An Economic Evaluation of Life-Style and Air-pollution-related Damages: Results from the BRFSS

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Abstract

This paper uses the Behavioral Risk Factor Surveillance System (2001) data in conjunctions with Environmental Protection Agency data to investigate on how individual health habits, air outdoor pollution and diseases combine to affect the likelihood of good health status and the amount of health investments. The environment is a second-best world characterized by uncertainty on the level of health, in which individuals are not able to avoid health shocks completely. Models are estimated using three different measures of overall health: a measure of self-assessed health and two health outcomes indicators (blood pressure and activity limitations due to health problems).

A multivariate probit approach is used to estimate recursive systems of equations for self-assessed health, health outcomes and life-styles.

JEL-Classification: I12, C31, D13, D81, Q25

Keywords: health production, multivariate probit, life-style, pollution, self-assessed health, health outcome.

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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 life-style, 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 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.

[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...*].

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 functional 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 A Model of Health Production

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

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

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

[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.

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

$$\Delta D_t = D_t - D_{t-1} = g(R, E, \Lambda, t) - \alpha D_{t-1} \tag{2}$$

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 $g(R, E, \Lambda, t)$.

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:

$$\delta_t = h\delta_0 \left(1+\bar{\delta}\right)^t \Psi_t^{\phi} + (1-h)\,\delta_0 \left(1+\bar{\delta}\right)^t \Psi_t^{\phi} D_t^{\gamma} \tag{3}$$

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.

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

[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).

[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.

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.

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 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

[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/.

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 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

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

[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.

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 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.

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, 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).

[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 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)

4 Estimation Strategies and Results

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:

$$\begin{aligned} y_{1i}^* &= \beta_1' x_{1i} + \varepsilon_{1i} \\ &\cdot \\ &\cdot \\ &\cdot \\ y_{Mi}^* &= \beta_M' x_{Mi} + \varepsilon_{Mi} \end{aligned} \tag{4}$$

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

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

$$y_m = \begin{cases} 1 & if \quad y_m^* > 0 \\ 0 & otherwise \end{cases} \tag{5}$$

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 (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:

$$
\begin{aligned}
&y_{1i}^{*} = \beta_1' x_{1i} + \varepsilon_{1i} \\
&y_{2i}^{*} = \beta_2' x_{2i} + \varepsilon_{2i} \\
&y_{3i}^{*} = \beta_3' x_{3i} + \varepsilon_{3i} \\
&y_{4i}^{*} = \beta_4' x_{4i} + \varepsilon_{4i} \\
&y_{5i}^{*} = \beta_5' x_{5i} + \varepsilon_{5i} \\
&y_{6i}^{*} = \beta_6' x_{6i} + \varepsilon_{6i} \\
&y_{7i}^{*} = \beta_7' x_{7i} + \varepsilon_{7i} \\
&y_{8i}^{*} = \beta_8' x_{8i} + \varepsilon_{8i} = \delta_{81} y_{1i} + \delta_{82} y_{2i} + \delta_{83} y_{3i} + \delta_{84} y_{4i} + \\
&+\delta_{85} y_{5i} + \delta_{86} y_{6i} + \delta_{87} y_{7i} + \alpha_8' z_{8i} + \varepsilon_{8i} \\
&y_{9i}^{*} = \beta_9' x_{9i} + \varepsilon_{9i} = \delta_{91} y_{1i} + \delta_{92} y_{2i} + \delta_{93} y_{3i} + \delta_{94} y_{4i} + \\
&+\delta_{95} y_{5i} + \delta_{96} y_{6i} + \delta_{97} y_{7i} + \delta_{98} y_{8i} + \alpha_9' z_{9i} + \varepsilon_{9i}
\end{aligned}
\tag{6}
$$

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 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

[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) 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.

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.

4.1.1 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 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 encour-

age 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 pollution 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 dis-

[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.

turbance of smoke equation that is consistent with the above results.

4.1.2 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.

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 measured 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.

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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