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Air quality and morbidity:
Concentration-response relationships for Sweden[•]

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The study investigates the morbidity impacts of air pollution when pollution may affect both the likelihood and duration of respiratory problems. The relationship between comparatively low pollutant levels and respiratory ailments is estimated using Swedish data, and the change in respiratory-related restricted activity days (RRAD) due to a unit change in NO_2 is calculated. The analysis pays particular attention to overdispersion, the high proportion of zero values and the peak in the RRAD distribution, problems that have not been addressed thoroughly in previous studies. Our results suggest that it is a challenging task to set air quality standards for environments where modest increases in pollutant concentrations may significantly prolong respiratory health problems for the most vulnerable individuals.

Key words: air quality, concentration-response, health, respiratory restricted activity days

JEL classification: C24, I12, Q53

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1. Introduction

It has been estimated that about 3 million people die and many more suffer serious health effects each year because of air pollution (WHO 2003). When inhaled, air pollutants affect the lungs and respiratory tract but can also be absorbed and transported throughout the body by the blood stream, causing additional damage. The impact of pollution on health depends on levels of exposure and the susceptibility of the exposed population. However, it is difficult to distinguish air pollution from other factors affecting health.

The derivation of Concentration-Response Relationships (CRRs), often referred to as dose-response relationships, involves estimating physical or medical relationships linking both socio-economic and environmental variables, such as ambient concentrations of air pollution, to observable health effects. Health effects are divided into mortality impacts, where the primary endpoint is death, and morbidity impacts, where the endpoint is a nonfatal illness. Mortality effects are measured as changes in the probability of dying, and morbidity effects as changes in hospital admissions, symptom frequency or labor productivity, such as work loss or restricted activity days.

The quantification of CRRs is crucial to evaluating the economic impacts of air pollution in terms of labor productivity. In recent contingent valuation studies on air pollution, the household production model has been applied to value morbidity impacts (e.g., Alberini et al 1997, Alberini and Krupnick 1998, Navrud 2001), but the estimated benefits from lowering pollutant levels are not explicitly based on CRRs linking air pollution to morbidity. Our purpose is to augment the approaches in these analyses by showing the importance for valuation of appropriately estimating concentration response functions. In the spirit of Grossman (1972), who first used the household production model to examine health decisions, we assume that health determines the total amount of time a person can spend producing monetary earnings and commodities and that both market and nonmarket time are relevant. Accordingly, productivity loss is easier to assess if, instead of using symptoms, one employs a measure of reduced labor productivity, such as the number of days a person is affected by the health impacts, as the health endpoint in the CRR when valuing the morbidity effects of air pollution.

Several studies (e.g., Ostro 1983, 1987, Hausman et al. 1987) have identified CRRs for US data, using restricted activity days or work loss days as a measure of the health impact of pollution. That this work is still cited in the literature (e.g. Hansen and Selte 2000, Holland et al 1999, Ostro and Chestnut 1998, and Zuidema and Nentjes 1997) indicates that more recent research serving comparative purposes is lacking. Be this as it may, one must bear in mind

that the data in these studies, collected in the 1970s and 1980s, are now dated, and the estimation methods used can be considered unsuitable in many respects. Yet, the results of the studies have been recently used for estimating productivity loss and assessing health impacts from pollution for other regions. For example, a recent research project of the European Commission, ExternE, used US studies to evaluate the external costs of different fuel cycles in European countries (See JOULE 1998). Other studies using results from US CRRs in Europe are Markandya and Pavan (1999), who assessed health effects from air pollution for four European countries, and Bellander et al (1999), who linked exposure data to hospital admissions for Stockholm.

The short-term effects of air pollution on mortality and hospital admissions were studied in a research project including 12 European countries (APHEIS 2003, Katsoyanni 1996), but the health endpoints used did not capture less severe morbidity effects. In two recent European studies, Zuidema and Nentjes (1997) estimate pollution effects on work loss days for the Netherlands with aggregate data without including individual specific variables, and Hansen and Selte (2000) estimate pollution effects on general sick leaves for a single large company in Norway.

In this study, we address two concerns that have not received sufficient attention in previous analyses. The first is the transferability of CRRs, for the health situation, medical care, population distribution and the pollution situation vary between countries. Previously, Alberini and Krupnick (1997) have drawn attention to the same problem by studying whether CRR results from the city of Los Angeles could be applied to three cities in Taiwan.[1] Their conclusion was that the transfer of concentration response is questionable. To find more evidence on the reliability or potential biases of transfers, we estimate a CRR based on Swedish national health survey data and use *number of respiratory-related restricted activity days* (RRAD) as a dependent variable. A RRAD is any day on which a person's activities are impaired, although the impairment need not prevent her from going to work. It is a more encompassing measure of sick time than work loss days which is relevant only for members of the labor force and is determined by disability insurance and sick leave arrangements that vary from country to country. Since Swedish air quality is rather good in international perspective, our analysis could be useful in understanding the relation between comparatively

[1] The data for Los Angeles were from 1978-79, those for Taiwan from 1991-92. The explanatory variable was an indicator (dummy) of the presence or absence of any one of the nineteen symptoms listed for the participants of the study. The coefficients of the pollutants were not significant for the Taiwanese cities despite the higher concentration levels of PM10, SO2 and NO2 in Taiwan vis-à-vis Los Angeles.

low pollutant levels and respiratory ailments.[2] As will be shown, our results indicate that people in regions with modest absolute levels of pollution are more sensitive to air pollutants. In particular, sensitivity to the concentration of pollutants is captured by the prolonged duration of episodes of pollution-related respiratory problems.

A second important finding from the literature on CRRs is that the estimation method highly influences the coefficients of the variables used to measure pollution (see Zuidema and Nentjes, 1997). The analyses most similar to ours are Ostro (1987 and 1990), Ostro and Rothschild (1989) and Hausman et al (1984), who used number of RRADs or work loss days as a dependent variable; these data were elicited using a questionnaire surveying the general population that asked how many RRADs respondents had had during the past two weeks. These analyses motivated the use of a Poisson regression model for its suitability when the dependent variable includes a large number of zero values. Yet, the model assumes equidispersion, or equality of conditional variance and conditional mean, and the raw data were overdispersed in these studies. Even if the inclusion of regressors may eliminate some overdispersion, it is clearly a problem. Overdispersion will cause the computed maximum likelihood *t* statistics to become considerably overinflated (Cameron and Trivedi, 1998). To improve the reliability of estimation results, we pay special attention to overdispersion that has not been addressed thoroughly in previous studies.

Our econometric modeling is based on a theoretical model which takes into account the fact that pollution can directly affect an individual's health status, i.e., the incidence of respiratory problems, and/or the duration of the ill health status. In several previous CRR analyses, these two impacts have not been separated, but estimated simultaneously, and where they have been separated, CRRs have not been used in the final valuation of health impacts. Our empirical results indicate that combining these two effects may have led to underestimation of pollution impacts. In including the different health responses in the same framework, our theoretical model clarifies the relevant components of the total valuation.

The paper is organized as follows. In the section to follow, we present a theoretical model that shows how the assessment of change in pollution should be carried out when pollution affects both the likelihood of respiratory problems and their duration. Section 3 presents the econometric models used. Sections 4 and 5 describe the data and the estimation results, respectively. The concluding section discusses the results.

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[2] During the period studied, the yearly average level of NO_2 level did not exceed the standard, 40 $\mu g/m^2$, implemented in 1999 to conform to EU standards according to ordinance SFS2001:527. For Sweden, other health endpoints such as symptom frequency have been studied using diaries focusing on an asthma population. The results indicate an association between negative health effects and the pollutants such as black smoke, nitrogen dioxide and sulphur dioxide. See Forsberg et al (1993, 1997a, 1997b, 1997c).

2. The theoretical model

Our theoretical model elaborates certain features of the household production models of Harrington and Portney (1987) and Berger et al. (1987) to show how the estimates of concentration-response functions affect valuation when health outcomes are uncertain and pollution may affect both the likelihood of respiratory problems and their duration. Our econometric estimations rest on decomposition of these two effects, an approach that aims at avoiding the problems of overdispersion, which previous CRR studies have not accounted for adequately.

Consider an individual with a utility function of the form

$$U = U(C, l, D), \tag{2.1}$$

where C represents goods consumed, l is leisure time, and D is time spent ill. We assume that duration of respiratory problems (D) depends on mitigating behavior or medicine used to shorten the duration (M)[3], on air pollution (P), and on health and other attributes of the individuals and their surroundings (x) such that $D=D(M;x,P)$ and $\partial D/\partial M=D_M<0$. In our empirical model, duration, D, is measured by the number of *RRADs*. When an individual's activities are not impaired by illness ($D=RRAD=0$), we express her utility as $U^0=U(C,l,0)=U^0(C,l)$, otherwise $U^1=U^1(C,l,D(M;x,P))$. As the individual does not know with certainty whether she will have incidence of respiratory problems ($RRAD>0$), we have to specify the probability of $RRAD>0$, or $p(x,P)$, such that the probability, p, depends on attributes x, and air pollution, P. The individual maximizes her expected value of utility ($U_C>0$, $U_l>0$, $U_D<0$)

$$\max_{C,l,M} E(U) = p(x,P)\cdot U^1\left[C, l, D(M;x,P)\right] + \left(1 - p(x,P)\right)U^0(C,l) \tag{2.2}$$

with respect to her budget constraint

$$\begin{aligned} I + w\left(T - l - D\left(M;x,P\right)\right) &= C + p_M M \quad \text{if } U=U^1 \\ I + w\left(T - l\right) &= C + p_M M \quad \text{if } U=U^0 \end{aligned} \tag{2.3}$$

[3] The variable M could, for example, be antihistamine taken to reduce nasal discharge or visit to a doctor.

where I is non-wage income (from capital or transfer payments), w is the wage rate, T is total time available, and p_M is the cost of mitigation activities such as medical expenses. Eliminating C by including the budget constraint in equation (2.2), the expected utility can be rewritten as

$$V(I,P,w) = p(x,P)\cdot U^1\left[(I + w(T - l - D(M;x,P)) - p_M M), l, D(M;x,P)\right] + (1 - p(x,P))U^0\left[(I + w(T - l) - p_M M), l\right]. \tag{2.4}$$

The first order conditions for l and M are

$$\frac{\partial V}{\partial l} = p(x,P)U_l^1 + (1 - p(x,P))U_l^0 - \mu w = 0 \tag{2.5}$$

$$\frac{\partial V}{\partial M} = p(x,P)(U_D^1 - U_C^1 w)D_M - \mu p_M = 0\,, \tag{2.6}$$

where $\mu = p(x,P)U_C^1 + (1 - p(x,P))U_C^0$, which is the weighted average of the expected marginal utility. Equation (2.5) simply states the marginal utility of leisure in optimum. Equation (2.6) says that mitigation measures are taken up to a point where the costs of these measures equal their marginal benefit, or the avoided loss of wage income and disutility from extra time spent ill (prolonged duration of illness).

We denote the maximum utility obtainable for a given set of parameter values in equation (2.4) by $V(I^*(P),P)=V_0$ where I^* is implicitly defined as a function of P holding V constant (see Harrington and Portney, 1987). The marginal value of a marginal change in pollution for given I and w, or $dI^*(P)/dP$, can be derived as

$$\frac{dI^*(P)}{dP} = -\frac{V_P}{V_I}\,.$$

Computing

$$V_P = \frac{\partial p}{\partial P}U^1 + p(x,P)(U^1{}_D - wU^1{}_C)\frac{\partial D}{\partial P} - \frac{\partial p}{\partial P}U^0$$
$$V_I = \mu$$

implies

$$\frac{dI^*(p)}{dP} = \overbrace{\underbrace{\frac{\partial p}{\partial P}}_{2}\frac{\left(U^0 - U^1\right)}{\mu}}^{a} + \overbrace{\left(\underbrace{p(x,P)}_{1}\frac{(wU^1{}_C - U^1{}_D)}{\mu}\right)\underbrace{\frac{\partial D}{\partial P}}_{3}}^{b}. \qquad (2.7)$$

This result provides the theoretical basis for our econometric analysis. Equation (2.7) states that the marginal value of an exogenous marginal change in pollution is the sum of two terms: (a) the product of the change in the probability of impairment in normal activities and the difference in utility between the healthy state and sick state (with respiratory problems) expressed in monetary terms by dividing by the expected marginal utility; and (b) the product of the expected marginal value of extra time spent ill, or the wage rate and the marginal direct disutility, and the change in the duration of the illness. Market data can be used for estimates of the value of lost work, and non-market valuation methods, such as contingent valuation, can be used for valuation of direct disutility. In addition, equation (2.7) suggests the applicability of concentration-response functions to estimate 1) the probability of getting respiratory illness, *p(x,P)*, 2) the marginal impact of pollution on this probability, *∂p/∂P*, and 3) the marginal impact of pollution on the duration of the illness, *∂D/∂P*, in order to assess the total pollution impacts. This is what we do next.

3. The econometric models

To estimate a concentration-response relationship (CRR) requires data on the air pollution concentration to which the individual is exposed and data on the frequency of symptoms and variables affecting health status. The dependent variable in CRR studies is usually hospital admissions or deaths caused by respiratory illnesses. According to Bellander et al (1999), these variables capture only a part of the total effect of moderate air pollution, since most effects are less severe. We use a more broadly defined dependent variable, *RRAD*, measured for the two-week period prior to the survey. This variable captures days when a person is affected although not absent from work, as well as those when she is absent. It is then matched with individual-specific explanatory variables, such as socio-economic, demographic, health (chronic condition), and municipal data on pollutants and temperature for the same period.

There are two special features of the dependent variable that must be taken into account in the estimations. First, the dependent variable is typically constructed on the basis of

questionnaires on which respondents representing the general population indicate the number of *RRADs* they have had during the two-week (or other) period preceding the survey. Since only a small proportion of the population has respiratory problems, an even smaller proportion will have had problems during that period, whereby the dependent variable will take on a very large number of zero values. This is a common phenomenon in analyses such as these in which the general population is sampled (see, e.g., Ostro 1987, 1990). Second, there is often a peak at the end of the distribution of the dependent variable, *RRAD*. People who answer that they have had 14 *RRADs* might have had problems for 15 or more days, but since the question is restricted to a two-week period, they answer "14".

The dependent variable, *RRAD*, is summarized in Table 1, which presents the actual frequency distribution. As expected, there is a large number of zero values, 96.4 percent of the sample. Second, there is a peak at the last value, which can be explained by the fact that people with respiratory problems, especially bronchitis, are affected for long periods or most of the time. In sum, the peak represents people that have been restricted for at least 14 days.

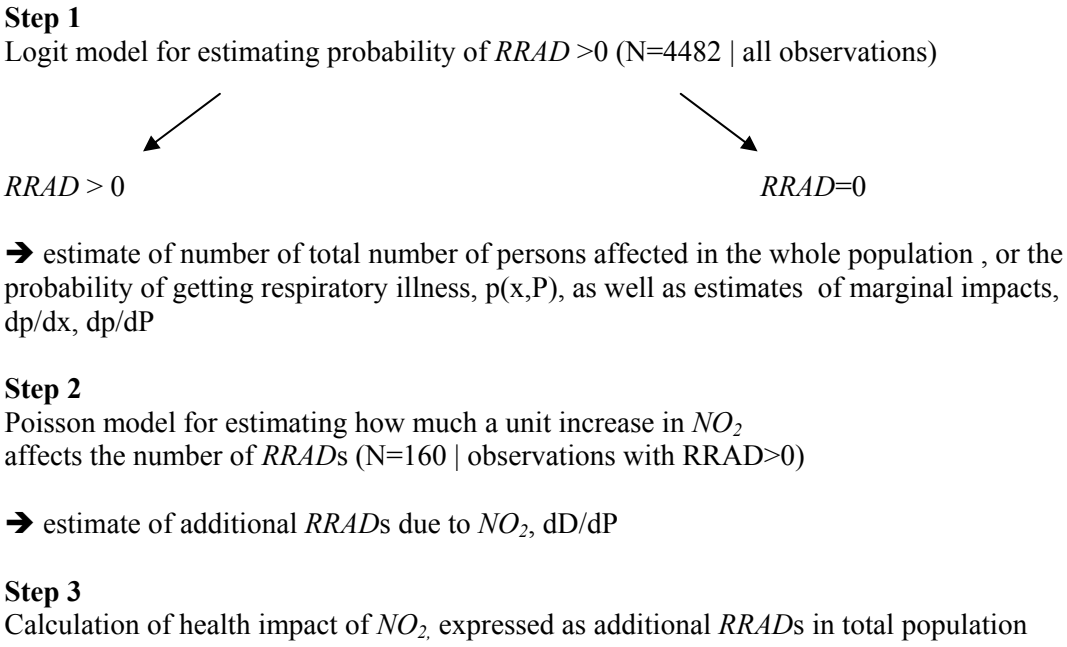
Table 1. RRAD: Actual Frequency Distribution

COUNT	FREQUENCY	RELATIVE FREQUENCY
0	4322	0.9643
1	11	0.0025
2	37	0.0083
3	27	0.0060
4	17	0.0038
5	17	0.0038
6	6	0.0013
7	12	0.0027
8	3	0.0007
9	0	0.0000
10	6	0.0013
11	0	0.0000
12	1	0.0002
13	0	0.0000
14	23	0.0051
Σ	4482	1.0000

The large percentage of zero values and the peak in the *RRAD* distribution have prompted us to apply estimation techniques that are better suited to these types of data than the traditional Poisson model. As shown in equation (2.7), we must first examine whether the concentration of air pollution affects the likelihood of an individual having *RRADs*, *p(x,P)*, which entails estimating a limited dependent variable model for the probability of *RRAD>0*. Second, we investigate whether air pollution prolongs the duration of restricted activity,

$\partial D/\partial P$. To link air pollution to *RRAD*, we employ a count data model to estimate the *RRAD* distribution, or $Pr(RRAD=D_i)$ where D_i indicates the observed number of days. In other words, we carry out two separate estimations, which are illustrated in Figure 1.

Figure 1. Health impact analysis applying concentration-response functions for respiratory-related restricted activity days (RRAD)



First, the whole data set is analyzed using the Logit model. The logistic distribution is defined as:

$$\Pr(RRAD > 0) = \frac{e^{\beta' z}}{1 + e^{\beta' z}}. \qquad (3.1)$$

where z is a vector $z=[x,P]$ containing the relevant explanatory variables. The Logit model truncates the dependent variable at zero and one by turning a frequency into a probability, $Pr(RRAD>0)$ or $p(x,P)$, from which also $\partial p/\partial x$ and $\partial p/\partial P$ can be derived for equation (2.7).

Second, we study the individuals with positive *RRAD*s separately, thus excluding all observations of $RRAD=0$ from the sample. The individuals with positive *RRAD*s are analyzed using a Poisson model, with the Poisson distribution providing the probability of the number of event occurrences, in this case the number of *RRADs*. The Poisson parameter, corresponding to the expected number of occurrences, is modeled as a function of the explanatory variables. Estimation makes use of the maximum likelihood method. This model

exploits and preserves the nonnegative and integer-valued aspect of the outcome (Cameron and Trivedi, 1998).

Assume that we have n independent observations, the i^{th} of which is (D_i, $\mathbf{z}_i$), where the dependent variable, D_i, is the number of *RRADs*, and $\mathbf{z}_i$ is a vector of linearly independent regressors that are hypothesized to determine D_i. The probability that D_i *RRADs* will occur in the two-week period surveyed is described by

$$\Pr(RRAD_i = D_i) = \frac{e^{-\lambda_i}\lambda_i^{D_i}}{D_i!}, \qquad D_i = 1,2,...,n \tag{3.2}$$

where λ_i is the expected mean rate of occurrence of *RRAD*s during this period. In a Poisson distribution, the conditional mean is equal to the conditional variance, $E(D_i|z_i) = V(D_i|z_i) = \lambda_i$. This property is called equi-dispersion. The regression model is produced by specifying the mean, λ_i, to depend upon a set of covariates. In the standard approach, the mean parameter is parameterized as $\lambda_i = \exp(z_i'\beta)$ to ensure that $\lambda>0$. Here, β is a vector of parameters to be estimated. These estimates are obtained by a maximum likelihood procedure, which seeks an estimator that maximizes the joint probability of observing the sample values, $D_1,...,D_n$.

The peak in the end of the distribution can be handled by estimating censored data. The observed data are said to be right censored when the observation $D_i=j$ may indicate that the true observation was at least, but not exactly, j. On our questionnaire, individuals were asked how many *RRADs* they had had during the previous two weeks. Those who answered 14 might equally well have had problems for more than 14 days. The Poisson model with right censoring is exactly as described in (3.2) except that for some integer, $\overline{D}$ (in our case 14 days), all values of y_i greater than or equal to $\overline{D}$ are reported as $\overline{D}$. A latent variable, $RRAD^*$, the underlying Poisson variable, is defined. The formulation of the Poisson regression model is then (Greene, 1993):

$$\Pr(RRAD^* = D_i) = \frac{e^{-\lambda_i}\lambda_i^{D_i}}{D_i!}, \qquad \ln\lambda_i = \beta' z_i \tag{3.3}$$

The observed variable is $D_i=Min(D, \overline{D})$, and

$$\Pr(D_i = j) = \Pr(D = j), \qquad if \qquad D < \overline{D}$$

$$\Pr(D_i = \overline{D}) = \Pr(D \geq \overline{D}) = 1 - \Pr(D < \overline{D}) = 1 - \sum_{j=0}^{\overline{D}-1} \Pr(D_i^* = j) \qquad (3.4)$$

The Poisson regression generates a probability distribution for occurrences of *RRADs*, and the coefficients can be used to derive the marginal impact of pollutants on the number of *RRAD*s, or $\partial D/\partial P$ in equation (2.7).

4. Data

The principal data source for individual-specific health data and socio-economic variables is the 1999 national environmental health survey (NMHE99) conducted by Institute of Environmental Health and Department of Environmental Medicine at Karolinska Institute. The survey questionnaire was sent out to 15,750 persons in Sweden in 1999. In each of the 21 country's counties, 750 persons between the ages of 19 and 81 years who had lived in Sweden for the last five years were randomly chosen. The overall response rate was 72.5 percent.[4] The Swedish environmental research institute (IVL), together with local environmental health offices, provided data on municipality-specific variables describing air quality. The relevant data on weather conditions were received from the Swedish Meteorological and Hydrological Institute (SMHI).

Table 2 summarizes the descriptive statistics for the variables included and the Swedish averages where these were available. Education (*EDU*) is measured as number of years in school and income (*INC*) is measured in 100 000 SEK (Swedish crown). Demographic variables are age (*AGE*), measured in years divided by 100, and sex (*SEX*), indicated by a dummy variable (0 for male and 1 for female). Previous studies have represented the health situation by a dummy for chronic condition.[5] Our data allow separation of respiratory health into three dummy variables, one for asthma (*ASTHMA*), one for bronchitis or emphysema (*BRONCH*), and one for hay fever or other nasal problems (*HAY*). The dummy variable receives a value of 1 if the respondent has, has had or has been diagnosed with asthma/bronchitis/hay-fever; otherwise the value is 0. Smoking (*SMOKE*) is included as number of cigarettes smoked per day.[6]

[4] Ideally, one would use time-series data. However, this was the first year when detailed questions about the respiratory system were asked on the Swedish environmental health questionnaire. Ostro (1987) ran his model for each year separately even though he had access to a six-year time series.

[5] See, for example, Cropper (1981), Krupnick, Harrington and Ostro (1990), Ostro (1983a, 1983b, 1987, and 1990) and Pourtney and Mullahy (1986).

[6] One could argue that smoking is a typical example of adverse mitigation that is determined endogenously. We have interpreted smoking as an exogenous variable, since addiction makes it difficult to quit smoking.

Table 2. Descriptive Statistics for the Whole Sample (N=4482) and Swedish averages.

Variable	Mean	Standard deviation	Min.	Max.	Swedish average
SEX, male=0, female=1	0.53	0.50	0.00	1.00	0.50[a]
AGE, years divided by 100	0.46	0.16	0.19	0.81	0.47[a]
EDU, years	11.87	2.83	3.00	16.00	11.03[a,b]
INC, 100000 SEK	1.70	1.20	0.00	26.57	1.80[a,c]
SMOKE, number of cigarettes	2.14	5.39	0.00	30.00	-
ASTHMA, dummy	0.09	0.28	0.00	1.00	6-10%[d]
BRONCH, dummy	0.04	0.20	0.00	1.00	-
HAY, dummy	0.34	0.48	0.00	1.00	-
TEMP, C-degrees	0.65	2.28	-10.40	3.50	-1.45[e]
NO_2, $\mu g/m^3$	15.99	6.51	7.00	35.00	15.73[f]
RRAD, days in 2 week period	0.19	1.27	0.00	14.00	-
RRAD, binary	0.04	0.19	0.00	1.00	-

[a] Statistics Sweden (2003).

[b] Refers to the educational level of persons aged 16 to 74 years.

[c] Calculated from averages for the age groups 20-64 years and 64+ years.

[d] Miljöhälsorapporten (2001).

[e] Calculated from the monthly averages for March at all 100 Swedish weather stations, Swedish Meteorological and Hydrological Institute (1999).

[f] Calculated from the monthly averages for March in the 73 Swedish municipalities that measure NO_2 concentrations.

Previous studies have used several indicators of exposure to assess the effects of air pollution.[7] Since pollutants often correlate with one another both in time and space, studies cannot strictly allocate observed effects to individual pollutants.[8] To avoid multicollinearity, Künzli et al (2000) recommend the use of only one pollutant as an indicator. We would have preferred to use data on particulates, but these were not systematically measured by the environmental authorities in 1999. The Swedish environmental research institute (IVL) had monthly data for the winter period (October1999-March 2000) on SO_2, NO_2 and soot, measured in $\mu g/m^3$, for the 39 Swedish municipalities that belong to its urban network. In addition, local environmental health offices in 34 municipalities measure SO_2 and NO_2. These

[7] The availability of data often determines how the pollutants are measured and which pollutants are included. US concentration-response studies have included the pollutants NO_2 (nitrogen dioxide), SO_2 (sulfur dioxide), O_3 (ozone), TSP (total suspended solids), IP (inhalable particles), FP (fine particles), and COH (coefficient of haze). In Zuidema and Nentjes (1997), air quality is measured by taking the annual average of SO_2, SO_4, Black smoke, particulates, ozone ($\mu g/m^3$) and ammonia (mol ha^{-1}), and work loss days are measured on an annual basis. Ostro (1983a and 1983b) also used annual average levels of the pollutants SO_4 and particulates ($\mu g/m^3$), although he used a two-week recall period for health outcomes. In Ostro (1987) and (1990), the RRADs measured for the two-week period prior to the survey were merged with the two-week average of particulate matter (in $\mu g/m^3$).

[8] The correlation between NO_2 and SO_2 in this study turned out to be as high as 0.76 when the counties in southern Sweden, where the fall-out of SO_2 from abroad is strongest, were excluded.

measurements indicate that urban air quality in Sweden has improved considerably during the past 15 years.[9]

Since the principal source of air pollution in Swedish urban areas is traffic, we use nitrogen dioxide (NO_2) as the indicator of urban air quality and traffic pollution (see e.g. Forsberg et al 1993, 1997a, 1997b, 1997c). In fact, fine particulates include nitrate and sulphate aerosols formed following the emission of NO_x and SO_2 (De Nocker et al 1999). The indicator, NO_2, should be interpreted as an approximate air quality index of local emissions from traffic. If a significant relationship with *RRADs* is found, it does not necessarily imply that NO_2 is causative but that a mixture of pollutants including NO_2, or merely associated with it, could have caused the correlation.

Most of the respondents filled out the questionnaire in March and April (92 respondents in March and 8005 respondents in April). Since the respondents were asked how many *RRADs* they had had in the previous two weeks, "March respondents" were mapped to data on air quality in February and "April respondents" to data on air quality in March. The analysis was limited to the 73 of 290 municipalities that measure air quality. This yielded 4482 observations. Since only densely populated municipalities measure air quality, this sample is more representative of the 56% of the Swedish population that live in these areas.

Since colder weather worsens air quality through inversion and there are synergistic effects on respiratory problems from temperature and air pollution, a temperature variable (*TEMP*) from an SMHI database is also included in the estimations. Since we do not have data on actual mitigation measures, we observe the health outcomes after a potential mitigation such as taking medicine has taken place.

Finally, fixed effects, in the form of county dummies, are introduced to control for differences between the counties that are not accounted for by explanatory variables. Hausman et al (1984) emphasize the importance of such variables in this type of analysis. They claim that a major source of uncertainty in interpreting the results of observational data on the effect of pollution on health is that pollution in an area may be correlated with other characteristics of the area that affect outcomes but are not controlled for in the analysis.

In Table 2 the minimum and maximum values of NO_2 represent the municipalities with the lowest and highest pollutant levels, respectively. The differences in concentration between municipalities are considerable. The maximum concentration is 5 times higher than the minimum value. None of the municipalities exceeded the maximum yearly average standard

[9] The average winter pollution levels in the urban areas have decreased during the period from 1986/87 to 2000/01; from 17 to 2 $\mu g/m^3$ for SO_2, from 31 to 17 $\mu g/m^3$ for NO_2, and from 11 to 7 $\mu g/m^3$ for soot (Swedish Environmental Protection Agency, 2002).

of 40 μg/m^2. Furthermore, we can see that 9 percent of the persons in our sample have asthma, 4 percent bronchitis or emphysema, and as many as 34 percent hay fever or other nasal problems. However, due to the short recall period (2 weeks) of the dependent variable, the mean number of *RRAD*s is low, or 0.19. The overall impression from Table 2 is that the sample is rather representative of the Swedish population. The average education level and age are approximately the same. Our data set includes slightly more females, and the average income is lower than the national counterpart.

Table 3 summarizes the descriptive statistics of the small sample comprising individuals with positive *RRADs* (160 observations). This sample includes a much larger share of women and people with asthma, bronchitis and hay fever/nasal problems. The average level of income is lower in this subsample than in the sample at large. However, the concentration of NO_2 is approximately the same. Tables A2a and A2b (in the Appendix) present the correlation coefficients of the variables included in the analysis.

Table 3. Descriptive Statistics for RRAD sample (N=160)

Variable	Mean	Standard deviation	Min.	Max.
SEX, male=0, female=1	0.57	0.50	0.00	1.00
AGE, years divided by 100	0.46	0.16	0.19	0.80
EDU, years	12.04	2.89	8.00	16.00
INC, 100000 SEK	1.59	1.01	0.00	6.96
SMOKE, number of cigarettes	2.22	5.82	0.00	30.00
ASTHMA, dummy	0.29	0.46	0.00	1.00
BRONCH, dummy	0.19	0.40	0.00	1.00
HAY, dummy	0.68	0.47	0.00	1.00
TEMP, C-degrees	0.71	2.16	-7.00	3.50
NO_2, μg/m^3	16.25	6.54	7.00	34.50
RRAD, days in 2 week period	5.36	4.16	1.00	14.00

5. Estimation, interpretation and transferability of concentration-response functions

As can be seen from Table 2, the raw data are clearly overdispersed, since the mean number of *RRADs* is 0.19 while the variance is 1.61. Not surprisingly, nearly all coefficients become significant when the Poisson model based on the whole data set is estimated; the *t* statistics are as high as 14.3 (See Appendix Table A3). Interestingly, the NO_2 coefficient was nearly the only insignificant variable in the estimation. Had we used a Poisson model as earlier studies have done, the conclusion would have been that there exist no pollution impacts on health in

the whole population.[10] As discussed above, we aim at handling overdispersion by analyzing seeparately with a Logit model what determines *RRAD* incidence and then analyzing the number of *RRADs* using a Poisson model. The results from the Logit and the censored Poisson models are shown in Table 4.

Table 4. Estimation results for Logit and Censored Poisson models.

Variable	Logit *RRAD* incidence		Poisson Positive counts	
	Coefficient	*t* ratio	Coefficient	*t* ratio
Constant	-4.623	-7.572	-0.969	-1.837
SEX	0.075	0.437	0.485	5.605
AGE	0.193	0.305	1.782	5.707
EDU	0.030	0.890	0.023	1.443
INC	-0.068	-0.753	-0.042	-0.911
SMOKE	0.000	0.003	0.021	3.508
ASTHMA	0.857	4.169	-0.326	-3.364
BRONCH	1.194	4.829	0.426	4.056
HAY	1.116	6.143	0.146	1.678
TEMP	0.011	0.305	-0.122	-1.833
NO_2	0.008	0.618	0.032	2.533
D1, Stockholm	-	-	0.394	0.676
D2, Uppsala	-	-	1.010	1.915
D3, Södermanland	-	-	0.088	0.125
D4, Östergötland	-	-	0.678	1.165
D5, Jönköping	-	-	0.696	1.186
D6, Kronoberg	-	-	1.275	2.230
D7, Kalmar	-	-	3.403	0.420
D8, Gotland	-	-	1.270	2.302
D9, Blekinge	-	-	0.269	0.435
D10, Skåne	-	-	0.470	0.649
D11, Halland	-	-	1.160	1.697
D12, Västra Götaland	-	-	0.866	1.367
D13, Värmland	-	-	0.382	0.665
D14, Örebro	-	-	0.592	1.132
D15, Västmanland	-	-	1.136	2.110
D16, Dalarna	-	-	0.417	0.979
D17, Gävleborg	-	-	0.608	1.284
D18, Västernorrland	-	-	0.193	0.512
D19, Jämtland	-	-	0.616	1.982
D20, Västerbotten	-	-	-1.810	-2.833
-lnL	626		379	
Number of observations	4482		160	

[10] There are other models that specifically should handle a large share of zeros, for example the Zero-inflated Poisson model (ZIP). However for our data the estimations showed that the ZIP results are very sensitive to model specification, adding or deleting variables did change the sign and significance for several variables.

Likelihood ratio tests indicate that the fixed-effects county dummies should be excluded from the Logit but included in the Poisson. The variables *EDU* and *INC* turned out to be insignificant in both models. In the Logit model, only the three respiratory health indicators, *ASTHMA*, *BRONCH* and *HAY*, significantly determine the incidence of *RRAD*s. When analyzing the positive counts, the variables *SEX*, *AGE* and *SMOKE* significantly indicate that women, elderly people and smokers have more *RRAD*s. Only *ASTHMA* and *BRONCH* are significant indicators of respiratory health. However, the *ASTHMA* variable now changes sign. This indicates that, among individuals with *RRAD*s, those with asthma have a lower number of such days than others. A possible explanation for this is that most asthmatics are able to control episodes of restricted activity with on-demand medication. This contrasts with the situation of bronchitis patients, for whom there is no such treatment available. Indeed, in our data, individuals with bronchitis – if they have *RRADs* – are affected for longer-than-average periods. This shows how important it is to separate chronic conditions into different indicators of respiratory health more carefully than has been done previously. The NO_2 level does not affect *RRAD* incidence, which implies that the first term in equation (2.7) is zero, since $\partial p(x,P)/\partial P=0$. However, individuals who have *RRADs* and who live in municipalities with high levels of NO_2 have longer *RRAD* episodes. These results indicate the importance of analyzing the positive *RRAD*s separately.

To further take into account overdispersion, the property of equi-dispersion was relaxed with the NB2 variance function, which assumes that the variance is quadratic in the mean. New *t* statistics were calculated and they showed that NO_2 remained significant. To see how sensitive the results are for NO_2, the Poisson model was also estimated without the censoring at 14. This did not change the significance of the variables. The NO_2 coefficient became 0.030 with a *t* value of 2.383. To further see how dependent the NO_2 results are on the inclusion of specific variables, the insignificant variables were excluded one by one. The NO_2 coefficient ranged from 0.034 to 0.035 and remained significant in all the specifications.

The interpretation of the NO_2 coefficient is that if the level of NO_2 increases by one unit ($\mu g/m^3$), the number of *RRAD*s will increase by 3.2 percent. Assuming that our sample is representative of the Swedish population, the figures in Table 5 allow us to calculate the effect on the population of an increase in NO_2 by one unit. An increase of NO_2 by one unit ($\mu g/m^3$) will result in 685,637 extra *RRAD*s, which is our estimate for $\partial D/\partial P$ annually in Sweden.

Table 5. Figures for calculating effect on RRAD of one-unit increase in NO_2

Predicted probability of *RRAD*>0 (from Logit estimation, p(x,P))	2.6%
Swedish population between ages 19 and 81 years	6,488,846
Estimated number of people with *RRAD>0* in Swedish population (0.026*6,488,846)	
Conditional *RRAD* mean for two-week period (from Poisson estimation)	4.87
Mean *RRAD* for one year (4.87*26)	127
Total *RRAD* in Swedish population (0.026*6,488,846*127)	21,426,168
Poisson coefficient for P (NO_2)	3.2%
Extra *RRAD* due to NO_2 using Poisson coefficient, dD/dP (21,426,168*0.032),	685,637

Ostro (1987, 1990) and Ostro and Rotschild (1989) are, to the best of our knowledge, the only analyses that comparably link *RRAD*s to pollutants. The explanatory variables they used education, family income, race, chronic condition, temperature, sex, marital status, and labor market status. The statistical significance of these variables was presented only in Ostro (1987), where all variables significantly determined *RRADs* but probably because overdispersion was not accounted for. Our analysis included these same variables, with the exception of race, marital status, and labor market status. In the Logit specification, only the different chronic conditions became significant. In the Poisson model, *SEX*, *AGE*, *SMOKE, ASTHMA* and *BRONCH* were significant. However, Ostro (1987, 1990) and Ostro and Rotschild (1989) did not include NO_2 in their analyses, and it is not possible to compare the impact and significance of that pollutant with those found in our study. Depending on the model specification, Ostro (1987) received 0.7-2.2 percent for fine particles, Ostro and Rotschild (1989) received 1.02-1.81 percent for fine particles and Ostro (1990) received 0.83-1.74 percent for SO_4. However, due to overdispersion problems in these analyses, the coefficients should be interpreted with caution. Our coefficient for NO_2 seems much higher, but it was obtained using a sample with only positive *RRAD*s. To illustrate the differences in measured impacts, we can use 1.5% as an average of Ostro's coeffients and 4.94 as a mean number of *RRAD*, for one year from the large sample (mean *RRAD*s for the two-week period 0.19 times 26 = 4.94). The effect on the Swedish population would then be (1.5%*4.94*6,488,846=) 480,823 extra *RRAD*s. This would indicate that the Swedish population is more sensitive to air pollution than the American population, that the pollutants capture health effects from air pollution differently, or that there are differences between emissions.

Our results can be compared to those of an earlier study which found that residents of Los Angeles were much more sensitive to fluctuations in pollution levels than residents of Taiwan (Alberini and Krupnick 1997) despite the lower concentrations of air pollution in Los

Angeles. The mean NO_2 level was three times higher in Los Angeles than that in Swedish municipalities, but NO_2 had a negative and significant coefficient for incidence of acute respiratory illness. In our study, NO_2 does not seem to increase the probability of *RRAD* incidence among the general Swedish population. However, the impact of pollution on the average duration of the *RRAD* period is statistically significant. If the concentration increases by 1 $\mu g/m^3$, the number of *RRADs* will increase on average by 0.16. The prolonged duration explains why the Swedes seem to be more sensitive to air pollutants. As the healthcare benefit system is obviously more generous in Sweden than in many other countries, it should be emphasized that *RRAD*s include days not absent from work and is a more reliable measure for comparisons than work loss days.

We have now derived the relationship between an indicator of urban air quality and a measure of reduced labor productivity due to respiratory conditions. This result could be used as a first step in the valuation of the health effects of pollution in monetary terms. Next, one would need a translation of physical effects into monetary terms. The value of extra *RRAD*s resulting from a one-unit increase in NO_2 can be estimated in terms of disutility and loss of wage income, as shown in equation (2.7). Disutility from minor and major respiratory illness episodes has been valued for the Netherlands, Norway, Portugal, Spain and the UK in a European study (Ready et al., 2001) and also recently for Sweden (Samakovlis and Svensson, 2003). A task for future research is to use the results from these studies to complete the valuation of health impacts from air pollution in Sweden. This would involve identifying the symptoms associated with a typical *RRAD* in Sweden.

6. Conclusions

We have presented a theoretical model for how the valuation of health effects from pollution should be carried out when pollution may affect both the likelihood of respiratory problems and their duration. The components of health impacts derived in the theoretical model are examined by estimating the relationship between NO_2 as an indicator of urban air pollution and health effects in the form of respiratory-restricted activity days. Health effects are estimated using data on Sweden, where pollutant levels are low by international standards.

The dependent variable in this type of study is usually based on a questionnaire where individuals are asked how many work loss days, restricted activity days or respiratory restricted activity days (*RRAD*s) they had during a certain period. This results in a large proportion of zero values in the dependent variable and a peak at the end of the distribution.

Previous studies with similar specifications have not taken these data characteristics into account, which has resulted in overinflated *t* -values.

Our analysis has addressed these two shortcomings by separately analyzing the incidence of *RRAD*s with a Logit model and the number of *RRAD*s with a censored Poisson model. The analysis also introduces county-specific dummies to control for differences between the counties not accounted for by explanatory variables, and separates the customary chronic condition dummy into different chronic conditions. The results show that the chronic conditions asthma, bronchitis and nasal problems are the only variables that significantly determine *RRAD* incidence.

Regarding the number of days with respiratory problems, the results indicate that women, elderly people and smokers have more *RRAD*s. Asthma and bronchitis are significant indicators of respiratory health, but the sign of the asthma dummy became negative, indicating the importance of distinguishing different chronic conditions. The mean concentration of NO_2 in Sweden is relatively low, or 16 $\mu g/m^3$. The pollution variable, NO_2, significantly determines the number of *RRAD*s and indicates that if the level of NO_2 increases by one unit ($\mu g/m^3$), *RRADs* will increase by 3.2 percent. The effect on the population was calculated on the assumption that the sample is representative of the Swedish population.

Compared to previous findings, our results indicate that the Swedish population seems to be more sensitive to air pollution than the American population. This has interesting implications for setting standards for air pollution in environments where increases in modest pollution levels tend to significantly prolong the duration of respiratory health problems. In particular, harmonization of air pollution standards across countries – one of the goals of environmental policy within the European Union – may lead to non-optimal health outcomes.

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APPENDIX (Tables A1 – A3)

Table A1. Measures of Air Quality and Temperature by Municipality

MUNICIPALITY	MEAN NO_2, $\mu G/M^3$		MEAN TEMPERATURE, C	
	FEB	MARCH	FEB	MARCH
Stockholm county				
Huddinge(U)	14,4	8,8	-1,7	1,7
Botkyrka	Not available	19,5	-1,7	1,7
Sollentuna	22,5	21,5	-1,7	1,7
Stockholm(U)	28,1	22,7	-1,7	1,7
Södertälje(U)	21,8	19,0	-1,7	1,7
Lidingö	17,9	14,8	-1,7	1,7
Uppsala county				
Uppsala(U)	17,0	15,2	-2,6	1,2
Södermanland county				
Oxelösund	9,3	11,6	-0,8	1,1
Eskilstuna	12,3	8,5	-2,3	1,0
Östergötland county				
Finspång	*11,0	*12,0	-1,9	1,8
Linköping(U)	12,6	10,0	-2,2	1,4
Norrköping	18,0	16,0	-1,9	1,8
Söderköping	*11,0	*8,0	-1,9	1,8
Motala	*8,0	*7,0	-2,2	1,4
Jönköping county				
Jönköping(U)	18,6	15,5	-2,4	0,4
Värnamo(U)	12,6	9,2	-2,2	2,0
Vetlanda	*12,0	*12,0	-2,2	2,3
Eksjö	12,2	9,6	-2,2	2,3
Kronoberg county				
Älmhult(U)	12,9	11,4	-1,6	2,4
Växjö(U)	16,0	10,0	-2,2	2,0
Ljungby	*19,0	*14,0	-2,2	2,0
Kalmar county				
Torsås	*7,0	*9,0	-1,4	2,1
Västervik(U)	11,6	10,5	-1,1	2,5
Borgholm	*6,0	*8,0	-0,1	2,4
Gotland county				
Gotland	9,0	9,8	-0,9	1,7
Blekinge county				
Olofström	*9,0	*8,0	-0,9	2,6
Karlskrona	*22,0	*14,0	-1,3	2,2
Ronneby	*25,0	*31,0	-1,3	2,2
Karlshamn(U)	16,3	21,6	-0,9	2,6
Sölvesborg	*16,0	*24,0	-0,3	2,3
Skåne county				
Burlöv(U)	24,2	18,2	-0,5	3,5
Malmö	32,3	32,4	0,3	3,5
Lund	25,5	17,3	-0,5	3,5
Landskrona(U)	18,7	15,5	-0,2	3,3
Helsingborg	26,8	24,8	-0,2	3,3
Trelleborg(U)	24,1	22,0	1,1	3,1
Kristianstad(U)	13,6	15,2	-0,8	3,2

*Averages based on one-week values instead of one-month values, (U) Member of the urban project

Continuing Table A1. Measures of Air Quality and Temperature by Municipality

MUNICIPALITY	MEAN NO_2, $\mu G/M^3$		MEAN TEMPERATURE, C	
	FEB	MARCH	FEB	MARCH
Halland county				
Halmstad	18,9	17,2	-0,3	3,2
Falkenberg(U)	17,6	15,1	-0,1	2,8
Västra Götaland county				
Partille	25,9	26,2	0,1	2,9
Göteborg(U)	28,6	24,0	0,1	2,9
Mölndal	39,6	35,0	0,1	2,9
Kungälv(U)	20,9	16,6	-0,4	2,3
Borås	24,7	21,0	-1,7	1,5
Mariestad(U)	10,9	11,0	-1,2	1,4
Tidaholm(U)	11,0	9,5	-2,4	0,4
Värmland county				
Årjäng(U)	15,8	14,4	-2,3	-0,1
Karlstad(U)	27,0	16,6	-2,5	0,7
Örebro county				
Örebro(U)	20,5	15,8	-2,3	1,0
Karlskoga(U)	16,1	12,3	-2,3	1,0
Västmanland county				
Västerås	16,2	12,6	-2,6	1,2
Fagersta(U)	13,9	12,1	-2,6	1,2
Köping(U)	15,3	11,3	-2,6	1,2
Dalarna county				
Orsa(U)	16,3	8,7	-5,3	-1,4
Falun	22,0	13,6	-5,3	-0,5
Borlänge	26,8	23,0	-5,3	-0,5
Gävleborg county				
Hofors	23,6	14,1	-4,4	0,2
Gävle	21,4	11,2	-4,4	0,2
Sandviken(U)	17,1	11,3	-4,4	0,2
Bollnäs(U)	21,9	12,4	-6,8	-1,2
Hudiksvall(U)	24,9	19,5	-5,0	-0,5
Västernorrland county				
Timrå(U)	20,3	18,4	-6,9	-2,1
Sundsvall	29,0	28,0	-6,9	-2,1
Örnsköldsvik(U)	24,3	20,6	-5,1	-3,3
Jämtland county				
Hammarstrand(U)	13,2	8,8	-6,0	-3,0
Gällö(U)	10,3	9,1	-6,1	-3,5
Östersund(U)	17,0	16,7	-6,1	-3,5
Västerbotten county				
Lycksele(U)	Not available	15,1	-11,5	-5,4
Skellefteå	33,5	34,5	-7,7	-4,0
Norrbotten county				
Kalix(U)	15,3	Not available	-10,9	-5,2
Luleå	18,0	13,0	-10,4	-5,2
Piteå(U)	21,0	17,2	-10,3	-4,2
Kiruna(U)	15,2	8,9	-14,7	-7,0

*Averages based on one-week values instead of one-month values, (U) Member of the urban project

Table A2a. Correlation Coefficients (Whole sample, N= 4482)

	SEX	*AGE*	*EDU*	*INC*	*SMOKE*	*ASTHMA*	*BRONCH*	*HAY*	*TEMP*	NO_2
AGE	-0.02									
EDU	0.00	-0.44								
INC	-0.25	0.28	0.11							
SMOKE	0.01	0.04	-0.11	-0.00						
ASTHMA	0.04	-0.05	0.01	-0.05	-0.01					
BRONCH	0.01	0.12	-0.06	-0.01	0.06	0.26				
HAY	-0.00	-0.05	0.06	-0.03	-0.02	0.26	0.15			
TEMP	0.01	0.02	0.04	-0.00	0.02	-0.01	0.00	0.00		
NO_2	0.03	-0.00	0.05	0.02	0.01	0.00	-0.01	0.01	-0.13	
RRAD	0.03	0.03	0.00	-0.02	0.02	0.10	0.16	0.11	0.00	0.01
RRAD binary	0.02	-0.00	0.01	-0.02	0.00	0.14	0.14	0.13	0.00	0.01

Table A2a presents the correlation coefficients of the variables included in the analysis. Income correlates positively with age and education, but negatively with sex. Age correlates negatively with years in school, but positively with bronchitis. Education correlates negatively with smoking. Asthma, bronchitis, hay-fever and *RRAD* correlate positively with each other. Temperature correlates negatively with NO_2. Correlations among the other explanatory variables were low.

Table A2b. Correlation Coefficients (RRAD Sample, N=160)

	SEX	*AGE*	*EDU*	*INC*	*SMOK*	*ASTH*	*BRON*	*HAY*	*TEMP*	NO_2
AGE	-0.23									
EDU	0.16	-0.40								
INC	-0.32	0.34	0.12							
SMOK	0.04	0.10	-0.18	-0.05						
ASTH	0.04	0.06	-0.00	-0.10	0.03					
BRON	-0.08	0.26	-0.21	-0.08	0.13	0.41				
HAY	-0.12	-0.07	0.08	0.10	0.07	0.18	0.07			
TEMP	0.09	-0.10	0.20	0.02	0.09	-0.06	-0.07	0.09		
NO_2	0.08	0.08	0.07	-0.10	0.06	0.01	-0.03	0.06	-0.10	
RRAD	0.18	0.25	-0.08	-0.05	0.17	-0.04	0.21	0.04	-0.00	0.02

In Table A2b the correlations between the variables are similar to those for the large sample. The most striking difference is that *RRAD* exhibits a stronger positive correlation with age. The correlation between asthma and *RRAD* changed sign and is now negative. This indicates that, among individuals with respiratory problems, asthmatics are affected for shorter periods.

Table A3. Poisson Models for the Whole Sample

Variable	Poisson	
	Coefficient	*t* ratio
Constant	-4.854	-11.142
SEX	0.387	5.192
AGE	0.905	3.296
EDU	0.036	2.613
OCCU	-0.369	-4.660
INC	-0.044	-1.063
SMOKE	0.014	2.570
ASTHMA	0.562	6.450
BRONCH	1.341	14.343
HAY	1.110	14.161
TEMP	-0.174	-3.329
NO_2	0.017	1.638
D1, Stockholm	1.526	3.376
D2, Uppsala	2.084	5.107
D3, Södermanland	-0.183	-0.290
D4, Östergötland	0.456	0.986
D5, Jönköping	1.454	3.452
D6, Kronoberg	2.253	5.068
D7, Kalmar	1.710	3.276
D8, Gotland	1.794	4.181
D9, Blekinge	1.066	2.207
D10, Skåne	1.830	3.352
D11, Halland	1.658	3.187
D12, Västra Götaland	1.637	3.195
D13, Värmland	1.017	2.190
D14, Örebro	1.492	3.662
D15, Västmanland	1.693	4.149
D16, Dalarna	1.728	5.085
D17, Gävleborg	0.708	1.924
D18, Västernorrland	0.978	3.194
D19, Jämtland	0.932	3.538
D20, Västerbotten	-1.236	-2.170
-lnL	2802	
Number of obs.	4482	

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