# THE INDIRECT EFFECT OF FINE PARTICULATE MATTER ON HEALTH THROUGH INDIVIDUALS' LIFE-STYLE

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# The Indirect Effect of Fine Particulate Matter on Health through Individuals' Life-style.

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**Abstract**

Limited literature has been published on the association between environmental health indicators, life-style habits and ambient air pollution. We have examined the associations of asthma prevalence and the amount of health investments with daily mean concentrations of particulate matter (PM) with a mass median aerodynamic diameter less than 2.5 mm ($PM_{2.5}$) in 16 metropolitan areas in U.S. using the Behavioral Risk Factor Surveillance System (2001) data in conjunction with the Air Quality System data collected by the Environmental Protection Agency. A multivariate probit approach has been used to estimate recursive systems of equations for environmental health outcome and life-styles. A piecewise linear relationship has been postulated to describe the association between health outcome,health investments and pollution using the procedure *mkspline* from STATA 10. This model has allowed for fitting a"breakpoint" in the probit functions. We have assumed one change point at AQI value of 100 which corresponds to the US national air quality standard. The most interesting result concerns the influence of pollution on health-improving life-style

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choices: below a specified threshold concentration (AQI=100) a positive linear association exists between exposure to PM2.5 and health investments; above the threshold the association becomes negative. Hence, only if ambient pollution is in the 'satisfactory range' (AQI level at or below 100), individuals will have incentive to invest in health.

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

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# 1 Introduction

Under the 1970 Clean Air Act and subsequent amendments, introduced by the Environmental Protection Agency to limit the amount of air pollution, ambient air quality in the United States has improved dramatically. However, despite regulatory effort, fine particulate continues to be a matter for concern despite its falling level. The situation has been further aggravated by the fact that protection of public health is constrained by the inability of scientists to establish a safe level of $PM_{2.5}$ below which it poses little or no risks for human health. In fact, fine particulate even at much lower concentrations (below current US regulatory levels), has been associated with increased rates of mortality and morbidity in several cities in the United States, (in Europe and other developed countries, too) (Dume et al. 1998; Daniels et al., 2000; Bolin and Lindgren, 2002; Brunekreef and Holgate, 2002). Furthermore, the effect of particulate on health may be complex, as it may vary from one individual to another: scientists have to consider that individuals and groups are not equally vulnerable to air pollution health effects. Susceptibility factors could be strictly linked to fixed individual characteristics such as genetics, gender, age and race or to variable individual characteristics caused by the realities of life (Di Novi, 2009). Low socioeconomic classes, for instance, tend to be more susceptible to the adverse effects of air pollution because of other factors related to their life-styles: they are more likely to be uninformed over environmental health issues, to have an unhealthy diet, to smoke and drink alcohol, and in general to lead less healthy lives, with associated effects on their health (Grassman, 1996; Sexton, 1997). Hence, analysts must calculate changes in health outcomes by taking into account that the effect of pollution could easily be correlated with other factors that may be just as influential (Schwartz and Weiss, 1994 a, b; Brook et al., 2004)[1] .

[1]After age, height, body mass index, race, sex, cigarette smoking, and employment status were controlled for, Schwartz and Weiss (1994a, b) find that a diet rich in fish and vitamin C could have a protective effect on lung function; while cigarette smoking and

While, on the one hand, epidemiological studies have shown that pollution acts synergistically with tobacco smoking, alcohol consumption and unhealthy diet to induce respiratory illness such as asthma, lung cancer and cardiovascular diseases (Valavanidis et al., 2009) on the other hand there is little information on the extent to which quality of the environment may influence choices of life-style. This is an issue that has, in our view, received too little attention (see Cropper,1981; Erbsland et al.1994; Di Novi, 2009). An important contribution in this area was Cropper (1981), who explored the consequences of introducing pollution variables into the health production function. She considered changes in environmental conditions to influence the amount of health investments through the rate at which an individual's stock of health depreciates: Cropper assumes that when pollution increases, it becomes more costly to reduce the probability of a health shock. Individuals feel less healthy because they perceive the 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 the health depreciation rate and an indirect effect, described by Cropper (1981), by which individuals will invest less in health and display a higher probability of suffering from bad health.

The purpose of this study is to examine the influence that the quality of the environment, captured by the $PM_{2.5}$ level, may have on health investment decisions. The paper divides into two parts. The first part provides theoretical framework built on the basic concepts and ideas of the demand for health by Grossman (1972) and the subsequent contribution by Cropper (1981). In its second part the paper provides empirical support to the theoretical assumptions.

In the empirical part of the paper, in order to introduce a measure of

heavy alcohol consumption interacts with air pollution to increase mortality risks from cardiovascular diseases (Brook et al., 2004).

health stock, a dichotomous measure of asthma prevalence has been used. We choose asthma since it simultaneously represents a health outcome and an "environmental health indicator" (see WHO, 1999). Since we have included life-style variables as regressors in the health equations, a problem of simultaneity may arise. Hence, we try to correct the potential endogeneity of the behavioral variables by using a recursive multivariate probit model which is available in the literature although not so frequently used (Blaylock and Blisard, 1992; Contoyannis and Jones, 2004; Balia and Jones, 2008; Di Novi, 2009). A piecewise linear function has been employed to describe the relationship between health, health investments and pollution using the procedure *mkspline* from STATA 10 (Stata Corp., College Station, TX, USA). This model allows for fitting a "breakpoint" into the probit functions. We assume one change point at AQI value of 100 which corresponds to the U.S. national air quality standard.

The model is estimated using data from the 2001 Behavioral Risk Factor Surveillance System (BRFSS). BRFSS does not measure environmental quality but it can be used in conjunction with the 2001 Environmental Protection Agency's (EPA) Air Quality System (AQS) database. We merged data from the AQS with BRFSS data using the metropolitan area information. The EPA's Air Quality System (AQS) database contains measurement of six criteria pollutants: ozone ($O_3$), sulphur dioxide ($SO_2$), carbon monoxide (CO), and particulate matter ($PM_{2.5}$, $PM_{10}$). Because our study focused on $PM_{2.5}$ we used the daily AQI which reported daily air quality based on the concentration levels of $PM_{2.5}$. The daily $PM_{2.5}$. AQI represented the highest concentrations of $PM_{2.5}$ for that day. Ambient air measurements collected from a network of national, state, and local air monitoring stations were used to calculate the $PM_{2.5}$AQI.

The most interesting-and possibly surprising- result is the effect that pollution appears to have on health-improving life-style choices. This result partly contradicts what one should expect from Cropper's model, where pol-

lution makes the investments in health more costly. In order to rationalize the empirical result obtained, one should refer to the relationship between pollution and the investments in health as an inverse-V-shaped emission-health investments relationship with a threshold pollution point: only if air pollution is concentrated above this point individuals will no longer have incentives to invest in health-improving activities. This result may have an important policy implication: an intervention that reduces air pollution below the threshold pollution level, may have not only a direct effect on individuals' health status, but also an indirect health effect through a healthier life-style which seems to be one of the driving factors for good health.

The rest of the paper 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 empirical approach 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

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

$$\triangle H = H_{t+1} - H_t = f\left(\Lambda(E), t\right) - \delta_t H_t - \vartheta_t \tag{1}$$

where $\delta_t \in (0, 1)$ is the natural rate at which health deteriorates. $\vartheta_t$ 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 $\vartheta_t$ 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. The investments/disinvestments in health are captured by a household production function $f(\Lambda(E), t)$. Where $\Lambda$ 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. $E$ is the exogenous education level that is assumed to affect the productivity of producing health. Schooling helps people choose healthier life-styles by improving their knowledge of the relationship between health behaviors and health outcomes (Berger and Leigh, 1989; 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. $f(\Lambda(E), t)$ can increase or fall in individual behavior $\Lambda$. In particular $f(\Lambda(E), t)$ 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 life-style increases the stock of health capital, actions detrimental to health such as cigarette smoking and excessive alcohol consumption lower the stock of health capital.

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:

$$\delta_t = \delta_0 \left(1 + \tilde{\delta}\right)^t \Psi_t^{\phi} \tag{2}$$

Following Grossman (1972) and subsequent contribution by Cropper (1981) we assume that health depreciates over time at an increasing rate with age $(\tilde{\delta})$ and with the ambient air pollution to which an individual is exposed $(\Psi)$.

Pollution enters directly the rate of decay and physically alters the state of a person's health; its effect is measured by $\phi$.

As in Cropper's (1981) model, we assume that the individual behavior is influenced by environmental pollution. We assume that there is an optimal pollution level $\Psi^*$ to maximize health investments and healthy behaviors. An increasing level of pollution encourages health investments if it does not exceed a certain threshold. But if pollution level exceed the optimal threshold, a decrease of ambient air quality may lead individuals to invest less in health. Individuals may have no incentives to invest in health since they feel less healthy because they perceive $\delta$ 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 above the optimal threshold may have two effects on health: a direct effect which consists in an increase of $\delta$ 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. In Section 4, we will provide empirical support to this theoretical assumption and we will test if there exist an optimal threshold in the relationship between pollution and life-style variables.

# 3 Data and Variables

Data pertaining to health status and health-related characteristics were drawn from the 2001 Behavioral Risk Factor Surveillance System Survey[2]. The BRFSS is an ongoing state-based, landline cross-sectional telephone survey used to collect information on health risk behaviors, preventive health practices, access to and use of health care services primarily related to chronic conditions among U.S. adults aged 18 years or older. BRFSS does not directly

[2]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).

measure environmental quality, 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 data sets by metropolitan area[3].

The term metropolitan area is a geographic subdivisions formally defined by the U. S. Office of Management and Budget (OMB), for use by Federal statistical agencies in collecting, tabulating, and publishing Federal statistics. Following the OMB definition, the metropolitan area is "*an area containing a recognized population nucleus and adjacent communities that have a high degree of integration with that nucleus*" (OMB, 2000). The term metropolitan area refers collectively to metropolitan statistical area (MSA), primary metropolitan statistical area (PMSA), or New England county metropolitan area (NECMA):

- a MSA consists of one or more counties with a high degree of social and economic integration, the presence either of a city with 50,000 or more inhabitants, or an urbanized area (i.e. a contiguous area of relatively high population density), and a total population of at least 100,000;

- a PMSA consists of one county or group of counties that qualify as an MSA and have a total population of 1 million or more;

- because of the greater importance of towns over counties, for the New England states, metropolitan areas are defined by a collection of towns and cities instead of by counties with a presence a total population of at least 75,000.

We have excluded respondents with missing air pollution information as well as those with an unknown metropolitan area of residence. Finally, we have dropped all those living outside the continental USA. After correcting

[3]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.

for missing values, the final sample contains 6,760 individuals distributed in 16 metropolitan areas: Chicago, IL; Cincinnati, OH-KY-IN; Cleveland-Lorain-Elyria, OH; Columbus, OH; Des Moines, IA; Huntington-Ashland, WV-KY-OH; Las Vegas, NV-AZ; Louisville, KY-IN; Memphis,TN-AR-MS; Milwaukee-Waukesha, WI; Minneapolis-St. Paul; MN-WI; Nashville, TN; Omaha, NE-IA; Phoenix-Mesa, AZ; St, Louis, MO-IL; Tucson, AZ. Here, the term MSA refers to PMSAs.

Each respondent in the BRFSS was assigned an average annual $PM_{2.5}$ exposure based on his or her metropolitan area of residence[4]. Exposures are measurements taken at a network of national, state and local air monitoring stations which report the concentrations for $PM_{2.5}$. As $PM_{2.5}$ is measured in each metropolitan area at multiple sites, an annual mean has been computed and has been used in the analysis.

The EPA's Air Quality System database contains $PM_{2.5}$ AQI measurements. Formerly known as the Pollutant Standard Index(PSI)[5] the nationally uniform AQI is a single number used by state and local agencies for reporting the air quality with respect to its effects on the human health. To make the AQI as easy to understand as possible, EPA has divided the AQI into six categories that correspond to different level of health concern with a scale that runs from 0 to 500 associated with a color scheme that runs from green to maroon:

- 0-50 (green)- good, i.e. air quality poses little or no risks;

- 51-100 (yellow)- moderate, i.e. air quality is acceptable but may be some concern for very small number of people;

[4]An important limitation of our analysis concerns the particulate matter data which provide estimates for ambient air particulate matter at the metropolitan area level for a one-year period and not at an individual exposure level to the daily concentrations level.

[5]Based on available evidence that fine particles were particularly damaging, in 1999 the PSI was revised by US Environmental Protection Agency and and replaced by the Air Quality Index (AQI) to incorporate new $PM_{2.5}$.

- 101-150 (orange)- unhealthy for sensitive groups, i.e. members of sensitive groups (i.e. children, older adults, people with respiratory disease or heart disease) may experience health effects. The general public is not likely to be affected;

- 151-200 (red)- unhealthy, i.e. everyone may begin to experience health effects;

- 201-300 (purple)- very unhealthy, i.e. everyone may experience more serious health effects;

- 300-500 (maroon)- hazardous, i.e. the entire population is more likely to be affected and everyone should avoid all outdoor exertion.

In 1976 the U.S. EPA established the National Ambient Air Quality Standards (NAAQS) of 100: only if the AQI level is at or below 100 ambient air quality can be considered in the satisfactory range.

## 3.1 Environmental Health Indicator and Life-Style Variables

As "environmental health indicator" (WHO, 1999) a dichotomous measure of asthma prevalence has been used. Asthma is one of the most common chronic illness in the world and represents one of the most important cause of morbidity, economic cost and mortality in developed and developing countries alike: approximately 300 million people currently suffer from asthma worldwide. In North America about 1 person in 10 has been diagnosed with asthma. The asthma prevalence rates in the United States are approximately 10.9%, representing 35.5 million individuals (Masoli et al. 2004; Braman, 2006; Bateman et al., 2008;). Asthma has been associated with worse health-related quality of life among all adult (Ford et al. 2003).

Although understanding of many aspects of asthma has improved over the past decades, the fundamental causes of the tendency to develop asthma

remains largely unknown (Braman, 2006). The development of asthma seems to be related to certain asthma genetic factors and to individual exposures to certain life-style (in particular to smoking habits and obesity) and environmental factors (allergen exposure, tobacco smoking, outdoor air pollution ) (Basagaña, et al., 2004; GINA, 2006). Concerning outdoor pollution, there is evidence that long term exposure to substances found in the air, such as particles, may interact with genetic factors to develop and to determine the subsequent maintence of asthma (Bascom et al.,1996; Katsouyanni et al. 1997; Brunekreef and Holgate, 2002). In order to capture the effect of pollution on asthma,we have introduced a self-reported measure of asthma prevalence. To be classified as having asthma, the respondent must have answered "*yes*" to the following two fairly precise questions: *"Have you ever been told by a doctor, nurse, or other health professional that you have asthma?"* and "*Do you still have asthma?"*.

The life-style variables[6] indicate whether the individual is a smoker, consumes heavy alcohol, eats fruits and vegetables at least once per day, meets recommendations for physical activity[7], is obese, suffers from mental stress and depression, has had flu shot vaccination during the year of the interview. We use BMI (Body Mass Index) to compute an indicator of obesity[8]according to medical and WHO guidelines. Table 1 contains the full description of the life-style variables[9].

[6]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 study based on the Alameda County survey carried out in California in 1965.

[7]Based on the "Physical Activity Guidelines for Americans" (2008) exercise recommendations for healthy adults and older adults are 300 minutes a week of moderate-intensity, or 150 minutes a week of vigorous-intensity aerobic physical activity, or an equivalent combination of moderate- and vigorous-intensity activity (Physical Activity Guidelines for Americans, 2008).

[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

[9]The original variables are mostly discrete qualitative indexes but with more than two modalities; clearly it would be better to use this information, but the computational

TABLE 2a) 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 (40.7 % men) consists of individuals whose behaviors are mostly healthy with the exception of the variables that assess physical activity: only 25 per cent of individuals are current smokers, 5 per cent of individuals drink heavily, and 36 per cent of them suffer from stress; while 96 per cent of them follow a healthy diet and 22 per cent devote some time to physical activity.

## 3.2 Other Characteristics

The other independent variables in the model can be grouped into categories which are listed, together with the life-style variables, in TABLE 1. We consider the following categories: physical characteristics such as age, sex, race, prior health in order to capture health status at the beginning of the observation period[10]; a measure of leisure time physical activity (as a predictor of healthy behaviors); household composition; air pollution; socioeconomic variables such as health coverage (including HMO plan[11]), education, marital status, employment status. Despite many research findings have suggested a significant associations between income inequality and health, we do not include income among socioeconomic variables because of the unreliability of this information[12].

complexities would have increase considerably with uncertain benefits for the point of view of the estimation quality.

[10]We have included a measure of prior physical health with a time frame of 30 days, refers to a measure of self-reported physical health: respondents were asked if their physical health was not good in the thirty days before the interview.

[11]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.

[12]The BRFSS does not collect a continuous measure for income. The only measure available in the BRFSS is household income which is provided in the form of eight intervals, with the highest category listed as "$75,000 or more". Respondents are assigned a value

Concerning leisure time physical activity, the following item was used to create a binary outcome: "During the past month, did you participate in any physical activities such as running, calisthenics, golf, gardening, or walking for exercise". The response was "yes" or "no". Individuals answering "no" to this question were categorized as inactive, and those answering "yes" were categorized as "active". Leisure time physical activity has been shown to be a strong predictor of regular moderate physical activity or physical fitness necessary to improve health (Sandvik et al., 1993; Friede et al. 1997). In our sample, of those who reported leisure time physical activity (74.8 %), 28.22% meet recommendations in physical activity, while among those who initially reported no leisure-time physical activity (25.2%) only 3.99% exercise to meet health-related recommendations (Physical Activity Guidelines for Americans, 2008).

Recent studies have found that the absence of leisure time physical activity is a good measure for the tendency to sedentary behavior that is considered an important reason for obesity and for poor dietary habits: people who are active generally have healthier dietary habits than their sedentary counterparts (Sherwood and Jeffery, 2000). Moreover, sedentary behavior is strictly linked to smoking behavior and to the use of other substances as well, such as alcohol or high-fat foods (King and Brassington, 1997; Marcus et al., 1999).

The main variable of interest in the model is fine particulate. Particulate matter (PM) is an air pollutant consisting of dust, dirt, soot, smoke, and liquid droplets suspended in the air. Particles with aerodynamic diameters between 2.5 and 10 micrometers consist mainly of crustal particles

corresponding to the midpoint of their income interval. In general, missing data is not a large problem with the BRFSS questions: response is around 95% or higher for all questions with the exception of baseline household income which suffer a high rate of item non-response (around 20%). Because of the high rate of non-response, we have not included income given the potential for sample selection bias and the reduction in sample size induced by using only those individuals for whom we have this information.

mechanically generated from agriculture, mining, construction, road traffic, and related sources, as well as particles of biological origin. While particles with aerodynamic diameters 2.5 consist mainly of combustion particles from motor vehicles and the burning of coal, fuel oil, and wood, but also contain some crustal particles from finely pulverized road dust and soils. PM has been associated with a wide range of related human adverse health effects, including an increased incidence of chronic bronchitis and acute respiratory illnesses, exacerbation of asthma, impairment of lung functions and premature mortality (Dume et al. 1998; Bolin and Lindgren, 2002; Brunekreef and Holgate, 2002). The size of particles is directly linked to their potential for causing health problems (EPA, 2003). In particular smaller or fine particles ($PM_{2.5}$) seem to pose the greatest problems since they may penetrate more deeply into the lung and may reach the alveolar region.

# 4 Empirical Approach

## 4.1 Multivariate Estimation

From a methodological point of view, it should be noted that asthma equation is a structural equation since the health behavior inputs may be endogenous. Efficient and consistent estimation of the parameters in the health equation 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[13] (Contoyannis and Jones, 2004, Balia and Jones, 2008; Di Novi, 2009).

We identify two classes of dependent variables: the individual health behaviors and the health outcome.

The seven equations for the health behavior variables are modeled as

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

reduced-form equations. The asthma equation is the structural equation with the health behavior variables as explanatory factors. Hence, we construct and estimate a system of eight equations ($m = 8$) with seven reduced-form and one structural equation.

Thus:

$$\begin{aligned} y_{1i}^{*} &= \beta_1' x_{1i} + \varepsilon_{1i} = \delta_2 y_{2i} + \delta_3 y_{3i} + \delta_4 y_{4i} + \delta_5 y_{5i} + \delta_6 y_{6i} + \\ &+ \delta_7 y_{7i} + \delta_8 y_{8i} + \alpha' z_i + \ f(PM_{2.5}\ AQI) + \varepsilon_{1i} \\ &\vdots \\ y_{li}^{*} &= \beta_l' x_{li} + f(PM_{2.5}\ AQI) + \varepsilon_{li} \end{aligned} \tag{3}$$

where $x_{li}$ (with $l = 2, ..., 8$) and $z_i$ are vectors of exogenous variables, $\beta_l$ and $\alpha$ are parameter vectors, $\delta_l$ is a scalar parameter and $\beta_1' = (\delta_l, \alpha')$. $f(PM_{2.5}\ AQI)$ is some function of $PM_{2.5}\ AQI$. Two forms of $f(PM_{2.5}\ AQI)$ has been used in this article:

- linear function $\eta\ PM_{2.5}\ AQI_i$; and
- piecewise linear function with one change point:

$$f(PM_{2.5}\ AQI) = \begin{cases} \eta_{_1} PM_{2.5} AQI_i & \text{if } PM_{2.5} AQI < \theta \\ \eta_{_1}\theta + \eta_{_2}\left(PM_{2.5} AQI - \theta\right)_i & \text{if } PM_{2.5} AQI \geq \theta \end{cases}$$

where $\eta_n$(with $n = 1, 2$) are scalar parameters, and $\theta$ is the change point fixed at 100 which corresponds to the national air quality standard i.e. the $PM_{2.5}$ AQI value at which the slope of the piecewise linear function is allowed to change (see Robert, 2004).

$\varepsilon_{1i}, ..., \varepsilon_{li}$ are the error terms distributed as multivariate normal, each with a mean zero and a variance covariance matrix $\Sigma$. $\Sigma$ has values of 1 on the leading diagonal and correlations $\rho_{jk} = \rho_{kj}$ on off-diagonal elements (where $\rho_{jk}$ 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 (3) can be estimated separately as single probit model only in the case of independent error terms i.e. the coefficient $\rho_{jk}$ is not significantly different of zero.

The parameters of the equations are not identified if $z_i$ includes all the variables in $x_{li}$. Estimation requires some considerations for the identification of the model parameters. Maddala (1983) proposes that at least one of the reduced-form exogenous variables ($x_{li}$) 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[14] assuming that it has only an indirect effect on health through the life-style variables. Moreover, in order to address the identification problem, we include in the reduced form equation the measure of leisure time physical activity. In addition, we exclude from the asthma equation 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[15].

The reference individual in the model is female, married and employed.

[14]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 (1994), Contoyannis and Jones (2004), Balia and Jones (2008) 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.

[15]Family structure and composition may affect health behaviours. Takeda et al. (2004), for instance, find that an increasing number of women in households is associated with a strong presence of protective health behaviours (less smoking, less heavy drinking), but also with more sedentary behaviour, while the presence of men in household are associated with a higher probability of heavier smoking. Moreover Bakhshi et al. (2008)'s paper shows that there exists an association between the number of young children and obesity among men and women aged 20-75 years.

She is aged eighteen years old or more and she has attended elementary school. The estimation of a multivariate probit is carried out using the STATA 10 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.

## 4.2 Estimation Results

The first column of TABLE 3a) and TABLE 3b) shows coefficients for the structural asthma equation estimated in the full recursive model, using the multivariate probit specification.

Starting from life-style variables, we can observe that the results support the theoretical model: in the asthma equation smoking behavior, alcohol consumption, being stressed and being obese have the expected significant positive influence on the probability of suffering from asthma. Physical activity and diet variables do not contribute to explain the probability of reporting asthma. Concerning the preventive medical behavior, flu vaccination shows a positive and statistically significant coefficient in the asthma equation. One of the possible reasons for the ambiguous sign is that the utilization of the flu shot may be the result, rather than the cause of asthma. In fact, it is more plausible that an individual who suffers from asthma will receive preventive medical care and immunization by seeing a physician on a regular basis that will encourage him to have vaccinations. Influenza is an infectious disease with possible severe health consequences for the elderly (over 65) and non-elderly in poor health (Mullahy, 1999). The vaccination of adults and children with asthma or reactive airway disease is recommended every year by the Advisory Committee on Immunization Practices (ACIP) since they are at increased risk of complications from influenza infection (Advisory Committee on Immunization Practices, 2009).

Medical literature clearly indicates that asthma tends to be a disease of young people: the prevalence of asthma is highest in people younger than age 18 and tends to decrease with increasing age. Our model too, predicts that the probability of suffering from asthma decreases with age. Also gender affects the probability of suffering from asthma: according to the medical literature, in our sample asthma seems to be more prevalent in women (Dicpinigaitis and Rauf, 1998; De Marco et al. 2000; Eagan, et al., 2002).

Concerning the effect of pollution, first a linear function and then a piecewise linear function were fitted to fine particulate using the procedure *mkspline* function of STATA 10. While the relationship between $PM_{2.5}$ and asthma seems to be adequately described by a linear model[16], with a positive statistically significant effect on asthma prevalence, the effect of fine particulate on individuals' health-improving activities seems to be better represented in an inverse-V-shaped emission-health investments relationship with a threshold pollution point. The changing point at which the slope of the piecewise linear relationship changes is the AQI value of 100[17]. From our empirical results, it arises that when fine particulate is concentrated above national air quality standard, people start to disinvest in health. Above the "$PM_{2.5}$ AQI optimal point" if air pollution level increases along the downward-sloping portion of the curve, individuals will have no incentives to

[16]When the relationship between particulate matter concentration and health indicators have been evaluated, most studies have reported no evidence of a clear threshold concentration below which the harmful effect of fine particulate is less likely (Samet et al. 1995; Daniels et al.,2002; Dominici et al., 2002 ). According to these studies we can conclude that the relationship could be reasonably considered linear.

[17]An AQI value of 100 generally corresponds to the national air quality standard for the pollutant, which is the level that the EPA has set to protect public health. AQI values below 100 are generally thought of as satisfactory. When AQI values are above 100, air quality is considered to be unhealthy at first for members of sensitive populations (i.e. children, elderly and people who suffer from respiratory or cardiovascular diseases) then for everyone as AQI values go above 150. Many US larger cities have severe air pollution problems, and the AQI in these areas may exceed 100 even though AQI values higher than 200 are infrequent, and AQI values above 300 are extremely rare. For the metropolitan areas under consideration in this study $PM_{2.5}$ AQI displays values between 62 and 161 (with the higher value experienced by Milwaukee-Waukesha, WI).

invest in health. While a reduction in fine particulate along the downward-sloping portion of the curve may lead to an increase in the health-improving life-style choices. Thus, it arises that, concerning the effect of pollution on health investment decisions, the most important number in the AQI scale is 100 (the standard established under the Clean Air Act): only if the $PM_{2.5}$ AQI level is at or below 100, fine particulate will be in the satisfactory range and people will have an incentive to invest in health. According to our results while a higher concentration of $PM_{2.5}$ when fine particulate is in the satisfactory range would have a positive influence on healthy habits (in particular, a negative influence on smoking behavior, alcohol consumption, stress, and a positive effect on diet and flu vaccination) when $PM_{2.5}$ AQI values go above 100 an increasing level of fine particulate seems to lead individuals to invest less in health-improving activities with a positive effect on the probability of smoking, consuming heavy drinks and suffering from stress; in addition, it decreases the probability of following a diet rich in fruits and vegetables and having preventive care[18]. Our model predicts that people increase physical exercise in response to higher $PM_{2.5}$ level when pollution is outside the satisfactory range. However, this does not always seem to be a healthy choice, in particular when individuals exercise outdoors: when AQI values exceed 100, air quality is considered to be unhealthy, and is always accompanied by EPA advice to avoid harmful air pollution and especially those with medical conditions affecting the heart and lungs should consider limiting the type, duration and location of outdoor activity pursuing alternative physical activities, such as indoor activities. Activity outdoors at times when the air is polluted can harm the heart and lungs (Wen et al. 2009) and may lead to other serious health effects.

Concerning socioeconomic indicators, it arises that being outside the labor force (students, retired and homemaker) has a positive influence on developing asthma. This result can be explained by the fact that homemakers,

[18]These results confirm the findings of Di Novi (2009).

students and retired people spend more time indoors (95% of their time against 90% of their employed counterpart - 2008 American Time Use Survey) where the quality of air is often poor since it may be affected by indoor pollution (i.e. environmental tobacco smoke and other indoor allergens) that even at relatively low levels, may play a significant role in the development of asthma (Ernst, 2002). On the other hand, people outside the labor force show a higher probability of following healthier behaviors, with respect to the employed and those who are involuntarily unemployed: they smoke less, they consume heavy drinks less frequently and suffer less often from stress and obesity than people who are in the labor force (even though they more often tend to settle down to a more sedentary life). The other socioeconomic indicators seem to have no influence on asthma prevalence. In the empirical literature, social class (based on occupation, education and income) has been suggested to relate to asthma: some studies have shown increased asthma hospital admissions and asthma severity in low social class groups (see, for instance, Watson et al., 1996; Walters et al., 1995; Chen et al. 1999), but the association between socioeconomic deprivation and asthma prevalence is less clear and consistent evidence is still lacking. The tendency to develop asthma seems to be more related to certain asthma genetic factors (for instance, a family history of asthma may contribute to the development of asthma) and to individual exposure to certain life-style and environmental factors (Basagaña et al., 2004). On the other hand, socioeconomic status seems to affect health improving activities. There is a clear indication of the allocative effects of schooling, since schooling is related to life-style variables in a health promoting way: on the one hand, attending a college or being a college graduate has a negative impact on cigarettes and alcohol consumption, on stress, and on the risk of obesity. On the other hand, a higher degree of education is positively related to the probability of having the flu shot, meeting physical activity recommendations and following a healthy diet.

Our results do not offer significant evidence of racial disparity in asthma

prevalence between White and Black Americans. The disparity in asthma prevalence and treatment between races has been studied at length. From the literature it arises that the disparity in asthma hospitalization is greater than the disparity in asthma prevalence, which suggests that once asthma is established, the black/white gap in asthma seems to be explained by other factors (such as differential access to medical care, differential access to housing, differential patterns of medical care use). Instead our results show a black/white gap in obesity: being black increases the likelihood of obesity. Obesity has reached near epidemic proportions in the United States. Together with cigarette smoking, obesity is one of the leading causes of several chronic conditions and mortality in U.S. The prevalence of obesity is high among African Americans, particularly African American women (Stolley and Fitzgibbon, 1997).

As for the household composition variables, we can observe that the presence of children younger than eighteen years old is negatively correlated with alcohol consumption, but it has a negative influence on exercise and on preventive care and increases the probability of being obese. An increasing number of women in a household increases the probability of choosing a healthier diet and of reducing cigarette smoking, but has a positive impact on stress and a negative influence on exercise indicators.

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 unmarried (Espinosa and Evans, 2008). 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 for each other..." (Hu, Wolfe, 2002). Our model is consistent with the previous findings: in fact, from the empirical results, it arises that marital status has a significant influence on the life-style variables. In particular, marriage seems to be positively correlated to healthy habits while being divorced, separated,

never married, or an unmarried couple has a positive impact on smoking and on alcohol consumption and on bad habits in general.

As mentioned previously, we have estimated the eight equations together using the multivariate probit specification. The multivariate probit allows us to test for unobserved heterogeneity whose effect is captured by the correlation between the error terms from the single equation models. By estimating all eight equations at the same time and taking account of correlation in the error terms for the eight equations we are able to control for the effect on unobserved factors.

TABLE IV shows the correlation matrices for the full recursive models. The null hypothesis of exogeneity is rejected in eleven cases in the linear model and in nine cases in the piecewise linear model[19]. Starting from the asthma equation, the correlation parameters indicate whether and how unobservable factors jointly affect life-style decisions and health outcome. As we can note, there exists a negative statistically significant correlation between the disturbance of the asthma equation and the equation for alcohol consumption and the stress equation. The negative coefficient concerning alcohol and stress support weak evidence that individuals with poor health tend to have a healthier life-style.

From the correlation matrices it appears that unobservable factors, which affect the propensity to smoke are positively related to those that affect heavy drinks consumption and being stressed, and it is negatively related to the propensity of consuming fruit and vegetables. Alcohol consumption is positively related to stress and negatively related to a healthy diet. From these results, it emerges that the unobserved propensity for a unhealthy life-style seems to be explained by unobserved characteristics which determine whether an individual smokes and consumes alcohol.

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

# 5 Summary and Conclusions

Limited literature has been published on the association between environmental health indicators, health improving activities and ambient air pollution. We have examined the associations of asthma prevalence and the amount of health investment with daily mean concentrations of fine particulate ($PM_{2.5}$) in 16 metropolitan areas in U.S. using the Behavioral Risk Factor Surveillance System (2001) data in conjunction with the Environmental Protection Agency's Air Quality System data.

The theoretical framework is based on a Grossman (1972)-style health production model and subsequent contribution by Cropper (1981) set up in discrete time.

A multivariate probit approach has been used to estimate recursive systems of equations for environmental health outcome and life-styles. We have assumed that the relationship between investment in health and ambient air quality could be represented by an inversely V-shaped curve, which assumes the existence of an optimal pollution level at which health investments are maximized. Based on this assumption, a piecewise linear relationship has been employed to describe the association between health investment and pollution using the procedure *mkspline* from STATA 10. This model has allowed for fitting a "breakpoint" in the probit functions. In particular, we have assumed one change point at AQI of 100 which corresponds to the national air quality standard. The empirical results show that below the specified threshold concentration (AQI=100) a positive linear association exists between exposure to $PM_{2.5}$ and health improving activities: people may invest more in health to offset the higher decline rate. But, above the threshold the association becomes negative: if particulate concentration is above the AQI optimal level, an increase in $PM_{2.5}$ along the downward-sloping portion of the curve may lead individuals to invest less in health, since it becomes more costly to build up resistance against pollution. Because of the higher investment costs, individuals may have lower incentives to invest in health

and they may choose to maintain lower health stocks. Hence, only if ambient pollution is in the 'satisfactory range' (AQI level at or below 100), individuals will have incentive to invest in health. In this sense, a pollution concentration above the national air quality standard may have two effects on health: a direct effect which consists in an increase of health deterioration rate (with negative consequences on health stock) and an indirect effect (the same described in Cropper 1981), by which individuals will invest less in health and display a higher probability to suffer from health shocks.

In urban areas, diesel vehicular traffic, often one of the main contributors to pollution including highly damaging emissions of particulate matter which fall into the fine particulate range, is more dangerous for human health (in contrast to coarse particles). Hence, from a policy perspective, intervention that combines public education (for instance, by increasing the number of passengers per vehicle, reducing trips on poor air quality days, keeping vehicles well-maintained, purchasing and using low emission vehicles, using alternative fuels etc.) with other action that abates pollution in the transport sector, would be a key part of urban air quality management strategies in order to reduce fine particulate to below the "optimal threshold level"; such intervention may have not only a direct effect on individuals' health status, but also an indirect health effect through a healthier life-style which is, based on our theoretical and empirical results, one of the driving factors for good health.

This paper suffers from two major limitations: first, we have established a model of health production which is dynamic to show the theoretical relationship between individual life-styles and the rate of health deterioration due to air pollution. Evidently, cross-sectional design of this study involves the evaluation of the characteristics of the individuals and their health and health behaviors at the same point in time making it impossible to disentangle the temporal sequencing of individual behaviors and failing to take the dynamic nature of the health accumulation model into account. In order to

sort out these temporal sequences, long-term studies are needed. However, data of this type are rarely available. Moreover, where panel data do exist, they often lack the details on environmental or personal behaviors- such as cigarette smoking, alcohol consumption, dietary habits, and physical inactivity. So, despite the limitations of the cross-sectional data, we have used BRFSS survey since it provides rather detailed information about health status, diseases, life-style, education and other individual characteristics and it can be combined with the environmental information available from other sources, such as the Environmental Protection Agency and state and local monitoring networks, to compare measures of environmental quality and chronic disease.

Second, in estimating the relationship between particulate and health, we have used ambient levels of pollution as proxy for an individual's exposure to pollution. This approach may be oversimplified because individuals can undertake avoidance activities to reduce the effect of pollution: households can respond to an increasing level of particulate, 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 fine particulate concentration by staying inside or by increasing the avoidance behavior or by mitigating the effects, for instance, through 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). So, a limitation of our study is the absence of proxy for individual avoidance or mitigating behavior in the data set.

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# Appendix I: Tables

TABLE II a): Variables Name and Definition

| *Variables Name* | *Variables Definition* |
|---|---|
| asthma | 1 if has asthma, 0 otherwise |
| smoke | 1 if has smoked at least 100 cigarattes in his life and is current smoker, 0 otherwise |
| 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 meets recommendations for physical activity, 0 otherwise |
| obese | 1 if is at risk for overweight or obesity (BMI >25.0000), 0 otherwise |
| flushot | 1 if has flu shot in the 12 months before the interview, 0 otherwise |
| stress | 1 if mental health (including stress) was not good, 0 otherwise |
| inactive | 1 if is inactive, 0 otherwise |
| male | 1 if male 0 otherwise |
| age | Age in years |
| hmo | 1 if has health care coverage , 0 otherwise |
| element | 1 if elementary school or Kindergarden, 0 otherwise |
| high_sch | 1 if attend high school or high school graduate, 0 otherwise |
| 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 |
| out_labor_market | 1 if retired, homemaker, student, 0 otherwise |
| unemployed | 1 if unemployed, 0 otherwise |
| self_emp | 1 if employed or self-employed, 0 otherwise |
| white | 1 if White, 0 otherwise |
| black | 1 if Black, 0 otherwise |
| other_race | 1 if other race, 0 otherwise |
| nummen | Number of men living in household |
| numwomen | Number of women living in household |
| children | Number of children less than 18 years of age living in household |
| physhlth | 1 if during the past 30 days physical health was not good, 0 otherwise |
| pm2.5_aqi | maximum daily PM2.5 AQI |

TABLE II a): Summary Statistics

| | Means | St. Deviation |
|---|---|---|
| asthma | 0.1172 | 0.3216 |
| age | 46.6242 | 17.0868 |
| male | 0.4074 | 0.4914 |
| smoke | 0.2470 | 0.4313 |
| alcohol | 0.0518 | 0.2216 |
| diet | 0.9643 | 0.1854 |
| exercise | 0.2212 | 0.4150 |
| obese | 0.2231 | 0.4163 |
| flu shot | 0.3089 | 0.4621 |
| stress | 0.3596 | 0.4799 |
| inactive | 0.2521 | 0.4342 |
| hmo | 0.9018 | 0.2976 |
| element | 0.0271 | 0.1623 |
| high_sch | 0.375 | 0.4842 |
| collg | 0.3001 | 0.4584 |
| married | 0.5176 | 0.4997 |
| divorce | 0.1469 | 0.3540 |
| widow | 0.1 | 0.3 |
| seprd | 0.0222 | 0.1473 |
| never_married | 0.1843 | 0.3878 |
| unmar_couple | 0.0289 | 0.1678 |
| employed | 0.6368 | 0.4809 |
| out labor market | 0.3302 | 0.4703 |
| unemployed | 0.0329 | 0.1786 |
| white | 0.8361 | 0.3702 |
| black | 0.1071 | 0.3093 |
| other_race | 0.0568 | 0.2315 |

TABLE II b): Summary Statistics

| Metropolitan Area Name | Maximum Daily PM 2.5 AQI |
|---|---|
| Chicago,IL | 144 |
| Cincinnati,OH-KY | 132 |
| Cleveland-Lorain | 155 |
| Columbus,OH | 142 |
| Des Moines,IA | 92 |
| Huntington-Ashla | 134 |
| Las Vegas,NV-AZ | 132 |
| Louisville,KY-IN | 141 |
| Memphis,TN-AR-MS | 154 |
| Milwaukee-Waukes | 161 |
| Minneapolis-St. | 118 |
| Nashville,TN | 97 |
| Omaha,NE-IA | 122 |
| Phoenix-Mesa,AZ | 146 |
| St, Louis,MO-IL | 143 |
| Tucson,AZ | 62 |

TABLE III a): Estimatated Coefficients of Asthma-$PM_{2.5}$ Model with Threshold

| | asthma | smoke | alcohol | exercise | diet | obese | flu shot | stress |
|---|---|---|---|---|---|---|---|---|
| age | -0.0068 (0.000) | 0.0085 (0.000) | -0.0037 (0.118) | -0.0150 (0.000) | 0.0104 (0.000) | 0.0059(0.000) | 0.0264 (0.000) | -0.0208 (0.000) |
| male | -0.3484(0.000) | | | | | | | |
| physhlth | 0.3364 (0.000) | | | | | | | |
| smoke | 0.2346 (0.003) | | | | | | | |
| alcohol | 0.4246 (0.017) | | | | | | | |
| exercise | -0.0641 (0.482) | | | | | | | |
| diet | 0.0555 (0.761) | | | | | | | |
| obese | 0.2834 (0.002) | | | | | | | |
| flu_shot | 0.3999 (0.000) | | | | | | | |
| stress | 0.1407 (0.088) | | | | | | | |
| inactive | | 0.1096 (0.003) | - 0.1105 (0.090) | -1.067 (0.000) | -0.4166 (0.000) | 0.2605 (0.000) | -0.0641 (0.111) | 0.1044 (0.006) |
| hmo | 0.0019 (0.981) | -0.2174(0.000) | -0.03784 (0.658) | 0.0254 (0.691) | 0.1061 (0.232) | -0.1012 (0.086) | 0.3558 (0.000) | -0.0348 (0.524) |
| high_school | -0.0585 (0.276) | 0.1648 (0.000) | 0.0221 (0.720) | -0.1252 (0.007) | -0.1563 (0.027) | 0.0788 (0.054) | 0.0229 (0.577) | -0.0654 (0.090) |
| collg | -0.0392 (0.514) | -0.3161(0.000) | -0.1953 (0.004) | 0.1914 (0.000) | 0.1859 (0.032) | -0.1417 (0.002) | 0.1275 (0.003) | -0.1459 (0.000) |
| divorce | | 0.2490 (0.000) | 0.3084(0.000) | 0.0596 (0.309) | -0.2984 (0.001) | -0.0104 (0.849) | -0.0828 (0.131) | 0.1930 (0.000) |
| widow | | -0.1143 (0.074) | 0.0145 (0.906) | -0.0758 (0.406) | -0.0115(0.939) | -0.1546(0.031) | -0.1403(0.036) | 0.1521 (0.027) |
| seprd | | 0.1801(0.096) | 0.1396 (0.464) | 0.0286 (0.828) | -0.0922 (0.644) | 0.1726 (0.131) | -0.0498 (0.687) | 0.2001 (0.069) |
| never_married | | -0.0437 (0.397) | 0.2603 (0.001) | -0.0129(0.825) | -0.3928 (0.000) | -0.0552 (0.337) | 0.1609 (0.005) | 0.0271 (0.602) |
| unmar_couple | | 0.3199 (0.001) | 0.4395 (0.001) | -0.1525 (0.168) | -0.1784 (0.289) | -0.1045 (0.343) | -0.0480 (0.681) | 0.2838 (0.003) |
| out_labor_market | 0.1616 (0.003) | -0.1067(0.006) | -0.2338 (0.001) | -0.1299(0.005) | 0.0867 (0.267) | -0.1008 (0.021) | 0.2042 (0.000) | 0.1097 (0.006) |
| unemployed | -0.0351(0.787) | 0.0602 (0.503) | -0.0333 (0.813) | -0.2452 (0.028) | 0.1356 (0.385) | 0.1689 (0.070) | 0.0277 (0.786) | 0.3285 (0.000) |
| black | 0.0481(0.517) | -0.2812 (0.000) | -0.3342 (0.001) | 0.0365 (0.564) | -0.0235 (0.799) | 0.3055 (0.000) | 0.0117 (0.840) | -0.1877 (0.001) |
| other_race | -0.0214 (0.832) | -0.2397 (0.001) | -0.1705 (0.135) | 0.0895 (0.256) | 0.0187 (0.877) | 0.2560 (0.001) | -0.0697(0.382) | -0.0401 (0.565) |
| children | | -0.0574(0.138) | -0.2039 (0.002) | -0.0809 (0.066) | 0.0175 (0.812) | 0.0863 (0.044) | -0.1407 (0.001) | -0.0255 (0.515) |
| nummen | | 0.1052 (0.000) | 0.0816 (0.064) | 0.1163 (0.000) | 0.0896 (0.092) | -0.0357 (0.266) | -0.0302 (0.351) | -0.0925 (0.002) |
| numwomen | | -0.0900 (0.004) | 0.000(0.998) | -0.0829 (0.022) | 0.1258 (0.026) | 0.0077 (0.821) | 0.0310(0.359) | 0.0861 (0.006) |
| pm2.5_aqi<100 | 0.0016 (0.571) | -0.0045 (0.017) | -0.0118 (0.000) | -0.0075 (0.001) | 0.0117 (0.002) | 0.0014 (0.522) | 0.0052 (0.013) | -0.0081 (0.000) |
| pm2.5_aqi>100 | 0.0012 (0.389) | 0.0029 (0.002) | 0.0065 (0.000) | 0.0035 (0.002) | -0.0068 (0.001) | -0.000 (0.834) | -0.0018 (0.074) | 0.0034 (0.001) |

p-values in parentheses.

TABLE III b): Estimatated Coefficients of Asthma-$PM_{2.5}$ Linear Model without Threshold

| | asthma | smoke | alcohol | exercise | diet | obese | flu shot | stress |
|---|---|---|---|---|---|---|---|---|
| age | -0.0069 (0.000) | 0.0087 (0.000) | -0.0033 (0.171) | -0.0148 (0.000) | 0.0099 (0.000) | 0.0059 (0.000) | 0.0261 (0.000) | -0.0205 (0.000) |
| male | -0.3486 (0.000) | | | | | | | |
| physhlth | 0.3364 (0.000) | | | | | | | |
| smoke | 0.2337 (0.003) | | | | | | | |
| alcohol | 0.4129 (0.022) | | | | | | | |
| exercise | -0.0693 (0.446) | | | | | | | |
| diet | 0.0618 (0.734) | | | | | | | |
| obese | 0.2839 (0.002) | | | | | | | |
| flu_shot | 0.4004 (0.000) | | | | | | | |
| stress | 0.1345 (0.102) | | | | | | | |
| inactive | | 0.1029 (0.005) | -0.1235 (0.057) | -1.0736 (0.000) | 0.3992 (0.000) | 0.2619 (0.000) | 0.0578 (0.150) | -0.0939 (0.013) |
| hmo | 0.0018 (0.982) | -0.2213 (0.000) | -0.0472 (0.579) | 0.0194 (0.762) | 0.1103 (0.212) | -0.1004 (0.088) | 0.3584 (0.000) | -0.0406 (0.457) |
| high_schoc | -0.0585 (0.276) | 0.1611 (0.000) | 0.0152 (0.805) | -0.1314 (0.004) | -0.1432 (0.042) | 0.0794 (0.052) | 0.0254 (0.537) | -0.0704 (0.068) |
| collg | -0.0399 (0.506) | -0.3109 (0.000) | -0.1843 (0.007) | 0.1969 (0.000) | 0.1809 (0.036) | -0.1431 (0.002 | 0.1221 (0.005) | -0.1377 (0.001) |
| divorce | | 0.2526 (0.000) | 0.3111 (0.000) | 0.0649 (0.268) | -0.3072 (0.001) | -0.0112 (0.837 | -0.0869 (0.113) | 0.1981 (0.000) |
| widow | | -0.1132 (0.077) | 0.0148 (0.904) | -0.0733 (0.421) | -0.0145 (0.923) | -0.1549 (0.030) | -0.1417 (0.034) | 0.1536 (0.025) |
| seprd | | 0.1823 (0.092) | 0.1326 (0.487) | 0.0307 (0.815) | -0.0977 (0.623) | 0.1718 (0.132) | -0.0522 (0.673) | 0.2037 (0.063) |
| never_married | | -0.0366 (0.478) | 0.2765 (0.001) | -0.0033 (0.955) | -0.4084 (0.000) | -0.0568 (0.322) | 0.1534 (0.008) | 0.0377 (0.467) |
| unmar_couple | | 0.3374 (0.000) | 0.4764 (0.000) | -0.1329 (0.230 | -0.2240 (0.180) | -0.1089 (0.322) | -0.0654 (0.576) | 0.3098 (0.001) |
| out_labor_1 | 0.1612 (0.003) | -0.1029 (0.009) | -0.2272 (0.001) | -0.1225 (0.008) | 0.0802 (0.302) | -0.1015 (0.020) | 0.2009 (0.000) | 0.1147 (0.004) |
| unemployec | -0.0352 (0.787) | 0.0696 (0.438) | -0.0208 (0.883) | -0.2313 (0.039) | 0.1233 (0.431) | 0.1668 (0.073) | 0.0178 (0.862) | 0.3418 (0.000) |
| black | 0.0467 (0.527) | -0.2693 (0.000) | -0.3043 (0.002) | 0.0521 (0.409) | -0.0559 (0.539) | 0.3032 (0.000) | 0.0017 (0.977) | -0.1700 (0.002) |
| other_race | -0.0232(0.816) | -0.2087(0.002) | -0.1057 (0.349) | 0.1334 (0.087) | -0.0599 (0.613) | 0.2492 (0.001) | -0.0974 (0.218) | 0.0090 (0.896) |
| children | | -0.0580 (0.134) | -0.2036 (0.002) | -0.0831 (0.059) | 0.0177 (0.808) | 0.0864 (0.044) | -0.1401 (0.001) | -0.0269 (0.491) |
| nummen | | 0.1067 (0.000) | 0.0839 (0.056) | 0.1184 (0.000) | 0.0820 (0.120) | -0.0361(0.260) | -0.0321(0.322) | -0.0897 (0.002) |
| numwomen | | -0.0899 (0.004) | 0.0041 (0.930) | -0.0816 (0.024) | 0.1176 (0.036) | 0.0077 (0.822) | 0.0312 (0.356) | 0.0864 (0.006) |
| pm2.5_aqi | 0.0013 (0.095) | 0.0006 (0.212) | 0.0007 (0.424) | 0.0001(0.835) | -0.0012 (0.298) | 0.0003 (0.679) | 0.0003 (0.671) | -0.0000 (0.930) |

p-values in parentheses.

TABLE IV a): Estimated correlation coefficients of Asthma-$PM_{2.5}$ Model with Threshold

| | asthma | smoke | alcohol | exercise | diet | obese | flu shot | stress |
|---|---|---|---|---|---|---|---|---|
| **asthma** | 1 | | | | | | | |
| **smoke** | -0.0448 (0.269) | 1 | | | | | | |
| **alcohol** | -0.1242 (0.076) | 0.2672 (0.000) | 1 | | | | | |
| **exercise** | 0.0269 (0.536) | -0.0199 (0.365) | 0.0247 (0.391) | 1 | | | | |
| **diet** | -0.0611(0.372) | -0.0940 (0.012) | -0.0792 (0.121) | 0.0530 (0.202) | 1 | | | |
| **obese** | -0.0259 (0.063) | -0.0301 (0.144) | -0.0189 (0.501) | -0.1197 (0.000) | -0.0118 (0.695) | 1 | | |
| **flu shot** | -0.0725 (0.121) | -0.0089 (0.658) | -0.0421 (0.125) | -0.0361 (0.112) | 0.0821(0.006) | 0.0198 (0.363) | 1 | |
| **stress** | -0.0181 (0.660) | 0.0653 (0.001) | 0.0811 (0.002) | -0.0156 (0.463) | -0.0396 (0.147) | 0.0806 (0.000) | -0.0267 (0.191) | 1 |

p-values in parentheses.

TABLE IV b): Estimated correlation coefficients of Asthma-$PM_{2.5}$ Linear Model without Threshold

| | asthma | smoke | alcohol | exercise | diet | obese | flu shot | stress |
|---|---|---|---|---|---|---|---|---|
| **asthma** | 1 | | | | | | | |
| **smoke** | -0.0439(0.279) | 1 | | | | | | |
| **alcohol** | -0.1181 (0.095) | 0.2673(0.000) | 1 | | | | | |
| **exercise** | 0.0298 (0.492) | -0.0172 (0.435) | 0.0279 (0.333) | 1 | | | | |
| **diet** | -0.0638 (0.349) | -0.0970 (0.009) | -0.0851 (0.095) | 0.0509 (0.220) | 1 | | | |
| **obese** | -0.0262 (0.059) | -0.0306 (0.137) | -0.0196 (0.486) | -0.1201 (0.000) | -0.0123 (0.682) | 1 | | |
| **flu shot** | -0.0727 (0.119) | -0.0106 (0.601) | -0.0445 (0.103) | -0.0383 (0.092) | 0.0832 (0.005) | 0.0202 (0.356) | 1 | |
| **stress** | -0.0143 (0.728) | 0.0677 (0.000) | 0.0848 (0.001 | -0.0129 (0.546) | -0.0418 (0.125) | 0.0791 (0.000) | -0.0293 (0.151) | 1 |

p-values in parentheses.