Università di Torino

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On the Determinants of Human Health: An Economic Perspective

Cinzia Di Novi

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Ciclo XIX

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Acknowledgements: I wish to thank U. Colombino, S. Dalmazzone, M. Ferrero, G.Nicodano, T. Ota, G. Turati, the participants of the 2006 AIES annual conference in Venice, 2007 IIPF annual conference in Warwick and 2007 AIES annual conference in Florence. I also thank the audience of DSE workshops and two anonymous referees for helpful comments.

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INTRODUCTION

Since Arrow's (1963) seminal paper, interest in the economic aspect of health issues and in economists' opinion about how to deal with problems related to health and the health care sector has strongly grown. Arrow's paper announced the entry of health economics as a new discipline and became one of the most widely cited articles in the field of health economics. Today, health is considered one of the most valuable personal and universal rights:

> *[The enjoyment of the highest attainable standard of health is one of the fundamental rights of every human being without distinction of race, religion, political belief, economic or social condition.]*,

as stated in the preamble to the World Health Organization (WHO) Constitution; the determinants of health and health inequalities have became two issues of vital importance to health policy in establishing the extent to which the effect of various non-medical inputs such as income, education, social position, age, ethnicity, health-related behaviors and environmental quality contribute to modify the individual health status.

Grossman's (1972) contribution was the first formal economic model of the determinants of health and represented the first relevant theoretical and empirical work after Arrow's theoretical approach. Treating health as endogenous was a major difference between Grossman's model and the health models that preceded it. Drawing on the theory of human capital formulated by Becker (1965), Grossman constructed a model where individuals use medical care and their own time to produce health. Grossman interpreted a person's health as a capital stock that exogenously deteriorates at an increasing rate with age. To counteract this health deterioration, he assumed that individuals invest a

portion of their assets into health production each period. Hence, he did not consider individuals' health status as totally exogenous but as dependent, at least in part, on the resources allocated to its production.

In the last decades there has been increasing concerns about the possible adverse impact of pollution on human health. Public awareness on environmental health issues and their important economic repercussions has led to a pattern of substantial literature development on theoretical and empirical aspects of the economic valuation of environment-related health costs with the aim of promoting policies to improve environmental quality and human health.

The health production approach, first introduced by Grossman, has been successfully employed in the evaluation of health pollution related damages: subsequent contributions to Grossman's paper, by analyzing the decisions consumers make concerning the resources allocated to health production, tried to infer the value of health to the consumers and derived and estimated econometrically a measure of individual willingness to pay for a reduction in pollution (Gerking and Stanley, 1986; Cropper, 1981; Dixie and Gerking, 1991). An important contribution in this area was Cropper (1981). Cropper presented a simple model of preventive health care, similar to that of Grossman (1972). She takes changes in environmental conditions to influence the rate at which an individual's stock of health depreciates and used her model to define what a person would pay for a change in air quality.

The health production function approach is one of the several methodologies that environmental and health economists have developed to measure the value of pollution

health related damages. In the first chapter of our work, we critically review these methods and the research efforts that have been devoted to estimating pollution health related impacts. First, starting with Weitzman's (1974) seminal paper, we provide a short but comprehensive overview of the key literature on the choices faced by policy makers concerning price-based versus quantity-based instruments to regulate pollution and protect human health; then, we review the methods employed in estimating pollution abatement costs and pollution related health damages whose comparison (with reference to Weitzman's theoretical rule) form the basis for the choice among the price-based and quantity-based regulation instruments.

The aim of chapter 1 is to shed light on the issue concerning the difficulties that the analysts face in estimating the value of (marginal) pollution health damages or (marginal) benefits from reduced pollution. In fact, while measuring control costs seems relatively straightforward (market exists in principle in which pollution control equipment can be bought, and such equipment will reduce pollution by measurable levels), health damages (or benefits) are much harder to measure. First, analysts should accurately measure the health effects of pollution. Once they have determined those effects, they have to put a monetary value on them. However, valuing health is obviously controversial because each person may place a different value on it.

In 1977, Lave and Seskin published a pioneering work on the physical relationship between air pollution and health. Their analysis was conducted on aggregate data. The big problem with these data was that they did not allow to consider the influence of individual's specific choices (nutrition behavior, sport activities, smoking and alcohol habits,

sleeping, housing conditions etc.) and other several important factors. Moreover, the effects on health will also vary across individuals due to the so called "confounding factors": genetics, avoidance behavior, life-style, socioeconomic status. Hence, analysts have to calculate associated changes in health outcomes by taking into account that pollution could easily be correlated with other factors that may be just as influential. Based on this insight, in the second chapter when analyzing the relationship between air pollution and health, we control for socioeconomic variables and other important individual characteristics; in particular we control for life-style variables since individuals specific behavior represents another crucial determinant of the risk of illness. The analysis is focussed on how individual health habits and outdoor air quality combine to affect the likelihood of a good or bad health status, in a second-best world characterized by uncertainty on the level of health, where an individual may not able to avoid adverse health shocks completely. The framework is built on the basic concepts and ideas of the demand for health by Grossman (1972) and the subsequent contribution by Cropper (1981).

Following Grossman (1972) and Cropper (1981), we construct a model of health accumulation in which we assume that health depreciates at an increasing rate with age and ambient air pollution. The main differences with respect to Grossman and Cropper' models are that the level of health is uncertain and, for individuals who suffer or have suffered from a pathological condition, illness enters directly the rate of health depreciation too. As in Cropper's (1981) model, we assume that when pollution increases it becomes more costly to reduce the probability of suffering from health shocks. Individuals feel less healthy because they perceive health depreciation rate to be higher. Hence, they may

choose to invest less in their health and maintain lower health stock because of the higher net investment costs. In this sense, a higher pollution concentration may have two effects on health: a direct effect which consists of an increase of natural rate of depreciation, and an indirect effect, described by Cropper (1981), by which individuals will invest less in health and display a higher probability of suffering from health shocks. We will analyze this aspect in section 4 of this chapter focusing on the relationship between increasing pollution and health investment decisions. In addition we will examine whether chronic illnesses, by altering the rate to which health capital stock deteriorates, have any influence on individual health investment decisions too.

To estimate the health accumulation model and investigate the relation between health status, pollution, and health investment decisions, we use three different measures of overall health: dichotomous measures of blood pressure and functional limitations and disability are employed; moreover we take, as an indicator of health, a self-assessed health measure that is common in empirical research (Contoyannis and Jones, 2004, Balia and Jones, 2004 etc.). Since we have included life-style variable as regressors in the health equation a problem of simultaneity may arise. Hence we try to corrected the potential endogeneity of the behavioral variables by using a recursive multivariate probit model (Contoyannis and Jones, 2004; Blaylock and Blisard, 1992).

The model is estimated using data based on the 2001 Behavioral Risk Factor Surveillance System, which however does not measure environmental quality; environmental information at metropolitan area-level is available from EPA and can be used in conjunction with BRFSS data to compare measures of environmental quality and health. Data are

merged at the metropolitan area-level which is available in both the BRFSS and EPA data.

In chapter 2, we concentrate our attention on one of the main worldwide sources of air pollution: motor vehicle emissions. The most important standard concerning motor vehicles pollution is carbon monoxide. CO air concentration is generally high in areas with heavy traffic congestion therefore we consider carbon monoxide as a proxy for vehicle emissions (U.S., EPA 2000)

According to our results, a higher concentration of carbon monoxide has respectively a negative impact on the probability of enjoying good health and a positive influence on healthy habits. Then, concerning vehicular air pollution our results do not support Cropper's (1981) model: people living in polluted areas tend to invest more in health probably to counteract to the deterioration of a higher depreciation rate due to an increasing pollution. Arguably, people lead a healthy life-style to increase their health stock and build up resistance against pollution symptoms and future damages.

Grossman's model has become a cornerstone in the field of health economics. The model, however, is not undisputed. A key criticism has been that it fails to take into account the uncertainty on the future health status and the uncertainty on the returns from investments in health production. By investing in health, individuals do not determine with certainty their health status: environment and chance are two factors which may interfere. Grossman's model, however, did not account for uncertainty as it included neither explicit acknowledgment of uncertainty nor the description of illness, even though the fundamental relationship between health and uncertainty was established by economic theory (Arrow, 1963). Because of uncertainty, much of an individual's demand for health care is not

steady, but irregular and unpredictable. This implies that the costs of health care act as a random deduction from an individual's income. Therefore, under uncertainty, risk-averse individuals demand risk-bearing goods, such as health insurance, to safeguard their income against possible shocks (Cagatay, 2004).

It is well known, however, that health insurance coverage reducing an individual's marginal cost of medical care inputs, leads to use additional medical services: an insured individual, in fact, may consume more medical services and have a greater expenditure compared to an uninsured one (*moral hazard effect*, Leibowitz, 2004). Hence, health insurance biases health production decisions toward over-use of curative medical treatment at the expense of one's own preventive efforts.

Insurance choice itself may be affected by planned medical expenditure and expectations about medical care utilization (*adverse selection effect*). Chapter 3 focuses in particular on this last effect. In health insurance market, adverse selection may occur when consumers' true health-cost risk is private information: insurance companies may know that consumers vary in the level of risk, but, in principle, are not able to discern who are high and who are low risk profile individuals within a group of potential insured. (Akerlof, 1970; Rothschild and Stiglitz, 1976). Identifying risks accurately is not an easy task and requires that insurance company incurs some costs. Insured parties are heterogeneous in terms of expected costs and have more information about their risks. Naturally, high-risk individuals are not encouraged to "reveal" their risk to the insurance company; this asymmetry is a serious problem since may lead insurance company to face large differences in expected health costs due to heterogeneity in demographics and the incidence of illness.

In spite of the extensive theoretical interest on the adverse selection, there is little empirical evidence on the extent of the problem. The goal of the third chapter is to test empirically for adverse selection in the U.S. health insurance market. The test is based on the 2003/2004 Medical Expenditure Panel Survey – Household Component (MEPS-HC) data used in conjunction with the previous year's National Health Interview Survey (NHIS) data. The exercise is conducted by estimating the correlation between the completeness of insurance an individual buys and his ex-post risk experience, conditional on the observable characteristics which are used in pricing insurance policies.

Completeness of health insurance plan is measured by health insurance reimbursement that is the difference between total health expenditure and out-of-pocket expenditure on health care paid by consumers. Health insurance reimbursement, however, is only defined for a subset of individuals from the overall population since we observe it only for those who participate in insurance and face positive health care expenditure. Thus, the model may suffer from sample selection bias and straightforward regression analysis may lead to inconsistent parameters estimate. Another problem that arises from the estimation is the presence of unobserved heterogeneity in the equations of interest. In most of the studies which test for adverse selection two important estimation issues such as unobserved heterogeneity and selection bias, are traditionally treated separately. In our model, we control for selection bias and at the same time for unobserved heterogeneity issue by using Wooldridge (1995) two-step estimation procedure. We extend this estimation method to the case in which selectivity is due to two sources rather than one (participation in insurance and participation in health care expenditure).

We find no systematic relation between illness of individuals and insurance choice. We think that a possible explanation can be found in the so called "cream skimming" practise: health plans may have an incentive to alter their policy to attract the healthy and repeal the sick (Newhouse, 1996; Ellis, 1997). Then, insurers may practice a kind of "reverse adverse selection": they would try attempt to increase their profits by refusing to write policies for the worst risks in an insurance pool (see Siegelman, 2004). This strategic behavior can take a variety of forms including: designing insurance benefits packages in such a way as to be more attractive to healthy persons than unhealthy one for instance by excluding particular prescription drugs, offering numerous pediatrician (families with children are better risks) or by excluding cancer specialist visits. In such cases health plan may also refuse to sell an applicant insurance altogether. If health plans cream healthy individuals, those who are enrolled in health insurance are relatively healthy people and this lead to the failure of the correlation test.

Chapter 1

From Theory to Implementation of the Best Policy Instrument to Protect Human Health: a Brief Overview

1.1 Pollution Regulation to Protect Human Health

Human health problems related to environmental degradation and use of natural resources are potentially serious in many parts of the developed and developing world alike. These problems may have important economic repercussions: they may generate additional costs for patients and the social welfare system, they can lead to a loss of productivity and profits for firms and they often result in a loss of income for individuals. Then, im-

proving human health through measures addressed at preventing or reducing environmental pollution is a very important task for policy makers and government intervention through regulation is necessary to control pollution.

> *[...there are to many people damaged by most emissions of pollution for them to act as a single coordinated agent. Victims of pollution damages have different tastes, incomes, education so that they cannot agree how much to control pollution.* (This justifies) *government intervention in absence of which the market would fail to abate emissions...]* (Mendelsohn, 2002).

Government intervention to protect human health means regulations through standards or other mechanisms of pollution control. In the last decades, much of the debate on instruments for government regulation has centered on the use of market-based incentives (MBI) mechanisms as opposed to command and control approaches (CAC). In CAC regulatory mechanism, the regulator usually specifies a technology or emission standards with the aim of controlling the substances that can contribute to pollution (Ellerman, 2005). The problem with CAC mechanisms is that they do not abate pollution efficiently. The marginal cost of control is high for some firms and low for others, since a such as uniform regulations treat all firms the same way. Moreover, command and control approaches do not encourage polluters to do any better than the law demands (Gangadharan and Duke, 2001).

Among economists there is near unanimity in preferring market-based incentives since they encourage firms to abate pollution more efficiently. Among MBIs, the basic choice faced by policy makers concerns price-based versus quantity-based instruments, or in other

words, pollution taxes[1] and tradeable permits[2]. To establish a basis for comparison among these policy instruments, the traditional literature often relies on the following assumptions:

- the same amount of emissions from different sources have equal external costs;

- the literature ignores possible interactions with other markets;

- there is no uncertainty about the costs and the benefits of pollution control;

- a competitive structure prevails.

In this setting, it is easy to show that emission taxes and tradable permits are equivalent: the two approaches will lead to the same outcome that is the optimal level of emissions at minimum cost. In a world of perfect knowledge, marketable permits are in principle a fully equivalent alternative to unit taxes. Instead of setting the proper tax and obtaining the efficient quantity of emissions as a result, regulator could issue emissions permits. This symmetry between taxes and tradable permits, however, is critically dependent upon the assumption of perfect knowledge. In a setting of imperfect information concerning the marginal benefit and cost function, the outcomes under the two approaches can differ in important ways. When there is uncertainty either about the marginal benefits and the marginal costs the optimal level of emissions will typically not be achieved, and the goal of regulator than becomes to minimize efficiency losses (Cropper and Oates, 1992).

[1]The concept of pollution tax was developed by the British economist Arthur Pigou, in "The Economics of Welfare" (1920). The term pollution taxes otherwise known as externality taxes or Pigouvian taxes, by definition refers to a tax used to correct the misallocation of estimated damage.

[2]The term tradable permit was developed in the pioneering work of Dales (1968). Dales proposed a market of tradable permits as solution to pollution problems in which the government grants pollution rights, that should be tradable for a certain period , and in which government acts as broker for the trade monitoring the system.

The choice between quantity regulation and price regulation in terms of economic efficiency under imperfect information has been shown repeatedly in the pollution control literature. The classic article in this area is Weitzman (1974)[3]. He assumed linear marginal costs and uncertainty about the level of the marginal costs and benefits (not their slopes). Under these assumptions he reaches four main conclusions. Firstly, under full information it does not matter whether taxes or individual permits are used. Both instruments secure a first best optimum. Secondly, an error in estimating the benefits function has adverse effects on welfare but does not favor one policy instrument over the other: the efficiency losses will be exactly the same for the emission tax as the tradable permits system. Thirdly, if there is uncertainty about costs, emission tax is preferred over quantity regulation if the marginal costs are steeper than the marginal benefits function. Finally, transferable permits are preferred over taxes in the case of imperfect information about costs if the marginal benefits function is steeper than the marginal costs function. These results can be summarized as follows:

$$\bigtriangledown \approx \frac{\sigma_C^2 \left(B" + C"\right)}{2C"^2} \tag{1.1}$$

where $\bigtriangledown$ denotes the relative advantage of taxes over tradable permits measured in terms of welfare. If $\bigtriangledown > 0$ taxes are preferred over quantities regulation; while $\bigtriangledown < 0$ implies that tradeable permits is preferred over taxes. $B"$ denotes the marginal benefits slope (with $B" > 0$) while $C"$ denotes the curvature of marginal cost (with $C" > 0$). σ_C^2 represents the uncertainty on the cost function[4], while the sign $\approx$ is used to denote "a local approximation"

[3]Weitzman did not prescribe exact types of price or quantity instruments, but many authors see the issue as binary choice problem between taxes and a quantity-based regime of tradeable permits.

[4]The regulator perceives the cost function only as an estimate or approximation: $C(q, \theta)$ where q denotes the emissions reduction and θ is a disturbance term or a random variable. σ_C^2 denotes the variance of costs; as σ_C^2 shrinks to zero we move closer to the perfect certainty case where in theory the two pollution

in the traditional Taylor theorem sense.

Subsequent contributions to this topic fall into two categories:

- Modifying the assumptions in Weitzman's analysis (see Weitzman and Laffont, 1977; Malcomson,1978; Stavins, 1996; Stranlund and Ben-Haim, 2007[5])

- Comparing policy tools other than an emission taxes and tradeable permits (see Yohe, 1977; Baumol and Oates, 1988; Mckitrick, 1997; Williams, 2000; Montero, 2004).

Weitzman stresses that if the uncertainty on benefit and the uncertainty on cost function are simultaneously present and benefit and cost function are not independently distributed, the correct form of the above rule becomes:

$$\bigtriangledown \approx \frac{\sigma_C^2 \left(B" + C"\right)}{2C"^2} - \frac{\sigma_{BC}}{C"} \tag{1.2}$$

where σ_{BC} represents the covariance between benefits and costs. In order to explore the full implications of the above rule, Stavins (1996) rewrite equation (1.2) as:

$$\bigtriangledown \approx \frac{\sigma_C^2}{C"} \left(\frac{B"}{2C"} + \frac{1}{2} - \frac{\rho_{BC}}{\sigma_C} \sigma_B \right) \tag{1.3}$$

where ρ_{BC} is the correlation coefficient between benefits and costs, while σ_B and σ_C are respectively the standard deviation of benefits and costs. Based on the equation (1.3) Stevins made the following important observations:

regulation mechanisms perform equally satisfactorily.

[5]Weitzman and Laffont (1977) examine modifications to the relative-slopes criterion when these are also uncertain. Malcomson (1978) reexamine Weitzman's rule when local linear approximations to the benefits and costs function are not appropriate. Stanlund and Ben-Haim (2007) revisit Weitzman's original work under Knightian uncertainty that is when uncertainty cannot be modelled with known moments of probability distribution.

1. when benefits and costs are not correlated, so that $\rho_{BC} = 0$, an error in estimating the benefit function has adverse effects on the welfare, but the welfare loss does not differ under taxes and tradable permits regime;

2. Given $\frac{\partial \bigtriangledown}{\partial (\sigma_B \cdot \sigma_C)} = -\frac{\rho_{BC}}{C"}$, a positive correlation between benefits and costs tends to favor tradeable permits over taxes while a positive correlation tends to favor emission taxes.

3. Given $\frac{\partial \bigtriangledown}{\partial \rho_{BC}} = -\frac{\sigma_B \sigma_C}{C"}$, the greater the benefit or the cost uncertainty and the lesser is the slope of the marginal cost function and the greater is the influence of the correlation among benefits and costs on the choice of the the best policy instrument to regulate pollution.

4. Theoretically these effects can overwhelm the usual Weitzman's relative-slopes instrument recommendation.

5. The "instrument neutrality" identified by the equality between marginal benefits from pollution reduction and marginal abatement costs disappears when benefits and costs are not independently distributed; in fact by setting $B" = -C"$ Stevins showed that:

$$\bigtriangledown = -\frac{\sigma_{BC}}{C"} \tag{1.4}$$

a positive correlation favor quantities based instruments (tradeable permits) while if negative correlation between costs and benefits exists, price instruments would be optimal. Stavins presents various scenarios for statistical dependence between marginal benefits from environmental protection and marginal abatement costs. Many scenarios, however, provide examples of positive correlation, suggesting that quantity

instruments would be more attractive than otherwise. For instance, he considered the weather as generator of stochastic shocks that produce correlated impacts on marginal benefits and marginal costs of pollution control:

> [*... the increased ultraviolet radiation that reaches the ground level on sunny days means more ozone formation from oxides of nitrogen and volatile organic compounds. Hence the marginal cost of ambient concentration reduction (and risk reduction) would increase. Of course, on beautiful sunny days, people are more likely to be outside, exercising, and breathing the ozone-laden air; hence, the marginal benefits of ambient-reduction would also increase, yielding a positive correlation between the relevant marginal benefits and marginal costs...*] (Stavins, 1996).

Williams (2000, 2002) extends Weitzman's (1974) paper by developing a model of regulation of a group of pollution sources which investigates the relative efficiency of three regulatory instruments when there is uncertainty in the regulators's knowledge of firms' costs: an emission tax, fixed quotas and tradeable permits. The general structure is similar to the model in Weitzman, but differs in two important respects: Williams's paper compares three pollution regulation instruments (tradeable permits, taxes and fixed quotas), and considers the degree of substitutability between the pollution sources. Williams results can be summarized as follows:

$$\bigtriangledown^{TQ} = \frac{B" (1-\phi) + C"}{2C"} \left(\frac{N-1}{N}\right) \sum_i \sigma_{iC}^2 \tag{1.5}$$

where, $\bigtriangledown^{TQ}$ denotes the relative advantage of tradeable permits over fixed quotas in terms of welfare. N denotes the number of pollution sources distinguished by location, time period or both, while ϕ represents the degree of substitutability between the pollution sources. When abatement at one location is a perfect substitute for abatement at any other location, as in the case of globally mixed pollutants, tradable permits are preferred

over fixed emission quotas. However, when pollution is high localized and independent of emissions produced by other sources, tradable permits are dominated by emission taxes or fixed quotas.

Montero (2004) considers the optimal policy instruments choice when the regulator faces several information constraints: each firms have private information about its emissions, abatement costs and production costs. Montero develops a theoretical model for an industry of heterogeneous firms that produce output and undesirable by-products. The nature of Montero's model is similar to the one in Weitzman (1974) but with important differences: he compares the performance of two quantity instruments (tradeable permits and fixed standards) and considers the effect of cost heterogeneity across firms on instrument performance. He concludes that tradeable permits is preferred over CAC regulation when cost heterogeneity across firms is large, while when heterogeneity disappears the advantage of permits reduces in favor of standards. He also examine the advantage of a hybrid policy that optimally combines permits and standards.

Weitzman and the above described subsequent contributions fixed the conditions under which each of this two instruments is to be preferred to the other in a perfectly competitive equilibrium. However, many of major polluters in the real world are large firms in non-competitive industries (oil refineries, chemical companies, and auto manufacturers) where firms are not price takers in their output markets. Buchanan (1969) called attention to this issue by showing that the imposition of a Pigouvian tax may lead to a contraction in output that under monopoly regime is below the social optimum: a tax on a polluting monopolist will reduce the generation of external damages, but it may also cause the firm

to reduce further its output. Thus, there is a trade-off between the two distortions, one due to the monopolistic underproduction and the other due to negative externalities. A tax based only on negative externalities ignores the social cost of further output contraction by a monopolist whose output is already below an optimal level.

Barnett (1980) was the first to solve the problem of determining the second best optimal emission level and the corresponding second best emission tax to be imposed on a monopolist. He considers a polluter who produces a single product output q and who discharges smoke s generating external diseconomies $E(s)$. He finds that a tax rate for unit of smoke discharged which maximizes social welfare must be equal to:

$$T^* = \frac{\frac{df(p)}{dq}\frac{dq}{dT} \cdot q}{\frac{\partial s}{\partial q}\frac{dq}{dT} + \frac{\partial s}{\partial w}\frac{dw}{dT}} + \frac{dE(s)}{ds} \tag{1.6}$$

where $f(q)$ is the industry demand curve, while w denotes resources devoted to smoke treatment. He discusses two cases: one in which the only means to abate the external diseconomies represented by smoke is reducing output , and the second case is one in which end-of-pipe treatment is the only means of smoke abatement. In the first case, terms involving w disappear and an optimal tax is given by:

$$T^* = \frac{\frac{df(p)}{dq}\frac{dq}{dT} \cdot q}{\frac{\partial s}{\partial q}\frac{dq}{dT}} + \frac{dE(s)}{ds} \tag{1.7}$$

If the only means of smoke abatement is end-of-pipe treatment the polluter responds to tax by changing w, than $\frac{dq}{dT}$ is equal to zero and the optimal tax is given by:

$$T^* = \frac{dE(s)}{ds} \tag{1.8}$$

Only in this last case market structure is not relevant. But, market structure becomes relevant for the more general case where both w and q vary with T. Finally, Barnett

reformulate equation 1.6 introducing the price elasticity of demand $\mu = \frac{dq}{df(p)}\frac{f(p)}{q}$ and showing explicitly its role in determining the optimal second-best taxation:

$$T^* = \frac{-\frac{df(p)}{|\mu|}\frac{dq}{dT}}{\frac{\partial s}{\partial q}\frac{dq}{dT} + \frac{\partial s}{\partial w}\frac{dw}{dT}} + \frac{dE(s)}{ds} \tag{1.9}$$

He derives two main conclusions:

- when polluters are perfectly competitive μ approaches infinity, and the value of the optimal tax rate approaches marginal external damage $\frac{dE(s)}{ds}$.
- when polluters are imperfectly competitive μ is finite, and second best optimal tax rate may be less than marginal external damage to achieve an optimal trade-off between the external diseconomies and the welfare loss associated with monopoly output contraction.

The second best tax rate is equivalent to the combination of a Pigouvian emission tax and a subsidy on production, thereby correcting both distortions. Formally, the second-best tax rate can be negative if the social damage associated to pollution is very small compared to the distortion due to the market structure. The environmental problem becomes less significant and the regulator sets a negative tax (i.e. a subsidy on pollution) to induce firms to produce more.

Chen (1990) proposes another method for regulating monopolies and their production. He assumes that the planning authority would provide to the pollutant monopolist a subsidy equal to the expected value of the total benefit from emission reduction. The pollutant monopolist chooses the abatement level which maximizes profits given the subsidy

and its own private information on production costs. Chen compares, referring to Weitzman model, the abatement subsidy with two other planning mechanisms: quantity based instruments (tradable permits), price-based instruments (emissions taxes).

He derives two expressions for the comparative advantage of the subsidy relative to quantity based instruments and another for the comparative advantage of the subsidy relative to price based instruments respectively:

$$\bigtriangledown^{ST} \approx \frac{\sigma_C^2}{2\left(C" - B"\right)^2} > 0 \tag{1.10}$$

and

$$\bigtriangledown^{SP} \approx \frac{\sigma_C^2 B"^2}{2C"\left(C" - B"\right)^2} > 0 \tag{1.11}$$

where $\bigtriangledown^{ST}$ and $\bigtriangledown^{SP}$ denote respectively the relative advantage of subsidy over tradeable permits and the relative advantage of subsidy over price regulation mechanism in terms of welfare. Not only the abatement subsidy does dominate quantity based instruments , but also dominates price-based instruments. Chen concludes that we have the best mechanism when pollutant monopolist chooses the level of reduction in emission receiving, or internalizing the social benefits of his abatement. Subsidies however are often politically and financially infeasible and might deter the adoption of new abatement technology.

Instead, little work has been done to investigate emissions trading markets where one or more participants have market power. Much work has been done on tradeable permits as mechanisms that may themselves influence the market structure since they may be more susceptible to strategic behavior. The basic idea for the tradeable permits control is that firms will trade quotas among themselves; such trading could continue until firms have equal

marginal abatement costs and there is no further incentive to trade. In equilibrium, the price in such market should be equal to marginal abatement costs of each of the firms. In practice, however, the transactions costs in the market for permits might be high and this might reduce the number of transactions and prevent marginal abatement costs from being fully equalized. It is also possible that firms could behave in an anticompetitive fashion, for example hoarding permits in effort to drive polluting competitors out of business. (see Hahn, 1984; Misiolek and Elder, 1989; Mansur, 2006).

1.2 Implementation of Environmental Policy

While the theoretical literature has clarified the issue concerning the determination of the best policy instrument to regulate pollution and reduce health pollution related damages, there has been less empirical work (see Kolstad, 1986[6]; and Choi and Feinerman, 1995). Lack of appropriate data (as well as empirical research evidence) makes it difficult to quantify environmental health impacts and pollution social costs; hence, the choice of environmental instruments becomes more complicated than what would appear from the theoretical results[7].

[6]Kolstad (1986) was the first that empirically examined the fees vs. permits issue. He evaluated policies to control sulphur emissions from power plants by creating a stochastic model of regulatory design and industrial response for taxes and permits for air pollution regulation. He found that if marginal benefits from reduced sulphur emissions were constant a price instrument would be slightly preferable, but that a slight marginal benefits slope would be enough to make permits the more desirable option.

[7]As to the environmental charges and taxes, for example, the lack of information on the damage levels and problems related to their measurement constitute serious obstacles to the practical implementation. Consider, for instance, taxes paid by road transport: in addition to the morbidity and mortality caused by vehicular emissions, road traffic leads to noise stress, loss of quality of life, water pollution etc. Often, road traffic damage is greater than the additional tax paid by road transport. Then, road transport may not pay for the social costs it generates. This may lead to a pattern of transport development which may be accompanied by excessive impacts on the environment and health.

In response to these measurement obstacles, the literature has explored some second-best approaches to policy designs which have appealing properties. Baumol and Oates developed the "environmental charges and standards approach" in "The Theory of Environmental Policy". They suggested to first set a certain

One of the explanations for the gap in the empirical literature can be found in the difficulties that analysts encounter in estimating marginal pollution health damages or marginal benefits from reduced pollution. In fact, while measuring control costs is relatively straightforward (market exists in principle in which pollution control equipment can be bought, and such equipment will reduce pollution by measurable levels [8]), health damages caused by an increase of ambient pollution or marginal benefits from a reduction in pollution concentration are much harder to measure. Many difficulties derive from the fact that individuals have different susceptibilities toward pollution: the effects on health will vary across individuals due to genetics, avoidance behavior, life-style and other several factors. Hence, quantify exactly the extent of the health damages or health benefits deriving from increasing or decreasing pollution is a very hard task: we have to calculate the associated changes in health outcomes by taking into account that pollution could easily be correlated with other factors that may be just as potent. Once we have determined health pollution damages or pollution abatement benefits, we have to put a monetary value on

standard of pollution (emission, air and water quality, etc.) and then, through a process of trial and error, derive which level of taxes have proved to give certain outputs. Taxes would be set to achieve a certain acceptable standard rather than being based on the "unknown value of marginal damage". They further argued that such an approach would not result in Pareto optimality but that the

> [*..use of unit taxes to achieve specified quality standard is the least-cost method for the achievement of these targets...*] Baumol and Oates (1988).

The regulator can also move towards the use of voluntary approach. The regulator offers the firm a contract based on a certain level of abatement effort to achieve a certain standard of pollution and the firm can accept or refuse; if firm refuses, the regulator will impose an emission charge that is a fee levied on each unit of pollutant emitted. The emission charge is not a Pigouvian tax strictly speaking; it is not based on the estimated damage, but it can be considered a legitimate interpretation of the Pigouvian concept as it is a tax implemented to combat environmental pollution circumventing the problem of the damage level measurement.

[8] In order to compute and to evaluate producers' marginal abatement costs, we should to calculate shadow prices of pollution from the production technology that can be derived from the estimated output distance function (Shepherd,1970; Färe et al., 1993; Färe et al., 1997). Shadow prices derived from estimated output distance function do not directly reflect the value of abatement to society in terms of reduced morbidity but they could be compared to independent calculations of such marginal benefits in order to guide regulatory policy.

them. However,

> [*...valuing health is obviously controversial because each person may place a different value on health. The problem facing society with pollution control is that we must make decisions that are not specific to each person but rather apply to us all. It is therefore not surprising that there is such controversy about picking a single value for health...*] (Mendelsohn, 2002).

Concerning the quantification of health impacts scientists have performed several epidemiological studies on the linkages between air pollution and human health and have used the air pollution dose-response function[9] to estimate and evaluate the effects of a change in environmental quality on health[10]. Ostro (1994) presented the estimated health

[9]The dose-response function relates health effects to air pollution concentrations and other factors affecting health.

[10]Many of these epidemiological studies has been conducted in developed countries and used to estimate the effects of air pollution on health in developing countries.

Ostro (1994), for instance, uses the available epidemiology literature dose-response functions from the United States, Canada and Britain, to estimate the health impacts of conventional air pollutants (particulate matter, sulfur dioxide, nitrogen dioxide, ozone) and emissions of lead in Jakarta, Indonesia. Health effects of air pollutants (such as premature mortality, hospital visits and admissions, emergency room visits, restrictions in activity, acute respiratory symptoms, acute bronchitis in children, asthma attacks, IQ loss, and blood pressure changes.) are estimated by applying these functions to ambient air pollution leave.

Alberini and Krupnick (1997) use daily records from a diary-type epidemiologic study in Taiwan to fit logit equations predicting the probability of experiencing acute respiratory symptoms (and headaches) as a function of pollution and weather variables, individual characteristics, and health background and proxies for reporting effects. They find that the rate at which illnesses are reported follows the fluctuations in PM levels but remains unaffected by ozone concentrations. Their model predicts that the impact of the particulate matter effects is very small. Moreover, illness rates tend to be poorly predicted when the corresponding equation estimated for a similar study conducted in Los Angeles is used.

Alberini and Krupnick study stresses that great care must be taken in the application of these method since dose-response transfer might give very misleading results. Firstly, elderly are more sensitive to the life-shortening effects of air pollution and in particular to particulate matter. Extrapolations from the U. S. population to a population with a much younger age structure would likely lead to an overestimate of the effect of pollution on premature mortality (Cropper, et al., 1997). Secondly, measured particulate matter is a heterogeneous mixture of solids and liquid. Differences in the physical and chemical composition of particulate matter could lead to quite different relationships between measures of particulates and the health effects of concern across countries (Ostro, et al., 1996).

> [*...this approach neglects differences between the United States (and other developed countries) and the target country in pollution levels, baseline health, the age distribution of the population, medical care systems, sickleave policies, and cultural factors that might affect perceptions of illness and pollution and behavioral responses...*] (Alberini and Krupnick, 1997).

impact, for a given type of health risk, as follows:

$$dH_i = b \cdot POP_i \cdot dA \tag{1.12}$$

where: dH_i denotes change in population health risk, b_i denotes the slope from dose-response curve[11], POP_i is population at risk, dA denotes the change in air pollution. To complete the benefit or damage estimation for health effects, one would calculate the economic valuation V_i of this effect as well. Therefore the total change of the social value (dT) of the health effects due to the change in environmental quality under consideration can be represented by:

$$dT = \sum_i V_i dH_i \tag{1.13}$$

Even assuming that we can accurately measure health effects dH_i, putting monetary values V_i on that effects is rarely easy. The ability to place a monetary value on the consequences of pollution on health remains the crucial problem of the economic approach to human health problem related to environmental degradation (Hanemann, 1994).

Environmental economists have developed methodologies to measure the value of pollution health damages; these methods can be grouped in two broad categories. The first includes methods that measure only the loss of direct income (lost wages and additional expenditures). These approaches do not include discomfort, pain, losses in leisure, and other less-tangible impacts to individual and family well-being, moreover, may seriously understate or completely ignore the health costs of people who are not members of the labor force. Therefore, these methods provide only the lower bound of the social costs since tend to understate the total costs to individuals. The second category is based on the

[11]The slope of dose-response function measures the percentage change in the health outcome for a one unit change in ambient air pollution level.

willingness to pay (WTP) of some economic agents for avoiding pollution health damages. Willingness to pay reflects the individual's preferences and can be interpreted as a monetary measure of health damage.

In addition, following the conventional economic practice, we distinguish these methodologies on the basis of whether their primary focus concerns respectively nonfatal illness or rather death (or more specifically the change in the conditional probability of dying at each age, for an identified group of individuals at risk).

1.2.1 The Economic Value of Morbidity

Models that describe what an individual would pay to avoid illness associated to pollution are, by now, well established in the literature (Berger et al. 1987; Harrington and Portney, 1987; Cropper and Freeman, 1991). We start by sketching Berger et al.'s (1987) model in order to provide a framework for interpreting individuals' willingness to pay. Then, we critically review the methods and the research efforts that have been devoted to estimating the willingness to pay for reduced morbidity (see also Dickie and Gerking, 2002).

Berger et al. (1987), assume that a person's utility depends on the consumption of goods and services and the state of health:

$$U = U(c, q) \tag{1.14}$$

where U is utility, C is consumption and q is a vector of health characteristics. Individuals, however, do not know their health status with certainty . The probability of enjoying good health is influenced by choosing one's life-style, thus making better and worse health status

more or less probable, and by using medical advice, pharmaceuticals, hospital treatment, etc. Although one's current health status certainly provides some information about the likelihood of future health outcomes, the risk of getting a disease may also depend on other factors such as pollution exposure, smoking history, which are more or less independent of one's observable health state. Berger et al. (1987) assume that the probability density function for health status is:

$$h\left(q;X,E\right) \tag{1.15}$$

where X is preventive expenditure and E is any exogenous shift such as environmental quality change. They reasonable assume that the chances of survival can be expressed as function of health characteristics:

$$p = p\left(q\right) \tag{1.16}$$

In addition, they assume that health is a matter only of absence or presence of a deleterious condition and the density function $h\left(q;X,E\right)$ is discrete rather than continuos, with $q=1$ if individuals will enjoy good health and $q=0$ otherwise, thus:

$$\begin{aligned} h\left(q;X,E\right) &= H\left(X,E\right) \text{ if } q=0 \\ h\left(q;X,E\right) &= \left(1-H\left(X,E\right)\right) \text{ if } q=1 \end{aligned} \tag{1.17}$$

where $H\left(X,E\right)$ denotes the probability of contracting the disease. Berger et al. (1987) assume that a person will choose preventive expenditure X in order to maximize the expected value of utility:

$$\begin{gathered} max\ E(U) = U_0 P_0 (1-H) + U_1 P_1 H \\ subject\ to\ M = C + X + Z \end{gathered} \tag{1.18}$$

where M is the income, $U_0 = U(M - X, 0)$ is the utility if free of the disease; $U_0 = U(M - X - Z, 1)$ is the utility with the disease (where Z is the cost of illness that reduces consumption without providing utility). P_0 denotes the probability of survival if the individual is free from disease while P_1 denotes the probability of survival with disease.

From the above maximization problem Berger et al. (1987) derive a person's WTP for an exogenous reduction of the concentration of pollution. WTP can be defined as a change in income that would be required to keep the expected utility constant when there is an exogenous change. Berger et al. express WTP as sum of two terms:

$$-dM/dE = -\left[\left(U_0 P_0 - U_1 P_1\right)/\lambda\right](dH/dE) - (dX/dE) \tag{1.19}$$

the first term is the monetary value of the expected differences of the expected utilities between being healthy and ill by the change in health risk. The second term denotes the change in preventive expenditure due to an exogenous change in the environment. Finally, $\lambda = U_0' P_0 (1-H) + U_1' P_1 H$ and can be interpreted as the marginal utility of income.

Referring to the model above, Berger et al. (1987) explore two techniques aimed at measuring WTP: the cost-of- illness (COI) approach and the preventive expenditures (or averting expenditure) approach, that we review below.

The cost-of- illness (COI) *approach* is often used to value the cost of pollution related to morbidity. COI measures any loss of earnings resulting from illness (direct costs), medical costs such as for doctors, hospital visits or days, and medication, and any other re-

lated out-of-pocket expenses (indirect costs) (see Hodgson and Meiners,1982 for a complete description of the methodology).

A key criticism to COI approach has been that it fails to take into account individuals' preferences, disutility from illness (Harrington and Portney, 1987), averting behavior and averting expenditure (Cournat and Porter, 1981). This criticism is supported by the equation 1.19 that consists in two terms: a utility term which reflects the cost of illness and a second term reflecting preventive expenditure. Only if preventive expenditure does not exist or does not change with changes in environment ($dX/dE = 0$) or, in a less plausible case, if health status does not affect directly the individual utility function, equation 1.19 collapses to the first term and COI can be considered a measure of individuals' WTP.

In a similar model to the one presented above, Cropper and Freeman (1991) show that willingness to pay can be expressed as the product of the slope of a dose-response relationship times the marginal value of illness. But, since the marginal value of illness includes not only the loss in productivity and out-of-pocket expenditure but also pain and suffering, defensive expenditure, and loss in leisure time, it is likely to be higher than the COI measure. Hence, COI gives only a lower bound on willingness to pay (see also Dickie and Gerking, 2002).

The *preventive expenditures* (or *averting expenditure) approach* assumes that the link between environmental quality and health damages is affected by many human choices. In Berger et al. (1987) these choices are represented by consumption of X, which can be defined in several ways such as: whether to exercise on a day with high ozone level or to

install an air filter or to buy bottled water. The preventive expenditure approach infers the minimum amount people are willing to pay to reduce health risks through the amounts people living in polluted areas spend on averting measures: for instance, expenditures on air filters or bottled water can be used to infer the minimum value people are willing to pay to avoid respectively respiratory or waterborne diseases. This approach has received little attention in environmental literature since it presents many limitations (see Courant and Porter,1981). Firstly, referring again to the equation 1.19, we can observe that preventive expenditure represents only a lower bound of and individual's WTP since it does not consider explicitly the cost of illness. In addition, averting measures may be difficult to define for different types of pollution.

Abdalla et al. (1992) use the averting expenditure method for valuing environmental improvements and for approximating the economic costs of groundwater degradation to households in a southeastern Pennsylvania community. The decisions included in their study to test for averting behavior were: increased bottled water purchases among households buying it prior to the contamination, bottled water purchases by new buyers, installing home water treatment systems, hauling water from alternate sources and boiling water. The survey was conducted by mail and respondents were asked to report only those actions taken as a specific response to groundwater contamination. Their findings, obtained trough a logit specification, indicate that household's knowledge of contamination, perception of risk and presence of children determine whether they undertake averting actions and that their expenditure levels are higher if young children are present. Bresnahun et al. (1997) use panel data consisting of repeated observations on 226 Los Angeles area residents

during 1985-86 to explain defensive responses to air pollution using determinants predicted by an averting behavior model. Their empirical results indicate that people who experience smog-related symptoms spend significantly less time outdoors as ozone concentrations exceed the national standard. Many people also report making other behavioral changes to avoid smoggy conditions and the propensity to do so appears to increase if health symptoms are experienced. Other applications have investigated individuals' effort to reduce symptoms of air pollution exposure (Abrahams et al. 2000; Eiswerth et al. 2005). However most of these studies find it difficult to assign a cost to averting behaviors. There is no monetary price for many of these actions and no compelling reason to use wage rate for increasing time spent indoors since it may not be entirely lost.

Another method frequently used by the environmental economist for valuing reduced morbidity is the *contingent valuation method (CV)*. CV, first proposed by Ciriacy-Wantrup (1947) and first applied by Davis (1963), is a survey or questionnaire-based approach. This method can be thought of as an attempt to directly measure willingness to pay ($-dM/dE$ in the Berger et al. model presented above). In contingent valuation methods, randomly selected samples from the general population are given information about a particular problem. They are then presented with a hypothetical occurrence such as a disaster and a policy action that ensures against a disaster; they are then asked how much they would be willing to pay — for instance, in extra utility fees, income taxes, or access fees — either to avoid a negative occurrence or bring about a positive one. The actual format may take the form of a direct question ("how much?") or it may be a bidding procedure (a ranking of alternatives) or a referendum (yes/no) vote. Contingent valuation studies are

conducted as face-to-face interviews, telephone interviews, or mail surveys. The face-to-face is the most expensive survey administration format but is generally considered to be the best, especially if visual material needs to be presented. Non-response bias is always a concern in all sampling designs. In other words, people who do not respond have, on average, different values than people who do respond.

In principle, contingent valuation methods can be used to estimate the economic value of anything, even if there is no observable behavior available to deduce values through other means. Even though the technique requires competent survey analysts to achieve defensible estimates, the nature of CV studies and the results of CV studies are not difficult to analyze and describe. However, contingent valuation methods can be very expensive because of the extensive pre-testing and survey work. Moreover, contingent valuation methods suffer from a particular lack of accuracy: the presence of many biases (these include the way in which questions are phrased, the socioeconomic profile of respondents, the amount and type of information they are given etc.).

More fundamentally, contingent valuation approach is based on the assumption that individuals have well-defined preferences over all alternative states of the world. This assumption, however, in unreasonable for children, especially for infants. One approach to valuing the health effects on children is to make the assumption of "parental sovereignty" and to value these impacts according to the parents' willingness to pay for them (see Neidell, 2004). Freeman III (2000) however observes that

> [*... there is no clear reason for believing that parents' willingness to pay for changes that affect their children will be equal to the willingness to pay that the children would have for changes that affect their own well being. Some authors have noted that parents do not always seem to be the best judges of what is good for their children and sometimes engage in activities such as smoking and*

drinking that actually harm their children...].

Even thought contingent evaluation presents these limitations, has great flexibility, allowing valuation of a wider variety of non-market goods among which individuals well-being.

Many environmental economist use this method to evaluate health benefits from reducing pollution. For instance, Alberini et al. (1994) conduct a contingent valuation survey in three cities of the Republic of China (Taiwan) to estimate willingness to pay to avoid a recurrence of the episode of illness most recently experienced by the respondent. Alberini and Krupnik (2000) conduct a contingent valuation survey to estimate WTP to avoid minor respiratory illnesses. Then they compare cost-of-illness (COI) and willingness-to-pay (WTP) estimates of the damages from minor respiratory symptoms associated with air pollution using data from a study in Taiwan in 1991-92.

Several studies have relied on a new approach in estimating the willingness to pay for improved environmental quality by relying on the *health production approach* first introduced by Grossman (1972) (see Kiiskinen, 2003). Grossman interprets a person's health as a capital stock that exogenously deteriorates at an increasing rate with age. To counteract this health deterioration, he assumes that individuals invest a portion of their assets into health production each period. By analyzing the decisions consumers make concerning the resources allocated to health production such as medical care, time and a healthy life-style this method try to infer the value of health to the consumers and derive estimate econometrically a measure of individual willingness to pay for a reduction in pollution (Cropper, 1981; Gerking and Stanley, 1986; Dixie and Gerking, 1991; Cropper and Freeman III, 1991).

Cropper (1981), for instance, extends Grossman's model of health production and health demand to incorporate pollution and estimates willingness to pay for health risks related to an index of air pollutants. Gerking and Stanley (1986) estimate willingness to pay for health risks related to ozone exposure. They compute the value of a change in health by multiplying the cost of preventive activity by an estimated ratio of marginal products of inputs in the health production function. Harrington and Portney (1987) extended the household production function model introduced by Grossman to examine explicitly the relationships among willingness to pay for a reduction in pollution. Dickie and Gerking (1991) used a set of health symptoms in estimation of functions hypothesized to be associated with air pollution. Cropper and Freeman III (1991) develop a model of health production in which the health outcome of interest is the number of hours during a time period that a person spends in sickness. They show that willingness to pay can be expressed as the product of the slope of a dose-response relationship times the marginal value of sickness time.

The health production function approach suffers from an import limitation too. The estimation of a health production function is frequently based on instrumental variables since individual's life-style and averting expenditure/behavior inputs (that in the above model is represented by X) may be endogenous. Construction of instruments, however, usually can be done in a number of ways as theory often says little about how this problem should be handled and variables used for this purpose are chose depending on what information is available together with judgement of the investigator. Different choices of instrumental variables typically can produce different estimates of willingness to pay creating uncertainty.

1.2.2 The Economic Value of Mortality

Because some forms of pollution may increase mortality or shorten life expectancy, economists have to identify approaches for valuing life and the benefits of lifesaving activities. Since death is a more easily measured outcome than illness or injury (death is a one-dimensional event, whereas there are varying degrees of illness and injury) these methods are easier to apply.

In estimating the value of lifesaving, economists have followed two schools of thought. The first approach is based on measurements of the economic productivity of the individual whose life is at risk. This is referred as *human capital approach*. The second approach is based on the individuals' *WTP to reduce the risk of death* (Cropper and Freeman, 1991; Shepard and Zeckhauser, 1982; Berger et al. 1994; Johansson,1995).

In the standard human capital approach the value of preserving a life V_i (with reference to the equation 1.13) is equal to the discounted present value of lifetime earnings lost due to premature mortality. Formally the present value of lifetime earnings is given by:

$$V_i = \sum_{t=j}^{T} q_{j,t} \, (1+r)^{j-t} \, y_t \tag{1.20}$$

where $q_{j,t}$ is the probability of the individual surviving from age j to age t, y_t is the individual earnings at age t, r is discount rate and T is age at retirement from labor force. This approach has been criticized on a number of grounds: it considers individuals as units of human capital that produce goods and services for society. The values calculated are dependent on the age of death and on income, skill level, sex, race and country of residence. It omits the role of nonmarket production and because of earning differences by sex and race, it places a lower value on saving the lives of women and non whites than on saving the lives

of adult white males. Moreover, the human capital approach assigns zero value to people who are retired, handicapped or totally disabled and to children (Landefeld and Seskin, 1982). Another important objection is that the human capital approach is inconsistent with the fundamental premise of welfare economics by which individuals' preferences should constitute the cornerstone of the benefit-cost analysis(Cropper and Oates, 1992).

Individuals make decisions everyday that reflect their preferences and how they value health and mortality risks, such as driving an automobile, smoking cigarettes or living in polluted areas. Many of these choices involve market decisions. Using evidence on these market choices, which involve implicit trade-offs between risks and money, economists have developed estimates of the individuals' WTP to reduce risk of death. The *willingness-to-pay approach* is based on the assumption that changes in individuals' economic welfare can be valued according to what individuals are willing (and able) to pay to achieve that change. According to this assumption, individuals treat longevity like other consumption good and reveal their preferences through the choices that involve changes in the risk of death and other economic goods whose values can be measured in monetary terms.

One of the most used approach to measuring willingness to pay to reduce the risk of death is to infer the value from *compensating wage differentials* in the labor market (see Viscusi and Aldy, 2003 for a complete treatment). The theory behind this approach is simple:

> [*...The basic idea behind compensating wage differentials is that jobs can be characterized by various attributes, including risk of accidental death. Workers are described by the amount they require as compensation for different risk levels, while firms are characterized by the amounts they are willing to offer workers to accept different risk levels. The matching of wage offers and acceptances determines the hedonic wage equation, which describes the compensation received*

for bearing risk in market equilibrium...] (Simon et al., 1999)

Simon et al. (1999) express the individual's willingness to substitute risk for income in the labor market as the compensation C that he would require to work at various risk levels, holding utility constant. Formally:

$$(1-\rho)(1-\phi)U(C+I)=k \tag{1.21}$$

where ϕ is the risk of death on the job, and ρ is the risk of dying from all other causes while I denotes the non-labor income. The worker's choice of risk level, ϕ, occurs where a marginal change in required compensation, $C\prime(\phi)$, equals a marginal change in the wage offered in market equilibrium, $w\prime(\phi)$, or, equivalently, where the compensation function is tangent to the hedonic wage equation, $w(\phi)$. Equilibrium in the labor market is given by the locus of tangency points between various required compensation and offer curves. This locus is the hedonic wage function, and its derivative with respect to risk of death measures the value of a small change in risk to the worker:

$$\frac{dw}{d\phi}=\frac{(1-\rho)U(w+I)}{(1-\rho)(1-\phi)U'(w+I)}=V_i \tag{1.22}$$

Equation 1.22 gives the rate at which a worker is willing to substitute income for risk $\frac{dw}{d\phi}$ that is equal to his expected utility if he survives risk of death on the job, $(1-\rho)U\prime(w+I)$, divided by his expected marginal utility of income, $(1-\rho)(1-\phi)U'(w+I)$. Hence, equation 1.22 is a measure of the risk premium that a worker receives to compensate him for risk of death on job (we label this relationship, V_i with reference to the equation 1.13). Then, the compensating wage approach by providing a value V_i that is a measure of individuals WTP to avoid death risks could be used in the equation 1.13 to measure total change

of the social value (dT) of the health effects due to the change in environmental quality. Cropper and Freeman (1991) and Cropper and Oates (1992) stress that the compensating wage approach presents at least three problems: the first problem concerns the fact that compensating wage differentials exist only if workers are informed of job risks. A second problem related to this approach is that compensating differentials seem to exist only in unionized industries than it may provide estimates of the value of a risk reduction only for certain segments of the population. Finally, if workers have biased estimates of job risks market wage premium will yield biased estimates of the value of a risk reduction.

Moreover, compensating wage differentials approach presents other important limitations. Firstly, it tends to focus on the value adults in the prime of their life place on reducing their risk of dying, even though according to the epidemiological literature, the significant correlation between air pollutants and deaths occur among people over 65 (Ostro, 1994; Schwartz and Dockery, 1992). In addition, this method tends to focus only on immediate risk changes. However, when an environmental policy program reduces exposure to a carcinogen, while the costs of doing so are often incurred in the present, mortality risks are reduced in the future, following a latency period.

Difficulties in measuring the individual's WTP using labor market compensating wage approach and its limitations have led to the use of CV to measuring willingness to pay. As we have already seen in the evaluation of morbidity cases (section 1.2.1), CV presents great flexibility but also a number of limitations (for a complete treatment of these problems see Diamond and Hasuman, 1994).

1.3 Summary and Conclusions

There is a substantial amount of literature on theoretical and empirical aspects of the economic valuation of policies and instruments to improve environmental quality and human health. Here, starting with Weitzman's (1974) seminal work, we have provided a short but comprehensive overview of key literature on the choices faced by policy makers concerning price-based versus quantity-based instruments to regulate pollution and protect human health. We have reviewed the methods employed in estimating pollution abatement costs and pollution related health damages whose comparison (with reference to Weitzman's theoretical rule) should form the basis for the choices among the price-based and quantity-based regulation instruments.

The aim of this chapter is to shed light on the difficulties that analysts face in estimating the value of pollution health damages or benefits from reduced pollution. In fact, while measuring control costs seems relatively straightforward, measuring and valuing the health impacts of pollution is a very complex task: we have shown that the available methods of economic analysis are often rudimentary and the answers vary greatly depending on the method used. In recent years, however, considerable progress has been made, especially with respect to air and water pollution.

Chapter 2

An Economic Evaluation of Life-Style and Air Pollution-Related Damages: Results from the BRFSS

2.1 Introduction

Air pollution is a major environmental problem affecting the developing and the developed countries alike. Various studies by economists and epidemiologists have tried to understand the relationship between health and air pollution and other relevant factors: the effects of air pollution on health are very complex as there are many different pollutants and their individual effects vary from one to the other. Despite this, the World Health

Organization (WHO) estimates that every year 800,000 people die prematurely from lung cancer, cardiovascular and respiratory diseases caused by outdoor pollution. Other adverse health effects include increased incidence of chronic bronchitis and acute respiratory illness, exacerbation of asthma and impairment of lung function.

In analyzing the relationship between air pollution and health, it is important to consider the influence of the individual's specific behavior too, since individual life-style is another crucial determinant of the risk of illness. Concerning the individual health and health behaviors, the economic literature has often relied on the assumption that individuals treat health as exogenous and has not recognized that they may undertake actions that increase or reduce health risks. Only in the last thirty years the health economics literature, following Grossman's (1972) seminal paper, has recognized health as an outcome of a production process which involves medical care and depends on several factors including individual behaviors.

Grossman (1972) interprets a person's health as a capital stock that exogenously deteriorates at an increasing rate with age. To counteract this health deterioration, he assumes that individuals invest a portion of their assets into health production each period. Hence, the level of health of an individual may be not totally exogenous but it can depend, at least in part, on the resources allocated to its production like medical care, time and a healthy life-style.

The demand-for-health model by Grossman has become a cornerstone in the field of health economics. The model, however, is not undisputed. A key criticism has been that it fails to take into account the uncertainty of the future health status and the uncertainty

of investments in health production. By investing in health, individuals do not determine with certainty their health status – environment and chance are two factors which may interfere – but rather they influence it quite substantially. Grossman's model, however, does not account for uncertainty as it includes neither explicit acknowledgment of uncertainty nor the description of illness, even though the fundamental relationship between health and uncertainty has been established by economic theory (Arrow, 1963). Subsequent contributions analyze individual health behavior when health status is uncertain and governed by a stochastic process (Cropper 1977, Dardanoni and Wagstaff ,1991, Selden, 1993, Zweifel and Breyer,1997).

In fact, the probability of having good health is influenced by choosing one's lifestyle, thus making better and worse health status more or less probable, and by using medical advice, pharmaceuticals, hospital treatment, etc. in order to restore good health. Although one's current health status certainly provides some information about the likelihood of future health outcomes, the risk of getting a disease may also depend on other factors such as pollution exposure, smoking history, which are more or less independent of one's observable health state[1].

In the next sections we focus on how individual health habits, outdoor air quality and the presence of a pathological condition combine to affect the likelihood of a good or bad health status, in a second-best world characterized by uncertainty on the level of health and

[1]For instance Carbone et al. (2005) stressed that

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

in which an individual is not able to avoid adverse health shocks completely. The framework is built on the basic concepts and ideas of the demand for health by Grossman (1972) and on Cropper's (1981) model that extends Grossman's model to incorporate pollution.

We construct a model of health accumulation in which, following Grossman (1972) and subsequent contribution by Cropper (1981), we assume that health depreciates at an increasing rate with age and air pollution. The main differences here are that the level of health is uncertain and for individuals, who suffer or have suffered from a pathological condition, illness enters directly the rate of health depreciation too. As in Cropper's (1981) model, we assume that when pollution increases it becomes more costly to reduce the probability of facing health shocks. Individuals feel less healthy because they perceive health depreciation rate to be higher. Hence, they may choose to invest less in their health and maintain lower health stock because of the higher net investment costs. In this sense, a higher pollution concentration may have two effects on health: a direct effect which consists in an increase of natural rate of depreciation and an indirect effect, described by Cropper (1981), by which individuals will invest less in health and display a higher probability of suffering from health shocks. We will analyze this aspect in the section 4 focusing on the relationship between increasing pollution and health investment decisions. In addition we will examine if chronic illnesses, by altering the rate to which health capital stock deteriorates, have any influence on the individuals' health investment decisions too.

To estimate the health accumulation model and investigate the relation between health status, pollution, and health investment decisions, we use three different measures of overall health: a dichotomous measure of blood pressure; a dichotomous measure of func-

tional limitations and disability; a self-assessed health measure that is common in empirical research (Contoyannis and Jones, 2004, Balia and Jones, 2004 etc.). Since we have included life-style variables as regressors in the health equation, a problem of potential simultaneity may arise. Hence we try to correct the potential endogeneity of the behavioral variables by using a recursive multivariate probit model (Contoyannis and Jones, 2004, Blaylock and Blisard, 1992).

The model is estimated using data based on the 2001 Behavioral Risk Factor Surveillance System (BRFSS), which however does not measure environmental quality; environmental information at the metropolitan area-level is available from the 2001 EPA's Air Quality System (AQS) database and can be used in conjunction with BRFSS data. Data are merged by the metropolitan area-level which is available both in BRFSS and EPA data.

In particular, we concentrate our attention on one of the main sources of air pollution worldwide, i.e.motor vehicle emissions. The most important standard related to motor vehicles pollution concerns carbon monoxide. CO air concentrations are generally high in areas with heavy traffic congestion. Therefore we can consider carbon monoxide as a proxy for vehicle emissions (U.S., EPA 2000)

According to our results a higher concentration of carbon monoxide has respectively a negative impact on the probability of enjoying good health and a positive influence on healthy habits. Then, concerning vehicular air pollution our results do not support Cropper's (1981) model: people living in polluted areas tend to invest more in health, probably in order to counteract to the deterioration of a higher depreciation rate due to an increasing

pollution. Arguably, people lead a healthy life-style to increase their health stock and build up resistance against pollution symptoms and future damages.

The chapter is organized as follows: section 2 introduces a model of health production. Section 3 describes the data and the variables for the analysis. Section 4 presents the estimation strategies and the econometric results. Section 5 concludes with a discussion. The definition of the variables, descriptive statistics and tables with estimation coefficients are in Appendix .

2.2 A Model of Health Production

Assume that each individual is endowed with a stock of health capital H_t that evolves according to:

$$\triangle H_{t+1} = H_{t+1} - H_t = f\left(P, \Lambda, E, t\right) - \vartheta_{t-1}\Delta D_t - \delta_t H_t - \vartheta_t \tag{2.1}$$

where $\delta_t \in (0, 1)$ is the natural rate at which health deteriorates. ϑ_t is a random shock. We assume that the shock could be any injury which causes a reduction in the current state of health. Moreover, we assume that ϑ_t can take a value of zero when the shock does not occur and a positive value $\vartheta_t > 0$ when it does occur. The transition probability of having a shock next period is assumed to be inversely related to the stock of health. Then, the size of health is important since it affects the probability for an individual of enjoying good or bad health. Individuals can affect the probability of bad or good health next period by "investing" or "disinvesting" in health.

For an individual who has not suffered from a health shock in the past ($\vartheta_{t-1} = 0$)

the investments/disinvestments in health are captured by a household production function $f(P, \Lambda, E, t)$, where P is preventive medical cares such as regular exams, screening tests designed to catch a disease before it has the chance to spread or immunization such as flu shot vaccine. E is the exogenous education level that is assumed to affect the productivity of producing health[2]. Λ indicates the individuals behavior. We distinguish between healthy and unhealthy behavior. A proxy for healthy behavior consists, for instance, in a healthy diet (fruits and vegetables consumption etc.) or in sport activities practice, while a proxy for unhealthy behavior includes consumption of hazardous goods like alcohol consumption or cigarettes smoking. $f(P, \Lambda, E, t)$ is increasing in preventive medical care, in education and it can increase or fall in individual behavior Λ. In particular $f(P, \Lambda, E, t)$ is increasing in a healthy behavior and decreases if individuals disinvest in their health by consuming, for instance, hazardous goods. It follows that while a healthy lifestyle increases the stock of health capital, actions detrimental to health such as cigarette smoking and excessive alcohol consumption lower the stock of health capital.

If a health shock has occurred in the past ($\vartheta_{t-1} > 0$) the stock of illness D_t will

[2]Based on the theory of the demand for health (Grossman, 1972), we expect that schooling plays an important role in influencing the productivity of health inputs: individuals who choose higher levels of schooling are observed to be healthier than those choosing lower level of schooling. One explanation of this empirical regularity is that education increases the productivity of producing health i.e. more health can be produced for the same inputs (Gerdtham et al., 1999, Berger and Leigh, 1989). Schooling helps people choose healthier life-styles by improving their knowledge of the relationship between health behaviors and health outcomes (Kenkel, 1991). A more educated person may have more knowledge about the harmful effects of cigarette smoking, pollution exposition, alcohol consumption or about what constitutes an appropriate, healthy diet. Furthermore, schooling increases information about the importance of having regular exams or screening tests to prevent an illness or at least to minimize disease.

Grossman and Kaestner (1997) present an overview of studies on the relation between education and health. This survey shows that higher educated people are less likely to smoke, exercise more and are more likely to participate in screening programs for breast cancer and cervix cancer. They discuss three broad explanations of the relationship between education and health. The first is that education improves health, the second that education and health are related through their relationship to a third variable, and the third explanation for why education and health are related is that health causes education: we do not consider the issues of reverse causation in this paper. We will assume that a higher education affects the individual health status by leading people to choose healthier behaviors.

affect directly the health accumulation. The stock of illness is characterized by the following law of motion:

$$\Delta D_t = D_t - D_{t-1} = g\left(R, E, \Lambda, t\right) - \alpha D_{t-1} \tag{2.2}$$

where $\alpha \in (0, 1)$ is the natural rate of depreciation of illness stock caused by the antibody activities.

If an adverse shock affects the stock of health, individuals can operate to reduce illness: illness is decreasing in recuperative medical care R, in education and in healthy behavior, while it increases because of adverse behavior. This concept is captured by a household production function $g\left(R, E, \Lambda, t\right)$.

We assume that an increase in the stock of disease $\Delta D_t > 0$ will gradually reduce health by increasing the probability of health shock in next period while a decrease in the stock of illness will decrease the probability of encountering a shock in the future. Reduced illness, from a steady state level, trough curative medical care and reduction in hazardous goods consumption can be considered an investment in health.

As we can note the marginal products of curative medical care and of a healthier behavior increase with the size of the shock, which can be considered a measure of the severity of illness. In terms of health it means that the larger is the shock the more severe is the illness and the more dangerous is, for instance, to consume goods like alcohol or tobacco.

In order to introduce the impacts of the environment, our analysis takes changes in environmental conditions to influence the rate at which an individual's stock of health depreciates. Following Grossman (1972) and subsequent contribution by Cropper (1981) we

assume that health depreciates over time and with the ambient air pollution. However, we partly modify Cropper's (1981) assumption allowing the stock of illness to enter the rate of depreciation. In particular we assume that:

$$\delta_t = h\delta_0 \left(1+\bar{\delta}\right)^t \Psi_t^{\phi} + (1-h)\,\delta_0 \left(1+\bar{\delta}\right)^t \Psi_t^{\phi} D_t^{\gamma} \tag{2.3}$$

where h is and indicator function which takes value 1 if $\vartheta_{t-1} = 0$ and value zero if $\vartheta_{t-1} > 0$. Ψ is the air pollution concentration to which an individual is exposed. Pollution enters directly the rate of decay and physically alters the state of a person's health.

Illness increases the health depreciation rate; to counteract this deterioration, individuals can invest a portion of their assets into healthy behavior or in curative medical care in order to reduce the stock of illness and restore the initial rate of depreciation.

As in Cropper's (1981) model, when pollution increases it becomes more costly to reduce the probability of a shock. Individuals feel less healthy because they perceive δ to be higher. Hence, they may choose to invest less in their health and maintain lower health stock because of the higher net investment costs. In this sense, a higher pollution concentration may have two effects on health: a direct effect which consists in an increase of δ and an indirect effect, described by Cropper (1981), by which individuals will invest less in health and display a higher probability of suffering from health shocks. Cropper, however, has not deeply studied this aspect in her paper. We will analyze in the section 4 the relationship between pollution and life-style variables and we will examine if chronic illnesses, by altering the rate at which health capital stock deteriorates, have any influence on the individuals' health investment decisions.

2.3 Data and Variables

To analyze how individual life-style, pollution and health shocks combine to affect the likelihood of a good health status and the amount of investment in health we will use data based on the 2001 Behavioral Risk Factor Surveillance System Survey[3]. The BRFSS is the world's largest cross-sectional telephone survey conducted every year since 1984 by health state departments in collaboration with the Centers for Disease Control and Prevention. Fifteen states participated in the first survey in 1984. The number of participating states grew to thirty-three in 1987, to forty-five in 1990 and to all fifty-one States (including the District of Columbia) in 1996.

Data on preventive health practices and risk behaviors were collected from a random sample of adults (18 years of age or older) living in households through monthly telephone survey[4]. The BRFSS contains rather detailed information about health status, diseases, life-style, education and other individual characteristics. It is designed to monitor the prevalence of the major behavioral risks among adults (tobacco use, alcohol consumption etc.) associated with chronic diseases, and premature mortality.

Pollutants in the environment have been linked to chronic diseases such as cancer, asthma, and cardiovascular health problems too. Although the BRFSS does not directly measure environmental quality environmental information at the metropolitan area-level is available from the 2001 EPA's Air Quality System (AQS) database and can be used in

[3]Centers for Disease Control and Prevention (CDC). Behavioral Risk Factor Surveillance System Survey Data. Atlanta, Georgia: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention (2001).

[4]We have to take into account that the BRFSS is a survey of private households and it may be prone to selection bias in terms of assessing health and its interaction with behavioral indicators, as those individual with severe or chronic health problems and disabilities are "more likely to be in a hospital, or otherwise unavailable for interview". (Cox et. al., 1987, Cropper, 1981).

conjunction with BRFSS. Thus, we have merged the two datasets by metropolitan area[5].

The EPA's Air Quality System (AQS) database contains measurements of criteria pollutants such as ozone (O3), sulfur dioxide (SO2), carbon monoxide (CO), and particulate matter (PM2.5, PM10) concentrations at sites in all 50 states, plus the District of Columbia, Puerto Rico, and the Virgin Islands. Ambient measurements are collected from a network of national, state and local air monitoring stations, and used to create the Air Quality Index (AQI)[6].

After correcting for missing values, the sample contains 4,913 individuals.

2.3.1 Health and Life-Style Variables

The model is estimated using three different measures of overall health: a measure of blood pressure, a measure of disability and a self-assessed health measure. Berger and Leigh (1989), in analyzing the relationship between school and good health, introduce blood pressure as a dependent variable representing overall health. Many pollutants produce harmful effects on the blood and the coronary system and may be one of the cause of cardiovascular diseases. Since blood pressure is the most important predictor of cardiovascular disease, which is the greatest killer in the U.S., we expect that high blood pressure is related to air pollution. Following Berger and Leigh we create a binary variable (BLOODPRESSURE) that takes value one if respondents report that they suffer from high

[5]Because the environmental data from the AQS were collected during 2001 and reported at the metropolitan area-level, they should only be used in conjunction with 2001 BRFSS data that have been re-weighted for metropolitan area-level analysis.

[6]The AQI is used to report daily air quality based on levels of the criteria pollutants. The AQI scale runs from 0 to 500. It is categorized into the following six groups: 0-50 = Good; 51-100 = Moderate; 101-150 = Unhealthy for Sensitive Groups; 151-200 = Unhealthy; 201-300 = Very Unhealthy; 301-500 = Hazardous. Additional information on the AQI is available at http://airnow.gov/.

blood pressure and zero otherwise. We include, as a measure of health, a binary variable (AC_LIMIT) that takes value one if respondents are limited in any activities because of health problems and zero otherwise. This variable is traditionally used by the economists to represent the presence of work-preventing or work- limiting disabilities due to health problems. Moreover, following Contoyannis and Jones (2004) we also use, as an indicator for health, the self- assessed health (SAH), which is a five category variable rating from poor to excellent. We construct a binary indicator with the value one if individual report that their health is excellent, very good or good, and zero otherwise (fair or poor).

Following U.Schneider and S. Schneider (2006) we distinguish between health outcome and self-assessed health. Health outcomes such as high blood pressure and disabilities are objective measures of health[7], which are themselves influenced by the health behavior and that are also proxies for pathological conditions. Self-assessed health measures the individual's perception of her health capital stock. It is a function of health outcome and health behavior.

The endogenous behavioral variables employed are those which cover as much as possible the life-style categories used by Belloc and Breslow's (1972) epidemiological studies of around 7000 individuals conducted in Alameda County, California: the so called "Alameda Seven". These seven categories are: diet, smoking, alcohol, physical exercise, sleep, weight (for height) and stress to which we add preventive medical care. Weight (for height) is included using an indicator related to the body mass index (BMI). BMI can be

[7]In the BRFSS survey the objective measures of health are self-reported too. Then they may be subject to measurement errors.

considered as a measure of obesity[8] and is defined as weight in kilograms divided by height in meters squared (Kg/m^2). According to the World Health Organization (WHO) persons with BMI $\geq 30 Kg/m^2$ are classified as obese. We do not include sleep among the life-style variables because of the lack of a reasonable proxy in the BRFSS data set.

As a measure of diet, we use a binary variable (DIET) that takes value one if respondent consumes fruits and vegetables at least once per day and zero otherwise.

To measure smoking behavior we also employ a binary variable (SMOKE) that takes value one if respondent is everyday smoker or someday smoker and zero if she is a former smoker or non- smoker. Again we employ a binary variable (ALCOHOL) which is equal to one if an individual is at risk for heavy drinking and zero if she is not. This categorization is gender specific: drinking is defined heavy if it is greater than two drinks per day for men and one per day for women.

To measure the exercise habit we employ again a binary variable (EXERCISE) which equals one if an individual participates in any level of leisure time exercise or physical activity in the thirty days before the interview (other than as part of a regular job) and zero otherwise.

The variable that we use to measure (the presence of) obesity is based on BMI. This variable (OBESE) takes the value one if respondent is at risk for overweight, or obese (BMI equal or greater than 25.0000) and it takes value zero if respondents are not at risk (BMI less than 25.0000).

Stress was also recognized as behavioral variable which affects health in the Alameda

[8]Obesity is considered a risk factor for several diseases. It is often associated with aspects of an individual's life-style such as insufficient exercise and inappropriate diet or nutrition. Those who are obese are expected to have poorer health.

study. STRESS takes value one if during the thirty days before the interview respondent's mental health (which includes stress and depression) was not good, 0 otherwise.

To measure preventive medical care utilization we include again a dummy variable (FLUSHOT) which takes value one if an individual had a flu shot in the year before the interview and takes value zero otherwise. We do not include a proxy of recuperative medical care because of the lack of good proxy in the data set.

TABLE 2 shows a simple descriptive analysis, which presents sample means and standard deviations for the variables used in the models. It is worth noting that the sample (that comprises 42 per cent men and 58 per cent women) is made up of individuals whose behaviors are mostly healthy: only 27 per cent of individuals are current smokers, only 4.5 per cent of individuals consume drinks heavily and only 28 per cent of them suffer from stress; while 97 per cent of them follow a healthy diet and 77 per cent devote time to physical activity.

2.3.2 Other Characteristics

The exogenous variables in the model can be grouped into categories which are listed, together with the life-style variables, in TABLE 1. As can be seen from the table, we consider the following categories: health coverage (including HMO[9] plans), prior health in order to capture health status at the beginning of the observation period, education, marital status[10], employment status, race, physical characteristics, household composition,

[9] A health maintenance organization (HMO) is a type of managed care plan that provides health coverage in the United States to its members through a network of doctors, hospitals, and health care providers. HMOs are popular alternatives to traditional health care plans offered by insurance companies because they can cover a wide variety of services, usually at a significantly lower cost.

[10] In the past decade many empirical findings have documented a potential health benefit of marriage: married people (including those who cohabit) appear to be healthier and to have a longer life expectancy

air pollution.

Arguably the principal source of air pollutants worldwide is motor vehicle emissions, although many other sources have been found to contribute to the ever growing problem. The most important standard relating to motor vehicles pollution is for carbon monoxide. CO air concentrations are generally high in areas with heavy traffic congestion then we can consider carbon monoxide as a proxy for vehicle emissions (U.S., EPA 2000). Carbon monoxide is a colorless, odorless and tasteless gas that is a product of the incomplete combustion of carbonaceous material used as fuels for transportation. The major health concerns associated with exposure to CO are its strong tie with the hemoglobin molecule, forming carboxyhemoglobin (COHb). COHb impairs the oxygen-carrying capacity of the blood, this can impact on the brain, nervous tissue, heart muscle and other tissues that require large amounts of oxygen to function. The most susceptible to the health effects of ambient air exposure to CO include those with ischemic heart disease and other form of cardiovascular disease (Ostro, 1994). Since carbon monoxide remains one of the major air pollutant of concern, we will use, as proxy of air pollution, the daily maximum level of carbon monoxide air quality index (AQI).

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than the non-married. Some of the most convincing evidence is consistent with the marriage protection hypothesis, which assumes that "... married individuals engage in low-risk activities, share resources and enjoy caring from each other..." (Hu, Wolfe, 2002)

2.4 Estimation Strategies and Results

2.4.1 Multivariate Estimation

The theoretical model describes the relationship between health status, life style and pollution variables. An important question is whether life-style follows from health status or if health status follows from life-style. In the theoretical model we have assumed that not only individuals' behaviors may impact on health status but that health status in turn, by influencing the health rate of depreciation, may impact on the health investment decisions. Then, from a methodological point of view, it should be noted that the perceived health and the health outcome equations are structural equations since the health behavior inputs may be endogenous. Efficient and consistent estimation of the parameters in the health equations requires a model that takes account of the nature of the variables used. The potential simultaneity, which can arise with the inclusion of life-style variables as regressors, can be corrected by using a recursive multivariate probit model[11] (Contoyannis and Jones, 2004, Blaylock and Blisard, 1992). Following Cappellari and Jenkins (2003) the multivariate probit model can be described by the following equations system:

$$\begin{aligned} y_{1i}^{*} &= \beta_1' x_{1i} + \varepsilon_{1i} \\ &\cdot \\ &\cdot \\ &\cdot \\ y_{Mi}^{*} &= \beta_M' x_{Mi} + \varepsilon_{Mi} \end{aligned} \tag{2.4}$$

[11]The multivariate probit model with endogenous dummies belongs to the general class of simultaneous equation models (see Maddala, 1983).

Here we have $m = 1, ..., M$ equations and $i = 1, ..., N$ observations. We have M latent variables y_m^* (with $m = 1, ..., M$) and M observed dummy indicators y_m. For the latent variable we assume that

$$y_m = \begin{cases} 1 \quad if \qquad y_m^* > 0 \\ \\ 0 \qquad otherwise \end{cases} \tag{2.5}$$

The latent variables are assumed to be linear function of the vector of exogenuous variables x_{mi} where β_m is the associated parameters vector. $\varepsilon_{1i}, ..., \varepsilon_{Mi}$ are the error terms distributed as multivariate normal, each with a mean zero and a variance covariance matrix Σ. Σ has values of 1 on the leading diagonal and correlations $\rho_{jk} = \rho_{kj}$ on off-diagonal elements (where ρ_{jk} is the covariance between the error terms of equation j and k).

In the above setting, the exogeneity condition is stated in terms of the correlation coefficient, which can be interpreted as the correlation between the unobservable explanatory variables of the different equations. All the equations in (2.4) can be estimated separately as single probit models only in the case of independent error terms ε_{mi} i.e. the coefficient ρ_{jk} is not significantly different of zero (Maddala, 1983).

Following U.Schneider and B.Schneider (2006), we identify three classes of dependent variables: the individual health behaviors, the health outcomes and the self-assessed health.

The seven equations for the health behavior variables are modeled as reduced-form equations. The health outcome equations are structural equations with the health behavior variables as explanatory factors. Last, in the self-assessed health equation health behavior and health outcomes are included as regressors. Therefore, we construct and estimate two

systems of nine equations ($m = 9$) with seven reduced-form and two structural equations. One of the two structural equations is always represented by the SAH equation and the other one by one of the two different health outcomes: blood pressure and disability. Thus:

$$
\begin{aligned}
y^*_{1i} &= \beta'_1 x_{1i} + \varepsilon_{1i} \\
y^*_{2i} &= \beta'_2 x_{2i} + \varepsilon_{2i} \\
y^*_{3i} &= \beta'_3 x_{3i} + \varepsilon_{3i} \\
y^*_{4i} &= \beta'_4 x_{4i} + \varepsilon_{4i} \\
y^*_{5i} &= \beta'_5 x_{5i} + \varepsilon_{5i} \\
y^*_{6i} &= \beta'_6 x_{6i} + \varepsilon_{6i} \\
y^*_{7i} &= \beta'_7 x_{7i} + \varepsilon_{7i} \\
y^*_{8i} &= \beta'_8 x_{8i} + \varepsilon_{8i} = \delta_{81} y_{1i} + \delta_{82} y_{2i} + \delta_{83} y_{3i} + \delta_{84} y_{4i} + \\
&+ \delta_{85} y_{5i} + \delta_{86} y_{6i} + \delta_{87} y_{7i} + \alpha'_8 z_{8i} + \varepsilon_{8i} \\
y^*_{9i} &= \beta'_9 x_{9i} + \varepsilon_{9i} = \delta_{91} y_{1i} + \delta_{92} y_{2i} + \delta_{93} y_{3i} + \delta_{94} y_{4i} + \\
&+ \delta_{95} y_{5i} + \delta_{96} y_{6i} + \delta_{97} y_{7i} + \delta_{98} y_{8i} + \alpha'_9 z_{9i} + \varepsilon_{9i}
\end{aligned}
\tag{2.6}
$$

where x_{li} (with $l = 1, ..., 7$) and z_{hi} (with $h = 8, 9$) are vectors of exogenous variables, β_l and α_h are parameter vectors, δ_{ho} (with $o = 1, ..., 8$) are scalar parameters and $\beta'_h = (\delta_{ho}, \alpha'_h)$.

Estimation of the above described multivariate probit model requires some considerations for the identification of the model parameters. Maddala (1983) proposes that at least one of the reduced-form exogenous variables is not included in the structural equations as explanatory variables. Following Maddala's approach we impose exclusion restrictions. For the reduced form, we use marital status[12] and employment status variables assuming

[12]To balance statistical fit of the model we use the Bayesian information criterion proposed by Schwarz (1978). This criterion suggests the exclusion of the variables that describe marital status from the health outcomes and the SAH equation. Kenkel (1995), Contoyannis and Jones (2004), Balia and Jones (2004)

that they have only an indirect effect on health through the life-style variables. In addition, we exclude from the self-assessed health and the health outcome equations the variables that indicate the number of adults and children living in the household which are considered to influence to a certain extent individual's preferences and decisions about health. Moreover, for the outcome equations, the variables physical pain and chronic symptoms are excluded to avoid causality problems with the dependent variables.

The reference individual in the model is female, married and employed. She is aged eighteen years old or more and she has attended high school or is high school graduated.

The estimation of a multivariate probit is carried out using the Stata software which applies the method of Simulated Maximum Likelihood estimation. Stata provides the statistic $z = \hat{\rho}/S_{\hat{\rho}}$ to test the hypothesis $H_0 : \rho = 0$. If the error terms are independent, the Maximum Simulated Likelihood estimation is equivalent to the separate Maximum Likelihood probit estimation.

SAH and Activity Limitations Equations

The first two columns of TABLE 3a show partial effects for the structural SAH and activity limitations equations estimated in the full recursive model, using the multivariate probit specification.

Starting from the life-style variables we can observe that in the health outcome equation (ac_limit equation) smoking behavior has the expected significant positive effects

exclude marital status from the health and the death equation claiming that marital status influence only indirectly the probability of good or bad health and the probability of death, through the life-style habits: smoke, alcohol, diet etc.

on activity limitations as well as stress and obesity, while diet variable and alcohol consumption do not contribute to explain the probability of suffering from disability. In the SAH equation,with exception of the variables alcohol and diet, all life-style variables are statistically significant too. Their partial effects on health lead to the conclusion that unhealthy habits decrease the probability of enjoying good health. Immunization is statistically significant only in the SAH equation with a negative partial effect. One of the possible reasons for the ambiguous sign is that health status and immunization, in this cross section study, are observed at the same point in time, so the utilization of flu shot vaccine may be the result, rather than the cause of poor health. In fact, it is more plausible that an individual with poor health status will receive preventive medical care and immunization by seeing a physician on a regular basis that will encourage him to have preventive test or vaccinations. Then, when interviewed, those who had flu shot display a higher probability of suffering because of bad health. Moreover, the model predicts that the probability of bad health status increases with age and for individuals who faced health problems in the thirty days and in the year before the interview. On the other hand age affects positively the probability of having a healthier life-style.

Vehicular air pollution presents a direct negative impact on the probability of enjoying good health but it has not the expected negative indirect effect on health investment. In fact it turns out that people react to a higher natural health depreciation rate, due to increasing pollution concentration, by investing more in their health. For most healthy people the symptoms of air pollution exposure usually go away as soon as the air quality improves. However, certain groups of people are more sensitive to the effects of air pol-

lution than others. People with heart or lung disease also react more severely to polluted air. During times of heavy pollution, their condition may worsen to the point that they must limit their activities or even seek additional medical care. Probably, individuals, in particular the frailer ones, lead a healthy life-style to increase their health stock to reduce the air pollution symptoms and future damages.

Schooling is positively related to perceived health: a higher degree of education increases the probability of feeling well but it has no significant impact on the probability of suffering from health impairment. Schooling affects health behavior too. There is a clear indication of the allocative effects of schooling, since schooling is related to the life-style variables in a health promoting way: attending a college school, or having a college school degree affects positively exercise and the probability of following an healthy diet. A higher degree of education has a negative impact on cigarettes and alcohol consumption and on the obesity risk.

Marital status has a large impact on the life-style variables. In particular, marriage seems to influence positively healthy habits while being divorced, separated, never married, or an unmarried couple has positive impact on smoke and on alcohol consumption and in general on bad habits.

People in the labor force show a higher probability of enjoying good health and a higher probability of following better health behavior, while those who are involuntarily unemployed exhibit adverse health activities: they smoke more and suffer stress more often than people who are in the labor market. Retired individuals, indeed, follow healthier behaviors.

Referring to the household composition variables we can observe that the presence of children less than eighteen years old has is negatively correlated to cigarettes and alcohol consumption. An increasing number of men in a household is negatively correlated to the healthy habits: it increases the probability of people choosing to drink heavily and the probability of being overweight or obese but has a positive impact on exercise.

Last, health insurance increases the probability of good health perception. It has a significant impact on the individual behavior: it decreases the probability of smoking and of following an unhealthy diet it reduces the probability of being stressed and it encourages the use of preventive care.

TABLE 5 shows the estimated statistically significant correlation coefficients between the disturbance of the nine equations system. The null of exogeneity is rejected in seventeen cases[13]. As we can note there exists a statistically significant correlation between the disturbance of the health impairment equation and the equation for smoke, diet, exercise and stress. Then, unobservable that increase the likelihood of bad health, increase the probability of doing physical exercise and the probability of following a correct diet with fruits and vegetable, while it decreases the probability of smoking and of being stressed.

The negative coefficients concerning smoke and stress and the positive correlation coefficients on exercise and diet show that individuals with poor health tend to adopt healthier behaviors with respect to individuals with better health who tend to adopt an unhealthy behavior. Moreover, there exists a positive correlation between SAH equation disturbance and the disturbance of smoke equation that is consistent with the above results.

[13]The statistically significant correlation coefficients suggest that the null hypothesis of nine univariate probit model or the hypothesis of independence across the error terms of the nine latent equations, can be rejected, and multivariate probit model is a better model for the observed data.

SAH and Blood Pressure Equations

TABLE 4a-4b present the results for the system in which perceived health is measured again by SAH and health outcome is measured by another important indicator of overall health that is blood pressure. Starting with the endogenous variables, regular exercise has the expected significant positive effect on the probability of feeling well, while it has a negative but not significant impact on the probability of suffering from high blood pressure. Smoking behavior and alcohol consumption decrease the probability of perceiving good health. Smoke does not influence the likelihood of suffering from high blood pressure, while alcohol has a positive significant effect on this health condition. This result seems surprising since blood pressure is often related not only to the adverse health effects of alcohol but also of smoking behavior: the nicotine in cigarettes and other tobacco products causes blood vessels to constrict and heart to beat faster, which temporarily raises blood pressure. It is well known that quitting smoking can significantly lower the risk of heart disease and heart attack, as well as help lower blood pressure. Obesity and stress variables show a significant negative effect on SAH and increase the likelihood of suffering from high blood pressure. In this model, as the previous one, flu shot variable shows a negative coefficient on SAH and a positive coefficient on blood pressure but the coefficients are not statistically significant.

The probability of perceiving bad health increases with pollution. Again pollution has a positive impact on the health investments: a higher pollution concentration decreases the probability of smoking, of being obese and of suffering because of stress or mental problems. Moreover, if outdoor pollution increases individuals will spent more time doing

physical exercise and will consume more preventive medical care. Then, this model confirms that an increasing level of ambient air pollution will have negative direct effect on the likelihood of good health but it will have a positive impact on healthy behavior.

The effect of schooling on health is similar across the two health models: those with more schooling are observed to display a higher probability of perceiving good health but a higher degree of education has no significant impact on blood pressure. This model also shows that a higher degree of education helps individuals to choose healthier life-style: more schooling increases the probability of vigorous physical activities and increases the probability of following an healthy diet. On the other hand it has a negative influence on the probability of consuming hazardous goods and of being obese.

Referring to predisposing variables, the probability of enjoying good health decreases with age due to higher health depreciation rate and to higher morbidity risks. On the other hand age has a positive impact on healthy habits. Being white relative to other race is associated with a greater probability of perceiving good health and a lower probability of suffering from high blood pressure. Being female has not significant effect on SAH and blood pressure outcome.

Concerning the other estimated coefficients we find similar results to the SAH- activity limitations model: being married and being in the labor force leads to more healthy habits. Young children is negatively correlated the probability of hazardous goods consumption whereas the presence of man is positively correlated to it. Men have a positive influence on the probability of doing regular exercise but also a positive influence on the probability of being obese or overweight and a negative impact on having immunization.

TABLE 6 shows the statistically significant estimated correlation coefficients between blood pressure, SAH and life-style variable equations. A positive and a negative significant correlation exists respectively between the SAH equation disturbance and the disturbance of the equation for smoke and diet: some unobservable that increases the likelihood of perceiving good health increases the probability of consuming cigarettes while unobservable that increases the probability of feeling well decreases the likelihood of a healthy diet. The negative correlation coefficient concerning diet and the positive correlation coefficient between SAH and smoke disturbances show that people who enjoy good health tend to behave in an unhealthy way and to invest less in their health than frailer people. This result is consistent with the findings of the previous model. We can conclude that individuals with poor health status try to counteract to the greater deterioration of their health, due to a higher health depreciation rate, by behaving in a healthier way, encouraged by the fact that the marginal product of their investment in health will be higher the more the illness or the pathological condition is severe.

2.5 Summary and Conclusions

This chapter develops and applies a Grossman-style health production model set up in discrete time to explain how environmental pollution, life-style, and chronic conditions combine to affect the health capital stock and health investment decisions. The quality of the environment turns out to affect both health capital and health investments. According to our results a higher concentration of carbon monoxide has respectively a negative impact on the probability of enjoying good health and a positive influence on healthy habits.

Then, concerning vehicular air pollution our results do not support Cropper's (1981) model: people living in polluted areas tend to invest more in health probably to counteract to the deterioration of a higher depreciation rate due to an increasing pollution. Arguably, people lead a healthy life-style to increase their health stock and build up resistance against pollution symptoms and future damages.

What may at first seem surprising is that the partial effect of CO on health is relatively small. However, in estimating the relationship between vehicular pollution and health, we have not considered that pollution exposure may be endogenously determined: people with high preferences for clean air may choose to live in areas with better air quality and far from areas in which vehicular traffic is more intense. On the other hand households can respond to an increasing level of outdoor pollution, for instance, by avoiding exposure or mitigating the effects of the exposure once they occur (Cropper and Oates, 1992). If people respond to a higher pollution concentration by increasing the avoidance behavior or by mitigating the effects, for instance, trough curative care to the point that health actually improves, not controlling for this aspect may yield estimates that are lower bounds of the true effect (Neidell, 2004).

Suffering from a pathological condition affects both health stock and health investments. We can conclude that individuals with poor health status, react to the greater deterioration, due to a higher health depreciation rate, by behaving in a healthier way. The investments are encouraged by the fact that the marginal product of their investments will be higher the more illness is severe.

The theoretical and the empirical results support the idea that life-style, as mea-

sured by smoking, alcohol consumption, dietary habits, physical activity, prevention, obesity and stress, is one of the driving factors for good health. Healthier habits are associated to a higher probability of enjoying good health in both SAH-activity limitations and SAH-blood pressure model.

Schooling represents a fundamental factor in determining the individual health too: the empirical results show that more educated individuals are significantly less likely to report a perceived bad health status. Moreover, education has a heavy impact on the health behaviors: more educated individuals are often informed about the long-term consequences of smoking, of lack of exercise of a bad nutrition. Hence, schooling helps people to choose a healthier life-style by improving their knowledge of the relationship between health behaviors and health outcomes. Then, additional education trough education programs would have positive effects on the overall health of the population.

Another important factor that the above models predict is that family structure has a great importance for individual behavior: those married are found to have healthier life-styles than singles or divorced. Married men and women are less likely to have drinking problems, are less likely to smoke and develop mental problems. These results are consistent with the marriage protection hypothesis that states that the actual process of living with a spouse confers benefits to both partners; the married state involves environmental, social, and psychological factors that make it a healthier state than an unmarried one.

Appendix I: Tables

Table 1: Variables Name and Definition

Variables Name	*Variables Definition*
ghealth	1 if current health is excellent, very good or good health, 0 otherwise
bloodpressure	1 if has high blood pressure, 0 otherwise
ac_limit	1 if has limited in any activities because of health problems, 0 otherwis
smoke	1 if is current smoker, 0 if does not smoke
alcohol	1 if is at risk for heavy drinking, 0 otherwise
diet	1 if consumes fruits/vegetables at least once per day, 0 otherwise
execise	1 if participates in physical activity in the last 30 days, 0 otherwise
obese	1 if is at risk for overweight or obesity (BMI >25.0000), 0 otherwise
stress	1 if mental health (including stress) was not good, 0 otherwise
flushot	1 if has flu shot in the 12 months before the interview, 0 otherwise
hmo	1 if has health care coverage , 0 otherwise
element	1 if elementary school or Kindergarden, 0 otherwise
education	1 if had high_school, master or PhD degree
high_sch	1 if attend high school or high school graduate, 0 otherwise
expenditure	total annual health care expenditure
collg	1 if attend college or college graduate, 0 otherwise
married	1 if married, 0 otherwise
divorce	1 if divorced, 0 otherwise
widow	1 if widow, 0 otherwise
seprd	1 if sepatated,0 otherwise
never_married	1 if never married, 0 otherwise
unmar_couple	1 if member of an unmarried couple, 0 otherwise
alcohol	1 if unable to work, 0 otherwise
retd	1 if retired, 0 otherwise
stdnt	1 if student, 0 otherwise
home_make	1 if homemaker, 0 otherwise
out_work	1 if out of work, 0 otherwise
self_emp	1 if self-employed, 0 otherwise
white	1 if White, 0 otherwise
black	1 if Black, 0 otherwise
	1 if other race, 0 otherwise

Table 2: Summary Statistics

	Means	St.Deviation
ghealth	0.8585	0.3485
bloodpressure	0.2573	0.4372
ac_limit	0.1508	0.3579
smoke	0.2369	0.4252
alcohol	0.0458	0.2091
diet	0.9668	0.1791
exercise	0.7689	0.4215
obese	0.5513	0.4974
stress	0.2801	0.4491
flushot	0.3513	0.4774
hmo	0.9259	0.2619
element	0.0228	0.1493
high_sch	0.3672	0 .4821
collg	0 .3173	0.4655
married	0.5268	0 .4993
divorce	0.1333	0.3399
widow	0.0995	0.2994
seprd	0.0236	0.1519
never_married	0.1997	0.3997
unmar_couple	0.0171	0.1296
unable	0.0324	0.1769
retrd	0.1993	0.39949
stdnt	0 .0318	0.1754
home_make	0 .0662	0.2486
out_work	0 .0256	0 .1581
self_emp	0.0679	0.2517
employed	0.5769	0.4941
white	0.6592	0.4740
black	0.0584	0.2345
other_race	0.2823	0.4501
age	46.8396	17.2957
male	0.4276	0.4948
children	0.3812	0.4857
nummen	0.8966	0.6767
numwomen	1.0071	0.5881
co_aqi	57.0767	17.7944
physhlth	0.3002	0.4584
chronic_symp	0.2123	0.4089

Table 3a) Estimatated Partial effects SAH- Activity Limitations Model

	1) gheatlh	2) ac_limit	3) smoke	4) alcohol
smoke	-0.1206 (0.000)	0.1668 (0.000)		
alcohol	0.0520 (0.298)	-0.0297 (0.684)		
exercise	0.1297 (0.000)	-0.3268 (0.000)		
diet	0.0142 (0.801)	-0.0135 (0.888)		
obese	-0.1002 (0.000)	0.1166 (0.001)		
flu shot	-0.0771 (0.007)	0.0582 (0.135)		
stress	-0.1049 (0.002)	0.2952 (0.000)		
co_aqi	-0.0031(0.047)	0.003 (0.001)	-0.003 (0.000)	-0.0007 (0.474)
hmo	0.0794 (0.000)	0.0049(0.884)	-0.1567 (0.000)	0.0202 (0.667)
ac_limit	-0.2287 (0.000)			
element	-0.2242 (0.000)	0.0272(0.585)	-0.0085 (0867)	0.0533 (0.539)
collg	0.0919 (0.000)	0.01 (0.631)	-0.1743 (0.000)	-0.0701 (0.015)
divorce			0.1296 (0.000)	0.1469(0.000)
widow			0.0589 (0.062)	-0.0736 (0.267)
seprd			0.1439 (0.001)	0.1803 (0.009)
never_married			0.0636 (0.004)	0.0814 (0.026)
unmar_couple			0.1362 (0.006)	0.1246 (0.119)
retd			-0.0998 (0.001)	0.0181 (0.719)
stdnt			-0.1753 (0.000)	0.0052 (0.274)
home_make			-0.018 (0.563)	-0.068 (80.274)
out_work			0.0907 (0.032)	0.0549 (0.433)
self_emp			0.0110 (0711)	-0.0248 (0.636)
unable			0.2058 (0.000)	-0.1935 (0.048)
black	-0.0051(0.865)	-0.0003 (0.992)		
other race	-0.0657 (0.003)	-0.1099 (0.000)		
age	-0.0134 (0.000)	0.0099(0.000)	-0.0049(0.000)	-0.0043(0.010)
male	-0.0174 (0.248)	0.0336 (0.057)		
children			-0.0499 (0.084)	-0.0848 (0.034)
nummen			0.0438 (00.18)	0.0635 (0.007)
numwomen			-0.0288 (0.167)	0.0000 (0.820)
physhlth	-0.1805 (0.000)			
chronic_symp	-0.0963 (0.000)			

p-values in parentheses.

TABLE 3b: Estimatated Partial effects SAH- Activity Limitations Model

	5) diet	6) exercise	7) flu shot	8) obese	9) stress
smoke					
alcohol					
exercise					
diet					
obese					
flu shot					
stress					
co_aqi	0.0008(0.626)	0.0029(0.001)	0.0043(0.000)	-0.0039(0.000)	-0.0089(0.000)
hmo	0.0559 (0.030)	0.0493(0.017)	0.121(0.000)	0.0053 (0.815)	-0.0549(0.040)
ac_limit					
element	0.0107 (0.866)	-0.1288 (0.001)	-0.2421(0.080)	-0.2351 (0.998)	-0.245 (0.895)
collg	0.0742 (0.000)	0.1030 (0.000)	-0.004 (0.787)	-0.0549 (0.000)	-0.0256 (0.105)
divorce	-0.0087(0.733)	-0.0129(0.482)	-0.0758 (0.001)	-0.0273(0.150)	0.0579 (0.009)
widow	-0.0197(0.668)	-0.0015 (0.946)	-0.0421 (0.122)	-0.0799 (0.001)	0.113 (0.000)
seprd	-0.0205 (0.721)	-0.0176 (0.636)	-0.0215 (0.642)	0.0633 (0.087)	0.0728 (0.097)
never_married	-0.1019 (0.000)	-0.0243 (0.182)	-0.0048 (0.822)	-0.0109 (0.539)	-0.0212 (0.329)
unmar_couple	-0.0369 (0.581)	0.07 (0.122)	-0.0364 (0.529)	-0.0372 (0.414)	0.133 (0.006)
retd	0.0924 (0.005)	0.0236 (0.246)	0.1216 (0.000)	0.0603 (0.007)	-0.0261 (0.367)
stdnt	0.0947 (0.030)	0.0736 (0.038)	0.0396 (0.341)	-0.0799 (0.026)	-0.0002 (0.996)
home_make	0.1047 (0.009)	0.0064 (0.782)	-0.0658 (0.030)	-0.0797 (0.001)	0.0339(0.225)
out_work	-0.0207 (0.666)	-0.0643 (0.080)	-0.0411 (0.378)	0.0339 (0.352)	0.1637 (0.000)
self_emp	0.0441 (0.217)	0.0516 (0.024)	-0.0989 (0.001)	0.0061 (0.794)	0.0093 (0.743)
unable	-0.0806 0.107)	-0.3121 (0.000)	0.0338 (0.408)	0.0738 (0.032)	0.286 (0.000)
black					
other race					
age	0.0024(0.416)	-0.0075(0.000)	0.0121(0.000)	0.0058(0.000)	-0.0107(0.000)
male					
children	0.01816 (0.798)	-0.0584 (0.126)	-0.1118 (0.000)	0.0693 (0.024)	0.0036 (0.900)
nummen	-0.0017 (0.968)	0.0683 (0.008)	-0.0455 (0.026)	0.0695 (0.001)	-0.035 (0.066)
numwomen	0.0339 (0.497)	-0.0213 (0.437)	0.0056 (0.792)	-0.0252 (0.253)	0.0394 (0.060)
physhlth					
chronic_symp					

p-values in parentheses.

TABLE 4a: Estimatated Partial Effects of SAH- Blood Pressure Model

	1)gheatlh	2) blood pressure	3) smoke	4)alcohol
smoke	-0.1478 (0.000)	-0.0313 (0.434)		
alcohol	0.0644 (0.182)	0.1405 (0.040)		
exercise	0.1381 (0.000)	-0.0766 (0.060)		
diet	0.0072 (0.900)	0.101 (0.192)		
obese	-0.0896 (0.001)	0.2155 (0.000)		
flu shot	-0.0672 (0.020)	0.1545 (0.000)		
stress	-0.1095 (0.001)	0.0977(0.007)		
co_aqi	-0.004 (0.012)	0.0003 (0.974)	-0.003 (0.000)	-0.0007 (0.470)
hmo	0.0767 (0.000)	-0.0078 (0.816)	-0.1576 (0.000)	0.0187 (0.690)
bloodpressure	-0.0947 (0.003)			
element	-0.2171 (0.000)	0.0455 (0.331)	-0.0040 (0.938)	0.0555 (0.521)
collg	0.0849 (0.000)	-0.0694 (0.000)	-0.1760 (0.000)	-0.071 (0.014)
divorce			0.1259 (0.000)	0.1446 (0.000)
widow			0.0602 (0.058)	-0.0742 (0.264)
seprd			0.1415 (0.001)	0.1811 (0.008)
never_married			0.062 (0.005)	0.0803 (0.028)
unmar_couple			0.1362 (0.006)	0.1297 (0.104)
retd			-0.1086 (0.000)	0.0125 (0.803)
stdnt			-0.1798 (0.000)	-0.0772 (0.260)
home_make			-0.0183 (0.560)	-0.0686 (0.262)
out_work			0.0821 (0.053)	0.051 (0.466)
self_emp			0.0089 (0.767)	-0.0253 (0.628)
unable			0.1736 (0.000)	-0.2044 (0.031)
black	0.0081(0.784)	0.1206 (0.000)		
other race	-0.041(0.048)	0.0241 (0.270)		
age	-0.0126 (0.000)	0.0185 (0.000)	-0.0048 (0.000)	-0.0042 (0.012)
male	-0.0263 (0.080)	0.02 (0.212)		
children			-0.0544 (0.059)	-0.0845 (0.034)
nummen			0.045 (0.016)	0.0649 (0.006)
numwomen			-0.0271 (0.194)	0.0000 (0.830)
physhlth	-0.2202 (0.000)			
chronic_symp	-0.1656 (0.000)			

p-values in parentheses.

TABLE 4b: Estimatated Partial Effects of SAH- Blood Pressure Model

	5)diet	6)exercise	7) flu shot	8) obese	9) stress
smoke					
alcohol					
exercise					
diet					
obese					
flu shot					
stress					
co_aqi	0.0009 (0.596)	0.0029(0.001)	0.0044 (0.000)	-0.0039 (0.000)	-0.0089 (0.000)
hmo	0.0566 (0.027)	0.0526 (0.011)	0.1179 (0.000)	0.0042 (0.853)	-0.0583(0.030)
hgbloodpress					
element	0.0084 (0.895)	-0.139 (0.000)	-0.2357 (0.078)	-02284 (0.947)	-0.2383 (0.760)
collg	0.0753 (0.000)	0.1046 (0.000)	-0.0042 (0.775)	-0.0557 (0.000)	-0.0373 (0.086)
divorce	-0.0057 (0.847)	-0.0049 (0.790)	-0.0779 (0.001)	-0.0297 (0117)	0.0506 (0.024)
widow	-0.0232 (0.615)	-0.0016 (0.994)	-0.0415 (0.125)	-0.0797 (0.001)	0.1167 (0.000)
seprd	-0.0127 (0.824)	-0.0141 (0.710)	-0.019 (0.680)	0.0632 (0.088)	0.0686 (0.123)
never_married	-0.1048 (0.000)	--0.0238 (0.197)	-0.0083 (0.694)	-0.0122 (0494)	-0.0238(0.279)
unmar_couple	-0.0276 (0.677)	0.0663 (0.149)	-0.0306 (0597)	-0.0345 (0.450)	0.1357 (0.006)
retd	0.0948 (0.004)	0.0387 (0.053)	0.1167 (0.000)	-0.657 (0.003)	-0.0418 (0.152)
stdnt	0.095 (0.028)	0.079 (0.026)	0.0383 (0.356)	-0.0826 (0.022)	-0.0077 (0849)
home_make	0.1027 (0.010)	-0.0053 (0.822)	-0.0656 (0.029)	-0.0797 (0.001)	0.0339 (0.231)
out_work	-0.0107 (0.820)	-0.0437 (0.233)	-0.045 (0.331)	0.0284 (0.436)	0.1505 (0.000)
self_emp	0.0443 (0.215)	0.0528 (0.022)	-0.1027 (0.001)	0.0042 (0.858)	0.0057 (0.843)
unable	-0.0428 (0.322)	-0.2236 (0.000)	0.0336 (0.378)	0.0583 (0.076)	0.2464 (0.000)
black					
other race					
age	0.0024 (0.430)	-0.008 (0.000)	0.0123 (0.000)	0.006 (0.000)	-0.0106 (0.000)
male					
children	0.0273 (0.698)	-0.0463 (0231)	-0.1112 (0.000)	0.0670 (0.000)	-0.0049 (0.864)
nummen	-0.004 (0.927)	0.0652 (0.012)	-0.044 (0.030)	0.0711 (0.001)	-0.0327 (0.088)
numwomen	0.3 (0546)	-0.2468 (0.375)	0.0069 (0.745)	-0.0235 (0.745)	0.0425 (0.044)
physhlth					
chronic_symp					

p-values in parentheses.

TABLE5: Estimated Correlation Coefficients SAH-

-Activity Limitations Model

correlation	coefficients
rho31	0.1249 (0.0019
rho32	-0.1784 (0.004)
rho52	0.3249 (0.000)
rho92	-0.2795 (0.000)
rho43	0.2763 (0.000)
rho53	-0.1086 (0.014)
rho63	-0.1113 (0.000)
rho73	-0.1206 (0.000)
rho83	-0.0755 (0.000)
rho93	0.0762 (0.000)
rho74	-0.73 (0.022)
rho94	0.917 (0.007)
rho56	0.2488 (0.000)
rho76	0.0716(0.005)
rho86	-0.0656 (0.005)
rho96	-0.745(0.004)
rho97	-0.0511(0.044)

p-values in parentheses.

TABLE 6: Estimated Correlation Coefficients SAH-

-Blood Pressure Model

correlation	coefficients
rho31	0.1408 (0.008)
rho51	-0.1088 (0.043)
rho72	-0.1758 (0.004)
rho43	0.2838 (0.000)
rho53	-0.1705 (0.000)
rho63	-0.1057 (0.000)
rho73	-0.1299 (0.000)
rho83	-0.081 (0.000)
rho93	0.0734 (0.004)
rho74	-0.0772 (0.019)
rho94	0.0878 (0.007)
rho56	0.2505 (0.000)
rho76	0.0779 (0.002)
rho86	-0.0618 (0.008)
rho96	-0.0606 (0.018)
rho98	-0.0517 (0.041)

p-values in parentheses

Chapter 3

On the Anatomy of Adverse Selection in Health Insurance Market: Evidence from the MEPS

3.1 Introduction

Since the seminal papers by Akerlof (1970) and by Rothschild and Stiglitz (1976) greater attention has been devoted to the problem of asymmetric information among agents. An important form of asymmetric information between consumers and insurers is adverse selection. In health insurance market adverse selection may occur when consumers' true health-cost risk is private information: insurance companies may know that consumers vary in the level of risk, but, in principle, are not able to discern who are high and who are low risk profile individuals within a group of potential insured. Identifying risks accurately is

not an easy task and requires that insurance companies incur some costs. Insured parties are heterogeneous in terms of expected costs and have more information about their risks. Naturally, high-risk individuals are not encouraged to "reveal" their risk; this asymmetry is a serious problem since may lead insurance companies to face large differences in expected health costs due to heterogeneity in demographics and the incidence of illness.

As the insurers has imperfect information on the individuals' health status, the cover and the premium will be set somewhere between what is required by the low and the high risk profile users. However, low risk users may feel they are paying too much with respect to their needs. Low risk individuals tend to drop out of the risk pool, then, the average risk in the pool rises causing premium to rise and yet more people to drop out and so on. This may leave to the case in which only high risk profile individuals buy insurance and pay "average" rate.

To counteract to this problem, insurance companies may offer separate contracts with different coverage and prices, making claimant pay part of the claim (with coinsurance rate, deductible etc.) so that individuals should reveal their risks. Hence, risky individuals who expect high health care costs would tend to purchase insurance with higher premium but lower excesses since they are more likely to be claiming on a regular basis. On the other hand lower risk users, who expect low costs, would prefer a less complete insurance, with a lower premium and a higher excess in the unlikely event that they have to claim.[1] The phenomenon described above is known as *ex ante adverse selection*" (Fang et.al, 2006)[2].

[1]This form of allocation has been proved superior (in terms of economic efficiency) to that in which a mean price is paid by all individuals. The main work in this area is attributed to Rothschild and Stiglitz (1976).

[2]This is also known as adverse selection effect à la Rothschild-Stiglitz: high risk agents, knowing they are more likely to have an accident, self-select by choosing contracts entailing a more comprehensive coverage.

The "*positive correlation property*" between the individual riskiness and the completeness of a health insurance plan, which characterize this phenomenon, forms the basis for our empirical test for adverse selection. This test is conducted by using data from the 2003/2004 Medical Expenditure Panel Survey – Household Component (MEPS-HC) in conjunction with the previous year's National Health Interview Survey (NHIS). Many empirical work use information on coinsurance rate, health insurance benefits, stop-loss and deductible to measure generosity of health insurance plan (see, for instance, Browne and Doerpinghaus, 1993). Unfortunately our data do not contain information about the insurance contract ; hence, we measure health insurance plan completeness by using health insurance reimbursement that is the difference between total health expenditure and out-of-pocket expenditure on health care paid by consumers.

Health insurance reimbursement, however, is only defined for a subset of individuals from the overall population since we observe it only for those who participate in insurance and have positive health care expenditure. Thus, the model may suffer from sample selection bias and straightforward regression analysis may lead to inconsistent parameters estimate. Another problem that arises from the estimation is the presence of unobserved heterogeneity in the equations of interest. Wooldridge (1995) has proposed an estimator which deals with both sources of estimation bias. We extend this estimation method to the case in which selectivity is due to two sources rather than one (participation in insurance and participation in health care expenditure). The nature of the test is similar to the one in Browne and Doerpinghaus (1993).

We find no systematic relation between illness of individuals and insurance choice.

We think that a possible explanation can be found in the so called "cream skimming" practise: health plans may have an incentive to alter their policy to attract the healthy and repeal the sick (Newhouse, 1996; Ellis, 1997). Then, individuals enrolled are relatively healthy people and this lead to the failure of the correlation test.

The remainder of this chapter is organized as follows. Section 2 briefly surveys the empirical related literature. Section 3 describes the data and variables. In Section 4 we perform the empirical analysis, explain the test in detail and present our main results. Section 5 concludes the paper with a discussion. The definition of the variables, descriptive statistics and tables with estimation coefficients are in Appendix .

3.2 Related Literature

There is substantial empirical literature examining adverse selection in health insurance markets. However, there is conflicting evidence on the presence of adverse selection: the results are mixed. We briefly summarize these studies here.

Cameron and Trivedi (1991), for instance, use Australian data to estimate a logit model for the choice between a standard package and a more generous insurance plan. They find no significant effect of health condition variables on insurance choice. Browne and Doerpinghaus (1993) find evidence for adverse selection: their results show that low and high risks purchase a pooling insurance policy and low risks subsidize the insurance purchase of high risk insured individuals. This supports the prediction by Miyazaki's (1977) theory of adverse selection. Cardon and Hendel (2001) test for a correlation between health care

spending and insurance coverage using a two-stage model of the demand for health insurance. In their setup, individuals first receive a private signal that is correlated with their future health. Based on this signal individuals make their choice about how much insurance to purchase. In the second stage, individuals consume health care. Their empirical analyses revealed that the joint insurance/health care consumption decision is largely explained by observed characteristics (such as income, education etc.) rather than unobserved health status. Thus, they conclude that apparently there is no private information that insureds can use against the insurers and hence no adverse selection.

Bajari et al. (2006) use the Health Retirement Study to estimate a structural model of the demand for health insurance and medical care. They find evidence of moral hazard but not of adverse selection. Goldman et al. (2006) estimate independent effects of medical and drug benefits on plan selection. They find that while generosity of the medical benefit played an important role in choosing a plan, choices did not vary significantly by health status. In contrast, their data support a significant correlation between health status and plans with generous drug benefits: sicker individuals tend to enroll in plans with generous drug benefits, while healthier choose less generous plan. Based on their findings, they assert that drug coverage may be more susceptible to adverse selection than medical coverage .

In insurance markets other than health, evidence for adverse selection is considerably contradictory too. Puelz and Snow (1994) presented empirical evidence of adverse selection in the market for automobile collision insurance. Using data from a private insurer, they find strong evidence of adverse selection in the insurer's portfolio. Chiappori

and Salanié (2000) use data on contracts and accidents to examine the extent of asymmetric information in the French market for automobile insurance. They examine a relatively homogenous group of drivers with less than four years' driving experience. Their test do not reveal evidence of risk-related adverse selection. They find that when choosing their automobile insurance contracts, individuals behave as though they had no better knowledge of their risk than insurance companies, which is in contrast with what the adverse selection hypothesis would require.

Cawley and Philipson (1999) test for adverse selection in the market for life insurance; they first show that the death rate among those who purchase life insurance is lower than those who do not, moreover they find that who expect to die soon do not buy more complete life insurance plan. This is clearly in contrast with the basic adverse selection theory.

Finally, Makki and Somwaru (2001) analyze farmers' choices of crop insurance contracts. Their analysis offers empirical evidence of adverse selection by showing that high-risk farmers are more likely to select revenue insurance contracts and higher coverage levels with respect to low–risk farmers.

Most of the studies we have come across have used discrete choice models to represent the health insurance purchase decision. They have used logit or probit specifications to analyze a decision where the dependent variable has two outcomes: buying or not buying health insurance. Few studies have gone to the next level and tried to explain which factors affect the extent of insurance purchase. Moreover, in most of the studies that test for adverse selection two important estimation issues such as unobserved heterogeneity and

selection bias, are typically treated separately[3]. The aim of this paper is to find factors which affect the extent of insurance purchase with particular attention to individuals' risk profile. In our model, we control for selection bias and at the same time for unobserved heterogeneity issue. The simultaneous account taken of both possible sources of bias seems to be relatively new for this kind of application.

3.3 Data and Variables

We use data from the 2003/2004 Medical Expenditure Panel Survey – Household Component (MEPS-HC) and 2002 National Health Interview Survey (NHIS). MEPS is an on-going survey sponsored by the Agency for Healthcare Research and Quality (AHRQ). MEPS provides a nationally representative sample of US civilian non-institutionalized population. MEPS is self-reported and contains detailed information on health care consumption and demographics including age, sex, marital status, income, work status and geographic location. In addition data contain information on the respondents' health status, health conditions, health charges and payments, access to care, health conditions, health insurance coverage.

Each year's sample for MEPS is drawn from respondents to the previous year's NHIS that is conducted annually by the National Centers for Health Statistics (NCHS), Center for Disease Control and Prevention (CDC). NHIS provides rather detailed information about health status, diseases, life-style, education and other individual characteristics.

[3]It is often mathematically complex to combine these two issues together, a large burden of computer programming and a set of strong distributional assumptions are needed for the combination. The model presented in this paper, however, is estimated with the common statistical software STATA 9. Also the statistical assumptions needed for Wooldridge's model in this paper is relatively weaker than the other methods.

We use the 2002 NHIS in conjunction with 2003/2004 MEPS with MEPS as our primary database because it contains information on health insurance reimbursement, which is the dependent variable of interest in this paper, as well as detailed information on health insurance.

After correcting for the missing values, the sample contains 890 individuals resulting in 1780 observations. Observations containing veterans and individuals who are covered by Champus/ ChampVa insurance are removed from the data set since their medical services demand and access to medical services differ distinctly from the general population[4].

Table 2 presents summary statistics for demographics and health insurance information. The sample of 1780 individuals is divided into insured and uninsured. Only 8% of the sample is uninsured. As showed in Table 2, uninsured are younger, and poorer. Health care expenditure is important relative to total income, around 11% for insured and 13% for uninsured. The average expenditure in the whole sample is 4,300 $. The distribution of the expenditures is highly skewed, as expected. Insured spent 50% more in health care than uninsured (4314$ versus 2001 $).

Table 2 shows that 89% of insured report that their health is good versus the

[4]The health care system in US is characterized by: private insurance, Medicare and Medicaid and Military health insurance.

Private medical insurance is the largest component of the health care system: insured pay a fee-for-service reimbursement basis; they pay directly the medical treatments and be reimbursed at a later date by the insurer. Medicare is a program funded by the government through social security payments. It was created mainly for people 65 years of age and older, some disabled people under 65 years of age, and people with end-stage renal disease. This scheme is extremely basic with very few services offered with much of the cost still having to be met by the patient. Since Medicare has a number of gaps in coverage, most of the enrolled buys own supplemental insurance coverage.

Medicaid is funded jointly by the federal and state authorities and is available for individuals of all ages and families with low incomes and resources who cannot afford proper medical care.

Champus (now known as Tricare) is a health care benefits program for active duty and retired members of the military.

ChampVa is a health care benefits program for permanently disabled veterans and their dependents.

82% of uninsured. Insured behave in a healthier way: the percentage of smoker and the percentage of heavy alcohol consumers are lower; on average they present a lower BMI, and they practice physical activity more often than uninsured.

At first sight, it seems that there are no symptoms consistent with adverse selection: a substantial fraction of the sample is insured and among insured about 90% of individuals enjoy good health[5].

3.3.1 Risk Profile Variables

To perform the correlation test, first we classify individuals as being high and low risk profile individuals. Individuals are classified as being low-risk if their health status is good. As a measure of health status we use two indicators: a subjective and an objective one. In particular, following Berger and Leigh (1988), we choose blood pressure as an indicator of overall health, since it is the most important predictor of cardiovascular disease which is the greatest killer in the U.S. We create a binary variable (hypertension) that takes value one if respondents suffer from high blood pressure and zero otherwise. We classify individuals as high-risk profile individuals if they report that they suffer from hypertension. Moreover, we use as a measure of overall health SAH (self-assessed health)[6], which is a five

[5]A possible explanation of the higher percentage of healthy individuals among insured can be found in the insurance plan characteristics. Plans may have been designed to distort their offering to attract the healthy and repel the sick.

Seeking favorable risk is often referred as cream skimming. These strategic behavior can take a variety of forms including designing insurance benefits packages in such a way as to be more attractive to healthy persons than unhealthy by, for instance, excluding particular prescription drugs or offering health club memberships which appeal to the low risks. The result is that individuals enrolled in health insurance are relatively healthy people.

[6]Self- reported health status is a very good indicator of overall health. It has been showed to be an important predictor of subsequent mortality and medical services use, and is widely used as a measure for the stock of health in pervious studies that analyze empirical determinants of health. (Contoyannis and Jones 2004, Contoyannis et al. 2004).

category variable rating from poor to excellent. We construct a binary variable (health) with the value one if individuals report that their health status is excellent, very good, good and zero otherwise (fair or poor). Then, we classify as high-risk individuals those whose self-reported health is fair or poor.

In addition, individuals are classified as being characterized by a high-risk profile if they follow an unhealthy life-style. Life-style variables measure the effort that individuals use to prevent an illness and at the same time they are good predictor of future illness. The behavioral variables employed are indicator of smoking, alcohol consumption, physical activity practice and BMI[7]. Individuals are classified as being characterized by a high risk profile if they smoke, usually consume heavy drinks, practice vigorous physical activity less than once per week and if their reported BMI is higher than 25.0000.

3.3.2 Other Characteristics

In addition to the health and life-style indicators, the independent variables, used to control for differences in policy, can be grouped in the following categories: demographic variables (age, sex, race), socioeconomic variables (education, marital status, employment status, income) preferences (risk aversion). Moreover, we control for total annual expenditure, out-of-pocket annual premium and whether individuals suffer from any form of disabilities that limit their activities (such as working, studying etc.).

Because older individuals tend to use more medical services and may have higher medical expenditure, we expect a positive relationship between age and the amount of

[7]BMI (Body Max Index) is used as measure of obesity. Obesity is considered a risk factor for several diseases. It is often associated with aspects of an individual's life-style such as an insufficient physical activity and inappropriate nutrition. Those who are a BMI $>$ or equal than 25.0000 are overweight and at risk of obesity and are expected to have poorer health.

reimbursement. Since males tend to use less medical services than females we expect a negative correlation between males and health insurance reimbursement. A positive relationship between the variables black and other race and the completeness of coverage is expected because of the higher need of medical services among non whites caused by a higher morbidity rate.

According to the "marriage protection hypothesis" (which states that the actual process of living with a spouse confers health benefits to both partners) we expect that married people tend to use less medical services. Thus, we expect a negative correlation between the variable "married" and the dependent variable that measure the generosity of health plan.

The variables which are indicators of education, employment status and income are included in the analysis to account for differences, other than risk type, which may affect the amount of insurance purchase by the insured. We expect a negative relationship between the degree of education and the amount of insurance purchased: individuals with a higher level of schooling are observed to be healthier than the others[8]. Hence, we expect that individuals with a higher degree of education use less medical services and purchase a less complete insurance plan. Similarly, the coefficients for income and employed are expected to be negative.

We include also a measure of risk aversion. Higher risk aversion translates into

[8]One explanation of this empirical regularity is that education increases the productivity of producing health i.e. more health can be produced for the same inputs (Gerdtham et al., 1999, Berger and Leigh, 1989). Schooling helps people choose healthier life-styles by improving their knowledge of the relationship between health behaviors and health outcomes. (Kenkel, 1991). A more educated person may have more knowledge about the harmful effects of cigarette smoking, alcohol consumption or about what constitutes an appropriate, healthy diet. Furthermore, schooling increases information about the importance of having regular exams or screening tests to prevent an illness or at least to minimize disease.

a willingness to pay more to eliminate financial risk. For a given premium, we expect a positive coefficient for the variable that measures risk aversion since more risk-averse insured will be willing to tolerate higher deductible, coinsurance rate, stop-loss than someone who is less risk-averse[9].

The variable that we use as indicator of limited activity controls for the portion of risk observable to the insurer. The activity limitations indicator is expected to be positively related to the generosity of the health insurance plan, because being limited increases the likelihood of need for medical care.

3.4 Estimation Strategies and Empirical Results

3.4.1 Wooldridge Two-Step Estimation

To test for differences in insurance purchases by high and low risk profile individuals we use as a measure of completeness of coverage the natural logarithm of health care reimbursement (total health care expenditure paid by private insurance, Medicare and Medicaid. The assumption of lognormality better fits the expenditure reimbursement and has precedents (see, for example, Keeler et al., 1977, Browne and Doerpinghaus, 1993).

Health insurance reimbursement is only defined for a subset of individuals from the overall population since we observe it only for those who participate in insurance and face positive health care expenditure. Hence the model suffers from sample selection bias

[9]Chiappori and Salaniè in their recent work "Testing for Asymmetric Information in Insurance Markets" stressed the importance of including risk aversion among explanatory variables:

[... *more risk averse drivers tend to both buy more insurance and to drive cautiously; this would even suggest a negative correlation between insurance coverage and accident frequency...*]. Then, if do not control for individuals risk aversion, we may obtain spurious correlation between individuals' risk profile and completeness of coverage.

and straightforward regression analysis may lead to inconsistent parameters estimate.

Another problem that arises from the estimation is the presence of unobserved heterogeneity in the equations of interest. Wooldridge (1995) has proposed an estimator which deals with both sources of estimation bias; this estimator requires panel data and produces consistent parameter estimates under a set of assumptions. It does not impose distributional assumptions about the error terms but requires specifying the functional form of the conditional mean of the individual effects in the structural equation. We extend this method to the case in which selectivity is due to two sources rather than one.

We start by sketching Wooldridge's (1995) sample selection model with one selection criterion, then we present a specification of the model in which the selection process is based on two selection criteria rather than one.

Following M.Rochina-Barrachina (1999), we consider the following problem:

$$\begin{aligned} d_{it}^{*} &= z_{it}\gamma + \mu_i + u_{it} \\ d_{it} &= 0 \quad if \quad d_{it}^{*} \leq 0 \\ d_{it} &= 1 \quad if \quad d_{it}^{*} > 0 \end{aligned} \tag{3.1}$$

$$\begin{aligned} y_{it}^{*} &= x_{it}\beta + \alpha_i + \varepsilon_{it} \\ y_{it} &= y_{it}^{*} \quad if \quad d_{it} = 1 \\ & y_{it} \ not \ observed \ otherwise \end{aligned} \tag{3.2}$$

where equation (3.1) defines the selection rule while equation (3.2) is the primary equation. i $(i = 1, ...n)$ denotes the individuals while t $(t = 1, ..., t)$ denotes the panel. x_{it} and z_{it} are vector of exogenous variables. The dependent variable in the primary equation, y_{it}, is observed only for the observations satisfying the selection rule. Terms μ_i and α_i are

fixed effects[10]. Wooldridge suggests employing Chamberlain (1980) characterization, by assuming the conditional mean of the individual effects in the selection equation as a linear projections on the leads and lags of observable variables:

$$\mu_i = z_{i1}\delta_1 + ... + z_{it}\delta_t + c_i \tag{3.3}$$

where c_i is a random component. By substituting Chamberlain characterization into the selection equation yields:

$$d_{it}^* = z_{it}\gamma + z_{i1}\delta_1 + ... + z_{it}\delta_t + v_{it} \tag{3.4}$$

where $v_{it} = c_i + u_{it}$. v_{it} is distributed independently of z_{it} and is normally distributed with zero mean and σ^2 variance. The regression function of α_i on z_{it} and v_{it} is linear, accordingly:

$$E\left[\alpha_i \left| z_{it}, v_{it}\right.\right] = x_{i1}\psi_1 + ... + x_{it}\psi_t + \phi_t v_{it} \tag{3.5}$$

We do not observe v_{it}, but only the binary indicator d_{it}. Then, we replace $E\left[\alpha_i \left| z_{it}, v_{it}\right.\right]$ with:

$$E\left[\alpha_i \left| z_{it}, d_{it} = 1\right.\right] = x_{i1}\psi_1 + ... + x_{it}\psi_t + \phi_t E\left[v_{it} \left| z_{it}, d_{it} = 1\right.\right] \tag{3.6}$$

Wooldridge assumes that ε_{it} is mean independent of z_{it} conditional on v_{it} and its conditional mean is linear on v_{it}:

$$E\left[\varepsilon_{it} \left| z_{it}, v_{it}\right.\right] = E\left[\varepsilon_{it} \left| v_{it}\right.\right] = \rho_t v_{it} \tag{3.7}$$

By the Law of Iterated Expectation:

$$E\left[\varepsilon_{it} \left| z_{it}, d_{it} = 1\right.\right] = \rho_t E\left[v_{it} \left| z_{it}, d_{it} = 1\right.\right] \tag{3.8}$$

[10]The individual effects are assumed to be the fixed effets rather than the random effects.

From the above assumption, Wooldridge derives an explicit expression for

$$\begin{aligned} E\left[\alpha_i + \varepsilon_{it} \,|z_{it}, d_{it} = 1\right] = E\left[\alpha_i \,|z_{it}, d_{it} = 1\right] + E\left[\varepsilon_{it} \,|z_{it}, d_{it} = 1\right] = \\ = x_{i1}\psi_1 + ... + x_{it}\psi_t + (\phi_t + \rho_t)\, E\left[v_{it} \,|z_{it}, d_{it} = 1\right] \end{aligned} \tag{3.9}$$

where

$$E\left[v_{it} \,|z_{it}, d_{it} = 1\right] = \lambda\left(z_{i1}\gamma_1 + ... + z_{it}\gamma_t\right) \tag{3.10}$$

So, for each period, Wooldridge suggests to estimate a cross-sectional probit model for participation and compute the Inverse Mills Ratio (IMR), then, estimate the structural equation:

$$y_{it} = x_{i1}\psi_1 + ... + x_{it}\psi_t + x_{it}\beta + (\phi_t + \rho_t)\,\lambda\left(z_{i1}\gamma_1 + ... + z_{it}\gamma_t\right) \tag{3.11}$$

by using fixed effect OLS or pooled OLS for the sample for which $d_{it} = 1$ (Vella, 1998).

Concerning the health insurance reimbursement model, we consider the following characterization of Wooldridge's sample selection model where selectivity bias is a function of two indices:

$$\begin{aligned} d^*_{it_1} &= z_{it_1}\gamma_{_1} + \mu_{i_1} + u_{it_1} \\ d_{it_1} &= 0 \quad if \quad d^*_{it_1} \leq 0 \\ d_{it_1} &= 1 \quad if \quad d^*_{it_1} > 0 \end{aligned} \tag{3.12}$$

$$\begin{aligned} d^*_{it_2} &= z_{it_2}\gamma_{_2} + \mu_{i_2} + u_{it_2} \\ d_{it_2} &= 0 \quad if \quad d^*_{it_2} \leq 0 \\ d_{it_2} &= 1 \quad if \quad d^*_{it_2} > 0 \end{aligned} \tag{3.13}$$

$$
\begin{aligned}
y_{it}^{*} &= x_{it}\beta + \alpha_i + \varepsilon_{it} \\
y_{it} &= y_{it}^{*} \quad if \quad d_{it} = 1 \\
& y_{it} \ not \ observed \ otherwise
\end{aligned}
\tag{3.14}
$$

Let d_{it_1} be an unobserved variable denoting insurance participation decision and d_{it_2} an unobserved variable denoting health care expenditure participation decision. z_{it_1} , z_{it_2} and x_{it} are vector of exogenous variables. y_{it} denotes the natural logarithm of health insurance reimbursement. y_{it} is observed only for the sample for which $d_{it_1} = 1$ and $d_{it_2} = 1$. Terms μ_{i_1}, μ_{i_2} and α_i are fixed effects.

Sample selection is now based on two criteria. The method of estimation relies crucially on the relationship between v_{it_1} and v_{it_2}[11], in particular, the estimation depends on whether the two error terms are independent or correlated, that is whether or not $Cov\left(v_{it_1}, v_{it_2}\right) = 0$. The simplest case is when the disturbances are uncorrelated (Maddala,1983, Vella, 1998). If $Cov\left(v_{it_1}, v_{it_2}\right) = 0$ we can easily extend Wooldridge's two-step estimation method to this model. The correction term to include as regressor in the primary equation is:

$$
\begin{aligned}
E\left[\varepsilon_{it} \left| z_{it}, d_{it_1} = 1, d_{it_2} = 1\right.\right] = \rho_{t_1}\lambda_1\left(z_{i1_1}\gamma_{1_1} + ... + z_{it_1}\gamma_{t_1}\right) + \\
+\rho_{t_2}\lambda_2\left(z_{i1_2}\gamma_{1_2} + ... + z_{it_2}\gamma_{t_2}\right)
\end{aligned}
\tag{3.15}
$$

Then, we estimate the following model:

$$
\begin{aligned}
y_{it} = x_{i1}\psi_1 + ... + x_{it}\psi_t + x_{it}\beta + \left(\phi_{t_1} + \rho_{t_1}\right)\lambda_1\left(z_{i1_1}\gamma_{1_1} + ... + z_{it_1}\gamma_{t_1}\right) + \\
+ \left(\phi_{t_2} + \rho_{t_2}\right)\lambda_2\left(z_{i1_2}\gamma_{1_2} + ... + z_{it_2}\gamma_{t_2}\right)
\end{aligned}
\tag{3.16}
$$

[11] From Chamberlain trasformation of the individual effects: $v_{it_1} = c_{i_1} + u_{it_1}$ and $v_{it_2} = c_{i_2} + u_{it_2}$

The procedure consists in first estimating, for each period, by two single a cross-sectional probit model, the selection equation one and the selection equation two. Than, the two corresponding Inverse Mills Ratio can be imputed and included as correction terms in the primary equation. Thus, by fixed effect or pooled OLS[12], estimate of the resulting primary equation corrected for selection bias can be done for the sample for which $d_{it_1} = 1$ and $d_{it_2} = 1$.

In the case v_{it_1} and v_{it_2} are correlated, so that $Cov\left(v_{it_1}, v_{it_2}\right) = \sigma^2$, [*... the expression get very messy...*] (Maddala, 1983) and we have to use for each period cross-sectional bivariate probit methods to estimate γ_{it_1} and γ_{it_2}. Further,

$$E\left[\varepsilon_{it} \left| z_{it_1}, z_{it_2} d_{it_1} = 1, d_{it_2} = 1 \right.\right] = \rho_{t_1} M_{12} + \rho_{t_2} M_{21} \tag{3.17}$$

where $M_{ij} = \left(1 - \sigma_{12}\right)^{-1} \left(P_i - \sigma_{12} P_j\right)$ and

$$P_j = \frac{\int_{-\infty}^{z_{it_1}\gamma_{t_1}} \int_{-\infty}^{z_{it_2}\gamma_{t_2}} v_{it_1} v_{it_2}\ f\left(v_{it_1}, v_{it_2}\right) dv_{it_1} dv_{it_2}}{F\left(z_{it_1}\gamma_{t_1}, z_{it_2}\gamma_{t_2}\right)}.$$ [13]

Bivariate Probit Model for Care Expenditure and Insurance

To test whether v_{it_1} and v_{it_2} are correlated we run for each year a "preliminary" bivariate probit between insurance and health care expenditure participation. In our model the dependent variable employed to predict the probability of facing positive health care

[12] In this analysis fixed effect however presents a significant limitation with the respect to pooled OLS : we can not assess the effect of variables that do not vary very much within group: i.e. degree of education, race, region, etc. that can impact significantly the health insurance reimbursement. Also, explanatory variables whose change across time is constant – e.g. age – can not be included.

[13] There is only one cross-sectional example in the literature that is due to Fishe et. al.(1981). They estimated the selection equations by bivariate probit method and evaluated the above expression by numerical methods.

expenditure is a binary variable that takes value one if individuals incur in positive health care expenditure during the year of interview, and zero otherwise.

The independent variables employed can been categorized into three dimensions: need for care (need to see a specialist or have treatments or tests and an indicator of health status[14]), predisposition to use health services (age, sex, marital status, race) and enabling factors (education, insurance, income, employment status, region and residential location[15]). Among enabling factor, we consider insurance participation. An insured individual, in fact, may consume more medical services and have a greater expenditure compared to an uninsured one (*moral hazard effect*, Arrow, 1963; Pauly, 1968). In this study, the situation is further complicated by the fact that insurance participation itself may be affected by the likelihood of having positive health expenditure. The choice of insurance coverage may be affected by planned medical expenditure and expectations about medical care utilization (*adverse selection effect*).

To test the potential endogeneity of health insurance and at the same time whether the covariance between health insurance choice and health expenditure participation is significantly different of zero, we run for each year a cross sectional recursive bivariate probit models (Maddala, 1999).

For each period, the recursive structure builds on a first reduced form equation for the potentially endogenous dummy measuring insurance participation and a second

[14]We adopt as indicator of health status the objective measure of health that is "hypertension" since it seems to work better.

[15]The variables MSA (Metropolitan Statistical Area) and the indicators of regions control for medical cost differences between metropolitan and no-metropolitan statistical area, as well as by region of the country.

structural form equation determining the expenditure participation:

$$d^*_{it_1} = z_{i1_1}\gamma_{1_1} + ... + z_{it_1}\gamma_{t_1} + v_{it_1} \tag{3.18}$$

$$\begin{aligned} d^*_{it_2} &= z_{i1_2}\gamma_{1_2} + ... + z_{it_2}\gamma_{t_2} + v_{it_2} = \\ &= z_{i1_2}\gamma_{1_2} + ... + d_{it_1}\zeta + w_{it}\xi + v_{it_2} \end{aligned} \tag{3.19}$$

where $d^*_{it_1}$ and $d^*_{it_2}$ are latent variables, and d_{it_1} and d_{it_2} are dichotomous variables observed according to the rule:

$$\begin{cases} d_{it_j} = 0 \quad if \quad d^*_{it_j} \leq 0 \\ d_{it_j} = 1 \quad if \quad d^*_{it_j} > 0 \end{cases} \; ; \; j = 1, 2 \tag{3.20}$$

z_{it_1}, the lags of z_{it_j} and w_{it} are vector of exogenous variables, γ and ξ are parameter vectors, ζ is a scalar parameter. The dependent variable d_{it_1} used to predict the probability of being insured is again a dummy variable that takes value one if respondents are insured and zero otherwise. The vector of explanatory variables z_{it_1} used to predict the probability of being insured includes both exogenous variables that are determinants of health expenditure and personal attributes that are only determinative of health insurance choice[16] (i.e. risk aversion). We assume that, for each period, the error terms v_{it_1} and v_{it_2} are distributed as bivariate normal, with zero mean and variance covariance matrix Σ. Σ has values of 1 on the leading diagonal and correlations $\rho_{12} = \rho_{21}$ as off-diagonal elements:

$$\begin{pmatrix} v_{it_1} \\ v_{it_2} \end{pmatrix} \sim IIDN \left(\begin{bmatrix} 0 \\ 0 \end{bmatrix}, \begin{bmatrix} 1 & \rho_{12} \\ \rho_{21} & 1 \end{bmatrix} \right) \tag{3.21}$$

[16]Estimation of a recursive bivariate probit model requires some considerations for the identification of the model parameters: at least one of the insurance equation exogenous variables has not to be included in the expenditure equation as explanatory variable (Maddala, 1983). Following Maddala's approach we include among explanatory variables in the insurance equation a measure of risk aversion assuming that risk aversion has direct effect on insurance choice while it has only an indirect effect on health care expenditure through insurance participation. In addition we exclude from insurance participation equation "need for care" variable to avoid causality problems with the dependent variable.

In the above setting, the exogeneity condition is stated in terms of the correlation coefficient, which can be interpreted as the correlation between the unobservable explanatory variables of the two different equations. The two selection equations can be estimated separately as single probit models only in the case of independent error terms v_{it_1} and v_{it_2} i.e. the coefficient ρ_{jk} is not significantly different of zero ($k = 1, 2$). If the error terms v_{it_1} and v_{it_2} are independent we can deal with the above model as independent equations (Maddala, 1983) and apply the model in the equation (3.16)[17].

Table 3 shows the correlation coefficients and the p-value for each year sample: the null hypothesis of $Cov\left(v_{it_1}, v_{it_2}\right) = 0$ is not rejected; hence, we can deal with the model in the equation (3.16) and compute Inverse Mills Ratio by using the two selection equations as single probit models. Moreover, $Cov\left(v_{it_1}, v_{it_2}\right) = 0$ (the error terms v_{it_1} and v_{it_2} are independent) does not support adverse selection in insurance choice: no unobservable that affect the health care expenditure significantly affect insurance participation, while being insured has a positive influence on the probability of facing positive health care expenditure(see Tables 4 and 5). It is worth noting that while socioeconomic variables influence the probability of being insured they do not impact significantly the probability of positive expenditure (see Tables 4 and 5)[18].

[17]The estimation of the model is carried out using STATA 9 software by which it is possible to run a bivariate probit with the command **biprobit**. STATA provides the statistics $z = \frac{\hat{\rho}}{S_{\hat{\rho}}}$ to test the hypothesis $H : \rho = 0$. If the error terms are independent the bivariate probit estimation is equivalent to the separate probit estimations.

[18]We have tested for multicollinearity in both probit models (health care expenditure and insurance model) by using the Variance Inflation Factor (VIF) and Tolerance(1/VIF)(Wooldridge, 2000). We find that VIF for all the independent variables in both the equations are quite low. Therefore, we can safely assume that there are no problems of multicollinerity.

3.4.2 Structural Equation Estimation

Tables 6, 7 and 8 show the coefficients for the structural insurance reimbursement equation estimated using pooled OLS specification. The test for completeness of insurance coverage purchased by high risk profile individuals includes three pooled OLS models each of which contains a different measure of risk: a subjective measure of health (self-assessed health), an objective measure of health (hypertension) and independent variables that measure the individual life-style[19]. In each model the dependent variable is the natural logarithm of health insurance reimbursement.

We find a little evidence for adverse selection: table 6 shows that the coefficient estimate for the variable "health" is negative but is not statistically significant. Life-style variables do not influence the choice of health plan with exception of the variable "exercise" that, however, presents a positive coefficient. Table 7 shows that the variable that measures whether individuals suffer from high blood pressure is positively and significantly correlated with the health insurance reimbursement. The reason of this positive correlation may be found in the fact that more than half of all hypertensive in our sample are covered by Medicaid or Medicare. Medicare and Medicaid are essentially universal health insurance programs for this segment of the population. However, these programs present a number of gaps in coverage: for instance, despite Medicare and Medicaid have a prescription drug benefit, often people face restrictions in the number of covered medications. Since these restrictions, many people will exceed the initial drug benefit cap and may remain at risk for inadequate blood pressure control. (Duru et al., 2007). Hence, many hypertensives are

[19]We have constructed three different sub-models since the three measures of risk are strongly correlated and may generate problems of multicollinearity.

forced to buy their own supplemental insurance coverage which offers hypertensive prescription drugs; normally, those plans are more comprehensive than Medicare and Medicaid.

As expected the variable that measures whether respondents suffer from disabilities which limit their activities presents a positive and significant coefficients. The variable that measures individual risk aversion presents a positive sign but the coefficient is not statistically significant.

Concerning the variables that measure total income, education and employment status, the parameter estimates have the expected sign, but only the parameter for education is statistically significant. In the empirical literature we can observe that higher educational degree is often associated to a better health status; in particular it seems that education improves indirectly health status by helping people choose healthier life-styles and by improving their knowledge of the relationship between health behaviors and health outcomes (Kenkel, 1991). Then, people with more schooling tend to choose less complete insurance plans since they tend to enjoy good health.

Other than regular variables, two independent variables here are the IMR (Inverse Mills Ratio) which have been estimated from the first and second probit selection equations. When added to the outcome equation as additional regressors, they measure the sample selection effect due to lack of observations on the non-health insurance purchasers and non-health expenditure participants. These variables should be statistically significant to justify the use of Wooldridge two-step estimation. Since in our models they are statistically significant there may be sample selection problem in the data and we need to use Wooldridge method (Bath and Jain, 2006).

3.5 Summary and Conclusions

We have used the 2003/2004 Medical Expenditure Panel Survey in conjunctions with the 2002 National Health Interview Survey to asses whether US health insurance market is affected by adverse selection. We have conducted a positive correlation test which estimates the correlation between the amount of insurance an individual buys and his ex-post risk experience. We have employed three measures of risk: perceived health status, blood pressure and individuals' life-style. In addition, we have controlled for a number of enrollee characteristics including age, sex, race, education and family size which are used in pricing insurance policies. As indicator of generosity and completeness of health plan, we have employed health care expenditure reimbursement which measures the difference between total health care expenditure and out-of-pocket expenditure on health care paid by consumers. Since health insurance reimbursement is only defined for those who participate in insurance and have positive health care expenditure the model is estimated using Wooldridge's (1995) two step estimation procedure. We have extended this method to the case in which selectivity is due to two sources rather than one.

The evidence for adverse selection seems to be lacking. Our findings do not support the existence of a systematic relation between illness of individuals and insurance choice. There is no separating equilibrium: high risk individuals do not purchase more complete insurance than low risk profile individuals.

The absence of correlation between individuals' risk-profile and completeness of health insurance can be explained by the fact that individuals may choose a health insurance plan based not only on their expected health status but also on their preferences such

as the geographic location, whether they can continue to see doctors with whom they have already established relationships, whether friends recommended plans etc. If such preferences exert sufficient influence, risk-based selection is a minor consideration; as they become less important, adverse selection increases.

Arguably, another explanation for these results may be found in health plans risk selection practise. The distribution of health expenditure is highly skewed: only a small fraction of individuals account for most of health care spending. Because of this, insurers may have a strong incentives to distort their offering to avoid enrollment of high cost individuals. Then, insurers may practice a kind of "reverse adverse selection": they would try to increase their profits by refusing to sign contracts with the worst risks in an insurance pool (see Siegelman, 2004). These strategic behavior can take a variety of forms including: designing insurance benefits packages in such a way as to be more attractive to healthy people than unhealthy ones for instance by excluding particular prescription drugs, offering numerous pediatrician (families with children are better risks) or by excluding cancer specialist visits. If health plans cream healthy individuals, those who are enrolled in health insurance are relatively healthy people and the correlation between risk- profile and the generosity of health insurance plans becomes insignificant.

Appendix I: Tables

Table 1: Variables Name and Definition

Variables Name	*Variables Definition*
age	age in years
male	1 if male, 0 otherwise
white	1 if white, 0 otherwise
black	1 if black, 0 otherwise
other_race	1 if other race, 0 otherwise
northeast	1 if lives in Northeast region, 0 otherwise
midweast	1 if lives in Midweast region, 0 otherwise
west	1 if lives in West region, 0 otherwise
south	1 if lives in South region, 0 otherwise
msa	1 if lives in Metropolitan Statistical Area, 0 otherwise
income	total annual income
employed	1 if employed, 0 otherwise
education	1 if had high_school, master or PhD degree when entered in MEPS, 0 otherwise
expenditure	total annual health care expenditure
reimbursement	total annual health care expenditure paid by insurance
family size	family size
married	1 if married, 0 otherwise
health	1 if current health is excellent, very good, good, 0 otherwise
activity limitations	1 if has limited in any activities because health problems, 0 otherwise
hypertention	1 if suffers from high blood pressure, 0 otherwise
smoke	1 if is current smoker, 0 otherwise
alcohol	1 ifcorrent consumes heavy alcohol, 0 otherwise
execise	1 if participates in vigorous physical activity at least once at week, 0 otherwise
obese	body max index >= 25.0000
need care	1 if needs for care during the year of interview, 0 otherwise
insured	1 if insured, 0 otherwise
risk aversion	1 if is not likely to take risk, 0 otherwise
mills1	mills ratio insurance partecipation
mills2	mills ratiohealth care expenditure partecipation

Table 2: Summary Statistics

	All	Insured	Uninsured
Age	48.18	48.58	43,30
Male	0.317	0.316	0.331
Income	38,062.86	39,924.13	15,563.32
Total health care expenditure	4,120.202	4,295.44	2,001.882
Annual premium		1,736.688	
Northeast	0.168	0.177	0.066
South	0.351	0.341	0.471
West	0.203	0.196	0.279
Midwest	0.278	0.285	0.184
White	0.859	0.869	0.728
Black	0.092	0.085	0.184
Other Race	0.049	0.046	0.088
Metropolitan statistical area	0.806	0.818	0.669
Health status	0.892	0.897	0.824
Hypertension	0.262	0.265	0.221
Activity limitations	0.318	0.314	0.360
Smoke	0.167	0.159	0.272
Alcohol	0.056	0.047	0.169
Bmi	27.44	26.96	33.18
Exercise	0.479	0.493	0.309
Risk aversion	0.788	0.799	0.662
Number of observations	1780	1644	136

Table 3: Bivariate Probit Correlation Coefficients

Dependent Variables	*pho*	*p-value*
Positive Expenditure/ Be Insured 2003	-0.5299	0.260
Positive Expenditure/ Be Insured 2004	-0.9496	0.541

Table 4: Cross-Sectional Bivariate Probit Estimation Coefficients

(p-value in parentheses)

	Expenditure 2003	Be Insured 2003
intercept	0.9643 (0.282)	
age	0.0168 (0.183)	0.0151 (0.014)
male	-0.9162 (0.000)	-0.1509 (0.357)
black	-0.2153 (0.503)	-0.5149 (0.018)
other_race	-0.1419 (0.769)	-0.3913 (0.190)
family size	-0.2158 (0.011)	0.0514 (0.447)
msa	-0.1917 (0.529)	0.4337 (0.010)
northeast	0.2356 (0.482)	0.4700 (0.097)
midwest	0.7171 (0.066)	0.0817 (0.676)
west	1.0350 (0.025)	-0.3043 (0.123)
insured	2.0831 (0.010)	
income	2.48e-06(0.608)	0.0000 (0.000)
employed	-0.9426 (0.073)	0.2692 (0.169)
education	-0.1600 (0.685)	0.7078 (0.000)
married	0.0911 (0.749)	0.4618 (0.007)
need care	0.1413 (0.361)	
hypertension	-0.0309 (0.922)	0.1954 (0.312)
activity limit.	-0.0227 (0.945)	0.0006 (0.997)
risk aversion		0.2727 (0.125)

Note: sample size 890; statistically significant at the 0.05 level.

Table 5: Cross-Sectional Bivariate Probit Estimation Coefficients

(p-value in parentheses)

	Expenditure 2004	Be Insured 2004
intercept	1.5073 (0.090)	
age	0.0040 (0.716)	0.0177 (0.003)
male	-1.064 (0.000)	-0.0717 (0.655)
black	-0.4799 (0.235)	-0.4255 (0.068)
other_race	-0.0838 (0.868)	-0.3941 (0.176)
family size	-0.2521 (0.005)	0.0495 (0.530)
msa	-0.4831 (0.126)	0.3653 (0.027)
northeast	-0.1615 (0.706)	0.4407 (0.094)
midwest	-0.3098 (0.702)	0.2263 (0.247)
west	-0.3386 (0.288)	-0.2170 (0.260)
insured	2.0491 (0.005)	
income	8.62e-06 (0.045)	0.0000 (0.000)
employed	-0.6465 (0.100)	0.0262 (0.892)
education	-0.1996 (0.690)	0.5928 (0.002)
married	0.1268 (0.693)	0.3238 (0.074)
need care	0.2761 (0.059)	
hypertension	0.6086 (0.150)	0.0780 (0.683)
activity limit.	0.2282 (0.516)	0.0733 (0.665)
risk aversion		0.2129 (0.208)

Note: sample size 890; statistically significant at the 0.05 level.

Table 6: Pooled OLS Regression Results.

Risk Variable: Self-Assessed Health.

Preidictor Variables	*Coefficients*	*p-values*
intercept	6.710288	0.000
age	0.0021	0.479
male	-0.1328	0.111
married	-0.0544	0.452
black	-0.0849	0.505
other race	0.0123	0.932
education	-0.4279	0.002
income	-2.33e-06	0.078
employed	-0.0418	0.671
premium	-0.0000	0.004
expenditure	0.0001	0.000
activity limitations	0.2518	0.001
health	-0.1613	0.162
risk aversion	0.1370	0.113
mills1	-2.2891	0.000
mills2	-1.1936	0.001

Note: sample size 1613; $R^2 = 0.4239$; Adjusted $R^2 = 0.4185$;

statistically significant at the 0.05 level.

Table 7: Pooled OLS Regression Results.

Risk Variable: Hypertension

Preidictor Variables	*Coefficients*	*p-values*
intercept	6.5331	0.000
age	0.004	0.887
male	-0.1535	0.066
married	-0.0442	0.541
black	-0.1352	0.292
other race	0.0114	0.944
education	-0.4367	0.001
income	-2.09e-06	0.115
employed	-0.0185	0.851
premium	-0.0000	0.008
expenditure	0.0001	0.000
activity limitations	0.2405	0.001
hypertension	0.2514	0.002
risk aversion	0.1379	0.109
mills1	-2.1702	0.000
mills2	-1.0837	0.004

Note: sample size 1613; R^2 =0.4265; Adjusted R^2 = 0.4212;

statistically significant at the 0.05 level.

Table 8: Pooled OLS Regression Results.

Risk Variable: Life-Style Indicators

Preidictor Variables	*Coefficients*	*p-values*
intercept	6.3921	0.000
age	0.0034	0.260
male	-0.1537	0.069
married	-0.619	0.393
black	-0.0529	0.680
other race	0.0507	0.752
education	-0.4623	0.001
income	-2.52e-06	0.058
employed	-0.7782	0.436
premium	-0.0000	0.006
expenditure	0.0001	0.000
activity limitations	0.2672	0.001
smoke	0.1384	0.137
obese	0.0393	0.570
alcohol	-0.2403	0.118
exercise	0.1982	0.004
risk aversion	0.1458	0.091
mills1	-2.3287	0.000
mills2	-1.1903	0.001

Note: sample size 1613; $R^2 = 0.4277$; Adjusted $R^2 = 0.4212$;

statistically significant at the 0.05 level.

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