respectively, imply that $EI_{s\alpha}, EI_{sm} < 0$.

3.4. Concluding remarks

To sum up, we have analysed the relation between health interventions and individuals' healthy behaviour (i.e. self-insurance) under the following two scenarios. First, the State-provided NHS can only reduce the probability that ill-states occur - so-called State-protection. Second, in addition to the State-protection role, the NHS can effectively act as insurer by reducing the size of income loss should an ill-state occur - so-called State-insurance. In both cases we have shown that the relation between medical services and the care individuals take of themselves is an ambiguous one.

The occurrence of moral hazard (i.e. substitution of self-care with medical services) depends on particular relations between self-care itself and the quantity and effectiveness of medical services. The predictions of the model would become even more difficult to interpret once individuals are assumed to both protect and insure themselves.

As the relations between self-care and the quantity and effectiveness of medical services depend on individuals' preferences, the moral hazard hypothesis remains an empirical question that we will address in the following sections of this paper.

4. Data and descriptive statistics

This descriptive section is divided into five parts. Firstly, we provide an overview of the Health Survey for England (HSE) in subsection (4.1). Secondly, we describe the characteristics of the sample in subsection (4.2).

[6]See Appendix A for a more detailed derivation.

Finally, we present the measures of State-protection and self-care together with a simple analysis of their correlation in subsections (4.3-4.5).

4.1. The Health Survey for England (HSE)

The Health Survey for England (HSE) comprises a series of annual surveys beginning in 1991. It is designed to be nationally representative of people of different age, sex, geographic area and socio-demographic circumstances. The HSE series is part of an overall programme of surveys commissioned by the Department for Health and designed to provide regular information on various aspects of the nation's health. Since 1994 onwards the survey has been carried out by the Joint Survey Unit of the National Centre of Social Research and the Department of Epidemiology and Public Health at University College London. All surveys have covered the adult population aged 16 and over living in private households in England. Children have been included in every year since 1995.

The Health Survey combines information on diagnoses, health behaviours and prescribed medicines.

The Health Survey for England contains a "core" sample which is repeated each year and each survey year has one or more modules on subjects of special interest and a "boost" sample. The "core" part includes questions on general health and psycho-social indicators, smoking, alcohol, demographic and socio-economic indicators. It also includes information on prescribed medicines. In each year of the "boost" sample, the Health Survey for England focuses on a different demographic group and looks at such health indicators as cardiovascular disease, physical activity, eating habits, oral health, accidents, and asthma. In particular, there is a focus on cardiovascular diseases and associated risk factors in the following waves of the data: 1991-1994, 1998, 2003 and 2006.

4.2. The characteristics of the sample

In order to have a consistent sample size, the empirical analyses in the following sections of this paper will focus on specific waves of the HSE where the largest sample of cardiovascular disease (CVD)[7] patients is available (i.e.

[7]More specifically, people diagnosed with heart murmur, diabetes, blood pressure, angina, heart attack, irregular heart rythm, stroke or other heart condition.

1993, 1994, 1998, 2003, 2005 and 2006)[8]. The descriptive statistics in this section only refer to some of these waves, namely, 1998, 2003 and 2006.

In Table (1) we provide some simple descriptive statistics for specific waves of the HSE. Over 50 percent of the sample of people with or without CVD is female. On average, people with CVD are older than people without CVD, where the age gap exceeds 15 years. There are substantial differences in the proportion of people with any CVD problem across the country over the years. The proportion of people living in the North East and West Midlands with CVD has increased, respectively, by ten and 13 percentage points from 1998 to 2006. In the North-East there is the highest CVD rate across the years.

Turning to socioeconomic measures, we find that the proportion of manual workers who have CVD has increased from 27 percent in 1998 to 31 percent in 2006. Unsurprisingly, as these conditions are most likely to appear at later stages in life, the majority of retired people have CVD. There are marked differences in the proportion of people with CVD problems across the distribution of income. Up to 2003, the majority of ill people are located in the lower tails of the income distribution (i.e. either in the first or second quintile). In 2006, there is an improvement in the bottom income quintile and a shift of ill people to the middle and higher quintiles.

4.3. Measures of State-protection/insurance in the HSE

State-protection/insurance in health can be provided by offering information to the patient or by delivering medical services such as prescription of medicines.

We focus on the information regarding smoking cessation advice contained in the HSE. From the late 1990s considerable effort has been put by the UK government to increase smoking cessation campaigns and the availability of services for those willing to quit. In Table (2) we report some demographic and economic characteristics of those, amongst the patients with CVD problems, who receive and fail to receive smoking cessation advice.

The proportion of people with CVD who do not receive smoking cessation advice is higher than those who do receive advice. CVD patients who receive smoking cessation advice are usually younger than those who do not receive

[8]All the years but 2005 contain a boost sample of CVD patients. In 2005 the large sample of people with CVD is due to the fact that the boost sample is composed by older people (who are also more likely to have CVD).

Table 1: Sample characteristics

	1998		2003		2006	
	No CVD	*CVD*	*No CVD*	*CVD*	*No CVD*	*CVD*
Demograph characteristics:						
Sample Size	(15,210)	(4,423)	(13,584)	(4,939)	(8,582)	(3,953)
Gender (=1 if female)	0.53	0.55	0.54	0.55	0.56	0.53
Age	34 (21.1)	58 (17.4)	33 (21.4)	59 (16.9)	41 (15.4)	56 (16.2)
Ethnicity (=1 if white)	0.92	0.96	0.94	0.90	0.88	0.94
Regions:						
North East	0.74	0.26	0.70	0.30	0.64	0.36
North West	0.78	0.22	0.75	0.25	0.69	0.31
Yorkshire % Humber	0.77	0.23	0.74	0.26	0.68	0.32
East Midlands	0.78	0.22	0.73	0.27	0.67	0.33
West Midlands	0.78	0.22	0.72	0.28	0.66	0.35
East of England	0.78	0.22	0.73	0.27	0.67	0.33
London	0.79	0.21	0.76	0.24	0.74	0.26
South West	0.77	0.23	0.72	0.28	0.67	0.33
South East	0.77	0.23	0.73	0.27	0.70	0.30
Social class:	(10,904)	(4,254)	(9,342)	(4,760)	(7,951)	(3,830)
Manual worker	0.73	0.27	0.68	0.32	0.69	0.31
Economic status:	(11,446)	(4,419)	(9,833)	(4930)	(8,550)	(3,947)
Employed	0.82	0.18	0.77	0.23	0.76	0.14
Unemployed	0.88	0.12	0.83	0.17	0.84	0.16
Retired	0.46	0.54	0.39	0.61	0.38	0.62
Other economic inactive	0.69	0.31	0.65	0.35	0.69	0.31
Income quintiles:	(13,313)	(3,748)	(11,782)	(4,119)	(6,987)	(3204)
1^{st} quintile	0.25	0.30	0.15	0.17	0.13	0.17
2^{nd} quintile	0.19	0.24	0.19	0.27	0.15	0.23
3^{rd} quintile	0.21	0.20	0.19	0.18	0.18	0.17
4^{th} quintile	0.19	0.14	0.22	0.18	0.23	0.19
5^{th} quintile	0.16	0.12	0.25	0.20	0.31	0.23

Note: S.D. when continuous variable and number of observations in parenthesis when dummy variables. The figures for regions, social class and economic status are row percentages. The figures for female, ethnicity and income quintiles are column percentages.

advice. The average income of those who receive smoking cessation advice is

Table 2: Smoking cessation advice to patients with CVD who have ever smoked

(a) With smoking cessation advice			
	1998	**2003**	**2006**
Sample size	0.33 (917)	0.31 (917)	0.33 (758)
Age	56 (16.0)	57 (15.5)	55 (15.3)
Female	0.47 (917)	0.45 (917)	0.46 (758)
HH income	16,277 (16244)	20,474 (19937)	23,326 (25957)
(b) Without smoking cessation advice			
	1998	**2003**	**2006**
Sample size	0.67 (1,892)	0.69 (2,068)	0.67 (1,517)
Age	60 (16.8)	61 (16.5)	55 (15.3)
Female	0.49 (1,892)	0.50 (2,068)	0.50 (1,517)
HH income	18,658 (16424)	24,365 (23922)	27,311 (26337)

Note: Number of observations in parenthesis if dummy and S.D. if continuous variable.

lower than the one without advice.

The HSE also contains information on medicines prescribed to people with a cardiovascular disease. Figure (1) shows the proportions of statins prescriptions for men and women in the waves of the HSE data between 1991 and 2006[9].

According to Figure (1) the prescription rates for people with CVD is higher in men than it is in women. Whilst the prescription rates in 1991-1994 for female and male are similar, the gap in statins prescriptions between men and women widens in 2003-2006. The rates of statins prescriptions increase over time. In line with National Guidelines, the rates of statin prescriptions increase to more than 40 percent in 2006.

4.4. Measures of self-care in the HSE

As outlined in section (3), people can reduce the size of income loss should illness occur by adopting healthier lifestyles. We focus on particular measures of self-care available in the HSE that are consistent across waves. More specifically, Table (3) reports measures of healthy and unhealthy behaviour.

We find that approximately 55 percent of CVD patients who drink low-fat milk are female. We also find that more than 50 percent of smokers who

[9]In figure (1) we have considered all the HSE years where the definition of CVD is available regardless of the boost sample. We take the average of the proportion of CVD patients (whether female or male) in the defined years intervals.

have CVD are women[10]. Patients with CVD who quit smoking tend to be older than those who do not quit. But CVD patients that drink low-fat milk are

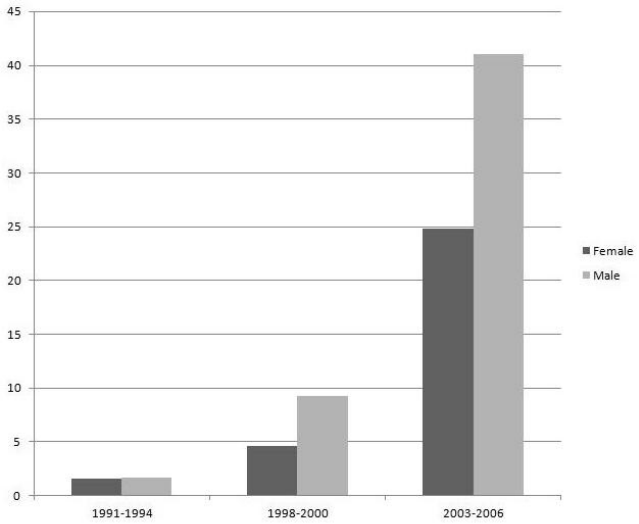

Figure 1: Trends in statins prescriptions

younger.

4.5. The relation between State-protection and self-care in the HSE

Do NHS interventions (i.e. State-protection) increase the probability of quitting smoking (i.e. self-care)?

We observed in Figure (1) that the prescription of statins has increased over time. In this section we want to analyse whether the increased medical treatment (proxied by the prescription of statins) corresponds to an increase in self-care (proxied by smoking cessation) for those with diagnosed CVD.

Table (4) shows the proportions of ex-smokers who have or have not been prescribed with statins[11]. The Table suggests that CVD patients on statins are much more likely to have quit smoking.

[10]In Table (3) we compare ex-smokers with current smokers after taking never smokers out of the sample.

[11]Here we consider the same boost samples used for the empirical analysis.

Table 3: Sample characteristics of self-care measures for patients with CVD

(a) Measures of healthy behaviour			
	1998	**2003**	**2006**
Quit Smoking:			
Sample size	0.65 (1,834)	0.64 (1,740)	0.62 (1,295)
Age	63 (15.0)	64 (13.9)	61 (14.7)
Female	0.45 (1,834)	0.43 (1,740)	0.45 (1,295)
HH income	18,384 (16346)	22,783 (21683)	26,396 (24839)
Low-fat milk:			
Sample size	0.75 (3,142)	0.82 (2,956)	0.86 (2,349)
Age	57 (16.8)	58 (16.1)	57 (15.4)
Female	0.55 (3,142)	0.55 (2,956)	0.54 (2,349)
HH income	20,477 (19,139)	25,178 (23083)	27,739 (25386)
(b) Measures of unhealthy behaviour			
	1998	**2003**	**2006**
Smoking:			
Sample size	0.35 (986)	0.36 (964)	0.38 (799)
Age	50 (17.0)	51 (16.7)	49 (15.2)
Female	0.54 (986)	0.55 (964)	0.52 (799)
HH income	16,909 (16402)	22,483 (24273)	23,550 (25029)
Full-fat milk:			
Sample size	0.25 (1,073)	0.18 (638)	0.14 (398)
Age	61 (18.9)	60 (18.6)	53 (18.2)
Female	0.52 (1,073)	0.46 (638)	0.46(398)
HH income	14,405 (14402)	20,480 (24556)	21,358 (25536)

Note: Number of observations in parenthesis if dummy and S.D. if continuous variable.

Table 4: Percentages of respondents with CVD with and without statins that have quit smoking

	Ex-smokers with statins	**Ex-smokers without statins**	**Difference**
1993	73	69	4
1994	84	61	23
1998	78	70	8
2003	78	67	11
2005	82	81	1
2006	72	62	10

5. The empirical model and results

We start our analysis with univariate pooled and cohort panel models in the waves of data between 1993 and 2006 of the HSE. More formally, we estimate the probability that an individual with CVD quits smoking ($q_{it} = 1$) as follows:

$$q_{it}^{*} = \beta X_{it} + \gamma s_{it} + \epsilon_{it} \tag{16}$$

where we (the researchers) can only observe $q_{it} = 1.[q^*_{it} > 0]$ depending on q^*_{it}, the latent propensity to quit smoking of individual i at time t. X_{it} is a vector of demographic and socioeconomic characteristics; s_{it} is a measure of State-protection proxied by the prescription of statins and ϵ_{it} is a random error assumed to be normally distributed.

More specifically, the vector X_{it} contains the following variables. Demographic characteristics include age and its squared value, gender and its interaction with age, number of children, marital status, ethnicity and an indicator of formal qualification. Socioeconomic determinants are denoted by the availability of a car, the number of bedrooms in the house, social class, employment status (i.e. proxied by a dummy indicating whether the individual ith is employed or not) and income. The value of income has been equivalised by household size and deflated by using the consumer price index (UK National Statistics). We also include regional and year dummies to control for macro policy variations across years and regional differences within England.

Results are reported in Table (5). Four models have been estimated. The first model pools the 1993-2006 HSE data. In the second model we include socioeconomic indicators in the years 1998-2006 of data where this information is available. The third model adds regional dummies. The final model is a pseudo-panel estimation including 12 birth cohort dummies disaggregated in five year bands (with the birth cohort born in 1933-1940 being the reference group) to allow for generational effects [Deaton (1997); Wooldridge (2001)].

We find no statistically significant evidence of crowding out of self-care. Table (5) shows that those who have been prescribed statins are between three and four percent more likely to quit cigarettes smoking. This result becomes more significant once socioeconomic characteristics, regional dummies and birth cohorts are included in the model[12].

Considering the vector of demographic characteristics, we find that those with CVD who are not single are ten percent more likely to stop smoking cigarettes in model IV. There is also evidence of a very significant and positive effect of education on the quitting smoking behaviour.

[12]We have also used other measures of self-care. We found a positive association between drinking low-fat milk and the prescription of statins. No statistically significant effect has been found on physical activity. The results whilst not displayed in this paper are available from the authors on request.

Table 5: Maximum likelihood estimates of the probability of quitting smoking for people with CVD

	Model I	Model II	Model III	Model IV
Statins†	0.03*	0.03*	0.04**	0.04**
	(0.02)	(0.02)	(0.02)	(0.02)
Age	0.00	-0.00	-0.00	0.00
	(0.00)	(0.00)	(0.00)	(0.01)
Age squared	0.00	0.00	0.00	0.00
	(0.00)**	(0.00)***	(0.00)***	(0.00)
Children	-0.03	-0.03	-0.03	-0.03**
	(0.01)**	(0.01)**	(0.01)**	(0.01)
Female†	-0.11	-0.12	-0.11	-0.11
	(0.07)	(0.07)	(0.07)	(0.07)
Female*Age	0.11	0.12	0.12	0.12
	(0.11)	(0.11)	(0.11)	(0.11)
White†	0.05	0.04	0.04	0.04
	(0.05)	(0.05)	(0.05)	(0.05)
Non single†	0.14***	0.10***	0.10***	0.10***
	(0.02)	(0.02)	(0.02)	(0.02)
Qualification†	0.10***	0.05***	0.05***	0.05***
	(0.02)	(0.02)	(0.02)	(0.02)
Car†	-	0.07***	0.06***	0.06***
		(0.02)	(0.02)	(0.02)
Bedrooms	-	0.02***	0.03***	0.03***
		(0.01)	(0.01)	(0.01)
Non manual†	-	0.03	0.03	0.03
		(0.02)*	(0.02)	(0.02)
Employed†	-	0.04*	0.04*	0.04*
		(0.02)	(0.02)	(0.02)
Log(equivalent income)	-	0.04***	0.03**	0.03***
		(0.01)	(0.01)	(0.01)
North East†	-	-	-0.05	-0.04
			(0.04)	(0.04)
North West†	-	-	-0.11***	-0.10***
			(0.03)	(0.03)
Yorshire & Humber†	-	-	-0.08**	-0.08**
			(0.03)	(0.03)
East Midlands†	-	-	-0.03	-0.03
			(0.03)	(0.03)
West Midlands†	-	-	-0.07**	-0.07**
			(0.04)	(0.04)
East of England†	-	-	-0.00	0.00
			(0.03)	(0.03)
London†	-	-	-0.04	-0.03
			(0.04)	(0.04)
South West†	-	-	0.01	0.01
			(0.03)	(0.03)
Years†	yes	yes	yes	yes
Birth Cohorts†	-	-	-	yes
N. observations	3971	3971	3971	3971
Log-likelihood	-2073.40	-2039.80	-2026.17	-2020.70

Marginal effects displayed and robust standard errors in ().†Dummy variables
$^{***}p < 0.01, ^{**}p < 0.05, ^{*}p < 0.1$.

Turning to the economic indicators, we find that wealthier individuals with CVD are less likely to keep smoking. For instance, having at least one car is associated with a higher likelihood of quitting smoking (the effect is between six and seven percentage points).

Models III and IV include regional dummies with South East as reference group. We find some evidence of regional differences in smoking behaviour. For instance, respondents in the North West, Yorkshire and Humber, and West Midlands regions are less likely to quit smoking compared to the South East.

Although we find a significantly positive relation between prescription of statins and quitting smoking behaviour, we should be cautious in making causal inference on the effect of NHS health interventions. There are at least three issues that should be considered. Firstly, the NHS can affect individuals' healthy choices directly, by providing more advice on healthy behaviour; and indirectly, by prescribing new pharmaceuticals (i.e. statins). In order to argue that there is indeed an indirect effect that is residual to the direct health advice provided by the NHS, we need to control for smoking cessation advice in our specification. The last two issues are related to the endogeneity problem. As the HSE is a repeated cross-section there is an issue of reverse causality between the prescription of statins and the decision to quit smoking. Finally and more importantly, whilst we (the researchers) cannot really observe the underlying individual propensity to stop smoking, doctors can acquire this information by frequent contacts with patients. They can thus tailor their decision to prescribe medicines according to their evaluation of the marginal benefit from the treatment.

To conclude, there are potential sources of endogeneity (either omitted variables or reverse causality) that need to be addressed in order to claim a causal relation between State-protection/insurance and self-insurance. The following subsections will examine each of these issues in turn.

5.1. State-protection/insurance and self-insurance: direct or indirect effect?

Can we argue that the prescription of statins has an effect on individuals' healthy behaviour independent of the effectiveness of information provided by doctors? In other words, do people with CVD substitute or complement self-care with medical treatment regardless of the information they are given on health lifestyles?

As our measure of State-protection, prescription of statins, can proxy contacts with the doctor in a broader sense, we need to disentangle the indirect

effect of statins from the direct effect of advice on health behaviour. Jones (1994) identified some special features of smoking such as health knowledge, addiction and social interaction. He used smoking cessation advice as a measure of health knowledge. However, he also pointed out the issue of response bias and simultaneity. First, medical advice can only be given to those who go to the doctor. Second, unobservable heterogeneity may bias the estimation because doctors may select those who, in their opinion, could benefit more from the advice.

The HSE provides data on whether the patient has been given smoking cessation advice by a doctor. It also contains information on the time when the individual stopped smoking and received smoking cessation advice from a medical practitioner. We can thus resolve reverse causality by defining the dependent variable as quitting smoking in the past 12 months and smoking cessation advice as a dummy variable that takes value one if advice has been given more than a year ago.

Table (6) displays the results of the cohort panel probit model of the quitting smoking behaviour. We include two measures of State-protection/insurance separately (i.e. prescription of statins and smoking cessation advice). The sample size is smaller because of the change in definition of the dependent variable[13].

We find that, whilst smoking cessation is not significant, there still is a significantly positive association between prescription of statins *per se* and quitting smoking behaviour. In particular, the prescription of statins increases the probability of quitting smoking in the last year by seven percentage points regardless of smoking cessation advice.

5.2. State-protection/insurance and self-insurance: reverse causality?

The second issue is the one of reverse causality. As the data are not longitudinal, the results shown in Table (5) cannot determine whether the decision to quit smoking has been taken after the diagnosis of CVD. Neither can we determine whether the doctor requires the patient to give up smoking before they are prescribed statins.

The HSE provides recall questions on the age of CVD diagnosis and the time when the individual stopped smoking[14]. The sample size is smaller be-

[13]See Table (B.9) in Appendix B where the sample sizes of some key variables across the HSE years are shown.

[14]For each of the CVD conditions - namely, heart murmur, diabetes, blood pressure,

Table 6: Maximum likelihood estimates of the probability of quitting smoking (including smoking advice before quitting smoking)

	Cardiovascular Disease
Statins†	0.07**
	(0.04)
Smoking cessation	-0.04
advice†	(0.02)
Age	-0.00
	(0.01)
Age squared	0.00
	(0.00)
Children	-0.03
	(0.02)
Female†	-0.12
	(0.10)
Female*Age	0.21
	(0.15)
White†	-0.04
	(0.08)
Non single†	0.03
	(0.03)
Qualification†	0.03
	(0.03)
Car†	0.01
	(0.03)
Bedrooms	-0.01
	(0.01)
Non manual†	-0.05**
	(0.02)
Employed†	0.02
	(0.03)
Log(equivalent	0.04**
income)	(0.02)
North East†	0.00
	(0.05)
North West†	-0.06
	(0.03)
Yorshire &	-0.00
Humber†	(0.05)
East	0.02
Midlands†	(0.05)
West	-0.03
Midlands†	(0.04)
East of	0.03
England†	(0.05)
London†	0.02
	(0.05)
South West†	0.00
	(0.05)
Years†	yes
Birth Cohorts†	yes
N. observations	786
Log-likelihood	-270.88

Marginal effects displayed and robust standard errors in ().†Dummy variables. $^{***}p < 0.01,^{**}p < 0.05,^{*}p < 0.1$.

cause of the change in definition of the dependent variable[15].

Equation (16) is estimated for the probability of quitting smoking after a cardiovascular disease has been diagnosed. The dependent variable, q_{it}, is equal to one whenever quitting occurs after the CVD diagnosis. Results are reported in Table (7).

After correcting for the timing of events, we still find a positive association between quitting smoking and the prescription of statins (the effect is significant at the five percent level). The magnitude of the effect is the same as the one in Table (6) We also find a very significant and positive relation between education and quitting smoking.

5.3. State-protection/insurance and self-insurance: omitted variables?

The results presented in the previous subsections provide evidence of a positive association between prescription of statins and quitting smoking behaviour (even after disentangling the indirect effect of medical treatment from the direct effect of advice and after resolving reverse causality). There is, however, a potential bias in these estimates due to the fact that doctors, who can observe the underlying individual propensity to quit smoking, may actually select the "best" patients as the ones who are thought to have greater benefits from the medical treatment.

We address this potential endogeneity by estimating the relation between prescription of statins and quitting smoking behaviour with a simultaneous bivariate probit model [Amemiya (1975); Greene (1997); Maddala and Lee (1976); Maddala (1983)]. Maddala (1983) defines this recursive, simultaneous equation with endogenous dummy as follows:

$$q_{it}^{*} = \beta X_{it} + \gamma s_{it} + \epsilon_{1it} \tag{17}$$

$$s_{it}^{*} = \beta_1 x_{1it} + \delta Z_{it} + \epsilon_{2it} \tag{18}$$

where the error terms, $(\epsilon_{1it}, \epsilon_{2it})$ are identically and independently distributed $(i.i.d.)$ with a bivariate normal distribution with zero mean and variance

angina, heart attack, irregular heart, stroke and other heart condition - the HSE contains a question on the age at diagnosis. We combine this information together with the age of the respondent and the time of quitting smoking to determine whether the decision of stopping smoking has been taken after the diagnosis.

[15]See Table (B.9) in Appendix B where the sample sizes of some key variables across the HSE years are shown.

Table 7: Maximum likelihood estimates of the probability of quitting smoking (after diagnosed CVD)

	Smoking cessation
Statins†	0.07**
	(0.03)
Age	0.00
	(0.01)
Age squared	0.00
	(0.00)
Children	-0.04*
	(0.02)
Female†	-0.12
	(0.11)
Female*Age	0.16
	(0.18)
White†	0.01
	(0.07)
Non single†	0.09***
	(0.03)
Qualification†	0.09***
	(0.03)
Car†	0.06**
	(0.03)
Bedrooms	0.01
	(0.01)
Non manual†	0.01
	(0.03)
Employed†	-0.02
	(0.04)
Log(equivalent income)	0.05**
	(0.02)
North East†	0.09
	(0.05)
North West†	-0.13***
	(0.04)
Yorshire & Humber†	-0.09*
	(0.05)
East Midlands†	0.00
	(0.05)
West Midlands†	-0.09*
	(0.05)
East of England†	0.01
	(0.05)
London†	-0.00
	(0.06)
South West†	0.03
	(0.05)
Years†	yes
Birth Cohorts†	yes
N. observations	1,700
Log-likelihood	-1010.69

Marginal effects displayed and robust standard errors in ().†Dummy variables. $^{***}p < 0.01,^{**}p < 0.05,^{*}p < 0.1$.

equal to one and with correlation ρ. q^*_{it} is the unknown propensity to stop smoking and s^*_{it} is the individual propensity to be prescribed with statins by doctors. The vector of socioeconomic characteristics, X_{it}, contains the same variables as in equation (16). In equation (18) we consider a partition of such vector, namely x_{1it}, that only considers demographic characteristics. We also include a vector of exogenous variables, Z_{it}.

Maddala (1983) argues that, unless equations (17-18) denote an intrinsically sequential system, no identification is possible without appropriate restrictions in one of the two equations (i.e. at least one explanatory variable should be contained in (18) and not in (17)). Greene (1997) and Wilde (2000) point out that the endogenous nature of the dummy variable in the first equation can be ignored in formulating the log-likelihood. The only problem in estimating the recursive, simultaneous system of equations - Wilde (2000) argues - is the availability of enough variation in the data. However, this condition can only be ensured by the (quite strong) assumption that each equation contains at least one varying exogenous regressor.

As we are concerned with potential biases caused by the doctors' decision to prescribe statins, we force equation (18) to determine the "objective" factors that should be taken into account when medical treatment is provided.

We resolve endogeneity by estimating a "statins equation" that includes the risk factors (as the ones defined by national guidelines) that doctors should take into account when deciding to prescribe statins, regardless of the latent patient's propensity (observed by the doctor, but unknown to us) to comply with the recommendations. Therefore we partition the vector X_{it} with demographic characteristics and exclude socioeconomic factors. In addition, we include a vector, Z_{it}, of risk factors such as longstanding illnesses (i.e. diabetes, other endocrine problems, stroke, heart attack/angina, other heart and kidney problems) and levels of cholesterol (i.e. high, low or unmeasured).

We estimate equation (18) on exactly the same sample of patients with cardiovascular diseases as in Table (5).

Table (8) reports the results. Interestingly, we find that there is a negative, albeit weak, correlation between the disturbances of the statins equation and the quitting smoking equation (i.e. the parameter ρ). The Wald test weakly rejects the hypothesis of no correlation between the two equations. This result can be interpreted as follows. Whilst the underlying unobserved propensity to quit smoking is negatively associated with the propensity of being prescribed with statins (and vice versa), once we control for the risk

Table 8: Recursive simultaneous equation model with an endogenous dummy

	(1) Quit smoking	(2) Statins
Statins†	0.09***	-
Age	0.004	0.01
Age squared	0.00004	-0.0001
Children	-0.03**	0.01
Female†	-0.10	-0.29***
Female*Age	0.11	0.40***
White†	0.04	-0.02
Non single†	0.10***	0.01
Qualification†	0.05***	-
Car†	0.06***	-
Bedrooms	0.03***	-
Non manual†	0.03	-
Employed†	0.04*	-
Log(equivalent income)	0.03***	-
North East†	-0.04	-0.01
North West†	-0.10***	0.01
Yorshire & Humber†	-0.08**	0.03
East Midlands†	-0.02	-0.01
West Midlands†	-0.07**	0.03
East of England†	0.003	-0.02
London†	-0.04	0.02
South West†	0.01	-0.0004
High cholesterol†	-	0.11***
Not measured cholesterol†	-	-0.23***
Low cholesterol†	-	0.08**
Diabetes†	-	0.12***
Other endocrine probl.†	-	0.13***
Stroke†	-	0.14***
Heart attack/angina†	-	0.25***
Other heart problems†	-	0.15***
Kidney problems†	-	0.16***
Years†	yes	yes
Birth Cohorts†	yes	yes
N. observations	3971	
Log-likelihood	-3593.15	
ρ	-0.15	$z = 0.08^*$
Wald test $\rho = 0$	$\chi^2 = 3.47$	$p = 0.063^*$

Marginal effects displayed.There is a direct marginal effect produced by explanatory variables in the quitting smoking equation and an indirect effect if the same regressor also appears in the statins equation. Therefore, the marginal effect in the quitting smoking equation is the sum of direct and/or indirect effects depending on the regressor being in the statins equation or not.
†Dummy variables.$^{***}p < 0.01,^{**}p < 0.05,^{*}p < 0.1$.

factors that should determine doctors' prescription decision, we find a positive effect of the statins dummy. In other words, this result can be taken as evidence that doctors compensate for patients' lack of effort in undertaking healthier lifestyles with medical treatment.

With regard to socioeconomic factors, we support the previous results showing that educated and wealthier people (i.e. who own at least a car, have higher income or a bigger house) are more likely to quit smoking. Also, people who live in the North-West, Humber and Yorkshire or West Midlands are less likely to quit smoking.

Turning to the statins equation, in line with the guidelines we find that high cholesterol increases the chances to be prescribed with statins by eleven percentage points[16]. Those who have not had their cholesterol measured are less likely to be prescribed with statins. Other risk factors such as longstanding diseases have a very significant and positive effect on the prescription of statins. For instance, patients who have had heart attack/angina are 25 percent more likely to be prescribed with statins.

Considering demographic factors, we find that whilst women are significantly less likely to be prescribed with statins, prescription rates increase with age.

6. Conclusions

This paper examines the relation between NHS interventions and the individuals' effort to undertake healthy lifestyles. Does the provision of State-protection/insurance substitute for individuals' choices on healthy lifestyles?

We have address this question with both a theoretical and an empirical model. We develop the insurance contracts framework provided by Ehrlich and Becker (1972) and Peltzman (1975) under the following two scenarios. First, the State-provided NHS can only reduce the probability that ill-states occur - so-called State-protection. Second, in addition to the State-protection role, the NHS can effectively act as insurer by reducing the size of income loss should an ill-state occur - so-called State-insurance. We show that higher levels of protection/insurance by the State (either through the increased avail-

[16]We also find that low cholesterol has a positive (although significant only at the five percent level) impact on the prescription of statins. This result could be affected by reverse causality that cannot be resolved as we do not have information on the timing of cholesterol measurements.

ability or effectiveness of medical care) can crowd out or reinforce self-care.

We then apply this model to the case of individuals in waves of the Health Survey for England between 1993 and 2006 with diagnosed cardiovascular diseases who must decide whether to quit smoking. There has been a considerable increase in the proportion of these individuals who receive prescriptions of statins from the State, a highly effective drug that reduces the probability of further heart attacks and premature death. Simple univariate probit models show a positive association between prescription of statins and quitting smoking, the effect ranges between three and four percentage points amongst those with CVD.

Several issues impeding causal inference on the results are then examined. First, the paper disentangles the direct effect of smoking cessation advice from the indirect effect of the prescription of statins. We find that the effect of statins on the decision of stopping smoking is still positive and significant regardless of the advice given. Second, we show that the results are robust to adjustments of the timing between quitting smoking and CVD diagnosis. Finally, we deal with the potential bias caused by the fact that doctors, who can observe the underlying individual propensity to quit smoking, may actually select patients who can benefit more from treatment. We estimate a recursive, simultaneous equation model with an endogenous dummy variable. Identification is resolved by including, in the statins equation, risk factors that according to national guidelines should affect doctors' decision to prescribe. We find that whilst patients' willingness to quit smoking makes them less prone to be prescribed with statins (and vice versa), the prescription of statins exclusively based on observed risk factors increases the chances to quit smoking. This is evidence that doctors compensate for the patient's lack of effort in undertaking healthier behaviour with increased medical treatment.

Appendix A. Derivation of the theoretical model

We can determine the effect of a change in the parameter α from the first order optimality conditions (6a-6b) and (13a-13b) as follows:

$$\frac{\partial^2 EI^*}{\partial m^2}\frac{\partial m}{\partial \alpha} + \frac{\partial EI^*}{\partial m \partial s}\frac{\partial s}{\partial \alpha} + \frac{\partial EI^*}{\partial m \partial \alpha} = 0$$
$$\frac{\partial EI^*}{\partial s \partial m}\frac{\partial s}{\partial \alpha} + \frac{\partial^2 EI^*}{\partial s^2}\frac{\partial s}{\partial \alpha} + \frac{\partial EI^*}{\partial s \partial \alpha} = 0$$

By the Cramer's rule we can write:

$$\begin{bmatrix} EI^*_{mm} & EI^*_{ms} \\ EI^*_{sm} & EI^*_{ss} \end{bmatrix} \begin{bmatrix} \frac{\partial m}{\partial \alpha} \\ \frac{\partial s}{\partial \alpha} \end{bmatrix} = \begin{bmatrix} -EI_{m\alpha} \\ -EI_{s\alpha} \end{bmatrix}$$

$$\frac{\partial m}{\partial \alpha} = \frac{\begin{vmatrix} -EI_{m\alpha} & EI^*_{ms} \\ -EI_{s\alpha} & EI^*_{ss} \end{vmatrix}}{|\Sigma|} = \frac{-EI_{m\alpha}EI^*_{ss} + EI^*_{ms}EI_{s\alpha}}{|\Sigma|} \tag{A.1}$$

and:

$$\frac{\partial s}{\partial \alpha} = \frac{\begin{vmatrix} -EI^*_{mm} & EI_{m\alpha} \\ -EI^*_{sm} & EI_{s\alpha} \end{vmatrix}}{|\Sigma|} = \frac{-EI^*_{mm}EI_{s\alpha} + EI_{m\alpha}EI^*_{sm}}{|\Sigma|} \tag{A.2}$$

where the determinant of the Hessian matrix:

$$|\Sigma| = \begin{bmatrix} EI^*_{mm} & EI^*_{ms} \\ EI^*_{sm} & EI^*_{ss} \end{bmatrix} = EI^*_{mm}EI^*_{ss} - EI_{ms}EI_{sm}$$
$$= EI^*_{mm}EI^*_{ss} - EI^2_{ms} > 0$$

by the necessary and sufficient second-order conditions for a maximum.

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From the first order conditions (13a-13b), we can derive the following

equations:

$$EI_{s\alpha} = -\pi_{\alpha}(\pi^e, \alpha, m)L_s(s, \alpha, m) - \\ - \pi(\pi^e, \alpha, m)L_{s\alpha}(s, \alpha, m) < 0 \quad \text{iff} \quad L_{s\alpha} > 0 \quad \text{(A.3a)}$$

and:

$$EI_{sm} = -\pi_{m}(\pi^e, \alpha, m)L_s(s, \alpha, m) - \\ - \pi(\pi^e, \alpha, m)L_{sm}(s, \alpha, m) < 0 \quad \text{iff} \quad L_{sm} > 0 \quad \text{(A.3b)}$$

where, by assumption, $L_s(s, \alpha, m)$ and $\pi_i(\pi^e, \alpha, m) < 0 \quad \forall \quad i = m, \alpha$. And:

$$EI_{m\alpha} = -\pi_{m\alpha}(\pi^e, \alpha, m)L(s, \alpha, m) - \\ - \pi_m(\pi^e, \alpha, m)L_{\alpha}(s, \alpha, m) - \pi_{\alpha}(\pi^e, \alpha, m)L_m(s, \alpha, m) - \\ - \pi(\pi^e, \alpha, m)L_{m\alpha}(s, \alpha, m) \quad \text{(A.3c)}$$

Note that $EI_{m\alpha} > 0$ if $-\pi_{m\alpha}L - \pi L_{m\alpha} > \pi_m L_{\alpha} - \pi_{\alpha} L_m$ given $\pi_{m\alpha}$ and $L_{m\alpha} < 0$.

Appendix B. Sample sizes for the robustness checks

In the first three columns of Table (B.9) we display the dependent variables of the empirical analyses shown in the paper for CVD patients. The first column contains the dependent variable "quitting smoking" used in the first specification of the empirical models where the largest sample is available. The difference in sample sizes between the first three columns depends on the fact that the dependent variables of the robustness checks constitute a subset of the dependent variable in the first column and on the fact that information on the timing of events has to be available.

Table B.9: Sample sizes for the robustness checks

	Quitting smoking	**Quitting smoking after CVD diagnosis**	**Quitting smoking in last year**	**Smoking cessation advice> 1 year**
1993	2,562	1,505	1,118	2,467
1994	2,165	827	839	2,263
1998	2,820	1,453	1,093	2,503
2003	2,704	1,485	1,102	2,682
2005	1,469	128	326	1,555
2006	2,094	1,175	918	2,003

The last column of Table (B.9) reports the sample size of the regressor smoking cessation advice given before the patient actually stopped smoking.

References

Acheson D. Independent inquiry into inequalities in health. Department of Health Report, 1998. Available at http://www.dh.gov.uk/en/Publicationsandstatistics/Publications/PublicationsPolicyAndGuidance/DH_4097582.

Acton J.P. Demand for health care when time prices vary more than money prices. Rand Corporation: R-1189-OEO/NYC, New York City Rand Institute (New York); 1973.

Amemiya T. Qualitative models. Annals of Economic and Social Measurement 1975; 4; 363-372.

Arrow K. Alternative approaches to the theory of choice in risk-taking situations. Econometrica 1951; 19; 404-437.

Balia S, Jones AM. Mortality, lifestyle and socio-economic status. Journal of Health Economics 2008; 27; 1-26.

Becker G.S. A theory of the allocation of time. Economic Journal 1965; 75 (299); 493-517.

Chang F.R., Trivedi P.K. Economics of self-medication: theory and evidence. Health Economics 2009; 12; 721-739.

Contoyannis P, Jones AM. Socioeconomic status, health and lifestyle. Journal of Health Economics 2004; 23(5); 965-995.

Courbage C. and de Coulon A. Prevention and private health insurance in the U.K. The Geneva Papers on Risk and Insurance 2004; 29(4); 719727.

Deaton A. The Analysis of Household Surveys: A Microeconometric Approach to Development Policy. World Bank Publications; 1997.

Debreu G. Theory of value: an axiomatic analysis of economic equilibrium, Wiley, London; 1959.

Department of Health. The National Service Framework for Coronary Heart Disease: Winning the War on Heart Disease. London, Progress Report 2004. Available at http://www.dh.gov.uk/en/Publicationsandstatistics/Publications/PublicationsPolicyAndGuidance/DH_4077154.

Department of Health. The NHS Plan: a plan for investment, a plan for reform. London, 2000. Available at http://webarchive.nationalarchives.gov.uk/+/www.dh.gov.uk/en/publicationsandstatistics/publications/publicationspolicyandguidance/dh_4002960.

Saving lives: Our healthier Nation. London, 1999 Available at http://webarchive.nationalarchives.gov.uk/+/www.dh.gov.uk/en/Publicationsandstatistics/Publications/PublicationsPolicyAndGuidance/DH_4118614.

de Preux L. B. Ex ante moral hazard and anticipatory behaviour: some evidence. HEDG Working Paper 10/13.

Ehrlich I., Becker G.S. Market insurance, self-insurance, and self-protection. The Journal of Political Economy 1972; 80 (4); 623-648.

Gill P. S. , Quirke T. P., Mant J. W. and Allan T. F. The use of lipid-lowering drugs across ethnic groups in the secondary prevention of ischaemic heart disease: analysis of cross-sectional surveys in England. British Journal of General Practice 2004; 54; 442-443.

Goddard M. and Smith P. Equity of access to health care services: Theory and evidence from the UK. Social Science and Medicine 2001; 53; 1149-1162.

Greene W. Econometric Analysis; Prentice-Hall, London; 1997.

Grossman M. On the concept of health capital and the demand for health. The Journal of Political Economy 1972; 80; 223-255.

Hillman A. Health maintenance organisations, financial incentives and physician judgements (editorial). Annals of Internal Medicine 1990; 112; 891-893.

Hippisley-Cox J. and Coupland C. Effect of statins on the mortality of patients with ischaemic heart disease: population based cohort study with nested case-control analysis. Heart 2006; 92; 752-758.

Isles C. G. and Norrie J. Lipid lowering drugs for patients who continue to smoke? Heart 2000; 83; 619-620.

Jones A.M. Health, addiction, social interaction and the decision to quit smoking. Journal of Health Economics 1994; 13; 93-110.

Lalonde M. A new perspective on the health of Canadians. Minister of Supply and Services, Canada. Available at http://www.hc-sc.gc.ca/hcs-sss/alt_formats/hpb-dgps/pdf/pubs/1974-lalonde/lalonde-eng.pdf.

Ma J., Sehgal N. L., Ayanian J. Z., Stafford R. S. National trends in statin use by coronary heart disease risk category. PLoS Medicine 2005; 2(5); 434-440.

Maddala G. Recursive models with qualitative endogenous variables. Annals of Economic and Social Measurement, 1976; 5(4); 525-545.

Maddala G. and Lee L.F. Limited Dependent and Qualitative Variables in Econometrics. Cambridge University Press, New York; 1983.

Office for National Statistics. Key health statistics from general practice 1998. London: HMSO, 2000.

Peltzman S. The effects of automobile safety regulation. Journal of Political Economy 1975; 83 (4); 677-726.

Ramsay S. E., Morris R. W., Papacosta O., Lennon L. T., Thomas M. C. and Whincup P. H. Secondary prevention of coronary heart disease in older British men: extent of inequalities before and after implementation of the National Service Framework. Journal of Public Health 2005; 27(4); 338-343.

Reid F. D. A., Cook D. G. and Whincup P. H. Use of statins in the secondary prevention of coronary heart disease: is treatment equitable? Heart 2002; 88; 15-19.

Stanciole, A. Health insurance and life style choices: Identifying the ex ante moral hazard. IRISS Working Papers, (2007-10).

Tonstad S., Rosvold E. O., Furu K. and Skurtveit S. Undertreatment and overtreatment with statins: the Oslo Health Study 2000-2001. Journal of Internal Medicine 2004; 255; 494-502.

Wanless D. Securing good health for the whole population: Final report. Department of Health Report, 2004. Available at http://www.dh.gov.uk/en/Publicationsandstatistics/Publications/PublicationsPolicyAndGuidance/DH_4074426.

Wilde J. Identification of multiple equation probit models with endogenous dummy regressors. Economics Letters 2000; 69; 309312.

Wooldridge J.M. Econometric Analysis of Cross Section and Panel Data, MIT Press; 2002.

Zweifel P., Breyer F. and Kifmann M. Health economics, Second Edition, Springer; 2009.